

# OPERATIVE OBSTETRICS

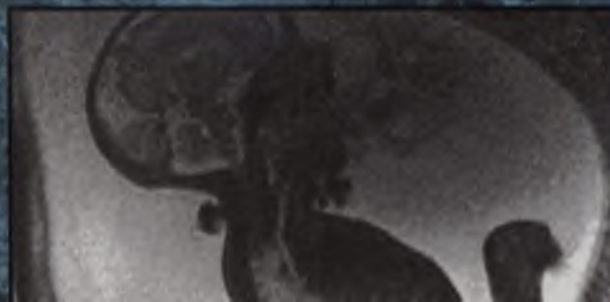
SECOND EDITION

*Edited by*

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CAMBRIDGE  
Medicine

# Operative Obstetrics

## *Second Edition*

Major changes in obstetric practice have occurred in the ten years since the publication of the first edition of *Operative Obstetrics*. Prospective clinical studies have improved clinical practice, and better techniques for antenatal fetal evaluation have been introduced. Yet, there are also less desirable trends. There has been a relentless increase in the rate of cesarean delivery, and persisting medicolegal and societal pressures continue to demand faultless performance. Our recognition of recent improvements in clinical practice and acknowledgement of the continuing challenges and limitations inherent in modern clinical management have prompted a new edition. This updated edition includes chapters on the important subjects of cesarean delivery, common surgical complications, ectopic pregnancy, birth injury, and instrumental delivery, among other topics. It features a new discussion of surgical procedures performed by non-physicians and a review of fetal surgery. The text also considers complicated and controversial subjects such as cervical insufficiency, pregnancy termination, and shoulder dystocia. In recognition of the realities of current practice, each of the four sections of the book has a chapter with an in-depth analysis of the legal issues underlying practice. An expanded appendix reviews general legal concepts pertinent to the practice of obstetrics.

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Every effort has been made in preparing this publication to provide accurate and up-to-date information that is in accord with accepted standards and practice at the time of publication. Nevertheless, the authors, editors, and publisher can make no warranties that the information contained herein is totally free from error, not least because clinical standards are constantly changing through research and regulation. The authors, editors, and publisher therefore disclaim all liability for direct or consequential damages resulting from the use of material contained in this publication. Readers are strongly advised to pay careful attention to information provided by the manufacturer of any drugs or equipment that they plan to use.

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*To JO: His son finally did become a surgeon. And to Molly for support and encouragement.*

JPOG

*For my family, Herakliusz, Stefania, Alexandria, and Madeline, for their patience and support during this endeavor.*

LBZ

*To Arlene, Alexis, and Matt. For your love and support.*

MLG

*To Marge and Bill, who sacrificed so much on my road to becoming an attorney.*

KG

---

*L*ooking back over a long clinical lifetime, one tends to forget or take for granted one's successes; it is the failures which stand out like keloid scars, never to be forgotten and, hopefully, a warning to others.

I have to recognise that if there is any classic mistake which I have not myself made it is simply because of the lack of time in which to commit it. It makes one wondrously sympathetic toward others in trouble. No apology is therefore made for the highly personal emphasis in this book.

Ian Donald (1910–1987)

*Practical Obstetric Problems*

London: Lloyd-Luke, 1979, p. viii.

### *Note to Readers*

The advancement of medical science brings continuous changes in management, methods of diagnosis and evaluation, and drug therapy.

The editors and contributors to *Operative Obstetrics* Second Edition, have closely reviewed the information included in this textbook, consulted appropriate literature, and conferred with experienced clinicians in the effort to provide accurate information and practice recommendations in accordance with the generally accepted standards of medical practice. The reader is cautioned, however, that owing to the rapid changes in the science of medicine and the possibility of human error, the authors of the various chapters, the editors, and the publisher cannot guarantee that all information included in this text is in every respect complete or accurate. We do not accept responsibility for errors, omissions, or results obtained from the use of these data. For these reasons, the reader is encouraged to confirm our practice suggestions with other standard sources. Relying on his or her experience, education, and unique knowledge of the individual patient, the attending physician or certified nurse midwife must determine the best treatment for a specific obstetric condition.

Recommended drugs and dosing schedules for various medical conditions do appear in this text. Before a drug is administered, however, clinicians should review standard compendia of drug information and package inserts for any changes in drug use or additional warnings of potential adverse reactions or other precautions. To ensure patient safety, caution is especially necessary when the drug in question is new to the practitioner, infrequently administered, or has the potential for serious side effects.

– The Editors

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## Foreword

THE PROCESS OF EVOLUTION affects not only the characteristics of a species but also the adaptive technology between a species and its environment. The practice of obstetrics is devoted to maximizing the ability of each human being to confront the environment and to be part of the creative, modulating path of evolution. It is almost, if not totally, impossible to discern evolutionary human changes within our own lifetimes; however, it is a different story with the technology of our interactions. Obstetrics has changed, and it has changed rapidly.

If the earth's lifetime were compressed into a single 24-hour day, humans would have appeared only 30 seconds ago. I cannot imagine what nanocalculation would be required to measure the history of operative obstetrics, yet that incredibly short measure of geologic time is packed with a geometrically increasing collection of events and stories. The interesting and comprehensive chapter on the history of operative delivery alone is worth the price of this book. Every contemporary obstetrician should know and learn from the history of obstetrics. Some might argue that this history is truly the past, and that operative obstetrics today is a matter of a few simple choices. Even that judgment, however, must be based on a critical analysis of the operative choices. Only then can the individual obstetrician understand the reasons behind modern decisions.

The modern focus on "evidence-based medicine" all too often fails to recognize the broad base of

knowledge that is the foundation of clinical decision making. This book is an excellent example of the fact that medical knowledge is more than what we read in the literature. Although medicine tests the worth of specific procedures with appropriately designed clinical studies, physicians also learn from each and every clinical experience and modify their decisions according to an understanding of the individual patient's needs. Nowhere is this more important than in operative procedures. The authors of this book have solidified their recommendations with a comprehensive survey of the literature, but they have filtered this knowledge through the valuable experiences of multiple clinicians, finally offering clinical advice that is meaningful and useful.

Obstetric decisions today are not simpler. They are actually more complex, requiring an ever-expanding knowledge base. This book provides a knowledge base of operative obstetrics derived from the accomplishments of the past and the experiences of the present. In so doing, it serves an important purpose: to assist obstetricians in achieving the objective of a successful pregnancy and a healthy newborn.

*Leon Speroff*  
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# Preface

*Obstetrics is not one of the exact sciences, and, in our penury of truth we ought to be accurate in our statements, generous in our doubts, tolerant in our convictions.*

James Young Simpson (1811–1870)

MUCH TO OUR SURPRISE, more than ten years have passed since the publication of the first edition of *Operative Obstetrics*. Since the initial text appeared in 1995, new tests, surgical procedures, and novel methods of medical education have been introduced to the practice of obstetrics. In addition, there has been an expansion of roles for non-physician personnel in the provision of care to pregnant women. There remain important unresolved controversies in the specialty, including elective or patient-choice cesarean delivery, trials of vaginal birth after cesarean, patient safety during hospitalization, pregnancy termination, and the recruitment and training of new practitioners, to list only a few. The influx of new ideas and the development of new techniques over the last decade have accompanied increasing demands by institutions, third-party payers, and governmental agencies for evidence-based, cost-efficient, and safe practice. Clinicians are thus pressured from many directions to rapidly incorporate new scientific advances into their management, rethink traditional concepts of best practice, follow increasingly restrictive protocols and practice guidelines, and even revisit basic ethical concepts. Because of the unresolved issues concerning appropriate practice and the risks associated with adverse outcomes, it is inevitable that medicolegal risks in obstetrics remain high and that increasingly few clinicians, with a decade or more of active practice, now escape litigation.

The stated goal of all recent textbooks is to define best practice by employing the techniques of evidence-based medicine. In fact, there is now a growing body of evidence-based data concerning obstetric practice, much to the improvement of the specialty; however, many areas of management have never been subjected to such systemic study. Experienced practitioners rapidly discover that there are obstetric and surgical practices and clinical problems that have not proved amenable to the rigid demands of evidence-based analysis. These observations emphasize the limitations of current methodologies and serve as a constant reminder of the incompleteness of physicians' knowledge and the need for continuous improvement through appropriately designed prospective studies.

This new edition required the amalgamation of data derived from quite different sources. Working with the editors, our many collaborators have strived to reconcile current scientific knowledge and data from evidence-based clinical studies with the rich heritage available from the past. Philosophically, the editors remain unrepentant advocates of combining essential elements of the art of traditional obstetrics and the accumulated experience of our predecessors with new concepts and methods of management derived from meta-analysis and other prospective and randomized clinical investigations. Reflecting the realities of modern practice, this new edition includes legal commentaries on areas of

special concerns, with recommendations for appropriate actions to help to avoid difficulty.

It is the editors' earnest anticipation that this new edition of *Operative Obstetrics* fulfills the demanding requirements of clinicians struggling with the many pressures of contemporary practice. Our aim is both to challenge and instruct our readers. The success of this endeavor will be measured by the extent to which we have constructively critiqued established ideas, fused the traditionally accepted with the scientifically proved aspects of practice,

and sustained the reader's interest. Our measure of success is simple. If this textbook proves helpful in the management of a single case, our original expectations will be met, and we will consider our intense labors and those of our coworkers to have been amply rewarded.

*John P. O'Grady*  
*Martin L. Gimovsky*  
*Lucy A. Bayer-Zwirello*  
*Kevin Giordano*

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# ANTEPARTUM

## Chapter 1 A HISTORY: OPERATIVE DELIVERY

John P. O'Grady

*Norwithstanding that I would use all my Endeavours to deter Men from the rash and imprudent Practice of instrumental Operations in Midwifery; yet it is not to be denied, but that such Operations are very useful and necessary, when undertaken with Caution, Skill and Prudence; . . .*

Fielding Ould (1710–1789)  
*A Treatise of Midwifery in Three Parts*  
Dublin: O. Nelson & C. Connor,  
1742: 111, pg 142.

Prolonged or obstructed labor, undeliverable fetal positions, maternal hemorrhage from retained products of conception, delivery of the second of twins, and the problematic extraction of large infants are among the recurring problems in human labor and delivery that do not resolve without intervention. Assistive techniques to manage these and other complications of human parturition are rooted deep in antiquity. Over many years, various manipulations and specialized instruments were developed to expedite delivery of viable infants or to remove the fetus and the other products of conception from the uterus in case of fetal demise or incomplete delivery. A brief historical review of the origins of operative delivery techniques increases the appreciation of modern practitioners for the complex roots of the science and art that have led to modern practice.

### THE HISTORY OF CESAREAN DELIVERY

#### Myth and Legend

Reports of the surgical removal of the fetus from the mother are common in history and legend. Such tales figure in the origin myths for important personalities from many cultures. For example, Brahma is described as emerging from his mother's umbilicus, and in 5636 B.C.E., Buddha is reported to have been delivered from his mother Maya's right flank [1]. Tall tales of preternatural or miraculous births are also common in our western Greco-Roman cultural heritage. Classic Greek mythology includes several descriptions of what could be termed cesarean deliveries of various gods, demigods, and mortals [2]. A representative example is the case of the inconstant princess Coronis. Upon receiving proof of her infidelity with another male suitor, her enraged paramour Apollo (Phoebus Apollo), god of prophecy, music, and archery, dispatched her with an arrow. In some versions of

this tale it is Apollo's twin sister, Artemis (Diana), daughter of Zeus and Leto, who was responsible for this murderous archery. In any event, Apollo next placed the body of the newly dead Coronis on a funeral pyre. As the flames leaped up, Apollo's rage rapidly changed to consternation for the fate of his unborn child. At Apollo's urgent request, Hermes (Mercury), the messenger of the gods and the patron of heralds, thieves, travelers, and merchants, intervened, and the infant was delivered from his mother's body by means of an abdominal incision. This child, who was the product of this unique perimortem delivery, was subsequently tutored in the healing arts by Chiron the centaur, son of Coronos and the nymph Philyra, and eventually became the most famous physician of antiquity, Asclepius. This tale has an ending that should serve as a warning to overly ambitious physicians. In his later life, Asclepius developed his medical abilities to the point where he could resurrect the dead. For his presumption in using his medical talents to thwart the will of the gods, Zeus killed him with a thunderbolt!

In another setting, Zeus prematurely delivered Dionysus (Bacchus), god of wine and ecstasy, from the abdomen of the dying Semele, the daughter of Cadmus and Harmonia. Zeus had actually fathered this child. Unfortunately, complications with the pregnancy led to disaster. In the sixth month of the pregnancy, malevolent advice was given to the young woman by the jealous Hera, Zeus's wife, who was masquerading as Semele's elderly nurse, Beroe. Under this influence, Semele refused Zeus her bed unless he would come to her in his true form. Zeus, trapped by her request, resumed his accustomed form as a thunderbolt, a dramatic process that proved fatal to the hapless Semele. Through the intervention of the ever-present Hermes, however, the unborn and premature Dionysis was removed from Semele's womb, sewn into the thigh of Zeus, and, through this unusual mechanism, carried to maturity as a bizarre type of ectopic pregnancy [3].

There are other unusual tales of obstetric interventions in Greek and Roman mythology. Adonis, famous for his great beauty, was born of his mother, Myrrha of Smyrna, after her transformation into a tree. Myrrha had conceived following an incestuous relationship with her father, Cinyras. Cinyras was a Cypriot king and originally one of the lesser suitors to Helen before her abduction and the beginning of the Trojan War. This unusual relationship

between father and daughter developed because of the enmity of Aphrodite, the goddess of love, who punished the unfortunate Myrrha because of her lack of devotion [2]. Aphrodite's intervention caused the poor Myrrha to fall in love with her own father. Under what proved to be a maleficent influence, Myrrha developed a subterfuge whereby she shared Cinyras' bed without his recognizing her. The god's punishment for Adonis's mother was her transformation into a myrrh tree, thus arresting her father's unacceptable advances. Her father's eventual fate was also severe. When he discovered that he had been tricked into impregnating his own daughter, Cinyras committed suicide.

In terms of drama, myth, and legend, classic theater also contains many stories of unusual births. Perhaps the most famous occurs in the denouement of the play *Macbeth*. Shakespeare's protagonist Macduff is free from mortal risk from Macbeth, because Macduff was "from his mother's womb untimely ripp'd . . ." [4] As he was not of woman born, Macduff fulfilled the prophecy of the witches and thus successfully defeated the regicidal Macbeth. This tale of ambition, greed, murder, and operative delivery has a long pedigree, with its origin well before the sixteenth century. Shakespeare had obtained the material for his tragedy from an earlier text, the *Chronicles of Holinshead*. From this reference, further sources for this Scottish tale can be traced to another text, *Scotorum, Historiae of Boece* (Paris, 1526); it can further be followed to a manuscript originally published in 1385! Doubtless, its roots are even earlier than the fourteenth century, in now lost sources.

History also includes many reports of unusual cesarean deliveries involving actual individuals. There are several well-documented cases in which women delivered themselves by conducting their own surgeries. Many if not most of these abdominal surgical deliveries would in current terminology be described as cesareans. Authentic reports from rural settings also describe traumatic deliveries when milkmaids were gored by cattle, the earliest dating back to 1647. In some of these latter cases, the mother, the infant, or both apparently survived [1].

### Derivation of Terms *Cesarean* and *Section*

In common parlance transabdominal surgical deliveries are termed *cesareans*. How this nomenclature

came to be employed for abdominal surgical delivery is a long and complex tale. The derivation of the term *cesarean* has been ascribed to several sources. Ancient historians, including Pliny the Elder are largely responsible for the widely believed myth that a Roman emperor or Caesar – either Scipio Africanus (237–183 B.C.E.) or more commonly, the most famous emperor, Gaius Julius Caesar (102?–44 B.C.E.) – was delivered from his mother via an abdominal incision. Unfortunately, it is unlikely that these historical figures or many of the other famous persons reputed to have been delivered by a surgical procedure were actually born in that manner. In reference to the historical Roman Emperor Gaius Julius Caesar, it is virtually certain he was *not* delivered surgically from his mother, since the term *cesarean* predates him by centuries. Furthermore, published letters of Julius Caesar indicate that he corresponded with his mother, Aurelia, while he was in Gaul. Finally, Aurelia is known to have lived until 54 B.C.E., when Caesar, who was then more than 40 years old, attended her funeral [5]. Her long-term survival after an unsterile abdominal surgery in the first century is distinctly improbable. The reports by Pliny and other classical writers of successful abdominal delivery of culturally important people such as the historical Emperor Julius Caesar lack historical support and are best viewed as political fables.

There are various interpretations but no clear evidence to explain how the family of Gaius Julius Caesar received the cognomen *caesar* and how this family name at some point became associated with a surgical procedure. The name of Caesar might derive from several literary sources, such as from the Latin *caedere/caedo*, meaning “to cut, fall, or kill; to cut down or to strike mortally as in conflict,” [6] possibly reflecting a traumatic or surgical delivery sometime in the family’s past [7]. It is also possible that a legend of an abdominal delivery became associated with the family name simply as an honor. Preternatural births were thought to confer on the child certain special virtues, powers, or abilities – exactly what might be expected of a world leader such as an emperor. After all, the Julian family was noble and from a patrician clan. Caesar’s father, once the governor of Asia, had served as praetor, the second most important post after consul [8].

Another possible origin of the term *cesarean* derives from legal responses to the problem of peri-

or postmortem delivery. The first law relating to postmortem delivery is reputed to have been promulgated by the quasi-legendary king of Rome, Numa Pompilius (715–673 B.C.E.), and termed the *lex regia* (and subsequently *lex caesarea*) [1]. This edict concerned the abdominal delivery of a child during an acute life-saving effort in the unusual circumstance of a dying or recently dead mother. The statute was a type of Good Samaritan law, requiring delivery of the unborn child from its mother and forbidding the burial of the dead woman until this was accomplished. The law also protected the person who performed such a perimortem procedure from an accusation of murder or manslaughter, assuming that the amateur surgeon acted in good faith.

Some English words with specialized meaning have their origin in the Latin roots that originally gave us the term *cesarean*. In musical notation, a *caesura* is a set of closely approximated parallel lines in the score that mark a sudden stop, or cut, in the course of the program. This term is also used to indicate an interruption, break, or pause between words within a metrical foot in poetry, or in the middle of a line of text. In a social/political context, both the titles of *Kaiser* and *Tsar* (*Czar*) have their origin in the original Latin *Caesar*. In English, both *Kaiser* and *Tsar* either describe an authority figure, usually a tyrannical one, or are used in their historical sense as the traditional titles for a Holy Roman, Austrian-German, or Russian Emperor, respectively.

Whatever the origin of the term, by the mid-sixteenth century, the term *cesarean* was used to describe abdominal surgical deliveries in medical literature. One of the earliest commentators or medical editors to refer to the abdominal delivery of an infant as a cesarean was Richard Jonas, who translated, edited, and expanded one of the many editions of the obstetric textbook usually termed the *Rossgarten*, which was originally authored by Eucharius Rösslin of Frankfurt-am-Main (discussed later in this chapter). First published in 1540 in its English editions as *The Byrth of Mankynde*, this text was thereafter frequently reprinted. In one of these reprintings, Jonas commented in reference to abdominal delivery “...that are borne after this fashion be called cesares, for because they be cut of theyr mothers belly, whervpon also the noble Romane cesar...of that name in Rome toke his name...” [9].

The second part of the usual term for obstetric abdominal surgery, *section*, probably has its origin in the Latin verb *secare/seco*, meaning “to light, strike, or reach,” or “to cut into, separate, divide, or part” [10]. Another possibility is *incidere/incido*, meaning “to fall or on, happen, or occur” [6,10].

At some indeterminate time in the past, the terms used to describe the surgical operation for abdominal delivery, *cesarean* and *section*, became inextricably linked. Over time, however, the terms used to describe the surgery for abdominal deliveries have changed. In modern times, such surgical delivery of the fetus was referred to as a *cesarean operation* until the early twentieth century, when the term *cesarean section* became popular [1]. Currently, the term *cesarean birth* is frequently used in both lay and professional literature. Because of the redundancy inherent in the term *cesarean section*, we prefer to describe the surgical operation for the abdomen delivery of a child as a *cesarean delivery*, a *cesarean operation*, or simply as a *cesarean*. These conventions are used in the current text.

### Cesarean Delivery in the Historical Record

Beyond the mythology of the origins of the cesarean-related terms is also a long historical record of successful and not-so-successful abdominal deliveries. The oldest reliably recorded operations date back to the Sumerians in the second millennium B.C.E. More than 1,000 years later, Gorgias (483–375 B.C.E.), a famous orator from Sicily, is reputed to have been delivered by a cesarean. Records from as early as the second century C.E. report the operation several times, and in early Jewish literature Maimonides (1135–1204) mentioned cesarean surgery and commented on technique. It was not until the seventeenth century, however, that thoroughly documented cesarean deliveries are known to have been performed on living women with occasional maternal or fetal survivals. Many of the earlier reports are incomplete, wildly improbable, or so warped and embellished by multiple retellings that they remain suspect.

Commentary concerning cesarean delivery appears early in obstetric literature; however, many of the classic medical authors fail entirely to mention the procedure, attesting to its rarity. As an example, Soranus of Ephesus (98–138 C.E.) does not include cesarean operations in his review of surgical pro-

cedures. Sonanus did describe the management of obstetric malpresentation by version and extraction but did not mention the use of instruments or abnormal surgery for delivery. Aurelius Cornelius Celsus (27 B.C.E.–50 C.E.) in his book *De Re Medica* (c. 30 C.E.) is also silent on abdominal delivery yet provided instructions for the extraction of dead infants by the use of a hook or crochet. Cesareans are also not a part of the corpus of Hippocratic writings. Eucharius Rösslin the Elder's (also Roeslin, Roesslyn, or Rhodion) important, early obstetric textbook *Der Swangern Frawen und Hebammen Rosegarten*, published in Strassburg in 1513 and widely known as *The Roszgarten* (also *Roszgarten* or *Rosengarten*) does not mention the cesarean operation. As earlier noted, however, one of the many later editors or revisers of this book, Richard Jonas, did make such a reference in a commentary included in one of the many subsequent English language reprints of this remarkably long-lived textbook.

There are various reports of cesarean deliveries from numerous sources before the seventeenth century. Unfortunately, most simply document the danger of the procedure and the extreme risk to the mother's life. In Sweden, a postmortem cesarean operation was first recorded in 1360. Scipio Mercurio (1550–1616?), a surgeon of Padua, claimed several successful cesarean operations in his textbook *La Commare o Riccoglitrice*, published in 1596. In 1578, Giulio Cesari Aranzio (1530–1589) reported a successful postmortem cesarean delivery on a mother who had died late in the third trimester. Jacques Guillemeau (1544–1612) was surgeon to Henry and a student of the noted barber-surgeon Ambroise Paré (1510–1590). Guillemeau included a chapter on cesarean delivery in an obstetric text that was later translated into English by Thomas Hatfield in 1612 and entitled *Childbirth or, The Happy Deliverie of Women* [11]. Guillemeau stated that he had seen the operation carried out by various surgeons on a total of five women, all of whom had died. In his discussion of the procedure in this book, Guillemeau was among the first to introduce the word *section* into the medical literature.

The most controversial of the early reports of successful operative deliveries is that involving Jacob Nufer, a sow-gelder who is reputed to have performed a successful cesarean on his own wife circa 1500. The Jacob Nufer story was first related by Caspar Bauhin (1550–1624), more than 80 years after

the supposed event, in the appendix and commentary to Bauhin's Latin translation of a text entitled *Traité Nouveau de l'hysterotomie ou l'enfantement Caesarienne* printed in Paris in 1581 and originally authored by François Rousset (1535–1590?), physician to the Duke of Savoy [12]. Rousset, although not himself a surgeon, recounted cases of cesarean deliveries performed by others and claimed to have been an observer in still more, including several with maternal and fetal survivals. He argued that a cesarean was not only "a feasible operation" but also could preserve the lives of both mother and infant. As the title of his text reflects, Rousset termed the procedure a *cesarean delivery* or "enfantement Caesarienne" presumably in homage to the legend involving the birth of Julius Caesar [13]. The Nufer story was retold as late as the mid-eighteenth century by the reviewer and critic John Burton (1710–1771) in his textbook of obstetrics, *An Essay towards a Compleate New System of Midwifry*, published in 1751 [14].

As the Nufer tale is usually related, both lithotomists and midwives were called in consultation when the labor of Nufer's wife was obstructed. None of these attendants was able to bring the child forth, however. In desperation, Nufer himself performed a surgical delivery. His wife is supposed to have not only survived the operation but also later to have delivered other children vaginally. Although this entire story is suspect, it might contain a kernel of hidden truth. Because of the nature of his work in animal husbandry, Nufer would have had rough surgical and birthing experience. Such people with a functional knowledge of delivery mechanics were occasionally called on in the sixteenth century to help manage obstructed human labors. This might explain his active involvement in his wife's confinement. But, can the rest of this remarkable story be believed? Perhaps what Nufer's wife had was an advanced abdominal pregnancy. This could explain both her survival following an unsterile laparotomy and her subsequent unimpaired fertility. What actually happened in that Swiss hamlet in 1500, and the degree to which the Nufer story has been embellished and distorted over time, cannot now be determined as no new information is likely to be forthcoming.

In 1610, a physician in Wittenberg, Jeremias Trautmann, conducted the earliest well-documented cesarean delivery [15]. Although a surgery is known

to have been performed and a child delivered, the clinical details remain confusing. It is possible that what Trautmann actually found was an anterior uterine sacculation or an abdominal pregnancy. In other accounts the pregnancy was normal and the reason for surgery was a large ventral hernia that precluded normal labor. In fact, whether a pregnancy was even diagnosed before the operation is uncertain, and the infant might have been an unexpected discovery during a surgical exploration to relieve acute abdominal symptoms. In any event, an abdominal procedure was conducted, a child was delivered and is presumed to have survived although the extant records are at best incomplete. Unfortunately, the mother died some 25 days after the original operation, presumably from infection.

From the inception of the operation, controversy concerning the propriety of cesarean delivery has characterized the medical literature. It was recognized very early that postmortem operations on mothers dying in labor or late in pregnancy would rarely result in a normal and surviving child. Owing to the state of development of surgical technique, a cesarean was a virtual death sentence for both mother and infant until the early nineteenth century. To operate on a living woman was thus shunned, owing to the profound maternal risk from surgery and the uncertainty of success in salvaging a living infant. When labor was obstructed, version and extraction, fetal destructive procedures, and later symphysiotomy were the accepted methods for delivery. Whereas the mother often survived these obstetric manipulations and destructive procedures for vaginal delivery, in almost all cases the infant did not.

With this background, including horrific reports in the literature and their own experience with disastrous cesarean results, most of the influential obstetric educators of the sixteenth and seventeenth centuries, including Ambroise Paré (1510–1590), Jacques Guillemeau (1550–1630), Pierre Dionis (1643?–1718), and François Mauriceau (1637–1709), advised strongly against performing a cesarean operation on living women. Mauriceau, the most celebrated obstetrician of the late seventeenth century, discussed known obstetric procedures in his textbooks, *Traité Les Maladies des Femmes Grosses, et Accouchées* (Figure 1.1) [16] and *Observations Sur la Grossesse et l'Accouchement des Femmes, et sur Leurs Maladies, & celles des Enfants Nouveau – Nez* [17].



FIGURE 1.1.  
Title page of the *Traité* of François Mauriceau (c. 1668).

Mauriceau argued that only postmortem cesareans should be performed. He was well experienced in serious obstetric complications and knew firsthand of the limitations imposed by the inability of physicians to conduct abdominal deliveries. His own sister had experienced a serious antepartum hemorrhage from a placenta previa. When her attendants recoiled from intervention, Mauriceau had delivered her himself by version and extraction. Unfortunately, she did not survive this procedure [18].

In contrast, some early medical authors did support cesarean delivery. Jean-Louis Baudelocque (1746–1810) and André Levret (1703–1780) advocated cesareans for a contracted pelvis, in preference to the usual procedures of embryotomy, decapitation, or cranial decompression. The maternal and fetal results of most early cesarean operations were disastrous, however, reinforcing the argument for those who opposed such surgeries. According to Baskett [11], on one occasion, the noted French accoucheur Baudelocque was forced to defend himself in court when a contemporary called him an assassin because of Baudelocque's favorable opinions concerning cesarean delivery!

Cesarean deliveries were sporadically reported in the medical literature from the eighteenth through the mid-nineteenth century with generally poor results and often the loss of both mother and infant. In the early to mid-1700s cesarean deliveries were performed in Paris at a rate of approximately 1 per 4000 births. Unfortunately, the associated maternal mortality was 70% to 80%! A few successful abdominal deliveries did occur outside of the French capital between 1760 and 1814, however [19]. There were similarly grim statistics from the British Isles. There was not a cesarean delivery with documented maternal survival in Ireland until 1738, when a midwife, Mary Donally, operated on a 33-year-old multipara. In this case, Donally made a right paraumbilical incision with a razor; the incision subsequently closed with a tailor's needle and silk thread. The patient survived but later developed a ventral hernia. A cesarean delivery following a 6-day obstructed labor is also known to have occurred in England in 1737, but neither mother nor infant survived. In fact, a cesarean operation in England in which the mother is known to have survived did not occur until 1793 when the first case was reported. The mother in this instance had been in labor for three days when a sur-

geon, James Barlow delivered a dead child through a left paramedian incision [1]. From the same era there is an incompletely documented report of a successful cesarean delivery from America. Dr. Jesse Bennett (1769–1842) is supposed to have performed the procedure on his own wife in 1794 in Staunton, Virginia, following an unsuccessful effort at vaginal instrumental delivery. The details of this case are sketchy, and the documentation is poor. Thus, this claim is not generally considered credible. The first well-documented American report dates from 1827, when Dr. J. Cambert Richmond (1785–1855) performed an operation on a nulliparous eclamptic woman. Although the mother survived, the infant did not [20]. Another cesarean with maternal survival was performed before 1821 (exact date unknown) by the physician and surgeon James Miranda Barry in South Africa. Barry holds the unique distinction of being both an Edinburgh graduate and a woman who successfully masqueraded as a man from 1809 until her death in 1865 [18]. Africa is also the source for a report of another successful cesarean delivery performed by an unknown indigenous surgeon. In 1879, R. W. Felkin, a Scottish medical traveler in what later became Uganda in East Africa, witnessed and later published his observations concerning a cesarean delivery [21]. Preoperatively the surgeon cleansed his hands and the mother's abdomen with banana wine. The same fluid was administered orally to the mother before the surgery began, presumably to induce a degree of insensibility. After the delivery, which the surgeon performed through a midline incision, the uterus was not sutured. The abdominal incision was pinned together with iron needles and then secured by a bark-cloth string. Bleeding was controlled by cautery. Felkin claimed that the woman made a full recovery and noted the apparent expertise of the surgeon, concluding that the procedure was well established in that part of Africa.

In the late eighteenth century and into the early years of the nineteenth century, because of the serious risks of surgery, symphysiotomy vied with cesarean delivery as the best procedure for obstructed delivery. Intentional incision of the pubic symphysis was introduced to medical practice in 1768, when Jean René Sigault (1740–18??) described the technique in a single case [1,11,25]. Sigault successfully delivered a multiparous woman (a Madam Souchot), whose first child was lost owing



to an obstructed labor and a fetal demise, eventually terminated by an embryotomy. Her other deliveries had been equally unfortunate, resulting in stillbirths. For his efforts, Sigault received both a medal from the Faculty of Medicine in Paris, and a government pension. A medal was given to his assistant, Alphonse LeRoy (1742–1816), and to complete the awards, a pension was provided for the patient, who, despite a rocky postpartum course, including abscesses and a vesicovaginal fistula, survived! Despite such occasional successes, because of the manner in which symphysiotomy was performed, maternal morbidity and mortality were high. For these reasons, the procedure soon fell into disfavor and was not revived until the twentieth century. Symphysiotomy is still occasionally performed in parts of the nonindustrialized world as an alternative to a cesarean [23,24].

Prior to the late nineteenth century, several serious technical problems precluded safe cesarean deliveries. First, the operation was viewed as the last resort. It therefore usually was not performed until after prolonged labor, multiple examinations, manipulations, and various unsuccessful efforts at vaginal instrumental delivery. Inevitably, many of these women were exhausted and dehydrated, and most were infected. Surgical procedures at that time were also primitive. Before the invention of inhalation anesthesia in the late 1840s, surgery needed to be rapid. Only laudanum and alcohol were available as analgesic agents and the patient had to be actively restrained during the procedure. Furthermore, nothing was known concerning aseptic methods of surgery, ensuring a serious risk of infection. In the usual technique, the maternal abdomen was opened by a vertical incision, lateral to the rectus muscle. Attendants restrained the mother and, once the abdomen was entered, endeavored to hold back the intestines with their hands. The uterus was incised vertically and the child removed. Usually, the uterine wound was specifically not sutured because sutures were believed to predispose to complications, but the edges of the abdominal wound were usually reapproximated. Because of the timing of the operation, the absence of aseptic technique, and the failure to close the uterus, mothers usually rapidly died of hemorrhage or, if they lingered for several days, of peritonitis.

Progress was slow. The first reported instance of the successful use of uterine sutures at a cesarean

was by the surgeon Jean LeBas (1717–1787). In a 1769 delivery, he applied silk thread sutures to a uterine incision to stop hemorrhage. The patient subsequently recovered. Inevitably, LeBas was heavily criticized by his contemporaries. After LeBas' report, several attempts at routine uterine suturing occurred in individual cases, usually with disastrous results [11].

From our vantage point, it is hard to understand why suturing of the uterine wound during a cesarean was considered inappropriate until almost the beginning of the twentieth century. This practice followed then-contemporary clinical experience and well-established surgical technique, however. A common reason given for not suturing the uterus routinely after a cesarean was the belief that rapid uterine involution would inevitably loosen any stitches, rendering them ineffective. Another problem was infection. In the eighteenth and well into the nineteenth century, sutures placed by a surgeon were routinely left long, protruding from the wound. This was believed necessary to facilitate drainage and to provide access for the eventual removal of the sutures, which usually were not absorbable and, of course, not sterile. Conventional wisdom and clinical observation held that deeply placed sutures invariably became infected, leading to abscess, cellulitis, or sepsis. A wound left open, with the suture ends exiting the skin, would eventually begin to develop what was termed laudable pus, however. With time, progressive tissue necrosis would eventually release the sutures. The usual practice was that several days after the surgery the surgeon would begin intermittently to pull gently on the suture ends. This process was subsequently repeated once or twice daily until local necrosis was sufficient to permit the extraction of the sutures without eliciting a hemorrhage. For patients who survived to the point of suture removal, eventual recovery was likely. After suture removal, the wound would slowly heal by secondary intention. Once the process of granulation was well advanced such wounds were quite resistant to infection and unlikely to lead to cellulitis or sepsis. Unfortunately, when such standard surgical techniques were used in cesarean deliveries, hemorrhage and infection were routine, with serious and usually fatal consequences for the mother.

When uterine reapproximation was finally introduced, silver wire became the initial suture material of choice, mirroring its use in nineteenth century

gynecology. Frank E. Polin of Springfield, Kentucky, first reported the use of silver wire in the closure of a uterine wound in 1852. Other than silver wire, many other types of suture were in use, derived from a wide range of materials including silk, carbolized gut, horsehair, and even hemp. What would now be considered as appropriate uterine approximation with nonpermanent suture materials was not introduced until the early 1880s.

Many important surgical innovations begun in the mid-nineteenth century eventually made safe cesarean deliveries possible. Ether was first used during labor in Boston in 1847 and subsequently popularized by the socially prominent New England obstetrician Walter Channing (1786–1876). The anesthetic properties of chloroform were discovered by James Young Simpson (1811–1870) and first employed by him in deliveries in Edinburgh beginning in 1847 [11].

A major breakthrough in the technique of cesarean surgery occurred in the early 1880s. Max Sänger (1853–1903), then an assistant to Carl Siegmund Franz Credé (1819–1892) in Leipzig, introduced an operative procedure in 1882 that is now considered the classic cesarean operation. In doing so, Sänger revolutionized standard cesarean surgical technique [26]. In a general review for a monograph concerning the cesarean operation, Sänger had collected published case reports of prior deliveries that he carefully reviewed and critiqued. Based on these data from the literature and his own experience, Sänger argued that operative complications from cesareans would occur less frequently if the myometrium were closed and a concerted effort made to avoid the spillage of intrauterine secretions into the peritoneal cavity [26]. His procedure featured a meticulous, water-tight reapproximation of the uterine wound, employing buried sutures. Sänger also exteriorized the uterus before delivering the infant and attempted to improve postoperative drainage by passing a drain from the fundus out through the cervix.

Although maternal morbidity and mortality from cesarean deliveries remained high even with Sänger's improvements, statistics were substantially better with his technique than the levels previously experienced. It was only after Sänger's 1882 paper that closure of the uterus was finally recognized as both a feasible and necessary part of cesarean technique [1].

Horatio R. Storer, of Boston, Massachusetts, first performed a cesarean hysterectomy in 1868, on a woman with a large leiomyoma that obstructed the birth canal. He removed the uterine corpus and adnexa during this procedure. The child was stillborn and "in an advanced state of decomposition." The mother died three days later. The first maternal survivor following cesarean hysterectomy occurred in 1876, when a woman with rickets and pelvic contracture was delivered by Eduardo Porro (1842–1901) [1,27]. What later was termed the *Porro operation* was a unique surgical procedure originally suggested by the Florentine surgeon Joseph Cavallini in 1768. Cavallini and later Porro had experimented with pregnant hysterectomy in animal models. Cavallini had operated on dogs and sheep; Porro had used rabbits. Each had proved to his satisfaction that the uterus was not necessary for life and that its surgical removal was technically possible.

In early 1876, Porro encountered a 25-year-old nullipara with a rachitic pelvis and a true conjugate of 4 cm or less, precluding vaginal delivery. Following careful consideration and preparations, including preliminary handwashing with carbolic acid, Porro performed a classic cesarean delivery by means of a midline abdominal incision, with the patient under chloroform anesthesia. After delivery of the baby, an iron-wire snare was passed around the uterus, tubes, and ovaries. All these structures were then amputated and the remaining cervical stump was brought out of the abdomen through the lower end of the midline incision. Drainage tubes were inserted and the abdominal wall was then closed around the residual stump with silver-wire sutures. The snare was removed on the fourth day and the sutures on the seventh. The exteriorized cervical stump and lower portion of the abdominal wound were then permitted to heal by secondary intention. Six weeks later, the woman left the hospital with her infant. Remarkably, she was the first to survive a cesarean delivery performed at that clinic!

The Porro operation rapidly gained acceptance in Europe because it radically solved the problems of both hemorrhage and infection. Maternal losses with the Porro operation remained high but were substantially below those experienced before the procedure was introduced. By 1884, approximately 140 of these operations had been reported in Europe, with a maternal mortality rate of 56%. After 1882, the classic cesarean operation without

hysterectomy as popularized by Max Sänger began to replace Porro's operation as the surgical technique of choice because the rates of maternal morbidity and mortality were lower. By the onset of the twentieth century, the Porro operation had been entirely superseded.

Despite these and other innovations, cesarean delivery did not gain popularity with practitioners until well after the introduction of aseptic technique by Joseph Lister (1827–1912) and others in the latter decades of the nineteenth century. Drawing upon the new discoveries in bacteriology and the development of the germ theory of infection, the combination of improved anesthesia and new surgical methods finally blunted the horrific rates of maternal morbidity and mortality associated with cesarean operations [28]. The great safety of cesarean delivery still awaited changes introduced during the twentieth century.

The rapidly falling mortality rate of cesarean hysterectomy expanded the potential indications for the operation. Cesarean hysterectomy became progressively popular during the period from the late 1940s to the mid 1960s, and was often performed for sterilization. In recent decades, because of the substantial morbidity of the operation, cesarean hysterectomy has fallen from favor as an elective method of sterilization. At present, this procedure is generally restricted to management of uncontrolled hemorrhage, the rare case of nonreparable uterine injury, or for other reasons of severe uterine or cervical pathology. In recent years, the availability of potent uterotonics and broad-spectrum antibiotics, the development of embolization techniques, and new methods of vessel ligation have markedly reduced the need for emergency cesarean hysterectomy, although it still remains an important and potentially lifesaving procedure (See Chapter 18, Cesarean Delivery).

Other innovations in surgical technique lessened the risks of surgery. Maternal complications from cesarean deliveries were reduced by the development of the lower-segment cesarean operation, a procedure originally suggested by Johann F. Osiander of Goettingen (1759–1822). In 1805, Osiander opined that entry into the uterus through a vertical lower-segment incision could avoid the complications of the usual surgical technique, which then involved a vertical incision in the upper and thicker portion. More than a century later, Bernard Krönig

(1912) revived this idea and proposed dissecting into the vesicouterine space and subsequently using the bladder serosa to cover the uterine incision, to protect the peritoneal cavity from exposure to the lochia. This combined technique of a lower-segment uterine entry and sequestration of the myometrial wound behind the peritoneum resulted in less immediate surgical morbidity and substantially reduced the risk of uterine rupture in subsequent pregnancies.

The extraperitoneal cesarean operation has an interesting history [20]. This procedure was first proposed by W. E. Horner in 1824. Such procedures were not performed until Alexander Johnston Chalmers Skene (1838–1900) successfully delivered a woman with a rachitic pelvis by this technique [7]. In 1909, the extraperitoneal operation gained support when Wilhelm Latzko of Vienna reported only two maternal deaths among thirty such procedures. Latzko's paravesical, extraperitoneal operation was later popularized in the years prior to World War I by E. G. Waters [29] and J. F. Norton [30]. The theoretical advantage of this operation was to isolate the entire operative site retroperitoneally and thus potentially avoid the risk of peritoneal contamination. The progressively increasing safety of the transperitoneal approach, the rapidly decreasing incidence of protracted, dystocic labors, and the advent of antibiotics markedly reduced the importance and advantage of the extraperitoneal operation, however. It is now uncommonly attempted.

In recent decades, additional modifications in cesarean operative technique have been introduced. New and less tissue reactive suture materials are now available. In routine operations contemporary surgeons now frequently omit the serosal or vesicouterine flap closure and closure of the parietal peritoneum in an effort to reduce adhesion formation. The standard methods for both opening and closing both the fascia and uterus also have changed, at least for many surgeons, replacing the traditional sharp entry by techniques of blunt dissection and employing running as opposed to interrupted sutures for closure. Perhaps the most marked change in cesarean practice in the last 75 years has not been in surgical technique, however, but in the remarkable reduction in serious maternal morbidity and mortality associated with the operation by the administration of prophylactic antibiotics, the rapid

development of medical therapies to treat complications, and general improvements in anesthesia. The overall mortality risk for unselected cesarean operations has fallen to 1 per 1,000 or less owing to these various advances and improvements.

### INSTRUMENTAL DELIVERY

The development of atraumatic delivery instruments is a complex and fascinating part of the history of obstetrics [31–36]. Beginning 200 years ago, a remarkably small group of innovators developed and perfected new types of obstetric instruments. Their trials, false starts, occasional successes, and many failures make for a rousing tale that involves trade secrets, professional jealousy, true altruism, a touch of scandal, and inevitably, the search for profit and fame. Beyond technical considerations concerning instruments or technique, practitioners of the past were also well aware of the potential risks and benefits of the use of instruments in obstetric practice and of the classic alternatives, either a cesarean or a destructive operation. They sought to develop vaginal delivery devices that were safe, effective, and ultimately lifesaving. The different approaches that contemporary accoucheurs have toward instrument-assisted delivery mirrors a two-century-old tension between contending philosophies of obstetric practice. This persisting and irresolvable controversy is between those willing to intervene versus those whose preferences are to wait and observe. The balance in the relative ascendancy between these positions is influenced by various advances in the field of obstetric practice, including the periodic publication of critical reevaluations of traditional obstetric procedures, the introduction of new instruments, the popularity of novel techniques or procedures, the complex pressures of society, and medicolegal trends.

Prior to the introduction of safe delivery instruments, intravenous fluid therapy, blood transfusion, potent antibiotics, and potent uterotonics, the options available to birth attendants when labor was obstructed were starkly limited. The mother could be permitted to continue to labor at high risk for her own injury and for the loss of her child in the hope of an eventual vaginal delivery. Alternatively, version and extraction, symphysiotomy, or a procedure destructive to the fetus could be performed. Such procedures might save the mother but often did so

at the cost of severe or fatal fetal injuries. Furthermore, before the late nineteenth century, attempts at any intervention were often delayed until the situation was nearly hopeless, effectively determining the outcome. Abdominal operations such as cesareans were uncommon prior to the latter part of the nineteenth century. Surgery was brutal, far from safe, and performed without anesthesia. As discussed in the previous section, cesarean delivery did not become an acceptable option until after the mid 1880s owing to the horrific risks of hemorrhage and infection and the limitations of anesthesia. It was in this formidable setting that nondestructive delivery instruments were first invented.

Modern obstetric delivery forceps are the highly modified descendants of instruments destructive to the fetus that date from antiquity [31,32,34]. The term *forceps* most likely takes its origin from a contraction of a Latin root word, either *ferriceps* (*ferum*, meaning “iron,” and *capio*, meaning “I take”) or *formus* (meaning “hot”) combined again with *capio*.

Although destructive instruments including hooks and other extraction devices are accepted as ancient, the date of invention for nontraumatic obstetric forceps is the subject of debate. Atraumatic instrumental delivery devices were unknown to the Greeks and probably to the Romans as well, although the latter is not completely certain. If the Romans ever had a nondestructive delivery forceps in their armamentarium, this device was lost over time and did not influence later developments. Destructive instruments, including cranial perforators, hooks and various cranial grasping devices, however, date to antiquity.

Various two-bladed, scissor-like metal instruments designed for obstetrical applications were in use by approximately 1000 C.E. and were known to Albucasis (1013–1106) and his contemporaries, Avicenna (c. 980–1037) and Maimonides (1135–1204). Jacob Rueff's (1500–1558) textbook *De Conceptu et Generatione Hominis* from 1544 illustrates such instruments (Figure 1.2). A surgeon and obstetrician in Zurich, Rueff drew his information largely from Soranus and from the previously mentioned text by Rösslin, usually entitled the *Rosengarten*, initially published in 1513. Unfortunately, devices depicted in this text were quite clearly designed for the destruction and removal of the fetus from the uterus and not to assist in the delivery of living infants. Atraumatic delivery required the

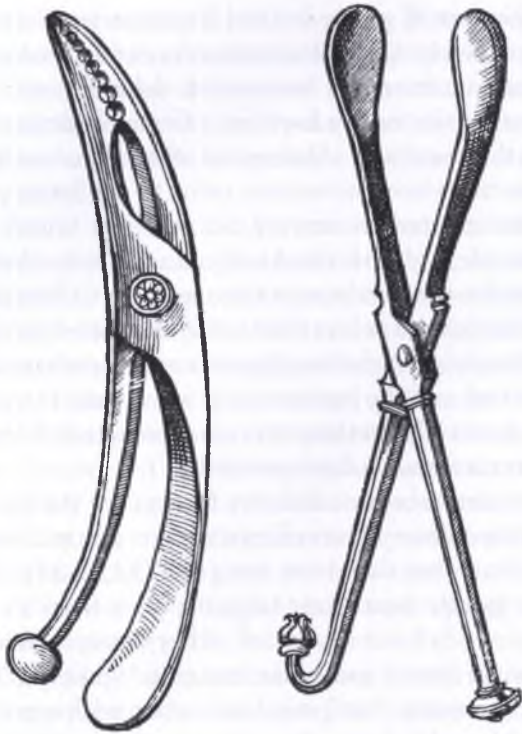


FIGURE 1.2.  
*Delivery instruments illustrated by Jacques Rueff in De Conceptu et Generatione Hominis (1554). (Courtesy of the Historical Division/Cleveland Health Sciences Library, Cleveland, OH.)*

development of instruments capable of two different but related tasks: grasping the fetal head securely and permitting cranial rotation and traction. Both of the tasks also had to be accomplished without resulting in serious maternal injury. Neither technical limitations nor the lack of surgeons delayed the development of safe delivery instruments, however. Europe had many talented medical fabricators in the flourishing armament industry of the sixteenth and seventeenth centuries who could easily have produced metal scissor-like instruments like forceps on demand. The problem was twofold: first, the requirement to identify the need for such instruments, and second, the recruitment of sufficiently skilled practitioners to direct the transformation of initially destructive instruments into atraumatic delivery instruments. These changes awaited the Chamberlens.

During the reign of Charles , religious persecution drove many Protestants from France, including William Chamberlen (c. 1540–1596), a medical practitioner who subsequently established his



FIGURE 1.3.  
*Chamberlen delivery forceps c. 1610 (facsimile). (Courtesy of the Dittrick Museum of Medical History, Historical Division/Cleveland Health Sciences Library, Cleveland, OH.)*

family in England [22,31,34,37]. By the late sixteenth century, the two sons of William Chamberlen were actively practicing medicine in London, working as barber surgeons and heavily involved in midwifery. Which of the brothers, Peter Chamberlen “the elder” (1560–1631) or Peter Chamberlen “the younger” (1572–1626) was the inventor of obstetric forceps is not clear, although Peter the Elder is usually give the credit. Although the process that led to the development of the Chamberlen instrument is unknown, it is believed that a practical forceps model was first developed after 1610 and then later modified several times based on clinical experience (Figure 1.3).

The Chamberlen delivery forceps were not released for general use after their invention, and for decades the instruments remained closely guarded as a family trade secret. The Chamberlen brothers and many of their descendants held themselves out as obstetric consultants. As such, they provided the public access to their secret method of delivery (the forceps) for a fee. Once they had been called in consultation, their “secret instrument” was delivered to the lying-in site in a large, gilded box [37]. All of the original birth attendants were then excluded from the room. The forceps were then removed from the box in such a fashion so as not to be seen by the parturient. As was usual continental practice, the delivery was conducted under the cover of a sheet that covered the parturient’s bed and was tied behind the accoucheur’s neck. His drape in place, the surgeon would sit at the end of the bed, grasp the forceps, and commence the procedure. Thus, both his manipulations and the delivery forceps were hidden under the sheet. After the delivery, the instrument

was replaced in its box and the delivery fee claimed. Because of this process, neither the woman nor her family or friends could attest to what had actually occurred, and thus the secret remained secure.

A later and somewhat unsavory member of the Chamberlen family, Hugh the Elder (1630–17??) was a notable entrepreneur and self-styled deal-maker. In 1670, hoping to raise money he went to Paris and offered to sell the family secret to the noted French obstetrician François Mauriceau for what was then a large sum of money [3,33]. Mauriceau provided a test case, a woman with a markedly deformed pelvis in obstructed labor. Despite Chamberlen's prolonged and heroic efforts, both the women and infant died. At a later postmortem examination, the uterus was found to be ruptured. Not surprisingly, this sale fell through. Despite this debacle, Chamberlen managed to secure an agreement from his French colleague to translate Mauriceau's textbook, the *Traité*, into English. On his return to London, Chamberlen published a version of this book in English, initially entitled *The Accomplish't Midwife* (1672). The text proved highly successful and at least eight subsequent editions were printed. This literary and professional coup was a substantial contribution to midwifery practice in England and improved Chamberlen's prominence in his profession while also helping to attract a large clinical practice, thus improving both his social and financial position [22].

Forever embroiled in political affairs and financial schemes, Hugh the Elder subsequently encountered sufficient difficulty in England to induce him to flee to Holland. During his five years on the Continent, it is suspected that he sold obstetric instruments to either Hendrik van Roonhuysen [also Roon-Huyse, Roonhuysen] (1615–1672) or more likely his son and successor Rogier van Roonhuysen (c. 1650–1709), both surgeons in Amsterdam [33,36]. This sale probably occurred after 1693 or perhaps 1695. Although details of this transaction are extremely sketchy, this commercial deal could have first introduced an atraumatic delivery device to Europe. It is also possible that no sale of an instrument actually occurred. The Amsterdam forceps might have been an independent invention. It is also possible that what van Roonhuysen received from Chamberlen was only the *idea* for a delivery instrument that he later independently refined, rather than an actual working model. Paralleling the example set

by the Chamberlen family, the sale also permitted Roonhuysen and his successors to hold the use of this instrument (or perhaps instruments) as a local monopoly for more than 50 years. With the payment of a substantial fee, practitioners who passed the examination for the Amsterdam Surgeon's Guild were permitted introduction to this secret delivery instrument.

Various modifications to the original Chamberlen design or one or more vectus blades independently developed either by Roonhuysen or his close associates, Jean (or Joannes) de Bruin (1681–1753), Paulus deWind of Middleburg, and Regner Bloom of Amsterdam, eventually came to public notice in the Netherlands after 1747. This occurred partly because several practitioners, including a disgruntled applicant to the Amsterdam Surgeons Guild, John Peter (or Jan) Rathaw (also, Rathlaw, Rathlauw; 1720–1791), and Van der Suam (or Swam), a former pupil of Rogier van Roonhuysen, wished to finally break the Amsterdam monopoly [33,36]. As published by Rathaw and later independently by another surgeon in 1747, Daniel Schlichtingting (1703–1765), the revealed van Roonhuysen secret instrument consisted of a type of forceps, quite different from the known Chamberlen models, with thin, bandlike parallel blades and no pelvic curve. This instrument was articulated only at the distal end of the handles.

On his deathbed in 1753, van Roonhuysen's closest pupil, Jean de Bruin, gave his original delivery instruments as a legacy to two friends, J. de Vischer and H. van de Poll. They subsequently published a description of one of these instruments in a text entitled *The Obstetric Secret of the Roonhuysens Discovered* (Leiden, 1753). What they revealed in this paper was a single-bladed device slightly curved at both ends and covered with dog leather. This instrument is best described as a modified lever or vectus blade.

The entire story of the van Roonhuysen's secret instrument(s), including what these instruments actually were, who was involved in the various transactions concerning these devices, and whether any of the "revealed" instruments were actually obtained from Hugh Chamberlen remains cloudy [36,38]. It is also uncertain if these forceps and vectus blades were actually invented independently by the van Roonhuysens or somehow inspired by their viewing an earlier model of the Chamberlen forceps.

Apparently, the Amsterdam group used two instruments, a vectus blade and a type of forceps. Part of the confusion lies in separating the “release,” or publication of the description of these separate delivery instruments, from their actual invention (or modification?). Owing to the various claims and counterclaims by the people involved and our distance from the actual events, no resolution concerning what the Amsterdam cartel either purchased from Chamberlen or independently invented seems likely. Apart from the quibbles concerning its origin, the van Roonhuysen extractors proved to be poor competition for the Chamberlen forceps. Forceps based on the Amsterdam model never became popular and had little influence on future developments. While possibly representing a true independent invention, the van Roonhuysen forceps remain now as a historical curiosity only. Of interest, the use of levers or vectus blades remained common in the Netherlands well into the nineteenth century. These instruments might be the only lasting obstetric contribution that can be ascribed to the Amsterdam group (Figure 1.4).

Other delivery instruments also became available in the early to mid-eighteenth century. Independently of the Chamberlens, Johannes Palfyn of Ghent (1650–1730), a surgeon and anatomist with an uncertain interest in midwifery, developed a two-bladed delivery instrument, his *tire-tête* or *mains de fer* [11,22,36]. This device was demonstrated in Paris, probably in 1720, at a meeting of the Academie Royale de Sciences. This instrument was also presented to the Medical Faculty of Paris in

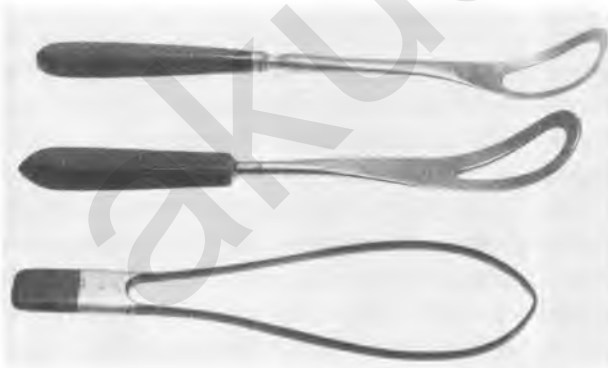


FIGURE 1.4.  
*Vectis blades and whale bone fillet c. 1850. (Courtesy of the Dittrick Museum of Medical History, Historical Division/Cleveland Health Sciences Library, Cleveland, OH.)*

1723. Unfortunately, Palfyn never published anything on either the construction of this instrument or its clinical use. All information about his forceps comes from the comments of his contemporaries and his critics. Palfyn might have derived the inspiration for his invention from a vectus blade instrument originally developed by the noted French surgeon Ambroise Paré (1510–1590). Palfyn’s innovation was to employ two blades, each with cephalic curve fitted to the sides of the fetal head. These blades were neither crossed nor otherwise articulated together, and they also lacked a pelvic curve. In its original description, the device was likened to a pair of “artificial hands” designed to assist the delivery of the fetal head. Thus their name, “iron hands” or *mains de fer*. Later, other practitioners including Guilles Le Doux of Ypres (c. 1710) and Grégoire the Elder (?–1730?) bound the two parallel blades together with a cloth tape or strap to try to increase their clinical utility. Parallel blades have a technical advantage over other forceps’ designs since they avoid the cranial compression inherent in the scissor-like articulated blades of most instruments, including those of the Chamberlens. As a parallel-blade device, however, the Palfyn instrument had major technical problems. The lack of a pelvic curve restricted its potential use. Even with the wrapping of the shanks, the instrument proved unstable and was largely ineffective in clinical use.

Palfyn’s device never achieved popularity owing to its technical limitations, marginal utility, and professional opposition from distinguished contemporaries. One of the most vocal critics, the noted accoucheur Guillaume-Manquest de la Motte (1665–1737), publicly denounced the *mains de fer* as both impractical and dangerous, which they most likely were. After this unfortunate trial presentation, nothing further was heard concerning Palfyn’s instrument, and it disappeared from obstetric history.

Instability is a design problem for all parallel-blade instruments because traction immediately drives the blades laterally, predisposing them to slippage. In addition, if parallel blades are unsupported by a firm locking mechanism they can be easily twisted, risking lacerations of the birth canal. Following redesign and crossing of the blades and the fitting of a screw-based lock (Duseë modification) a later modification of the Palfyn instrument actually was made clinically usable. This instrument never

gained popularity and had little influence on subsequent forceps design, however [36]. Many years later, once the problems of blade articulation and stability had been solved, Lafe, Shute, and others successfully revived the parallel-blade design for obstetric forceps [39–41].

In the mid-1730s, following the publication of several case reports and informal exchanges between several practitioners, obstetric forceps of varying types rapidly came into general use in England. Thus in 1733, in his text *A Treatise on the Improvement of Midwifery*, Edmund Chapman (1680–1756) mentioned that forceps were instruments well known to his contemporaries [22]. Other practitioners, including William Giffard (?–1731) and Benjamin Pugh (c. 1710–1775) also reported using forceps before 1750 [33,36]. Exactly how the secret of the forceps was revealed to these accoucheurs remains unknown. It cannot be simply a coincidence that the several physicians most involved in popularizing these early, Chamberlen-like instruments all worked in Essex, England, in reasonable proximity to the Chamberlen estate. Unfortunately, the details of this potentially fascinating part of the forceps story are now irretrievably lost.

Modifications to these early delivery instruments were required before they achieved popularity and utility. Both the Chamberlen and Palfyn forceps were short and straight and lacked a pelvic curve. Owing to these design limitations, they would have been useful only as low or outlet instruments. To improve performance, André Levret (1703–1780), William Smellie (1697–1763), and Benjamin Pugh (c. 1710–1775) independently added a pelvic curve [11]. This helped to accommodate the forceps blades to the birth canal, and the new instruments that incorporated this modification were capable of more accurate and less traumatic applications. This improvement was introduced at the same time as the French obstetrician Jean Louis Baudelocque (1746–1810) developed a technique for estimating pelvic capacity by taking external measurements with a large caliper or pelvimeter. His studies of pelvimetry demonstrated the importance of pelvic shape and various pelvic dimensions in the mechanism of labor, thus improving the understanding of how instruments should be used [22].

The newly modified cross-bladed forceps that incorporated the pelvic curve provided an attractive alternative to the dreary triad of heroically pro-

longed labors, attempted version and extraction, and destructive operations that had characterized earlier practice. Unfortunately, the indiscriminate use of instruments, often by the inexperienced, led to abuse. Knowledge of techniques for safe application and training to disseminate improvements in technique lagged well behind the enthusiastic application of these new devices.

Overuse of instruments provoked the expected response. An era of lively debate concerning the appropriate use of instruments ensued, much of which was strikingly similar to modern discussions. The result was that several of the best eighteenth century English practitioners, including William Smellie (1697–1763) and his student William Hunter (1718–1783), taught the conservative use of instruments. Although Hunter clearly knew how to use forceps, he took pride in noting that his pair was so little used that they were covered in rust. Practitioners of an even more conservative school of obstetric management, including Thomas Denman (1733–1815), William Osborn (1736–1808), and Richard Croft (1762–1818), favored extreme prolongations of labor rather than any resort to instrumental assistance [11]. In their view, the risks attendant to instrumentation outweighed any potential. The general guidelines for appropriate forceps use as designated by the conservative school would be quite unacceptable by modern standards [36]. These included

- No intervention is to be performed if any progress is noted, no matter how slowly, unless fetal demise is diagnosed;
- No intervention may be considered until the head has been on the perineum for >6 hours;
- Forceps are to be used only for the most urgent occasions, and then sparingly.

A famous and poignant reminder of the potentially serious error of failing to intervene despite strong indication was the childbirth death of George IV's only heir, Princess Charlotte, in 1817 [42]. The royal obstetrician, Sir Richard Croft, was a socially prominent and fashionably conservative practitioner. The Princess' labor lasted 50 hours, and the child was stillborn. Six hours following the delivery, Princess Charlotte died from what is now presumed to have been exhaustion, dehydration, and hemorrhage. Forceps were available but never



used. Later, in the face of intense public and professional criticism concerning his obstetric management, Croft committed suicide. Beyond the tragedy of these three related deaths, the event also presented a major political crisis. With the death of Charlotte, there was no legitimate heir for George. If no legitimate heir could be produced, the English crown would pass to a distant Hanoverian relative, the Duke of Brunswick, a young cousin of George. Eventually, after active intervention, a suitable bride of proven fertility for the king's brother was found. A successful pregnancy and delivery followed in 1819. Through this somewhat unusual mechanism, the English crown passed to the King's niece. In 1837, this woman assumed the English throne and was crowned as Queen Victoria, who proved to be the longest reigning of the English monarchs. The Princess Charlotte debacle and other similar events eventually discredited the ultraconservative school of obstetric management, and by the middle decades of the nineteenth century led to a more balanced view of the role of assisted delivery.

The extensive use of instrumental delivery was an event of the latter part of the nineteenth and the early twentieth century. Before the late 1840s, the incidence of forceps use both in England and the continent was 1% or less in large clinical services (Tables 1.1 and 1.2). Fleetwood Churchill was among the first practitioners to publish birth statistics. In his *Research on Operative Midwifery* (1841), he presented data summarizing experience in the late eighteenth and early nineteenth century (Table 1.1) [36]. These data indicate that both forceps and operations destructive to the fetus occurred in substantially fewer than 1% of all deliveries.

In 1875, T. More Madden of the Rotunda or Dublin Lying-in Hospital reported delivery data collected from 1787 to 1874 during the directorship of seven hospital masters (Table 1.2). As had been reported by Churchill, instrumental delivery was uncommon (0.5%) until midcentury. Thereafter, the rate rose from 1.6% for the interval 1847–1854 to 9.2% by 1868–1874.

During the interval from the eighteenth century until the latter decades of the nineteenth century, the percentage of procedures destructive to the fetus remained relatively stable, at approximately 0.4%. The increase in operative forceps deliveries probably reflects several factors:

**TABLE 1.1 Frequency of Forceps Use and Craniotomy or Operations Destructive to the Fetus in the Late Eighteenth and Early Nineteenth Centuries\***

Instrument Employed	Deliveries	Operations Performed (%)
<b>Forceps</b>		
British 1781–1840	42,196	120 (0.28)
French 1797–1831	44,736	277 (0.62)
German 1801–1837	261,224	1,702 (0.65)
Total	348,156	2,099 (0.60)
<b>Perforator and Crotchet</b>		
British 1781–1819	41,434	181 (0.45)
French 1797–1811	36,169	30 (0.08)
German 1801–1837	256,655	132 (0.05)
Total	334,258	343 (0.10)

\*As reported by Churchill, 1841. Modified from Hibbard [36], reprinted with permission.

**TABLE 1.2 Operative Deliveries at the Dublin Lying-in Hospital Under Various Masters: 1787–1874\*†**

Mastership	Deliveries	Forceps (%)	Perforator (%)
Joseph Clarke 1787–1794	10,387	14 (0.13)	49 (0.47)
Samuel Labatt 1815–1822	21,867	0	0
Robert Collins 1826–1833	16,654	24 (0.14)	118 (0.71)
Charles Johnson 1842–1833	6,702	18 (0.27)	54 (0.80)
Total	55,610	56 (0.10)	221 (0.40)
Robert Shekleton 1847–1854	13,748	220 (1.60)	54 (0.39)
A. H. McClintock 1854–1861	3,700	76 (2.05)‡	5 (0.14)
George T. Johnston 1868–1874	7,027	639 (9.1)	29 (0.41)
Total	24,475	935 (3.82)	88 (0.36)

\*As reported by More Madden, 1875.

†From Hibbard [36], with permission.

‡Includes vectis blade operations.

the availability of anesthetic agents after 1849, the development of new delivery instruments, and changing concepts of obstetric management. The rate of destructive procedures remained unaltered because of technical problems in ascertaining fetal condition and the inability of clinicians to perform cesarean delivery without extreme maternal risk.

Of interest, and as a reflection of the difficult cases presented to these practitioners, Hibbard [36] reports that the maternal mortality from forceps procedures varied from 4.8% (14/294; England) to 7.3% (35/479; Germany and France). In comparison, maternal losses from destructive operations to the fetus (predominantly perforation) were an astounding 21% (52/251)!

In the middle and late nineteenth century, obstetrics underwent rapid changes. Advances in therapeutics accompanied the development of many new delivery instruments and techniques. The introduction of anesthesia in the late 1840s and the development of new instruments and aseptic practices in the 1880s profoundly changed obstetric practice, permitting both sufficient time and relative safety for various surgical procedures.

In the latter part of the nineteenth century, instrumental delivery by forceps became more common and the procedures more extensive. Both more difficult and ever-higher procedures were progressively attempted, including operations performed before full cervical dilation. Hibbard [36] suggests that this more aggressive use of forceps arose from a then general belief that once the membranes ruptured, uterine inertia was common. In this setting some type of intervention was therefore thought to be appropriate.

The English obstetrician James Young Simpson (1811–1870) and his American contemporary George T. Elliot (1827–1871) were among the most prominent practitioners of the mid-nineteenth century [11,43,44]. Simpson, a highly regarded and influential obstetrician working in Edinburgh, developed not only a type of forceps but also the first effective obstetric vacuum extractor. His specially designed forceps were introduced in 1848, rapidly became popular, and are still in use. A man of many interests, Simpson authored papers on hospital design, mesmerism, acupuncture, and homeopathy, among other subjects. He also played a pivotal role in obstetric anesthesia, discovering the anesthetic properties of chloroform, which by 1848 he

had employed during deliveries and in the treatment of eclamptic seizures.

George T. Elliot (1827–1871) introduced his midwifery forceps in 1858. To limit compression of the fetal head, he included a setscrew in the instrument handle to control the degree to which the handles of the forceps could be approximated. Both his instrument and Simpson's remain among the most popular designs and are in common use today [32].

In the waning years of the nineteenth century, awakening interest in the mechanism of labor was reflected in the design of new instruments. Following earlier designs of Louis Joseph Hubert of Louvain (1810–1876) and his son E. Hubert and others, Etienne Stephane Tarnier (1828–1897) and Charles P. Pajot (1816–1896) introduced axis-traction forceps. These devices were developed to align the vector of traction with the pelvic curve, thus improving success and using force in a more judicious and less traumatic manner.

Friedrich Wilhelm Scanzoni (1821–1891) popularized rotational maneuvers, especially for management of occiput posterior positions [11]. Modifications of his grand rotation are still occasionally performed today (see Chapter 17, Instrumental Delivery).

Following an idea originally proposed in 1799, by Friedrich Oslander (1759–1822), solid-bladed forceps were popularized by James Woods McLane (1839–1912). To facilitate rotations, these forceps were modified with the addition of longer shanks by Ervin A. Tucker (1862–1902). Later, Ralph Herbert Luikart (1889–19??) modified these blades by selectively thinning the inner portion [45]. Such pseudo-fenestrated forceps blades retained the advantages of easy rotation inherent in the solid design yet maintained a firm grip on the fetal head. This modification remains popular and has been applied to several forceps types.

Rather than an inventor, the most important influence on American delivery practices in terms of instrumentation in the early part of the twentieth century was the medical educator, Joseph Bolivar DeLee (1869–1942). In the 1920s he described the prophylactic forceps operation [46]. Despite the lack of supporting data, DeLee championed the routine use of forceps combined with episiotomy for shortening the second stage once the fetal head had reached the pelvic floor, as a means of avoiding intracranial injury. This concept of routine operative

delivery for both maternal and fetal reasons – even though unsupported by data and based on theoretic concerns – was widely followed and strongly influenced North American practice for more than four decades.

Many other clinicians practicing in the early twentieth century designed modified forceps for specific clinical indications. These included the instruments introduced by Lyman Guy Barton (1866–1944) for transverse arrest, Arthur Holbrook Bill (1877–1961) for axis traction, Edmund Brown Piper (1881–1935) for breech delivery, and Christian Casper Gabriel Kielland (1871–1941) for mid-pelvic rotations. Recent years have seen the development of various new forceps designs, such as those of Laufe, Hays, Nargolkar, and Salinas, among others. These new devices attempt to improve maternal and fetal safety through specific aspects of their design.

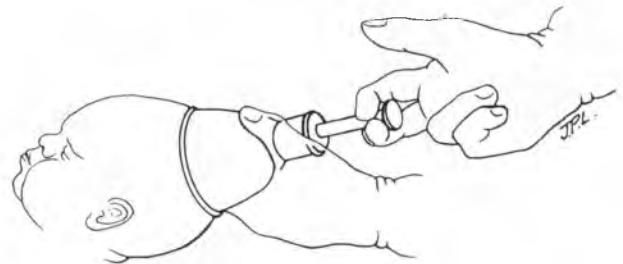
### **HISTORY OF THE VACUUM EXTRACTOR**

Vacuum delivery instruments have their origin in the very old practice of cupping [38,47]. In cupping, a metal or glass cup or globe is heated over an open flame and then pressed against the skin. As the cup cools, suction develops, extracting blood or other fluids. A century of experimentation with modifications of the vacuum principle inherent in cupping, combined with various technical advances of the nineteenth and twentieth centuries led to the development of modern obstetric vacuum extractors. Applications of cupping for assistance at deliveries were first reported in the late seventeenth century, when James Younge (1646–1721) and other surgeons performed vacuum deliveries using glass or metal cups [48]. These practitioners failed to publicize their successes, however, and nothing is known about either the construction of these instruments or the techniques used for application or traction [32].

Cupping faced serious technical limitations when the technique began to be applied to vaginal delivery. As this procedure was commonly performed, the cups were initially heated over an open flame before their application. Obstetric use, however, required both a vaginal application of the vacuum cup and a method for traction. Thus, a different technology was needed. Several important features had to be incorporated: easy insertion into the birth canal, the ability to form a firm seal to the fetal head,

a means of continuous regeneration of the vacuum as a result of imperfections of the seal, and finally, a practical method for applying traction.

The vacuum principle was the subject of both experimentation and speculation in the early nineteenth century. It was recognized that evacuating either glass or metal globes could result in substantial pressure and that such devices could be used for traction in several important applications. It was not long before medical applications were suggested [49]. Based on contemporary experimentation and crude commercial vacuum-based devices, James Young Simpson, who developed a several obstetric devices, including the forceps that bear his name, invented the first practical obstetric vacuum extractor in 1849 [44,50,51]. His new device, which he termed an *air* or *suction tractor*, was proposed as an alternative to forceps for use in both cephalic and breech presentations when assisted delivery was required [44,50,51]. Simpson's device consisted of a piston syringe, probably derived from a breast pump, attached to a deep and flexible rubber cup (Figure 1.5). In use, the cup was placed firmly against the fetal head and the syringe was rapidly evacuated. Once suction was achieved, traction was applied by simply grasping the pump cylinder and pulling downward. The extractor was simple and, despite its limitations, successfully employed in several cases. Technical problems with traction, maintaining the vacuum, and the inability of the instrument to accommodate the pelvic curve as a result of its design eventually proved insurmountable, however. After a brief trial Simpson abandoned his vacuum device. Thereafter, despite the occasional introduction of various new designs, vacuum extraction essentially disappeared as an obstetric technique for nearly 100 years.



**FIGURE 1.5.**  
*Simpson's "air tractor" vacuum extractor (1849).*  
(Reprinted from O'Grady JP: *Modern Vacuum Extraction*. Parthenon, 1995; with permission.)

Several vacuum extractors were invented in the century following Simpson's original report, but none achieved either popularity or commercial success until the 1950s, when Malmström introduced his stainless steel cup [52]. The Malmström extractor rapidly became popular, especially in Europe. The device was rugged, successful, and could be used as an alternative to forceps [52–54]. Despite European success, metal cup extractors had a variable reception in the United States. After widespread interest in the early 1960s, vacuum extraction promptly fell into disfavor, largely because of reports of serious scalp injuries and other complications. The popularity of vacuum extraction resumed two decades later only when the soft-cup devices were introduced. At this time clinicians proved more receptive to an alternative for forceps, new instruments were available, and better techniques had been developed for vacuum-assisted delivery.

Malmström's device incorporated several important features now found on all vacuum devices. A protective disk was fitted into the interior of the cup to avoid injury to the fetal scalp. There was a separate vacuum source capable of continuous vacuum production, protected by a collecting bottle or trap. In addition, a pressure gauge was fitted to determine the degree of force generated. Finally, a metal chain firmly attached the cup to an easily grasped handle, permitting easy traction. In later years, other obstetricians including Bird, Lovset, Party, O'Neil, Halkin, and others invented, modified, and improved metal vacuum cups [55–57]. These modified instruments were intended to reduce the likelihood of detachment, facilitate application, or better protect the fetal scalp. Among the rigid metal cups, Bird's modification, in which the vacuum tube is attached to a lateral suction port independent of the traction chain, has proved to be the most popular and useful [55,57]. New models of rigid plastic extractors largely reprise the construction of the Malmström cup, extending the popularity of the original design.

An unknown number of Malmström-type metal cup extractors, predominantly of Bird's modified design, still remain in use. For several reasons, however, most American practitioners prefer to use the plastic cups that have been introduced in recent years [58]. These new designs are disposable single-use devices, constructed of polyethylene and/or

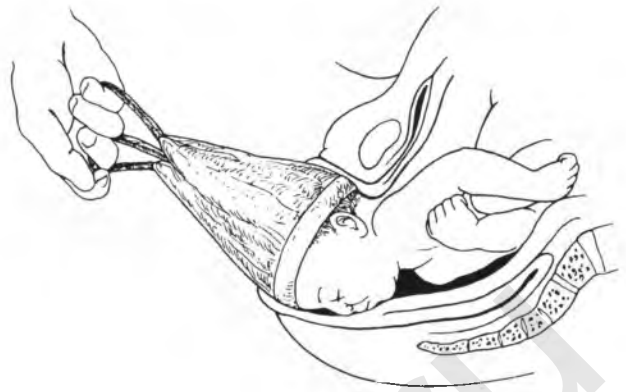


FIGURE 1.6.  
*Elliot's obstetric bonnet (1992). (From Elliott B, Ridgway LF, Berkus MD, Newton ER, Peairs W: The development and testing of new instruments for operative vaginal delivery. Am J Obstet Gynecol, 1992 Oct; 167(4 Pt 1): 1121–4; with permission.)*

Silastic polymer plastic. They are easy to use and have proven effective in most cases. As is always true when new devices become a commercial success, currently too many vacuum cup designs are available, with little if any significant difference between them.

Experimentation with various types of vacuum traction devices has not ceased. Elliot recently described a vacuum-based instrument consisting of a rubber or plastic "bonnet" that lacks either a suction or vacuum port [59]. This unusual-appearing device is designed to be unrolled or fitted onto the fetal head like an inverted parachute. Tension on the handle flattens the membrane around the fetal cranium, providing the force necessary to assist parturition (Figure 1.6). The concept of inserting a net or bag to grasp the fetal head is certainly not new, as strikingly similar examples have appeared fleetingly in the obstetric literature for over two centuries, the earliest perhaps being the *tire-tête* of Pierre Amand from 1715 [36].

An important development in the use of vacuum extraction has been major improvements in practitioner education. These efforts reflect the increasing use of vacuum devices, an appreciation of their potential risks, and the need to better train practitioners in best techniques. Vacuum extraction has become increasingly popular in recent years, and instrumental delivery by vacuum extraction is now more common in the United States than forceps operations [60].

In recent years a greater appreciation of the risks and benefits of all types of assisted delivery has developed. This has prompted increased clinical study to define the best obstetric practices. The continued requirement for some means to accelerate or assist parturition in selected circumstances short of cesarean delivery ensures the continued use of vaginal delivery instruments for the foreseeable future (See Chapter 17, Instrumental Delivery).

## REFERENCES

- O'Dowd M, Phillip EE: The History of Obstetrics and Gynaecology. 1994, New York: The Parthenon Publishing Group.
- Houtzager G: The Complete Encyclopedia of Greek Mythology. 2003, Edison, New Jersey: Chartwell Books, Inc.
- Gottschalk W: Dystocia on Mount Olympus. *Obstet Gynecol* 1959 Mar; 13(3): 381–2.
- Shakespeare W: *Macbeth*, 1606; V. vii. 44.
- <http://heraklia.fwsl.com>.
- <http://www.perseus.tufts.edu>, 2006.
- <http://catholic.archives.nd.edu>, 2006.
- Grant M: Julius Caesar. 1992, New York: M. Evans and Company.
- <http://www.wordorigins.org/wordorc.htm>, 2006.
- <http://catholic.archives.nd.edu>, 2006.
- Baskett TF: On the Shoulders of Giants: Eponyms and Names in Obstetrics and Gynecology. 1996, London: RCOG Press.
- Rousset F: *Traité Nouveau de l'Hystérotomie ou l'Enfantement Caesarienne, qui Est Extraction de l'Enfant par Incision Laterale de Ventre et Matrice de la Femme Grosse. Pouvant Autrement Accoucher, et ce sans Préjudicer a la Vie de l'un de l'autre ni l'Empêcher la Fécondité Maternelle par après.* 1581, Paris: Demeys Duval.
- Pottier Sperry F: Biography: François Rousset. *Hist Sci Med* 1996; 30(2): 259–68.
- Burton J: *An Essay towards a Compleat New System of Midwifry.* 1751, London: James Hodges.
- Gordon BL: *Medieval and Renaissance Medicine.* 1959, New York: Philosophical Library.
- Mauriceau F: *Les Maladies des Femmes Grosses et Accouchées.* 1668, Paris: Henavlt, D'Hovary, De Ninville and Coignard.
- Mauriceau F: *Observations sur La Grossesse et L'Accouchement des Femmes, et sur leurs Maladies, & celles des Enfants nouveau-nez.* 1715, Paris: La Compagnie des Libraires Associez.
- Drife J: The start of life: A history of obstetrics. *Postgrad Med J* 2002 May; 78(919): 311–15.
- Huard P: *sciences, Médecine, Pharmacie de la Révolution a l'Empire (1790–1815).* 1970, Paris: Les Editions Roger Dacosta.
- Speert H: *Obstetrics and Gynecology in America.* 1980, Washington, D.C.: American College of Obstetricians and Gynecologists.
- Chipfakacha V: Abdominal deliveries in Africa: Food for thought to scholars of the history of medicine. *Cent Afr J Med* 1989 Feb; 35(2): 333–6.
- Cutter IS, Viets HR: *A Short History of Midwifery.* 1964, Philadelphia: W.B. Saunders Company.
- Mola G: Symphysiotomy or caesarean section after failed trial of assisted delivery. *P N G Med J* 1995 Sep; 38(3): 172–7.
- Van Roosmalen J: Symphysiotomy – a reappraisal for the developing world. In *Progress in Obstetrics and Gynaecology*, J. Studd Editor. 1991, Edinburgh: Churchill-Livingstone. pp. 149–62.
- Lauverjat T-E: *Nouvelle Méthode de Practiquer L'Opération Césarienne, et Parallèle de cette Opération & de la Section de la Symphyse des Os Pubis.* Paris, 1788: pp. 150–1.
- Sänger M: *Der Kaiserschmitt.* *Arch Gynakol* 1882; 19: 370.
- Porro E: *Della amputazione utero-ovarica come complemento di taglio Cesario.* *Ann Univ Med Chirurg* 1876; 273: 289–350.
- Lurie S, Glezerman M: The history of cesarean technique. *Am J Obstet Gynecol* 2003 Dec; 189(6): 1803–06.
- Waters E: Retrovesical extraperitoneal cesarean section. *Am J Obstet Gynecol* 1940; 39: 423.
- Norton J: Latzko extraperitoneal caesarean section. *Am J Obstet Gynecol* 1935; 30: 209.
- Das K: *Obstetric Forceps: Its History and Evolution.* 1929, Calcutta: The Art Press.
- O'Grady J: *Modern Instrumental Delivery.* 1988, Baltimore: Williams & Wilkins.
- Radcliffe W: *Milestones in Midwifery and the Secret Instrument.* 1989, San Francisco: Norman Publishing.
- Speert H: The obstetric forceps. *Clin Obstet Gynecol* 1960 Sep; 3: 761–6.
- Drinkwater K: The midwifery forceps: Historical sketch. *Med Chir J* 1913; 64: 451–65.

36. Hibbard B: *The Obstetrician's Armamentarium*. 2000, San Anselmo, CA: Norman Publishing.
37. Speert H: *Obstetrics and Gynecology: A History and Iconography*. 1994, San Francisco: Norman Publishing.
38. Thiery M: Obstetric forceps and vectus: The roots. *Acta Belg Hist Med* 1992 Mar; 5(1): 4–20.
39. Shute W: Management of shoulder dystocia with the Shute parallel forceps. *Am J Obstet Gynecol* Oct 1; 84: 936–9.
40. Seidenschnur G, Koepcke E: Fetal risk in delivery with the Shute parallel forceps: Analysis of 1,503 forceps deliveries. *Am J Obstet Gynecol* 1979 Oct 1; 135(3): 312–7.
41. Shute W: An obstetrical forceps which uses new principle of parallelism. *Am J Obstet Gynecol* 1959 Feb; 72(2): 442–6.
42. Dewhurst J: *Royal Confinements: A Gynaecological History of Britain's Royal Family*. 1980, New York: St. Martin's Press.
43. Duns S: *A Memoir of Sir James Y. Simpson*. 1873, Edinburgh: Edmonston and Douglas. p. 288.
44. Chalmers J: James Young Simpson and the "suction tractor." *J Obstet Gynaecol Br Commonw* 1963; 70: 94–100.
45. Luikart R: A modification of the Kielland, Simpson and Tucker-McLane forceps to simplify their use and improve function and safety. *Am J Obstet Gynecol* 1937; 34: 686.
46. DeLee J: The prophylactic forceps operation. *Am J Obstet Gynecol* 1920; 1: 34–44.
47. O'Grady J, Gimovsky ML, McIlhargie CJ: *Vacuum Extraction in Modern Obstetric Practice*. 1995, New York: Parthenon Publishing Group Inc.
48. Younge J: An account of balls of hair taken from the uterus and ovaria of several women. *Philos Trans R Soc (London)* 1706–1707; 25: 2387.
49. Arnott N: *Elements of Physics, or Natural Philosophy, General and Medical, Explained Independently of Technical Mathematics, and Containing New Disquisitions and Practical Suggestions*, Vol 1. p. 543. 1831, Philadelphia: Carey and Lea.
50. Simpson J: On suction tractor or new mechanical power as a substitute for forceps in tedious labours. *Edinburgh Monthly J Med Sci* 1849; 32: 556–8.
51. Eustace D: James Young Simpson: The controversy surrounding the presentation of his Air Tractor (1848–849). *J R Soc Med* 1993 Nov; 86(11): 660–3.
52. Malmström T: The vacuum extractor: An obstetrical instrument. I. and the parturiometer, a tokographic device. *Acta Obstet Gynecol Scand* 1957; 36(Suppl): 5–82.
53. Malmström T, Jansson I: Use of vacuum extractor. *Clin Obstet Gynecol* 1965; 8: 893–913.
54. Sjostedt J: The vacuum extractor and forceps in obstetrics: A clinical study. *Acta Obstet Gynecol Scand* 1967; 46(Suppl 10): 1–208.
55. Bird G: Modification of Malmström vacuum extractor. *Br Med J: Online* 1969; 3: 526–9.
56. Vacca A: *Handbook of Vacuum Extraction in Obstetric Practice*. 1992, London: Edward Arnold.
57. O'Neil A, Skull E, Michael C: A new method of traction for the vacuum cup. *Aust N Z J Obstet Gynaecol* 1981 Feb; 21(1): 24–5.
58. O'Grady J, Pope CS, Patel SS: Vacuum extraction in modern obstetric practice: A review and critique. *Curr Opin Obstet Gynecol* 2000 Dec; 12(6): 475–80.
59. Elliott B, Ridgway LF, Berkus MD, Newton ER, Peairs W: The development and testing of new instruments for operative vaginal delivery. *Am J Obstet Gynecol* 1992 Oct; 167(4 Pt 1): 1121–4.
60. Demissie K, Rhoads GG, Smulian JC, Balasubramanian BA, Gandhi K, Joseph KS, Kramer M: Operative vaginal delivery and neonatal and infant adverse outcomes: Population-based retrospective analysis. *Br Med J* 2004 Jul 3; 329(7456): 24–9.

### Gabriel M. Cohn

*To some extent it may be said that the etiology of the transmitted foetal diseases is within our knowledge, and their diagnosis not altogether outside our grasp; with perseverance and skill their treatment will yet be hopefully undertaken by the well-informed physician.*

John William Ballantyne (1861–1923)

*Antenatal Pathology and Hygiene: The Embryo and the Foetus*  
Edinburgh: William Green and Sons, 1902.

The importance of genetics in clinical perinatal medicine has increased rapidly over the last three decades. Now, more than 6,000 genetic traits or disease entities have been identified (Figure 2.1) [1]. It is estimated that the incidence of genetic disease among newborns is 5%–6% [2]. Genetic disease has profound medical, financial, and societal consequences far greater than its actual numbers. With the progressive disappearance of many infectious and other diseases that in the past accounted for most hospital admissions, genetic disorders remain a serious contemporary social and medical problem. Studies examining inpatient pediatric admissions reveal that 33% to 52% of all pediatric hospitalizations result from complications of genetic disease [3]. If multifactorial disorders are eliminated, purely genetic diseases account for more than one in ten pediatric admissions [4]. Among adults, up to 11.5% of all inpatient admissions are due to genetically related abnormalities [5]. The contribution of genetic disorders to childhood mortality is edge with a substantial number of all pediatric deaths ascribed to genetic disease. Unfortunately, despite enhanced prenatal diagnostic capabilities and aggressive perinatal management, significant reductions in perinatal mortality associated with congenital malformations have not occurred [6].

In addition to its medical importance, genetic disease also has major financial and societal impact. Studies of patients with genetic diseases indicate that inpatient admissions for these patients are on average more common, more expensive, greater in duration, and, owing to geographic limitations of genetic services, require greater travel than similar treatment for persons with other types of illness. The loss of insurability following the diagnosis onset of genetic diseases doubles the likelihood that patients will pay out of pocket for medical services [3]. The societal cost is equally burdensome. Both years of work lost to impairment and years of life lost to disease span decades among persons afflicted with genetic disorders. In sum, the emotional, psychological, and financial impact of productivity lost

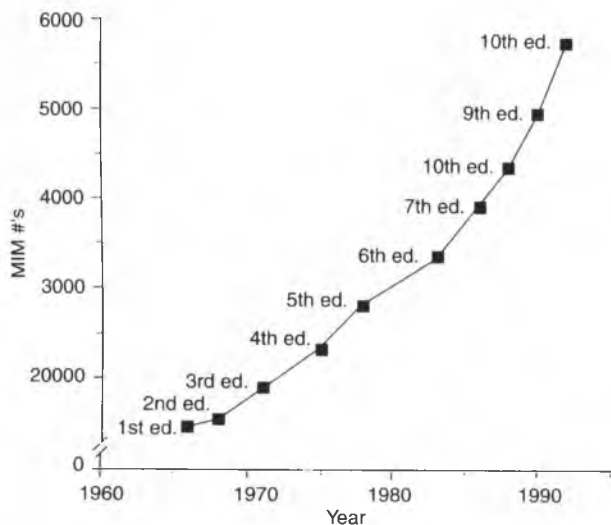


FIGURE 2.1.  
*Total number of entries in Mendelian Inheritance in Man. Since 1966 there has been a 3.8-fold increase in recognized human genetic traits and disorders.*

to genetic disorders is extensive and often poorly appreciated.

With the improvement and automation of techniques in the fields of cyto- and molecular genetics and the identification of many genetic diseases, an understanding of the molecular pathophysiology of many disorders is now possible. Several molecular genetic assays have been developed in kit form to assist clinicians in the early diagnosis of genetic disease. It is believed that with this increased understanding of the mechanisms underlying many genetic entities and with the increasing accuracy of case identification, both pharmacogenetic and gene transfer interventions will progressively evolve. The introduction of such enhanced diagnostic and treatment capabilities will place increased reliance on prenatal diagnosis techniques as the basis for the *prevention* of genetic disease through termination of pregnancy or as the basis for the *treatment* of genetic disease through in-utero medical or surgical interventions. The questions of best practices in genetic screening as well as potential treatment for congenital disorders remain both complex and controversial. [7–10].

This chapter focuses on the procedures and techniques currently available to clinicians to evaluate genetic disorders. As our knowledge in molecular and clinical genetics progressively expands and as more potential therapies become available, the

obstetrician plays a greater role in the identification of at-risk cases and in the prenatal diagnosis of genetic disease. Several currently investigative procedures that could become available for clinical use in the near future are also reviewed and their importance is discussed. For those desiring more extensive information, several excellent genetics resources are available free of charge.<sup>1</sup>

## HISTORY

The development of prenatal diagnostic techniques has closely paralleled the advances in clinical genetics. Amniocentesis was first introduced in the 1880s as a treatment for hydramnios [11–14]. It was not until 1960, however, that amniocentesis for X-chromatin evaluation was first described [15–17]. Amniotic fluid sampling subsequently proved useful for the diagnosis and management of Rh isoimmunization and more recently as a technique for direct evaluation of fetal pulmonary maturity [18]. With the advent of X-chromatin analysis, amniocentesis was demonstrated to identify fetal sex accurately, providing a technique for identifying fetuses at risk for X-linked recessive disorders, such as Duchenne muscular dystrophy and hemophilia [19,20]. In 1966, Steel and Berg [21] were successful in culturing and karyotyping amniocytes, suggesting that prenatal diagnosis of chromosomal aneuploidies was feasible. Jacobsen and Barter [22] subsequently introduced the first report of a fetal chromosomal anomaly identified on amniocentesis, a D/D translocation. In 1968, Valenti and coworkers [23] and Nadler [24] described the prenatal diagnosis of Down syndrome identified on amniocytes obtained by amniocentesis. A biochemical disorder, galactosemia, was similarly identified prenatally by

<sup>1</sup>Two invaluable Web sites include Online Mendelian Inheritance in Man (OMIM) (<http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=OMIM>) and the GeneTest Web site (<http://www.genetests.org>). OMIM is an extensive database searchable by disease, gene, or by clinical feature(s). This database can be particularly useful in generating a differential diagnosis based on ultrasound or other clinical findings, or alternatively in providing a synopsis of the key clinical features associated with a given genetic diagnosis. In addition, an extensive review of the literature is provided for approximately 6,000 known phenotypes, as well as several thousand genetic markers. The GeneTest Web site provides an up-to-date listing of genetic tests that are clinically available, labs and clinical centers that provide genetic services, as well as extensive reviews of several hundred common genetic disorders.



Nadler [24], indicating that prenatal diagnosis by amniocentesis was not strictly limited to chromosomal abnormalities. The first report of a clinical trial of genetic amniocentesis was published by Nadler and Gerbie [25] in 1970. This study demonstrated that prenatal diagnosis by amniotic fluid analysis was an accurate and a low-risk procedure when performed between 16 and 20 weeks.

Fetal visualization was another significant advance. In the 1930s, amniotic infusion of contrast media for amniography was introduced, a procedure used to visualize the fetus and the placenta. Further development of various radiographic and radioactive tracer techniques over the following decades assisted in utero diagnosis. These tests and procedures were promptly abandoned with the advent of high-resolution ultrasonography, however.

Despite its introduction into medicine soon after the Second World War, ultrasound scanning did not make a major impact on obstetric management until real-time machines of high resolution became widely available in the 1970s. Today, ultrasound scan is the primary screening method for many fetal abnormalities and is considered a necessity in many obstetric procedures (e.g., version, amniocentesis, and cordocentesis).

The initial experience with amniocentesis was performed without the benefit of ultrasonography. "Blind" procedures were performed by arbitrarily inserting a spinal needle 3 cm above the pubis symphysis, at no earlier than 15 weeks' gestation. Although considered a reliable technique, mid-trimester amniocentesis had the disadvantage of relatively late timing and hence late diagnosis. Societal pressures limiting pregnancy termination for genetic indications and medical considerations of the enhanced complications associated with mid-trimester abortions stimulated interest in methods of first-trimester prenatal diagnosis. Unfortunately, initial attempts at transvaginal amniocentesis at  $\leq 10$  weeks of gestation and first-trimester endoscopic chorion biopsy resulted in significant pregnancy loss as well as high failure rates [25, 26]. New methods were needed.

In 1968, the technique of chorionic tissue biopsy (i.e., chorionic villous biopsy [CVS]) for prenatal diagnosis was introduced by Mohr [27]. In this procedure, an endoscope of 6 mm in diameter was introduced by way of one of the vaginal fornices or transcervically. The endoscope was positioned against

the chorion, the optical device removed, and suction applied. A tubular knife was next introduced and used to biopsy tissue captured by the endoscopic suction. Success proved elusive, as only one half of the samples obtained were chorionic tissue, and many samples were found to contain amniotic membrane. Modifications of this technique were introduced, and a series of studies were performed on pretermination patients with only modest success [27–29]. Clinicians at Teitung Hospital in China [30] subsequently demonstrated that simple first-trimester placental biopsy was indeed feasible. In their original procedure, a 3-mm diameter metal cannula was blindly introduced through the cervix and advanced until "soft resistance" was encountered. A smaller-diameter inner catheter was then introduced to approximately 1 cm beyond the cannula tip, and tissue was then aspirated by syringe suction. This procedure was attempted in 100 pregnancies to determine fetal sex. In ninety-three patients, the appropriate fetal sex was assigned based on X-chromatin analysis. Of seventy continuing pregnancies, 4% were subsequently lost. Unfortunately, attempts to repeat this technique by other groups proved unsuccessful [31,32]. Attempts at procedure modification (e.g., endocervical lavage) were similarly without great success [33–35]. CVS remained investigational until new biopsy techniques and modified equipment were combined with modern ultrasonic visualization to improve both safety and success.

Direct ultrasonic visualization proved important in the development and acceptance of both amniocentesis as well as CVS. Combined with high-resolution ultrasonography, amniocentesis was directed either at a site identified as most suitable by a sonogram performed prior to the procedure (*ultrasound guided*) or by a sonogram performed during the procedure (*ultrasound monitored*). These modifications permitted localization of the placenta and allowed prenatal diagnosis prior to 14 weeks of gestation – the limit previously established by blind amniocentesis. In the early 1980s, Kazy [36] used ultrasonography to direct thin biopsy forceps (1.7 mm in diameter) at the chorion frondosum and successfully sampled a series of pregnancies. Among the patients studied were women carrying fetuses at risk for genetic disease. Thirteen such women who elected to continue their pregnancies after sampling experienced a successful pregnancy

outcome and the birth of a normal infant. Subsequently, Ward [37] and his London-based group introduced a method in which a blunt stainless steel malleable obturator served as a guide over which a 1.5-mm polyethylene catheter was threaded. Using continuous ultrasonographic guidance, this apparatus was introduced to abut the edge of the chorion frondosum. Once in position, the obturator was removed and a syringe applied. Chorionic villi were aspirated with negative pressure. Ward and coworkers [37] demonstrated that in pregnancies sampled between seven and 14 weeks, a 90% success in chorionic villus sampling was possible. Simoni and coworkers [38] subsequently compared four methods of villus biopsy, including blind insertion of the flexible catheter developed by Ward, blind insertion of an intravascular catheter, endoscopic sampling, and ultrasonographic introduction of the flexible Ward catheter. Using the first three approaches, maximal sampling success was limited to 76%, with bleeding occurring in 17% of attempts. With the Ward catheter used in conjunction with continuous ultrasonographic guidance, sampling success rates improved to 96%. In 1984, the technique of transabdominal CVS was introduced by Smidt-Jensen and coworkers (39) and proved to be a valuable alternative to transcervical sampling methods.

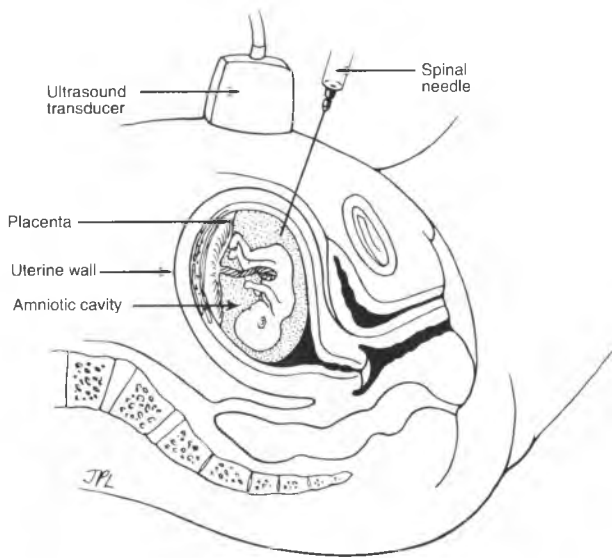
The role of CVS in early pregnancy diagnosis is developing. This issue is discussed in greater detail later, and the risk/benefit ratio is in the process of reconsideration. Recent evidence concerning possible fetal limb defects as a rare procedure-related complication continues to be closely analyzed.

Interest in transabdominal amniocentesis before 16 weeks was reawakened with the advent of improved ultrasound equipment. Subsequent evaluations have indicated, however, that early amniotic fluid sampling procedures ( $\leq 14$  weeks) entail more complications and these have largely been abandoned. The technologic advances that have had the most important influence on prenatal diagnosis, however, include new molecular and cytogenetic testing such as the polymerase chain reaction (PCR) and fluorescent in situ hybridization (FISH) studies. These and similar study methods, as well as other noninvasive techniques for the detection of genetic disease, are in the process of development. Such innovations include detection of fetal cells in maternal circulation, preimplantation diagnosis, polar body biopsy, and oocyte typing.

## AMNIOCENTESIS

### Transabdominal Procedures

Genetic amniocentesis usually is performed after 15 completed weeks of gestation. After ultrasonic study to confirm dates, fetal viability, fetal number, fetal anatomic survey and placentation, the patient is requested to empty her bladder. The abdomen is aseptically cleansed with a povidone-iodine or another antiseptic solution, and sterile drapes are applied. Ultrasound gel is applied to a transducer, which is subsequently inserted into a sterile surgical glove or sleeve. The sterile cover is tightly wrapped around the transducer and sterile surgical lubricant is applied onto the exterior of the gloved transducer. This permits the transducer to be applied to the maternal abdomen with minimal risk of contamination. A pocket of fluid free of fetus and placenta is next identified. If an area free of placenta cannot be found, an area containing the thinnest section of the placenta away from the cord insertion is localized. Once an ideal target is noted, the skin can be infiltrated with a local anesthetic, although the author has generally found this to be unnecessary. Thereafter, under direct visualization, a 22-gauge disposable spinal needle with stylet is passed through the patient's skin and into the amniotic cavity (see Figure 2.2) [40]. The length of the standard needle is 9 cm. A needle insertion of approximately 3.5 to 4.5 cm will usually suffice to tap fluid. Thus the standard needle is appropriate for most patients. In selected obese patients, however, a longer needle might be required. In these special circumstances, evaluation by an initial scan serves as a guide to estimating the required needle length. Once the sac is entered, 20 milliliters of fluid are generally withdrawn, using at least two separate syringes. The first few milliliters are discarded to avoid maternal cell contamination [41]. This initial aliquot can be used for alpha-fetoprotein evaluation, however. Once the complete sample is obtained, the needle is promptly withdrawn. The puncture site is then observed under real-time ultrasound for fetal hemorrhage and normal fetal cardiac activity confirmed. If all is normal, this author subsequently discharges patients to home, requesting that they report any fluid loss, lower abdominal pain, cramping, contractions, or fever. Strenuous activity or coitus is discouraged for the following 24 hours, and thereafter routine activity



**FIGURE 2.2.**  
*Ultrasound-guided amniocenteses. Following aseptic preparations, a 5-gauge needle is guided into the amniotic cavity with the aid of real-time ultrasonography. (See text for details.)*

may be resumed. Rh-negative patients subsequently receive Rh immunoglobulin (RhIG).

If, during the initial attempt, free-flowing amniotic fluid is not obtained, rotating the needle to reposition the bevel or minimal repositioning of the needle is often successful in achieving flow. Negative pressure should not be applied to the syringe during repositioning. If needle rotation or repositioning under ultrasonographic observation proves unsuccessful, a second tap attempt with a new needle is warranted. Whether repeat skin preparation is required for the second tap depends on the clinical circumstances. If a second attempt is unsuccessful, additional efforts are best postponed for 1 week [42,43]. Failure to obtain amniotic fluid is commonly due to needle misdirection, leiomyomas, uterine contractions, or membrane tenting [44,45]. The last problem occurs more frequently prior to 15 weeks of gestation.

### Complications

The recent review by Alfirevic and Sundberg has examined the fetal loss rate associated with mid-trimester amniocentesis and CVS [46]. To evaluate the clinical relevance of such studies, the baseline loss rate for ultrasonographically diagnosed viable

pregnancies at that same gestation, for woman of the same age, must be known. Maternal age is a major factor in the incidence of spontaneous miscarriage. In established pregnancies, the overall spontaneous fetal loss rate is 13.6% among women 40 years of age and older, 4.5% in women in the 35- to 39-year-old group, and 1.5% among women younger than 35 years of age [47–50]. Further, *preamniocentesis maternal-serum alpha-fetoprotein* (MSAFP) elevations, if present, are associated with a significantly higher pregnancy loss rate. Read and coworkers [51] compared the outcome of 212 pregnant women undergoing amniocentesis for MSAFP elevation to the outcome of 219 pregnant women in whom a prior pregnancy had resulted in a fetus with an open neural tube defect (ONTD). The spontaneous loss rate following amniocentesis among patients with MSAFP elevation was 8% versus 2.8% among patients with prior ONTD fetus. These data suggest that many patients who undergo prenatal diagnosis (i.e., advanced maternal age and elevated MSAFP) are at increased risk for spontaneous pregnancy loss *independent* of the procedure-related risk.

Most spontaneous losses occur early in gestation. Evaluation of pregnancy loss rate by gestational age indicates that, of patients awaiting preamniocentesis counseling, 1.2% spontaneously aborted between 12 and 16 weeks. Sant-Cassia and coworkers [52] reported a 1% pregnancy loss rate between 16 and 28 weeks among controls for an amniocentesis study. These data should be included when counseling patients prior to any invasive prenatal testing.

Based on these data presented above, to evaluate studies examining procedure-related loss rates, study designs must incorporate appropriately matched control patients *not* undergoing amniocentesis. There are a number of studies addressing amniocentesis [46]. In 1976, the National Institute of Child Health and Human Development (NICHD) reported a prospective study of 1040 subjects undergoing amniocentesis compared with 992 controls matched for race, socioeconomic conditions, parity, and age [53]. The results suggested no significant differences in the fetal loss rate (3.2% in the control group versus 3.5% in the amniocentesis group), elective second-trimester termination (2.1% in the control group versus 2.3% in subjects), birthweight, 5-minute Apgar Scores, congenital

anomalies, neonatal complications, or developmental problems. Immediate maternal complications (e.g., vaginal bleeding, leakage of amniotic fluid) were reported in 2.4% of patients undergoing amniocentesis. In this series, the risk of vaginal bleeding was significantly related to the number of needle insertions. The authors concluded that mid-trimester amniocentesis was both accurate and “highly safe” and did not significantly increase the risk of pregnancy loss. Interestingly, the loss rate observed in the control group was higher than that observed in other large series, suggesting the recruitment of a high-risk control population or perhaps imperfect matching of the control group.

Simpson and coworkers [54] reported the results of the Canadian Collaborative Group Study. A pregnancy loss rate of 3.2% was observed among 1,020 pregnancies in 900 women who underwent 1,223 amniocenteses. The immediate amniocentesis complication rate was 3.6%. A significantly higher fetal loss rate was observed in pregnancies sampled with needles of 19 gauge or larger, or when more than two needle insertions were undertaken in a single day. The authors concluded that amniocentesis was “... safe, accurate and reliable...” at about 16 weeks of gestation when carried out by an experienced clinician and monitored by ultrasound scan. This study lacked a control group, however.

The Working Party on Amniocentesis (U.K. Collaborative Study Group) [55] demonstrated a 2.4% spontaneous abortion rate among patients undergoing amniocentesis versus a 1.2% loss rate among matched controls. Furthermore, an increased risk of infantile respiratory difficulties and orthopedic abnormalities were seen among test subjects. There were, however, problems with study design; specifically, patient matching was imperfect. A significant fraction of patients in the amniocentesis group were selected on the basis of MSAFP elevation and were significantly older than the controls. In addition, in the data analysis, matched controls who spontaneously aborted were replaced with controls who had not aborted. Some controls also entered the study at older gestational ages than their matched subjects. These selection biases alone probably account for the observed differences between this study and the others previously described, and these data are to be interpreted with care.

Tabor and coworkers [56] performed a randomized, controlled study of amniocentesis on over 4,500 women aged between 24 and 34 years. Subjects and controls were matched for gestational age at entry, maternal age, prior induced and spontaneous abortions, stillbirths, low-birthweight infants, live births, smoking history and socioeconomic status. The loss rate was 0.7% in the control group and 1.7% in the subject group ( $p \leq 0.01$ ). The study suggested an increased pregnancy loss rate in patients estimated at approximately 1% undergoing amniocentesis.

Many smaller studies of the risk of amniocentesis also have been conducted, with findings suggestive of no minimal differences between patients undergoing amniocentesis and controls. Unfortunately, not all subjects were appropriately matched with controls, rendering these results difficult to interpret. The recent comprehensive review and meta-analysis of Mujezinovic and Alfirevic [57], which appeared in September of 2007, summarized MEDLINE data published after January 1, 1995, concerning both amniocentesis and CVS. These authors noted a wide range in reported risk for pregnancy complications from these diagnostic procedures. The pooled estimate for a pregnancy loss within 14 days of an amniocentesis was 0.6% (95% CI 0.5–0.7). This provides a reasonable benchmark for clinicians to use in counseling [58].

Blood-tinged amniotic fluid is detected in 2% of amniocentesis. This event is associated with an increased fetal loss rate and can be due to either maternal or fetal bleeding. Documentation of maternal blood in the sample is associated with an increased pregnancy loss rate from 1.7% (control population) to 6.6% (hemorrhage population) [59]. Fetal blood in amniotic fluid is associated with a loss rate of up to 14.3% [60]. Perhaps not surprisingly, transplacental amniocentesis has a significantly higher loss rate than non-transplacental amniocentesis (2.9% vs. 1.2% respectively, with the control group significantly lower than both at 0.4%) [56]. MSAFP elevation following amniocentesis is more common in sampled pregnancies with anterior placentas. Such elevations are believed to result from subclinical maternal-fetal hemorrhage and, if present, are associated with an increased fetal loss rate (14% vs. 1%) [59, 60].

Following second-trimester amniocentesis, Rh sensitization is a potential complication [61]. Both

Khalil and coworkers [62] and Golbus and coworkers [63] reported a decreased risk of sensitization in Rh-negative women who routinely received RhIG following amniocentesis. Tabor and coworkers [64] could not demonstrate a significant increase to Rh sensitization following amniocentesis in Rh-negative patients who had not received RhIG. Although there is no uniformity of opinion concerning RhIG administration following midtrimester amniocentesis, the consensus is that RhIG administration is indicated in at-risk pregnancies to prevent Rh sensitization. The American College of Obstetricians and Gynecologists recommends the administration of 300  $\mu\text{g}$  of RhIG following the procedure [65]. It has been this author's practice in the past to treat at-risk pregnancies (i.e., Rh negative and a negative maternal indirect Coomb's test), and such treatment remains the current recommendation of this author.

Up to 6% of the specimens resulting from midtrimester amniocentesis have green or brown discoloration of the fluid (53,67–69). Biochemical analysis of the pigment found in the discolored fluid reveals it to be breakdown products of hemoglobin [70]. It is highly unlikely that this fluid discoloration is from meconium passage, an event virtually restricted to late gestation. A more plausible explanation is that the staining results from occult intrauterine hemorrhage, with subsequent transmembranous passage of heme pigments.

Several studies have investigated the association between amniotic fluid discoloration and pregnancy loss. In a case-controlled study, Hankins and coworkers [68] could demonstrate no increased risks among patients found to have discolored amniotic fluid at the time of genetic amniocentesis. Nevertheless, several other studies have demonstrated a significant increase in risk of pregnancy loss in instances in which amniotic fluid discoloration is noted during mid-trimester amniocentesis [64,67,65–70]. In these studies, the spontaneous abortion rates in the control groups ranged from 1.5% to 1.6%, whereas pregnancy loss rates in the stained-fluid group ranged from 9% to 100%. Thus, amniocentesis specimens complicated by fluid discoloration (i.e., fresh blood or green/brown discoloration presumably from chronic or occult bleeding) identifies a group at increased risk for pregnancy loss. Patients with this finding should be so counseled.

Although amniocentesis is frequently performed in conjunction with real-time ultrasonography, no adequately designed prospective study has been performed to assess the value of ultrasound use in this procedure. Nonetheless, there are substantial amounts of data reflecting clinical experience. Several studies have demonstrated a reduction in the number of dry taps, needle insertions, bloody taps, failed cultures, and pregnancy losses when ultrasonography is used routinely [40,67,69–77]. Other studies have demonstrated no benefit to amniocentesis [55,78,79]. Unfortunately, most of these studies are flawed in their design, with inadequate controls, improper randomization, inappropriate crossing over, or different operators performing amniocentesis with or without ultrasound scan. Although the benefits of ultrasonographically monitored amniocentesis have not been scientifically demonstrated, in this setting the rule of reason must apply. No reports of an *increased* incidence of adverse outcomes of ultrasonically directed procedures have been published. The important assistance of direct ultrasonic guidance for intrauterine procedure is clearly recognized by clinicians. The ability of scanning to localize the pocket to be entered by the sampling needle makes the procedure easier and the operator more confident. It is this author's firm opinion that amniocentesis should *always* be ultrasound monitored.

Maternal infection following amniocentesis is rare. The risk of amnionitis has been estimated to range between 1 in 1,000–8,000 procedures. One maternal death following amniocentesis has been reported [80]. Given the extreme low risk, the use of prophylactic antibiotics for this procedure is not warranted [81].

Vaginal leakage or spotting is reported in approximately 3% of patients who undergo mid-trimester amniocentesis. Although fluid leakage can result in either oligohydramnios or pregnancy loss, successful pregnancy outcome following conservative measures and monitoring of such pregnancies is also possible. Patients must be made aware of the potential risks of such management [82].

Accidental needling of the fetus potentially resulting in skin dimpling and minor scars is believed to occur in up to 2% to 9% of procedures [83]. The magnitude of the risk for resulting skin lesions is unknown, but it is believed to be very low. Rarely, more significant anomalies have been reported

anecdotally and attributed to amniocentesis. Ultrasound monitoring of the procedure was not reported in any of these cases. Controversy exists in the literature supporting and refuting claims that neonates exposed in utero to amniocentesis are at increased risk for respiratory distress and orthopedic disorders such as talipes [84]. Although a specific risk has yet to be established, patients should be aware of these concerns and the limitations of the available data.

## CHORIONIC VILLUS SAMPLING

### **Procedure**

Prior to the attempt at transcervical biopsy (TC-CVS), a ultrasonic scan is performed to evaluate fetal viability, fetal number, placentation, and dating by crown-rump length (CRL) and gestational sac size. The latter two measurements are compared with the expected gestational age estimated from the patient's last normal menstrual period (LMP). Scheduling of procedures beyond the 9th week of gestation eliminates a substantial percentage of spontaneously aborting embryos. In addition, there are other reasons for performing procedures beyond 9 weeks, including a possible reduced incidence of fetal injuries (discussed later).

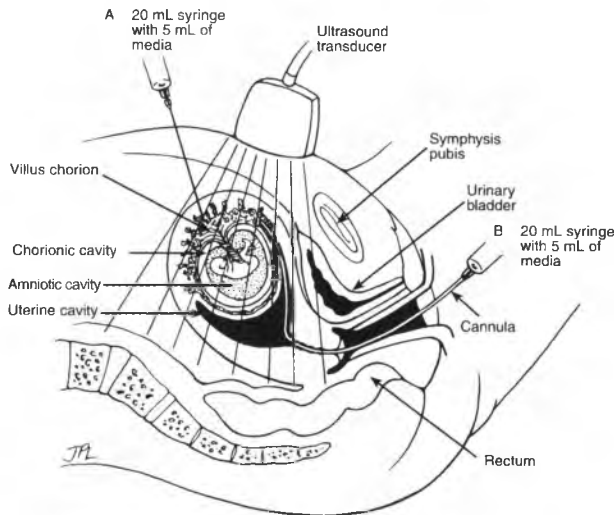
The accuracy of the reported LMP is important. Wapner and Jackson [85] reported that a discrepancy in crown-rump length more than 1 week less than predicted by menstrual dates is predictive of increased risk for spontaneous pregnancy loss. Furthermore, in patients considered for chorionic sampling based on advanced maternal age, over 10% were observed to already have an embryonic demise on the initial preprocedural ultrasound assessment.

In addition to embryologic evaluation, ultrasound scanning establishes the sites of both the chorion frondosum and of the cord insertion. Cervical and uterine anatomic relationships are also noted. Cervical manipulation and either bladder distention or emptying usually facilitate both successful visualization and completion of the procedures.

Once a thorough ultrasonic assessment is completed, the patient is placed in the dorsal lithotomy position. A sterile speculum is introduced, and the cervix is swabbed with an aseptic solution of the clinician's choice. Some operators grasp the ante-

rior lip of the cervix with a long Allis or other forceps as an aid in manipulating the uterus and directing the catheter to the chorion. Ultrasonic reevaluation confirms uterine position and the location of the chorion frondosum. For a successful biopsy procedure, the operator must coordinate his/her activity with that of the ultrasonographer. The operator next bends the distal portion of the catheter slightly to accommodate the demonstrated curvature of the lower uterine segment. The sampling catheter is then introduced through the endocervical canal beyond the internal os at which time a loss of resistance is perceived. Further advancement is delayed until the catheter tip can be visualized ultrasonographically. Once the tip is satisfactorily identified, the surgeon advances the catheter under direct visualization toward the homogenous, hyperechoic chorion frondosum. Where the location of the chorion is uncertain, identification of the cord implantation site serves as a valuable landmark. To assist in accurate placement, catheter rotation, cervical manipulation, or speculum adjustment all could be required. Once the catheter is positioned at the near distal end of the chorion frondosum, the obturator is removed, and a 20-ml syringe containing 5 ml of tissue culture media is attached to the catheter. Using continuous negative pressure on the syringe, the surgeon withdraws the catheter gradually. After a tissue examination confirms the presence of an adequate specimen, the instruments are removed, completing the procedure. As with routine amniocentesis, the patient is directed to call if fever, heavy vaginal bleeding or severe abdominal pain, or cramps develop. She is further instructed to avoid strenuous activity or coitus during the ensuing 24 hours; thereafter she may resume normal activities.

Transabdominal chorionic villus sampling (TA-CVS) is a procedure technically very similar to transabdominal amniocentesis. Two techniques were originally described. In both, a preliminary ultrasound survey is undertaken, and the patient's skin prepped in the usual manner. In the two-needle technique, an outer needle is used as a trochar. This is introduced into the myometrium adjacent to the sampling site. A thinner, inner needle is then guided through the outer needle toward the chorion frondosum under direct ultrasound guidance. Tissue is aspirated under negative pressure into a media-containing syringe. Several passes through the entire



**FIGURE 2.3.**  
*Chorionic villus sampling. Using either a transcervical (A) or a transabdominal (B) approach, the chorion frondosum is sampled under 15 ml to 20 ml of negative pressure. (See text for details.)*

length of the chorion are required to obtain an adequate sample.

The single-needle technique utilizes a regular 20-gauge spinal needle. The needle is simply inserted into the chorion frondosum under direct ultrasound guidance (See Figure 2.3). Once in the chorion, the stylet is removed, and a 20-ml syringe containing 5 ml of culture media is attached. Using continuous negative pressure, several passes through the entire length of the chorion frondosum are undertaken to obtain the specimen.

In both transabdominal and transcervical biopsy techniques, the specimen obtained is washed in a Petri dish and examined under sterile conditions, using a dissection microscope. An assessment of tissue type and quantity is performed immediately to ascertain the success of the biopsy. The specimen is subsequently submitted to the laboratory for processing.

There are certain contraindications to CVS. Absolute contraindications to transabdominal CVS include unavoidable myomas, a placenta not reachable through the maternal abdomen, and maternal intestines overlying the uterus. Relative contraindications include active bleeding, Rh isoimmunization, embryonic growth retardation, or maternal coagulopathy. Other possible contraindications includes low-lying myomas, a gestation greater

than 12 weeks, a multiple gestation, or an overly curved sampling pathway. Contraindications unique to transcervical sampling include vaginal infection, cervical stenosis, vaginismus, the presence of an IUD, and sampling failure following two passes.

Transcervical CVS is technically possible in the window from 6 to 7 weeks' to 12 to 13 weeks' gestation. Recent reports of the association of congenital limb anomalies with CVS, in particular before the 10th week of gestation, have resulted in a restriction of CVS to gestations greater than or equal to 10 weeks in duration, however. Early and mid-trimester transabdominal CVS procedures have gained popularity as alternatives to early amniocentesis or cordocentesis in selected conditions such as severe oligohydramnios [84,86].

The overall efficacy of TA-CVS and TC-CVS are comparable. Single sampling success rates range from 96.4% to 99.5% in the TC group, and 99.4% to 99.7% in the TA group [87-96]. Although both TC-CVS and TA-CVS are equally effective, centers offering CVS should be well versed in both techniques, with a particular approach taken based on the anatomy and condition of the patient being sampled as well as the expertise of the operator.

The accuracy of CVS compares favorably with that of amniocentesis [97-100]. Amniocentesis is generally considered 99.5% accurate, whereas CVS exhibits an accuracy rate of 97.5% to 99.7% [101,102]. Maternal cell contamination (MCC) was initially a significant concern with as many as 13% of cases reported to have this complication. The incidence of MCC is now reported to range from 0.1% to 1.3% [103-106]. Most cases of MCC are found in long-term cultures, although MCC has also been found in direct preparations of chorionic villi. It is believed that the risk of MCC is reduced by meticulous dissection of the villi under a dissection microscope, comparison of both direct preparation and long-term cultures of villi, and comparison of heteromorphisms of 46XX villus cells and maternal lymphocytes. Placental mosaicism has been observed in <2% [107] of all CVS samples. On repeat testing by mid-trimester amniocentesis, approximately two thirds of suspected mosaic pregnancies are cytogenetically normal. Mosaicism noted exclusively in villi is considered *confined placental mosaicism* and is associated with an increased

risk for pregnancy loss (8.6% versus 3.5%) [107–109]. Placental mosaicism has not been proven to be associated with growth retardation, gestational hypertension, preterm labor and delivery, or abruptio placentae.

When the cytogenetic diagnosis following CVS is in doubt, amniocentesis should be offered as a confirmatory study. Up to 3% of patients who undergo CVS will require a follow-up mid-trimester amniocentesis to evaluate mosaicism, maternal cell contamination, or a subsequently elevated mid-trimester MSAFP [110]. Patients who are CVS candidates should be counseled about this possibility prior to undergoing testing.

### Complications

Complications associated with CVS include vaginal bleeding, amniotic fluid leakage, infection, fetal-maternal transfusion, teratogenic effects, and fetal loss [110–113].

Vaginal spotting or bleeding occurs in up to 32% of patients with TC-CVS but is reported in only 4% to 5% of women undergoing transabdominal procedures. Intrauterine hematomas detectable at the first follow-up ultrasound scan have been noted in 3.1% to 4.0% of TC-CVS and 0.3% of TA-CVS cases. Fluid leakage is a rare complication of both procedures but is more common following transcervical operations [97]. Uterine infection ranges from 0.05% to 0.5% in TC-CVS cases. Infection has not been reported in TA-CVS. Changing of the sampling catheter with each passage through the cervix has reduced the incidence of maternal infection. Administration of 50  $\mu$ g RhIG is recommended following CVS procedure in Rh-negative patients. Perinatal risks including small for dates, preterm labor, low birthweight and perinatal mortality are not increased in patients undergoing either CVS procedure.

The fetal loss rate following CVS has been examined in several studies [46, 57, 84, 86, 99, 100, 111, 114–116]. In considering these studies, remember that as in mid-trimester transabdominal procedures, both the gestational timing and maternal age are important in establishing the a priori risk. An important additional factor is operator experience.

The Canadian Collaborative CVS study [114] was the first major prospective study to appear in

the literature. In this study, random assignment of 2,979 women to either CVS or to amniocentesis was undertaken. All centers performed transcervical procedures. The physicians were required to have performed a minimum of successful procedures prior to their acceptance as coinvestigators. The data revealed that the overall loss rate was 0.6% higher in the CVS group in comparison to the amniocentesis group. This difference was not statistically significant.

A similar study design was conducted by the NICHD [115]. In the NICHD study, however, acceptable randomization of the amniocentesis group could not be accomplished. The observed fetal loss rate was 7.2% for the TC-CVS group and 5.7% for the amniocentesis group. After the data for maternal age differences are adjusted, the TC-CVS group had a 0.8% higher loss rate, again not statistically significant.

The subsequent BMRC study [111] noted a significant difference in fetal loss rate between the CVS group and the amniocentesis group (13.6% and 9.0%, respectively). A careful evaluation of this study reveals that unlike the Canadian or American study, a standard method of villous sampling was not employed. Either TC-CVS or TA-CVS was performed according to operator preference. Furthermore, no criteria for operator inclusion based on experience was established in the study protocol. As TC-CVS has been shown to have a significant learning curve, this omission is believed to have influenced the results. A subsequent Danish study bears this out [116]. In this latter investigation, the fetal loss rate among patients undergoing amniocentesis was 6.4%, those undergoing TA-CVS 6.3%, and TC-CVS 10.9%. In the last group there was an inverse relationship between the fetal loss rate and the amount of tissue obtained. These data suggest that operator experience played a significant role in the observed increase in fetal loss.

In the major studies comparing TA-CVS to TC-CVS, therefore, a significant outcome difference was noted only in the Danish study. In the other studies, operators were skilled in TC-CVS before beginning the investigation, and no significant difference in outcomes for the two techniques was detected. Recent investigations with experienced practitioners have found similar outcomes [46, 57, 86, 99, 100].



### Special Issues

Several studies have reported an association between CVS procedures and limb anomalies and oromandibular-limb hypogenesis [112,113]. These complications were attributed initially to procedures performed between 56 and 66 days of gestation. Vascular compromise during limb development was believed to be the causative factor [129].

Kuliev and colleagues [117] reported the results of a WHO-sponsored registry established in Philadelphia. More than 80,000 of an estimated 150,000 CVS-exposed pregnancies worldwide were reported up to May of 1992. Although clusters of limb anomalies following CVS were reported from various centers, the overall incidence among CVS-exposed pregnancies was 6/10,000 live births, comparable with the background incidence rate reported by the British Columbia Registry (5.42/10,000 live births) [118].

A retrospective cohort study performed in five obstetric centers from 1984 to 1991 reported by Gruppo Italiano Diagnosi Embrio Fetal (GIDEF) [110] analyzed the association between CVS and transverse limb reduction defects (TLRD). Outcome data were available on 2,759 of 3,420 pregnancies exposed to CVS. Assuming normal outcome in all patients lost to follow-up, a minimum overall crude rate of TLRD was found to be four times higher in the CVS-exposed group (1/1,143) than in the general Italian population (1/4,458). The rate of limb defects was higher for CVS performed at 9 weeks' gestation (2.9/1000) than at 10 weeks (1/1,000). The rate of TLRD in CVS-exposed pregnancies with completed follow-up was 1/619, representing a sevenfold increase compared with the general population.

In 1995, the Centers for Disease Control and Prevention published the results of a study of CVS-exposed pregnancies from 1988 to 1992. It was found that the risk of limb defects among CVS-exposed pregnancies ranged from 0.03% to 0.10% and represented at least a sixfold greater risk than that in the general population (0.005%). The risk and severity of limb defects appears to correlate with the timing of CVS, with a 0.20% risk observed prior to 10 weeks' gestation and a 0.07% risk at 10 weeks' gestation or greater. Furthermore, most of the defects noted following CVS at 10 weeks or greater are limited to the digits [112].

To contrast CVS and amniocentesis, the increased risk of pregnancy loss and of limb anomalies should not preclude consideration of other important issues such as the desirability to obtain results with early gestation (particularly for women with medical conditions for which pregnancy poses a substantial risk), the advantages of first-trimester pregnancy termination (particularly interruption of pregnancy by the administration of antiprogestins and prostaglandins if early CVS is considered), and the type of genetic disorders in question.

### Early Amniocentesis

In the attempt to introduce first-trimester prenatal diagnosis in areas lacking a CVS program, several centers have investigated early amniocentesis as an alternative. Although technically similar to routine mid-trimester amniocentesis, early procedures are more likely to be complicated by the tenting of the amniotic sac. Needle insertion therefore must be more precise. Furthermore, most operators limit the amount of amniotic fluid aspirated to one milliliter per week of gestation. The initial experience indicated a fetal loss rate ranging from 0% to 4.7%. Success in obtaining fluid is as high as 95% with cytogenetic success rates of approximately 99%, comparing favorably with amniocentesis and CVS. Technically, amniocentesis becomes increasingly difficult before the 13th week of gestation and the rate of complications is increased, indicating that this method has a limited role in prenatal diagnosis, only partially bridging the gap between CVS and routine abdominal amniocentesis at the mid-trimester [57, 86, 99, 100].

### Cochrane Review and Comparison

The Cochrane Database of Systemic Review recently updated its comprehensive review and comparison of amniocentesis and CVS for prenatal diagnosis. The objective of this review was to assess the comparative safety and accuracy of CVS (TC and TA) and amniocentesis (early and second trimester). A total of fourteen randomized studies were included in the analysis [120].

In a comparison of amniocentesis versus control, the authors noted that an increase in spontaneous miscarriages of 0.8% among those undergoing amniocentesis relative to controls was statistically

significant (relative risk [RR] 1.6, 95% confidence interval [CI] 1.02–2.52). A 1% increase in total pregnancy loss was not statistically significant, however. Leakage of amniotic fluid was more common following amniocentesis (1.7% vs. 0.4%; RR 3.9, 95% CI 1.9–7.8), although there was no difference in vaginal bleeding between the two groups.

A comparison between early and second-trimester amniocentesis revealed that mid-trimester amniocentesis was technically less demanding and safer. Multiple needle insertions were required in 4.7% of early amniocentesis procedures, compared with only 1.7% for mid-trimester amniocentesis. Total pregnancy loss following early amniocentesis was 7.6% compared with 5.9% following mid-trimester amniocentesis (RR 1.29, 95% CI 1.03–1.61). The number of congenital anomalies (particularly the number of infants born with talipes equinovarus) was significantly higher in the early amniocentesis group (4.6% vs. 2.7%). Early amniocentesis was also associated with a higher rate of laboratory failures (1.8%) and a higher false-negative rate, compared with a 0.2% failure rate and no false-negative cytogenetic results following mid-trimester amniocentesis.

A comparison of CVS to second-trimester amniocentesis was evaluated in one of three ways: Second-trimester amniocentesis versus CVS (any route), second-trimester amniocentesis versus TC-CVS and second-trimester amniocentesis versus TA-CVS. In the comparison between second-trimester amniocentesis versus CVS by any route, the overall loss rate was significantly higher following CVS (11% vs. 8.2% RR 1.43, 95% CI 1.22–1.67). An increase in spontaneous miscarriages was the primary contributing factor to this increase (RR 1.51, 95% CI 1.23–1.85). CVS also presented greater technical challenges relative to amniocentesis. Repeated sampling was more common following TC-CVS (6.3% versus 0.2%), and laboratory failure occurred more frequently in CVS samples compared with amniocentesis samples (1.7% vs. 0.07%). There were more false-positive and false-negative results in the CVS group (2.2% vs. 0.2% and 0.3% vs. 0%, respectively) as well as more cytogenetic abnormalities confined to the placenta (2.3% vs. 0.4%). Maternal complications were uncommon following either procedure, and life-threatening complications were not reported. Vaginal bleeding was more common following TC-CVS, although no significant difference

was noted in the incidence of vaginal bleeding later in pregnancy. It appears that the route of CVS might contribute to the increased loss rate observed relative to amniocentesis. TC-CVS was associated with a higher risk of pregnancy loss and risk of spontaneous miscarriage compared with amniocentesis (14.5% vs. 11%; RR 1.40 CI 1.09–1.81 and 12.9% vs. 9.4%; RR 1.50, 95% CK 1.07–2.11). No statistically significant difference in the risk of pregnancy loss and miscarriage was found in a comparison between TA-CVS and second-trimester amniocentesis, however. Studies comparing TC-CVS and TA-CVS corroborate this observation. Overall, the risk of pregnancy loss, spontaneous miscarriages, vaginal bleeding, and rate of multiple insertions appears higher in the transcervical group compared with the transabdominal group. Finally, a comparison of early amniocentesis and transabdominal CVS revealed a higher risk of pregnancy loss and spontaneous miscarriage following early amniocentesis. Furthermore, the mean number of days required for culture was two to three days greater in the early amniocentesis group compared with the TA-CVS group.

On the basis of these findings, the authors concluded that second-trimester amniocentesis is safer than early amniocentesis or TC-CVS, but that if earlier diagnosis is required, TA-CVS is preferable. When TA-CVS cannot be performed, TC-CVS or second-trimester CVS are the “preferred options.”

## SPECIAL CONSIDERATIONS

### **Multiple Gestations**

The presence of a multiple gestation complicates prenatal genetic assessment and subsequent management. The ratio of dizygotic to monozygotic twins per 1,000 births approximates 2–3:1. The frequency of dizygotic twinning increases with parity and maternal age [121–125]. Based on these considerations, Rodis and colleagues [125] reconsidered the age-related risk of chromosomal aneuploidy in twin gestations, specifically the age-related risk that one or both fetuses is affected. In estimating the corresponding risks, the authors calculated that the percentage of dizygotic twinning was 80% in the population evaluated. Unfortunately, no adjustment for increased dizygotic twinning with increased age or parity was undertaken in these calculations. Hook's

[124] data on the risk of chromosomal abnormalities in singleton gestations was subsequently recalculated and weighted for the risk of monozygosity/dizygosity and for the possibility that neither, either, or both fetuses were aneuploid. In addition, a retrospective review of cytogenetic results from twin amniocentesis was undertaken.

According to the tables generated from this complex analysis, the risk to a patient at 33 years of age with a twin gestation of having at least one twin with Down syndrome is equivalent to that for a 35-year-old with a singleton pregnancy. In addition, the risk of having at least one twin with any chromosomal abnormality at the age of 32 years is 1:179 for live-born infants and 1:156 at the time of mid-trimester amniocentesis. This risk is higher than that estimated for the procedure-related fetal loss rate owing to standard mid-trimester amniocentesis in a singleton pregnancy (approximately 1:200) [125]. Although initial studies suggested the possibility of an increased risk of pregnancy loss in twin gestations because of amniocentesis [126,127], more recent data using more appropriate controls (i.e., using twin gestations, not singleton pregnancies, as the control group to assess the baseline pregnancy loss rate) indicates that there is no significant difference in fetal loss rate from amniocentesis in twin gestations, despite the need for at least two punctures. Specifically, Ghidini and coworkers [128] observed a baseline fetal loss rate of 3.2% among controls with twin gestations versus a 3.5% loss rate observed following amniocentesis of twin pregnancies. Interestingly, the loss rate of the amniocentesis group was similar to figures previously reported (3.2%–3.57%) [127], but that had previously compared unfavorably to the 0.6% loss rate observed among singleton gestations. Preliminarily, these data suggest that all patients with a twin gestation should undergo genetic counseling and be offered amniocentesis of both twins *beginning at a maternal age of 32*, because the risk of aneuploidy to at least one fetus is greater at that time than the procedure risk for amniocentesis. Unfortunately, with a background fetal loss rate of 3.2%, a study evaluating 2,300 patients in each group would be required to detect a 50% increase above baseline in the procedure-related fetal loss rate. Such studies have yet to be conducted. In counseling at the author's institution, we inform patients of these

risk/benefit calculations and generally offer amniocentesis for twin pregnancies based on the earlier maternal age.

Technically, amniocentesis of a twin gestation differs from that of a singleton gestation only in the importance of the identification of the specific fetus being sampled. The standard approach to twin sampling is to identify the membrane separating both fetuses and subsequently perform sequential needle insertions into each sac. With the first insertion, amniotic fluid is aspirated and indigo carmine dye is introduced. The dye serves as a marker when performing the needle insertion into what is believed to be the second sac. The aspiration of stained fluid with the second tap suggests an inadvertent reintroduction of the needle into the original sac. Alternatively, the presence of clear fluid indicates that the second sac is being sampled. Careful documentation of the fetal relationships is necessary because the specific identification of an affected versus an unaffected fetus might be required should selective termination be considered.

As an alternative to the two-stick-and-dye procedure, Jeanty and colleagues [129] proposed a single-puncture method. In this technique, the membrane separating both sacs is visualized. A single needle insertion is undertaken, and fluid is withdrawn from the first sac. Subsequently, the same needle is simply advanced through the separating membrane under direct ultrasonic guidance and into the second sac, which is then sampled. A prospective study comparing these two techniques for efficacy and safety has yet to be performed. There have been few published studies that have examined the safety of second-trimester amniocentesis in twins. In a controlled study in which the dual puncture and dye method was used, Ghidini and coworkers did not find a significant difference in outcome [128]. Yukobowich and associates published the results of their retrospective cohort analysis. Pregnancy outcomes at 4 weeks following mid-trimester amniocentesis in twins were compared with the 4-week outcomes of a control group of twin pregnancies in which amniocentesis was not performed, and compared with a control group of singleton pregnancies in which amniocentesis was performed. The rates of miscarriage for the three groups were reported to be 2.73%, 0.6%, and 0.67%, respectively ( $p = 0.01$  for both comparisons). These data suggest that the

risk of miscarriage associated with amniocentesis in twins is greater than the risk of miscarriage associated with twin pregnancy alone and is greater than the risk of miscarriage following amniocentesis in singleton pregnancies [130].

Twin sampling by CVS is reliably accurate only with a monochorionic placenta or with two distinct placentas. Difficulty is encountered in attempting to sample dichorionic twins with a fused placenta. In such situations, if sampling is attempted it is best done near each of the umbilical cord insertion sites. Under these circumstances, the sampling error ranges from 2.6% to 16.6%. This underscores the need to limit such sampling to the most experienced centers or to abandon the attempt at CVS except in unusual circumstances and to use conventional amniocentesis for genetic analysis at a more advanced gestational age [131].

The technique of prenatal testing in the higher multiple gestations is similar to that for twin gestation. Unfortunately, series sufficiently large to evaluate these methods are not available. Technical problems in sampling each sac of the higher multiples are common, and successful taps are usually difficult and tedious. The procedure(s) used must be individualized.

### Selective Pregnancy Termination

With genetic testing of twin pregnancies and the increased likelihood of detecting an abnormality in only one fetus, or with multifetal pregnancies from ovulation induction, the option of selective pregnancy termination needs consideration. As with all types of pregnancy termination, there are important ethical issues in selective reduction. Careful patient selection and counseling are required. Procedures, if attempted, should be performed only by experienced operators. Several methods are available, including intracardiac potassium chloride injection, cardiac tamponade, air embolization, and, most recently, percutaneous cord occlusion [132]. Although the data are limited, they do suggest relative safety for these procedures. Significant maternal complications have been reported, however. The safest and most efficacious approach to this problem awaits carefully controlled prospective studies. In the interim, selective termination can be considered in multiple gestations of four or more, or in

instances in which fetal abnormalities are diagnosed in one of a twin or a triplet gestation. Not surprisingly, the earlier the procedure can be performed, the lower the risk of complications.

### LABORATORY PROBLEMS

A detailed technical discussion of the laboratory aspects of prenatal genetic testing is beyond the scope of this text but is covered by several excellent reports [133–136]. Errors in prenatal diagnosis can arise in the laboratory from simple mislabeling of tubes or cultures, maternal cell contamination, pseudomosaicism, mosaicism, or misinterpretation of results. Similarly, CVS can be associated with misdiagnosis primarily because of maternal cell contamination, pseudomosaicism, and true mosaicism. Maternal cell contamination (MCC) has been reported to range from 0.1% to 1.3% of CVS specimens [134,135]. The rate of mosaicism on CVS specimens ranges from 0.6% to 0.8%. On repeat testing usually by mid-trimester amniocentesis, approximately three fourths of suspected mosaic pregnancies prove to be cytogenetically normal [114].

In cytogenetic results obtained from the randomized Danish study of TA-CVS and TC-CVS and amniocentesis, mosaicism was detected in 1% of all cases of CVS [136]. Of these, 90% were found to be confined to the placenta, 0.7% of mosaicism/pseudomosaicism were seen in amniotic fluid specimens, and only 40% of the latter were confirmed in the fetus. Pseudomosaicism was similar in cultures of chorionic villi and amniocytes (0.5 and 0.6%); MCC was more common following transcervical sampling (4.5%) compared with transabdominal sampling (1.5%).

Laboratory experience of early amniocentesis indicates that the overall culture success rates have been reported to range from 98.6% to 99.8%. Mean turn-around time ranges from 8.2 to 8.4 days compared with 6.59 to 8.3 days for amniocentesis performed after the 14th week of gestation [137, 138]. Kerber and Held [137] noted a significant increase in the number of numerical and structural aberrations in single cells and a significant increase in the frequency of numerical aberrations in multiple cells. Lockwood and Neu [138] reported abnormal karyotypes in 4.9% of their specimens but had no

clinical follow-up on these patients. Their experience is consistent with prior reports that found that 1.1% to 5% of early amniocenteses are associated with chromosomal abnormalities.

### **SERVICE ORGANIZATION AND QUALITY ASSURANCE**

As knowledge in genetics and its clinical application increases, greater demands will be placed on developing comprehensive prenatal genetics programs. These programs will be required to provide timely, accurate information, in a format that patients can readily understand and use to make informed decisions. Toward this end, a multidisciplinary team composed of persons skilled and specialized in genetics (i.e., geneticist, genetic counselors) maternal fetal medicine (i.e., perinatologists, perinatal nurses, ultrasonographers), neonatology (i.e., neonatologists and neonatal nurses), and grief and abortion counseling (i.e., social workers and gynecologic-specialty nurses) is needed to address the issues and complications raised by this new technology. In addition, a strong referral network must be established with qualified subspecialty physicians capable of managing unique diagnosable medical and surgical complications of the fetus or newborn (e.g., pediatric neurosurgeons, urologists, and others). Technical capabilities should include high-resolution ultrasound; the technical ability for early and routine amniocentesis; CVS and mid-trimester placental biopsy capabilities; and proficiency in cordocentesis. A close working relationship with a licensed, certified cytogenetics laboratory capable of providing timely cytogenetic, molecular genetic, and prenatal screening services is essential. Twenty-four hour emergency access to the system is also necessary.

Quality assurance is an essential part of maintaining a perinatal diagnostic service. A perinatal database allowing for population estimates of specific genetic and congenital anomalies and hence identifying areas of needed services addresses this issue. Furthermore, by using a regional perinatal database prospectively, the service can routinely evaluate and compare the sensitivity and specificity of services provided (i.e., genetic consultation, ultrasound, prenatal screening and testing) to national standards, so that specific areas of strength

or weakness can readily be identified and corrected. Patient satisfaction should be assessed regularly, and service adjustments made to better accommodate the special needs of families during such difficult times. Timely and effective communication among providers and between providers and the patient is essential. Systems must be in place to ensure that all relevant information is conveyed to all essential parties in a timely and accurate fashion while safeguarding the patient's confidentiality. Finally, establishing specific algorithms or protocols for certain high-volume clinical problems can standardize solutions to problems in management.

A continuous improvement model has been established by Boehm and coworkers [139] for a CVS program. Using a computerized database for the continuing analysis of complications and patient input, modifications in timing, type of procedure, and genetic counseling content were undertaken, with an overall improvement in outcome. Furthermore, areas of laboratory weakness were identified as well, with a subsequent improvement in specimen quality and turn-around time.

### **FUTURE TRENDS**

Recent reports have demonstrated evolving technologies and screening methods in prenatal diagnosis [140–143]. As an example, the detection of nucleated fetal red blood cells in maternal blood has generated great interest. With the enhancement of cell isolation and cell culture techniques, such cells could serve as a noninvasive source for prenatal diagnosis [144]. Currently, modifications in cell sorting and isolation, and the analysis techniques of FISH and PCR are being refined, to allow these technologies to serve as a prenatal screening tests. Such capabilities would permit the screening of a far larger number of abnormalities than is currently available and would help to better identify high-risk candidates for invasive prenatal testing. In brief, these techniques involve the use of fluorescent or biotinylated monoclonal antibodies directed at fetal cells. Using a fluorescent or magnetic activated cell sorter, the antibody-labeled cells are separated from unlabeled cells. Once isolated, tagged cells are subjected to genetic tests to assess whether specific chromosomal aneuploidies (e.g., trisomy 13, 18, or 21) are present. Recent reports have demonstrated

some limited clinical success with these techniques [144]. Problems and controversy continue to arise in the sensitivity of the technique, as well as concerns about the presence of fetal cells in maternal circulation for several years after each of a woman's several pregnancies [145].

In the field of assisted reproductive technology (i.e., in vitro fertilization [IVF] programs), the use of FISH and PCR holds great promise for preferentialization (polar body biopsy) and preimplantation (blastocyst biopsy) [146,147] diagnosis of genetic abnormalities. This could help identify ideal specimens for fertilization and implantation and potentially reduce the need for subsequent invasive prenatal testing. Several reports have been published in which post-IVF biopsy of one or two cells at the eight-cell blastocyst stage was undertaken. Using appropriate DNA primers and PCR, the identification of the sex of the embryos of couples at risk of having a child with an X-linked recessive disorder has been undertaken [148]. More recently, identification of cystic fibrosis in embryos of a couple at risk has been performed [149]. In all cases, embryos believed to be genetically unaffected were selected and implanted, with the subsequent birth of a normal child. Follow-up genetic testing has confirmed the preimplantation diagnosis.

The future for prenatal genetics is clearly one of high technology and continued controversy. Whether the specific technologies discussed here will develop is less important than the overall trend they represent. The aim for genetic services is to provide a maximum of genetic diagnostic capabilities for any given pregnancy, with a minimum of fetal risk. It is worthwhile to recall that technical ability commonly is far in advance of our ability to understand and manage the ethical, social, and personal implications of such studies, however. Physicians must continue to embrace new technologies while carefully weighing these abilities, to be certain that the advances and benefits for patient and practitioner are real and within our capacity to manage.

## REFERENCES

1. McKusik VA: A 40-year prospective on the evolution of a medical specialty from a basic science. *JAMA* 1993; 270:2351-2356.
2. Baird PA, Anderson TW, Newcombe HB, Lowry B: Genetic disorders in children and young adults: A population study. *Am J Hum Genet* 1988; 42:677-693.
3. Hall JG, Powers EK, McIlvaine RT, Ean VH: The frequency and financial burden of genetic disease in a pediatric hospital. *Am J Med Genet* 1978; 1:417-436.
4. Reich E, Wallace S, Ben-Yishay M, Schlesinger S, Marks J, Bloom A: Genetic disease in a pediatric hospital. *Am J Hum Genet* 1972; 26: 71A.
5. Emery AEH, Rimoin DL (eds): *Principles and Practice of Medical Genetics*, Second Edition. Edinburgh: Churchill Livingstone, 1990.
6. Nevin NC: Trends in prevalence of congenital abnormalities. In: Drife JO, Donnai D (eds): *Antenatal Diagnosis of Fetal Abnormalities*. London: Springer-Verlag, 1991; 3-11.
7. Liston R, Sawchuck D, Young D, Society of Obstetrics and Gynaecologists of Canada; British Columbia Perinatal Health Program: Fetal health surveillance: Antepartum and intrapartum, consensus guideline. *J Obstet Gynaecol Can* 2007; 29(9 Suppl 4):53-56.
8. Sekizawa A, Purwosunu Y, Matsuoka R, Koide K et al: Recent advances in non-invasive prenatal DNA diagnosis through analysis of maternal blood. *J Obstet Gynaecol Res* 2007; 33(6):747-764.
9. Williams C: Dilemmas in fetal medicine: Premature application of technology or responding to women's choice? *Social Health Iiin* 2006; 28(1):1-20.
10. Mennuti MT: Genetic screening in reproductive health care. *Clin Obstet Gynecol* 2008; 51(1):3-23.
11. Lambl D: Ein seltener fall von hydramnios. *Zent Fur Gynakol* 1881; 5:329-334.
12. Schatz F: Eine besondere Art von einseitiger polyhydramnie mit anderseitiger oligohydramnie bei einagueu Zwillingen. *Arch Gynakol* 1882; 19:329-369.
13. Prochownick L: Beitrage zur lehre vom fruchtwasser und seiner entsellung. *Arch Gynakol* 1887; 11:305-345.
14. Henkel M: Akutes hydramnion, leberkompression, enges becken, punktion des hydramnion. *Zent Fur Gynakol* 1919; 43:841-846.
15. Fuchs F, Riis P: Antenatal sex determination. *Nature*. 1956; 117:330.

16. Makowski EL, Perm K, Kaiser IH: Detection of sex of fetus by the incidence of sex chromatin in nuclei of cells in amniotic fluid. *Science* 1956; 123:542–543.
17. Shettles LB: Nuclear morphology of cells in human amniotic fluid in relation to sex of infant. *Am J Obstet Gynecol* 1956; 71:834–838.
18. Bevis DCA: The antenatal prediction of haemolytic disease of the newborn. *Lancet* 1952; 1:395–398.
19. Riis P, Fuchs F: Antenatal determination of foetal sex in prevention of hereditary disease. *Lancet* 1960; ii:180–182.
20. Serr DM, Margolis E: Diagnosis of fetal sex in a sex-linked hereditary disorder. *Am J Obstet Gynecol* 1964; 88:230–232.
21. Steele MW, Berg WR, JR: Chromosome analysis of human amniotic fluid cells. *Lancet*. 1966; 1:383–385.
22. Jacobson CB, Barter RN: Intrauterine diagnosis and management of genetic defects. *Am J Obstet Gynecol* 1967; 99:795–807.
23. Valenti C, Schutta EJ, Kehaty T: Prenatal diagnosis of Down's syndrome. *Lancet* 1968; 2:220.
24. Nadler HL: Antenatal detection of hereditary disorders. *Pediatrics* 1968; 42:912–918.
25. Nadler HL, Gerbie AB: Role of amniocentesis in the intrauterine detection of genetic disorders. *N Engl J Med* 1970; 282:596–599.
26. Johnson A, Gomilow L: Genetic amniocentesis at 14 weeks of less. *Clin Obstet Gynecol* 1988; 31:345–352.
27. Mohr J: Foetal genetic diagnosis: development of techniques for early sampling of foetal cells. *Acta Pathol Microbiol Scand* 1968; 73:73–77.
28. Hahnemann N: Early prenatal diagnosis; a study of biopsy techniques and cell culturing from extraembryonic membranes. *Clin Genet* 1974; 6:294–306.
29. Kullander S, Sandahl B: Fetal chromosome analysis after transcervical placental biopsies during early pregnancy. *Acta Obstet Gynecol Scand* 1973; 52:355–359.
30. Department of Obstetrics and Gynecology, Tietung Hospital of Anshan Iron and Steel Co. Anshan, China: Fetal sex prediction by sex chromatin of chorionic villi cells during early pregnancy. *Chinese Med J* 1975; 1:117–126.
31. Horwell DH, Loeffler FE, Coleman DV: Assessment of a transcervical aspiration technique for chorionic villus biopsy in the first trimester of pregnancy. *Br J Obstet Gynaecol* 1983; 90:196–198.
32. Liu DT, Mitchell J, Johnson J, Wass DM: Trophoblast sampling by blind transcervical aspiration. *Br J Obstet Gynaecol* 1983; 90:1119–1123.
33. Rhine SA, Cain JL, Cleary RE, Palmer CG, Thompson JF: Prenatal sex detection with endocervical smears: Successful results utilizing Y-body fluorescence. *Am J Obstet Gynecol* 1975; 122:155–160.
34. Rhine SA, Palmer CG, Thompson JF: A simple alternative to amniocentesis for first-trimester prenatal diagnosis. *Birth Defects* 1977; 12:231–247.
35. Goldberg MF, Chen ATL, Ahn YW, Reidy JA: First-trimester fetal chromosomal diagnosis using endocervical lavage: A negative evaluation. *Am J Obstet Gynecol* 1980; 138:436–439.
36. Kazy Z, Rozovsky IS, Bahkarev VA: Chorion biopsy in early pregnancy: A method of early prenatal diagnosis for inherited disorders. *Prenat Diagn* 1982; 2:39–40.
37. Ward RHT, Modell B, Petrou M, Karagozlu F, Douratsos E: Method of sampling chorionic villi in the first trimester of pregnancy under guidance of real-time ultrasound. *Br Med J* 1983; 286:1542–1544.
38. Simoni G, Brambati B, Daesino C, et al: Efficient direct chromosome analyses and enzyme determinations from chorionic villi samples in the first trimester of pregnancy. *Hum Genet* 1983; 63:349–357.
39. Smidt-Jensen S, Hahnemann N, Jensen PKA, Therkelsen AJ: Experience with transabdominal fine-needle biopsy from chorionic villi in the first trimester – an alternative to amniocentesis. *Clin Genet* 1984; 26:272–274.
40. Jeanty P, Rodesch F, Romero R, Venus I, Hobbins JC: How to improve your amniocentesis technique. *Am J Obstet Gynecol* 1983; 146:593–596.
41. Benn PA, Hsu LYF: Maternal cell contamination of amniotic fluid cell cultures: Results of a US nationwide survey. *Am J Med Genet* 1983; 15:297–305.
42. Verp MS, Gerbie AB: Amniocentesis for prenatal diagnosis. *Clin Obstet Gynecol* 1981; 24:1007–1020.
43. Turnbull AC, MacKenzie IZ: Second trimester amniocentesis and termination of pregnancy. *Br Med Bull* 1983; 39:315–321.
44. Finberg HJ, Frigoletto FD: Sonographic demonstration of uterine contraction during amniocentesis. *Am J Obstet Gynecol* 1981; 9:740–742.

45. Platt LD, DeVore GR, Gimovsky ML: Failed amniocentesis: The role of membrane tenting. *Am J Obstet Gynecol* 1982; 144:479–480.
46. Alfirevic Z, Sundberg K, Brigham S: Amniocentesis and chorionic villus sampling for prenatal diagnosis. *Cochrane Database Syst Rev* 2003; 3, CD 003252.
47. Wilson RD, Kendrick V, Wittmann BK, McGillivray BC: Risk of spontaneous abortion in ultrasonically normal pregnancies. *Lancet* 1984; 2:920–921.
48. Stein ZA: A woman's age: Child bearing and child rearing. *Am J Epidemiol* 1985; 327–342.
49. Gilmore DH, McNay MB: Spontaneous fetal loss rate in early pregnancy. *Lancet* 1985; 1:107.
50. MacKenzie WE, Holmes DS, Newton JR: Spontaneous abortion rate in ultrasonographically viable pregnancies. *Obstet Gynecol* 1988; 71:81–83.
51. Read AP, Donnai D, Harris R, Donnai P: Comparison of pregnancy outcome after amniocentesis for previous neural tube defect or raised maternal serum alpha fetoprotein. *Br J Obstet Gynaecol* 1980; 87:372–376.
52. Sant-Cassia LJ, MacPherson MBA, Tyack AJ: Midtrimester amniocentesis: Is it safe? A single-centre controlled prospective study of 517 consecutive amniocentesis. *Br J Obstet Gynaecol* 1984; 736–744.
53. NICHD National Registry for Amniocentesis Study Group: Midtrimester amniocentesis for prenatal diagnosis-safety and accuracy. *JAMA* 1976; 239:1471–1476.
54. Simpson NE, Dallaire L, Miller JR et al: Prenatal diagnosis of genetic disease in Canada: Report of a collaborative study. *Can Med Assoc J* 1976; 115:739–746.
55. Working Party on Amniocentesis: An assessment of hazards of amniocentesis. *Br J Obstet Gynaecol* 1978; 85(Suppl):1–41.
56. Tabor A, Philip J, Madser M, Bang J, Obel EB, Norgaard-Pedersen B: Randomised controlled trial of genetic amniocentesis in 4606 low-risk women. *Lancet* 1986; 1:1287–1289.
57. Mujezinovic F, Alfirevic Z: Procedure-related complications of amniocentesis and chorionic villus sampling: A systems review. *Obstet Gynecol* 2007 Sept 110(3):687–694.
58. American College of Obstetricians and Gynecologists: Invasive prenatal testing for aneuploidy Practice Bulletin Number 88, December 2007. Washington, DC: American College of Obstetricians and Gynecologists.
59. Zipursky A, Pollock J, Chown B, Israels LG: Transplacental fetal hemorrhage after placental injury during delivery amniocentesis. *Lancet* 1963; 2:493–498.
60. Mennuti MT, DiGaetano A, McDonnell A, Cohen AW, Liston RM: Fetal-maternal bleeding associated with genetic amniocentesis. *Obstet Gynecol* 1983; 62:26–30.
61. Hill LM, Platt LD, Kellogg B: Rh-sensitization after genetic amniocentesis. *Obstet Gynecol* 1980; 56:459–461.
62. Khalil MA, Tabsh K, Lobherz TB, et al: Risks of prophylactic anti-D immunoglobulin after second-trimester amniocentesis. *Am J Obstet Gynecol* 1984; 149:225–226.
63. Golbus MS, Stephens JD, Cann HM, et al: Rh-immunization following genetic amniocentesis. *Prenat Diagn* 1982; 2:149–156.
64. Tabor A, Jerne D, Bok JE: Incidence of rhesus immunization after genetic amniocentesis. *Br Med J* 1986; 293:533–536.
65. American College of Obstetricians and Gynecologists: Prevention of D Isoimmunization. Practice Bulletin. Number 4, May 1999. Washington DC: American College of Obstetricians and Gynecologists.
66. Karp LE, Schiller HS: Meconium staining of amniotic fluid at mid-trimester amniocentesis. *Obstet Gynecol* 1977; 50:47s–49s.
67. Cruikshank DP, Varner MW, Cruikshank JE, et al: Midtrimester amniocentesis: an analysis of 923 cases with neonatal follow-up. *Am J Obstet Gynecol* 1983; 146:204–211.
68. Hankins GD, Rowe J, Quirk JG, et al: Significance of brown and/or green amniotic fluid at the time of second-trimester genetic amniocentesis. *Obstet Gynecol* 1984; 64:353–358.
69. Dacus JV, Wilroy RS, Summit RL, et al: Genetic amniocentesis: A twelve years' experience. *Am J Med Genet* 1985; 20:443–452.
70. Zorn EM, Hanson FW, Greve LC, et al: Analysis of the significance of discolored amniotic fluid detected at mid-trimester amniocentesis. *Am J Obstet Gynecol* 1986; 154:1234–1240.
71. Milunsky A, Atkins L: Prenatal diagnosis of genetic disorders: An analysis of experience with 600 cases. *JAMA* 1974; 230:232–235.



72. Kerenyi TD, Walker B: The preventability of "bloody taps" in second-trimester amniocentesis by ultrasound scanning. *Obstet Gynecol* 1977; 50:61–64.
73. Crandall BF, Howard J, Lebhertz TB, et al: Follow up of 2000 second-trimester amniocenteses. *Obstet Gynecol* 1980; 56:625–628.
74. Benaceraff BR, Frigoletto FD: Amniocentesis under continuous ultrasound guidance: A series of 232 cases. *Obstet Gynecol* 1983; 62:760–763.
75. McDicken WN, Anderson T, MacKenzie WE, et al: Ultrasonic identification of needle tips in amniocentesis. *Lancet* 1984; 2:198–199.
76. Romero R, Jeanty P, Reece EA, et al: Sonographically monitored amniocentesis to decrease intraoperative complications. *Obstet Gynecol* 1985; 65:426–430.
77. Williamson RA, Varner MW, Grant SS: Reduction in amniocentesis risks under real-time guide procedure. *Obstet Gynecol* 1985; 65:751–755.
78. Gerbie AB, Shkolnik AA: Ultrasound prior to amniocentesis for genetic counseling. *Obstet Gynecol* 1975; 46:716–719.
79. Karp LE, Rothwell R, Conrad SH, Hoehn HW, Hickok DE: Ultrasonic placental localization and bloody taps in mid-trimester amniocentesis for prenatal diagnosis. *Obstet Gynecol* 1977; 50:589–593.
80. Thorp JA, Helfgott AW, King EA, King AA, Minyard AN: Maternal death after second-trimester genetic amniocentesis. *Obstet Gynecol* 2005; 105(5 Pt 2):1213–1215.
81. MacLachlan NA: Amniocentesis. In: Brock DJH, Rodeck CH, Ferguson-Smith MA (eds): *Prenatal Diagnosis and Screening*. Edinburgh: Churchill Livingstone, 1992; 17.
82. Simpson JL, Socol ML, Aladjem S, et al: Normal fetal growth despite persistent amniotic fluid leakage after genetic amniocentesis. *Prenat Diagn* 1981; 1:277–279.
83. Raimer SS, Raimer BG: Needle puncture scars from mid-trimester amniocentesis. *Arch Dermatol* 1984; 120:1360–1362.
84. Philip J, Silver RK, Wilson RD, Thom EA et al. for the NICHD EATA Trial Group: Late first trimester invasive prenatal diagnosis: Results of an international randomized trial. *Obstet Gynecol* 2004; 103(6):1164–1173.
85. Wapner RJ, Jackson L: Chorionic villus sampling. *Clin Obstet Gynecol* 1988; 31:328–344.
86. Cauqhey AB, Hopkins LM, Norton ME: Chorionic villus sampling compared with amniocentesis and the difference in the rate of pregnancy loss. *Obstet Gynecol* 2006; 108(3 Pt 1):612–616.
87. Dalpra L, Nocera G, Tibiletti MG, et al: "Late" chorionic villus sampling: Cytogenetics aspects. *Prenat Diagn* 1993; 13:239–246.
88. Smidt-Jensen S, Ludsteen C, Lind AM, Dinesen K, Philip J: Transabdominal chorionic villus sampling in the second and third trimester of pregnancy: Chromosome quality, reporting time and feto-maternal bleeding. *Prenat Diagn* 1993; 13: 957–969.
89. Bovicelli L, Rizzo N, Montacuti V, et al: Transabdominal chorionic villus sampling: Analysis of 350 consecutive cases. *Prenat Diagn* 1988; 8: 495–500.
90. Green JE, Dorfmann A, Jones SL, et al: Chorionic villus sampling: Experience with an initial 940 cases. *Obstet Gynecol* 1988; 71:208–212.
91. Jahoda MGJ, Pijpers L, Reuss A, et al: Evaluation of transcervical chorionic villus sampling with a completed follow-up of 1550 consecutive pregnancies. *Prenat Diagn* 1989; 9:621–628.
92. Leschot NJ, Wolf H, Van Prooijen-Knejt AC, et al: Cytogenetic findings in 1250 chorionic villus samples obtained in the first trimester with clinical follow-up of the first 1000 pregnancies. *Br J Obstet Gynaecol* 1989; 96:663–670.
93. Miny P, Basaran S, Pawlowitzki IH, et al: Validity of cytogenetic analysis from trophoblastic tissue throughout gestation. *Am J Med Genet* 1989; 33:136–141.
94. Brambati B, Lanzani A, Tului L: Transabdominal and transcervical chorionic villus sampling: Efficiency and risk evaluation of 2411 cases. *Am J Med Genet* 1990; 35:160–164.
95. USNICHD Collaborative CVS Group: Transcervical and transabdominal chorionic villus sampling are comparably safe procedures for first-trimester prenatal diagnosis: Preliminary analysis. *Am J Hum Genet* 1990; 47:A278.
96. Brambati B, Terzian E, Tognoni G: Randomised clinical trial of transabdominal versus transcervical chorionic villus sampling methods. *Prenat Diagn* 1991; 11:285–293.
97. Brambati B, Tului L, Simoni G, Travi M: Prenatal diagnosis at 6 weeks. *Lancet* 1988; 2: 397.

98. Monni G, Ibba RM, Lai R, Cau G, Mura S, Olla G, et al: Early transabdominal chorionic villus sampling in couples at high genetic risk. *Am J Obstet Gynecol* 1993; 168:170–173.
99. Brambati B, Tului L: Chorionic villus sampling and amniocentesis: Prenatal diagnosis: *Curr Opin Obstet Gynecol* 2005;17(2):197–201.
100. Wapner RJ: Invasive prenatal diagnostic techniques. *Semin Perinatol* 2005; 29(6):401–404.
101. Holzgreve W, Miny P, Gerlach B, Wensterdorp A, Ablert D, Horst J: Benefits of placental biopsy for rapid karyotyping in the second and third trimester (late chorionic villus sampling) in high-risk pregnancies. *Am J Obstet Gynecol* 1990; 162:1188–1192.
102. Williams J III, Medearis AL, Chu WH, Kovacs AD, Kaback MM: Maternal cell contamination in cultured chorionic villi: Comparison of chromosome Q-polymorphisms derived from villi, fetal skin and maternal lymphocytes. *Prenat Diagn* 1987; 7:315–322.
103. Roberts E, Duckett DP, Lang CD: Maternal cell contamination in chorionic villus samples assessed by direct preparation and three different culture methods. *Prenat Diagn* 1988; 8:635–640.
104. Wright DJ, Bradley BA, Koppitich FC, Drugan A, Johnson M, Evans MI: Interpretation of chorionic villus sampling laboratory results are just as reliable as amniocentesis. *Obstet Gynecol* 1989; 74:739–744.
105. Ledbetter DH, Martin AO, Verlinsky Y, et al: Cytogenetic results of chorionic villus sampling; high success rate and diagnostic accuracy in the United States collaborative study. *Am J Obstet Gynecol* 1990; 2:495–501.
106. Ledbetter DH, Zachary JM, Simpson JL, et al: Cytogenetics results from the US Collaborative Study on CVS. *Prenat Diagn* 1992; 12:317–345.
107. Boris BT, Ch R, Williams S III: Mosaicism in chorionic villus sampling; an analysis of incidence and chromosomes involved in 2612 consecutive cases. *Prenat Diagn* 1993; 13:179–190.
108. Johnson A, Wapner RJ, Davis GH, Jackson LG: Mosaicism in chorionic villus sampling: An association with poor perinatal outcome. *Obstet Gynecol* 1990; 75:573–577.
109. Wapner RJ, Simpson JL, Golbus MS, et al: Chorionic mosaicism: Association with fetal loss but not with adverse outcome. *Prenat Diagn* 1992; 12:347–355.
110. Ammala P, Hilesmaa VK, Liukkonen S, Saisto T, Teramo K, Vonkoskull H: Randomized trial comparing first-trimester transcervical chorionic villus sampling and second-trimester amniocentesis. *Prenat Diagn* 1993; 13:919–927.
111. Medical Research Council Working Party on the Evaluation of Chorion Villus Sampling: Medical Research Council European trial of chorionic villus sampling. *Lancet* 1991; 377:1491–1496.
112. Olney RS, Khoury MJ, Alo CJ, Costa P, Edmonds LD, et al: Increased risk for transverse digital deficiency after chorionic villus sampling: Results of the United States Multistate Case-Controlled Study, 1988–1992. *Teratology* 1995; 51(1): 20–29.
113. Golden CM, Ryan LM, Holmes LB: Chorionic villus sampling: A distinctive teratologic effect on fingers? *Birth Defects Res A Clin Mol Teratol* 2003; 67(8):557–562.
114. Canadian Collaborative CVS–Amniocentesis Clinical Trial Group: Multicentre randomized clinical trial of chorion villus sampling and amniocentesis. *Lancet* 1989; 1:1–6.
115. Rhoads GG, Jackson LG, Schlesselman SE, et al: The safety and efficacy of chorionic villus sampling for early prenatal diagnosis of cytogenetic abnormalities. *N Engl J Med* 1989; 320:609–617.
116. Smidt-Jensen S, Permin M, Philip J, et al: Randomised comparison of amniocentesis and transabdominal and transcervical chorionic villus sampling. *Lancet* 1992; 340:1237–1244.
117. Kuliev AM, Modell B, Jackson L, Simpson JL, Brambati B, Rhoads G, et al: Risk evaluation of CVS. *Prenat Diagn* 1993; 13:197–208.
118. Froster-Iskenius VA, Baird PA: Limb reduction defects in over one million consecutive live births. *Teratology* 1989; 39:127–135.
119. Mastroiacovo P, Tozzi AE, et al: Transverse limb reduction defects after chorionic villus sampling: a retrospective cohort study. *Prenat Diagn* 1993; 13:1051–1056.
120. Alfrevic, Z, Sundberg K, Birgham S: Amniocentesis and chorionic villus sampling for prenatal diagnosis [Review]. *The Cochrane Library* 2005; 4. Update Software, Oxford.
121. Waterhouse JAH: Twinning in twin pedigrees. *Br J Soc Med* 1950; 4:197–216.

122. Bulmer MG: Twinning rate in Europe during the war. *Br Med J* 1959; 1:29–30.
123. Myriantopoulos NC: An epidemiologic survey of twins in a large prospectively studied population. *Am J Hum Genet* 1970; 22:611–629.
124. Nicolaides KH: *The 11–14 weeks scan 2004*; London: Fetal Medicine Foundation. pg. 10.
125. Rodis JF, Egan JFX, Craffey MS, Ciarleglio L, Greenstein RM, Scorza WE: Calculated risk of chromosomal abnormalities in gestations. *Obstet Gynecol* 1990; 76:1037–1041.
126. Palle C, Andersen JW, Tabor A, Lauritsen JG, Bang J, Phillips J: Increased risk of abortion after genetic amniocentesis in twin pregnancies. *Prenat Diagn* 1983; 3:83–89.
127. Anderson RL, Goldberg JD, Golbus MS: Prenatal diagnosis in multiple gestation: 20 years' experience with amniocentesis. *Prenat Diagn* 1991; 11:263–270.
128. Ghidini A, Lynch L, Hicks C, Alvarez M, Lockwood CJ: The risk of second-trimester amniocentesis in twin gestations: A case-control study. *Am J Obstet Gynecol* 1993; 169:1013–1016.
129. Jeanty P, Shah D, Roussis P: Single-needle insertion in twin amniocentesis. *J Ultrasound Med* 1990; 9:511–517.
130. Yukobowich E, Anteby EY, Cohen SM, Lavy Y, Granat M, Yagel S: Risk of fetal loss in twin pregnancies undergoing second-trimester amniocentesis. *Obstet Gynecol* 2001; 98:231–234.
131. Brambati B, Tului L, Travi M, et al: First-trimester genetic diagnosis in multiple pregnancy: Principles and potential pitfalls. *Prenat Diagn* 1991; 11:767–774.
132. Quintero RA, Reich H, Puder KS, Bardneff M, Evans MI, Cotton DB, Romero R: Brief report: Umbilical cord ligation of an acardiac twin by fetoscopy at 19 weeks' gestation. *N Engl J Med* 1994; 330:469–471.
133. Hsu LYF: Prenatal diagnosis of chromosomal abnormalities through amniocentesis. In: Milunsky A (ed): *Genetic Disorders and the Fetus*. Third Edition. Baltimore: Johns Hopkins University Press, 1992; pp. 155–210.
134. Wright DJ, Brindley BA, Koppitch FC, Drugan A, Johnson MP, Evans MI: Interpretation of chorionic villus sampling laboratory results are just as reliable as amniocentesis. *Obstet Gynecol* 1989; 74:739–744.
135. Ledbetter DH, Martin AO, Verlinsky Y, et al: Cytogenetic results of chorionic villus sampling: High success rate and diagnostic accuracy in the United States collaborative study. *Am J Obstet Gynecol* 1990; 162:495–501.
136. Smidt-Jensen S, Lind AM, Permin M, Zachary JM, Windsteen C, Philip J: Cytogenetic analysis of 2928 CVS samples and 1075 amniocentesis from randomized studies. *Prenat Diagn* 1993; 13:723–740.
137. Kerber S, Held KR: Early genetic amniocentesis – 4 years experience. *Prenat Diagn* 1993; 13:21–27.
138. Lockwood DH, Neu RL: Cytogenetic analysis of 1375 amniotic fluid specimens from pregnancies with gestational age less than 16 weeks. *Prenat Diagn* 1993; 13:801–805.
139. Boehm F, Salyer SL, Dev VG, Reed CW: Chorionic villus sampling: Quality control – a continuous improvement model. *Am J Obstet Gynecol* 1993; 168:1766–1777.
140. Weisz B, Rodeer CH: An update on antenatal screening for Down's syndrome and specific implications for assisted reproduction pregnancies. *Hum Reprod Update* 2006; 12(5):513–518.
141. Ball RH, Caughey AB, Malone FD et al: FASTER research consortium. First and second trimester evaluation of risk for Down syndrome. *Obstet Gynecol* 2007; 110(1):10–17.
142. Natarajan G, Klein MD: The fetus as a patient: Prenatal diagnosis and fetal therapy. *Medicine (imedicine.com)* 2006, topic-2953.
143. Breathnach FM, Malone FD, Lambert Messerlian G, et al, FASTER research consortium: First and second trimester screening: Detection of aneuploidies other than Down syndrome. *Obstet Gynecol* 2007; 40(3): 651–657.
144. Simpson JL, Elias S: Isolating fetal cells from maternal blood: Advances in prenatal diagnosis through molecular technology. *JAMA* 1993; 270:2357–2361.
145. Hamada H, Arirami T, Hamaguchi H, Kubo T: Fetal nucleated cells in maternal peripheral blood after delivery. *Am J Obstet Gynecol* 1994; 170:1188–1193.
146. Navidi W, Arnheim N: Using PCR in preimplantation genetic disease diagnosis. *Hum Reprod* 1991; 6:836–849.
147. Marik JJ: Preimplantation genetic diagnosis. *imedicine (imedicine.com)* 2005, Topic-3520.

148. Hardyside AH, Kontogianni EH, Hathy K, Winston AML: Pregnancies from biopsied human perimplantation embryos sexed by 4 specific DNA amplification. *Nature* 1990; 344:768-770.
149. Handyside AH, Lesko JG, Tarin JJ, Winston RMC, Hughes MR: Birth of a normal girl after in vitro fertilization and preimplantation diagnostic testing for cystic fibrosis. *N Engl J Med* 1992; 327:909.

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Alisa B. Modena

Aileen M. Gariepy

Stuart Weiner

*... For a diagnostic technique to be acceptable it must not involve the patient in pain, indignity or hazard. Sonar meets these requirements admirably.*

Ian Donald (1910–1987)

*Practical Obstetric Problems*

London: Lloyd – Luke, 5th Edition, 1979, p. 1020

Traditionally, ultrasonography has been a means to guide antepartum obstetric management. Recently, investigators have focused on expanding its use in the labor and delivery suite, from the triage of high-risk patients to its assistance in the third stage of labor and postpartum evaluation. Additionally, the use of ultrasound scanning in the intrapartum assessment of patients in labor and its invaluable utility to guide invasive procedures are being examined. This chapter reviews several of these recent advances: cervical length evaluation as a predictor of preterm delivery and for the selection of appropriate induction of labor candidates; the evaluation of uterine bleeding; the monitoring of intrapartum fetal weight, presentation, and fetal well-being; and the guidance of invasive procedures. Furthermore, the authors wish to demonstrate the role of ultrasonography in aiding the understanding of aberrations from the expected course of descent and rotation of the fetus, choosing the proper operative delivery technique, guiding delivery of the second of twins, assessing retained placental tissue, and monitoring the maternal anal sphincter for nonapparent damage that could benefit from repair in the puerperium.

### CERVICAL LENGTH ASSESSMENT

Assessment of the uterine cervix has been accomplished with the assistance of ultrasonography for the past three decades. As technology has improved significantly, newer and more reliable techniques of evaluation have become the standard care for the evaluation of this portion of female anatomy. Why is accurate assessment of this particular structure important?

Determination of the length of the uterine cervix is vital to the evaluation of patients with obstetric pathologies. It is important in the assessment of the patient experiencing preterm uterine contractions and is crucial in the estimation of the risk of premature delivery in women with a history of prior preterm delivery. Sonographic assessment of the cervix has been established as a reliable means of measuring the cervical length and as an important component in the prediction of preterm labor.

It has been postulated and confirmed in the literature that as the cervix shortens in length, the risk for preterm birth increases [1]. Traditionally, digital examination was the method of choice to evaluate the cervix; unfortunately, this technique is limited by both significant variation among examiners and the inability to assess the proximal portion of the cervix and the internal os [2].

Cervical length measurement as a tool for predicting cervical readiness for induction of labor has also been evaluated. Investigation of cervical length to predict outcome in labor induction has demonstrated this parameter to be associated with the more accurate prediction of successful induction beyond more traditional methods, including the Bishop score [3–5]. Finally, sonography of the uterine cervix can delineate numerous anatomic features and establish relationships to other pelvic organs efficiently and reproducibly.

There are three ways to evaluate the cervix by ultrasound scan: *transabdominal*, *transvaginal*, and *transperineal*. Initially, sonographers imaged the uterine cervix by a transabdominal approach. Although not as challenging technically as other methods, accurate transabdominal imaging of the cervix requires the gravida have a full bladder to provide an acoustic window through which cervical length and anatomy can be seen. Visualization of the cervix by a transabdominal approach can be limited by partial fullness of the patient bladder, maternal body habitus, or shadowing from a fetal presenting part. Conversely, overdistention of the bladder can artificially extend and flatten the underlying lower uterine segment and cervix, yielding an incorrect assessment of its length and dilatation. Currently, transabdominal sonography of the cervix is used to screen the length of the cervix during an examination, whereas other techniques of measuring the cervix are used for more definitive evaluation. Owing to the difficulty in obtaining an accurate image for the reasons listed previously, transvaginal or transperineal sonography are typically preferred over transabdominal or are used as complementary examinations.

Transvaginal sonography replaced transabdominal sonographic evaluation of the cervix once it was established to provide a more accurate assessment of cervical length and pathology [6–8]. Transvaginal sonography can be performed with a variety of transducer types using higher sound frequen-

cies than transabdominal sonography because the region of interest is nearer to the probe. This characteristic confers better anatomic resolution of the cervix. It can be difficult at first for the sonographer to adapt to the more restricted field of view obtained with this type of probe, however. Initially, it was thought that inserting an ultrasound probe into the vagina could be potentially hazardous in specific circumstances. Several studies have looked at potential contraindications to transvaginal sonography, including preterm labor, chorioamnionitis in preterm ruptured membranes, and placenta previa, and found that there is no true clinical risk or contraindication to performing this procedure in these groups of patients [9–10].

In a transvaginal examination, the patient is placed in a supine position with hips abducted; a full bladder is not required. A 3.5- to 8-MHz transducer (properly sterilized according to the manufacturer's recommendations) sheathed in a probe cover is inserted halfway between the introitus and cervix. Initial insertion can be done by or with the assistance of the gravida herself. The operator should handle the probe lightly, taking care to avoid excessive pressure on the anterior cervical lip, which can lead to a falsely increased measurement of cervical length. Guidelines have been established for transvaginal sonography to reduce interobserver variability. The entire length of the cervical canal should be visualized (see Figure 3.1); the external os should appear symmetric, and the distance from the surface of the posterior lip to the cervical canal is equal to the distance from the surface of the anterior lip to the



FIGURE 3.1.  
*Normal cervix.*

cervical canal. Finally, the internal os should be visualized as a flat dimple or an isosceles triangle [11].

Transperineal or translabial sonography is typically well tolerated by the patient and should be considered when there are any concerns about the risk of infection or inciting bleeding, or when a vaginal probe transducer is not available. A 3.5- to 5.0-MHz sector or curvilinear transducer covered with a sheath is applied in the sagittal plane to the patient's perineum. Partial fullness of the bladder assists in the visualization of the cervix by conveying sound waves toward the cervix and by being an identifiable landmark. The cervical canal is generally oriented at a right angle from the distal vagina and can be assessed using the same landmarks described above for transvaginal sonography. Transperineal sonography has been observed to have a good correlation with transvaginal sonography for accurate cervical length measurement [12]; however, it remains technically more challenging because of the less familiar orientation of the anatomy and thus is less frequently performed.

Endocervical length varies by modality used. Transvaginal sonography has been shown to be the method most amenable to standardization, providing the highest degree of consistency for cervical measurement. Several studies described cervical lengths by transabdominal sonography (mean 3.2 cm–5.3 cm), transvaginal sonography (mean 3.2 cm–4.8 cm) and transperineal sonography (mean 2.9 cm–3.5cm) [8,12–14]. Additionally, cervical length can vary within different subpopulations; it depends on parity, obstetric history, maternal age, maternal nutritional status, and history of drug use [15].

To obtain an accurate measurement of the cervix, one should obtain a true longitudinal view, including both the internal os and the external os. The image should be enlarged to fill at least one half of the ultrasound screen and oriented, by convention, so that the patient's head (cephalad) is on the left side of the screen. Undue pressure on the cervix by a vaginal ultrasound probe can artificially increase its apparent length and can be avoided by first obtaining a satisfactory image, withdrawing the probe until the image begins to blur, and then reapplying only enough pressure to restore the image clarity. Amniotic fluid and the lowest edge of the empty maternal bladder should be identified as landmarks, and the presence of membranes in the cervical canal or

beyond the cervix should be noted. The *internal os* is often located just below the lowest edge of the bladder and is the point at which the cervical canal and the amniotic sac or fetal presenting parts meet. Proceeding from the internal os, one should then locate the endocervical canal and external os. The *external os* is the point within the vaginal canal at which the anterior and posterior ends of the cervix meet. The appropriate sagittal views are obtained by locating the V-shaped notch at the internal os, and the triangular area of echodensity at the external os. The canal appears as a faint line of echodensity or echolucency between the two.

The cervix should be measured at least three times; the *cervical length to record is the shortest one that clearly displays both the internal and external cervical os and the entire length of the cervical canal*. The sum of two separate measurements or the trace function of the ultrasound calipers might be required if the cervical canal is not straight. In the presence of funneling or dilation of the internal os (see Figure 3.2), the residual (i.e., closed) cervical length should be measured. The depth of a funnel and whether it is static or dynamic should also be noted. If a funnel does not develop spontaneously, one can be induced by asking the patient to perform the Valsalva maneuver or by applying moderate fundal pressure. Prolonged observation of the cervix for 3 to 5 minutes is recommended because dynamic changes manifesting as dilation of the internal os or funneling can occur during the course of an examination [16].



FIGURE 3.2.  
*Funneled cervix.*

When clinically indicated, cervical length measurement by ultrasound, used in conjunction with digital examination, is an invaluable tool. With digital examination only, the practitioner cannot obtain an accurate evaluation of the supravaginal portion of the cervix. The length of this segment of the cervix can be assessed by sonography. Furthermore, early cervical changes such as subtle dilation and funneling typically begin at the level of the internal os, which might not be perceived on digital examination. Using receiver operator curves, Iams and Gomez determined that cervical sonography was a better predictor of preterm birth than digital assessment of cervical dilation and effacement [1,17]. This advantage remains with even early changes of the uterine cervix. The overwhelming advantage of sonography over digital examination is chiefly confined to early labor, when cervical dilation is less than 3 cm [13].

The process of cervical effacement is visualized sonographically as funneling of the cervical canal, typically materializing once the amniotic sac begins to protrude into the cervical canal. On ultrasound scan, this can appear as a change from a closed cervix to a wedge-shaped opening of the internal os followed by progressive shortening of the cervix craniocaudally. The process of effacement described as initially V shaped and then U shaped on ultrasound examination depends on the descent of the fetal presenting part [18]. Criteria for defining a funneled cervix have been described in an effort to standardize measurement and reporting. *Funnel width* is described as the dilation of the internal os, whereas *residual cervical length* is the closed cervical length distal to the funnel. *Funnel length* is the length of an imaginary line that connects the apex of the funnel to the cephalad-most edge of the base of the funnel. The percentage of funneling is the total funnel length divided by the residual cervical length plus the funnel length, that is, the total length of the cervix [19]. A funnel percentage of greater than 40% is a significant predictor of preterm delivery [19].

Sonographic assessment of cervical length as a predictor of preterm delivery has been studied in low- and high-risk women who are asymptomatic or symptomatic. There are a multitude of prospective studies showing that cervical length assessment by ultrasonography is not only reproducible but is an extremely valuable technique in terms of its predic-

tive value. Normal cervical length is relatively stable in the late second and early third trimester but begins to shorten slightly with increasing gestational age during the third trimester. After 16 weeks' gestation, the median cervical length is between 40 mm and 45 mm. As pregnancy progresses, the median cervical length decreases to 35 mm to 40 mm at 24 to 28 weeks, and 30 mm to 35 mm after 32 weeks [1].

Standardization of cervical length using transvaginal sonography was performed in approximately 3000 women to determine these normal lengths [1]. Iams et al. described normal cervical length between 22 and 30 weeks' gestation by a bell-shaped curve; the 90 percentile is 45 mm; the 50 percentile is 35 mm; the 10 percentile is 25 mm; the 5 percentile is 20 mm. Furthermore, they established the lack of clinical utility in measuring cervical length prior to 16 weeks' gestation because cervical shortening and funneling typically do not occur prior to that gestational age.

In asymptomatic high-risk women (i.e., those gravidas with a history of a prior preterm birth), their risk of preterm delivery in the subsequent pregnancy correlates with the cervical length measured during that pregnancy [20]; furthermore, the relative risk of preterm birth increases as the cervical length decreases. Additionally, the degree of "shortness" of the cervix is associated with the gestational age at delivery in the previous preterm birth, that is, the earlier the gestational age of the previous delivery, the shorter the cervix in the subsequent pregnancy [20–21]. Conversely, there appears to be little correlation between cervical length on ultrasound examination and the risk of preterm birth in asymptomatic low-risk women. These women were analyzed in the Preterm Prediction Study for their risk for preterm delivery based on cervical length assessment at 24 weeks' gestation. Cervical length on ultrasound examination was found to have a low sensitivity in this population for predicting preterm delivery at less than 35 weeks [22].

Finally, symptomatic women, that is, those who report preterm uterine contractions, have been assessed to determine the utility of evaluating the cervical length on labor and delivery. There appears to be a significant benefit from sonographic evaluation of cervical length when managing women whose diagnosis of preterm labor is uncertain. Of *symptomatic* women whose cervical lengths are



measured with ultrasonography, those whose cervixes are found to be greater than 15 mm have a low likelihood of delivering within seven days of the examination [23–26]. Conversely, approximately 50% of symptomatic women with a cervical length of less than or equal to 15 mm will deliver within seven days of the examination [24]. The same group analyzed different risk factors and found that the only significant predictor of preterm delivery within 48 hours of initial examination was cervical length [25]. Furthermore, Gomez et al found that vaginal ultrasonographic examination of the uterine cervix was more accurate than digital examination of the cervix in the assessment of the risk for preterm delivery in patients with preterm labor and intact membranes [17]. The findings of these studies provide important clinical information to the obstetrician on labor and delivery in terms of assessing symptomatic patients and planning their management strategy.

Cervical length assessment for predicting the outcome of labor induction has been evaluated in numerous studies. There are mixed results in the literature; some authors have found a significant benefit to cervical length assessment [3–5,27–28], whereas others have found little additional assistance in guiding labor induction management [29–32]. In 2004, Rane et al. determined that preinduction cervical length was an independent predictor of the likelihood of vaginal delivery within 24 hours [27]. In this same cohort of more than 600 patients, those authors found that cervical length was an independent predictor of cesarean delivery. They concluded that sonographic parameters, including cervical length assessment, were superior to the Bishop score for the prediction of these labor outcomes. In separate studies by Yang, Ware, and Gabriel, cervical length performance was directly compared to the Bishop score [3–5]. All together these studies measured the cervical length in 350 patients prior to labor induction. They found a more significant relationship between vaginal delivery and cervical length than the Bishop score and concluded that cervical length measured by transvaginal ultrasonography was a useful and independent predictor of successful labor induction. Gabriel found that in women having an unfavorable Bishop score, a cervical length of less than 26 mm was associated with a lower risk of cesarean delivery and a shorter dura-

tion of labor, whereas Ware found that women with a cervical length of less than 30 mm were more likely to be delivered vaginally and had significantly shorter labors. Bartha et al. specifically looked at cervical length measurement as a means of defining cervical ripeness [28]. They employed a cervical length cutoff of greater than 30 mm for the use of prostaglandin for cervical ripening and found that using transvaginal cervical length significantly reduced the need for intracervical prostaglandin treatment for patients whose cervixes were deemed ripe on sonographic assessment.

Nevertheless, there are an equivalent number of studies that have not found cervical length ultrasound measurement to be more accurate at predicting the outcome of labor induction than digital examination or Bishop score. In postterm patients, Chandra et al. found that transvaginal ultrasound did not predict successful labor induction as well as digital cervical examination [31]. Additionally, several studies found the Bishop score to be a better predictor of the time interval from induction to delivery [29–30,32]. At this time, the use of cervical length in the assessment of preinduction cervical readiness should be used with caution.

## ASSESSMENT OF FETAL STATUS

### **Amniotic Fluid Index**

Evaluation of fetal amniotic fluid quantity is an essential fetal assessment tool for the obstetrician. Amniotic fluid serves numerous functions vital to the normal development of the fetus, including musculoskeletal and lung function. Pathologic quantities of amniotic fluid should alert the physician to the possibility of impending harm to the pregnancy. Traditionally, amniotic fluid volume has been a reliable predictor of perinatal outcome.

The origin of amniotic fluid during the first trimester is a combination of maternal plasma transudate through the chorioamnion and fetal plasma through permeable fetal skin. By the mid-trimester, amniotic fluid production and excretion are entirely managed by the fetus. Production of fluid occurs mainly in the fetal kidneys and lungs; removal of fluid occurs by the fetal gastrointestinal system and through the amniochorionic membrane, thus distributing it to the maternal circulation. Amniotic

fluid assessment is most typically accomplished by quantitative measurement of the volume of fluid, because this provides direct insight into the status of fetal physiology. In the average pregnancy, the mean amniotic fluid volume increases from 30 ml to 1000 ml between 10 and 37 weeks; the average volume remains stable up to 39 weeks, after which it declines to approximately 500 ml by 42 weeks. The rate of amniotic fluid production is also gestational age dependent; at 8 weeks' gestation, the rate increases by 10 ml/week; at 13 weeks it is 25 ml/week; and at 21 weeks the rate of production reaches a maximum of 60 ml/week. Postterm the volume declines at a rate of approximately 8% per week [33].

Many factors can influence amniotic fluid volume; fetal metabolism, fetal blood volume, maternal hydration, and certain maternal disease states have all shown associations with amniotic fluid volume. For example, it has been postulated that maternal hydration not only improves amniotic fluid volume by improving uteroplacental blood flow but also increases the transfer of water across the placenta into the amniotic cavity. Studies have shown that maternal rehydration can alter amniotic fluid volume significantly [34].

An accurate measurement of amniotic fluid volume is crucial to assist in clinical decision making. In lieu of the risk and difficulty in obtaining measurements through invasive means such as amniocentesis with dye-dilution studies, ultrasonography, a noninvasive technique, has become an objective and safe procedure to determine fluid quantity. Although experienced sonographers can make a subjective estimation of the amount of fluid, an objective measurement is preferable in most instances. In 1981, Manning et al. described a technique called the *maximum vertical pocket* (MVP), which involved the selection, under ultrasound guidance, of the single deepest amniotic fluid pocket [35]. The quantitative depth was determined as the largest dimension of this pocket measured with the ultrasound transducer perpendicular to the floor. In 1987, Phelan et al. devised a semiquantitative sonographic assessment of the amniotic fluid volume called the *amniotic fluid index* (AFI) [36]. The AFI is a measurement based on the division of the gravid uterus into four quadrants using the external landmark of the maternal umbilicus and linea nigra to divide the quadrants.

The AFI is obtained by the summation of the deepest vertical pocket in each quadrant that is free of umbilical cord or extremities. Many sonographers make use of color Doppler mapping, if available, to ensure an umbilical-cord-free pocket.

Studies have found modest differences in the assessment accuracy of AFI and MVP. In 1990, Moore found that AFI became less accurate at lower fluid volumes, decreasing the sensitivity for the detection of oligohydramnios to less than 50% [37]. Similarly, Miyamura et al. found that an MVP measurement of  $\leq 3$  cm was more useful for establishing oligohydramnios than AFI measurement [38]. In 2004, Chauhan found no difference in the two semiquantitative measurements in their ability to predict perinatal outcome; however, they did find that oligohydramnios was diagnosed more frequently with AFI than MVP [39]. The same author found, however, that both the AFI and MVP were relatively inaccurate predictors of oligohydramnios or polyhydramnios when compared with dye-dilution calculations of actual amniotic fluid volume [40]. Despite these limitations, AFI and MVP quantitative assessments are preferred over the highly invasive approach of amniocentesis with dye instillation since this latter approach confers a significant risk of pregnancy loss to the patient.

Regardless of which quantitative assessment of amniotic fluid volume the practitioner chooses, what is the relationship between an abnormal volume and poor perinatal outcome? There are numerous conditions that can cause decreased placental perfusion, thus decreasing the oxygen and substrate flow to the fetus and resulting in relatively hypoxemic blood flowing to the fetal organs. The fetus subsequently redistributes its cardiac output to ensure adequate oxygen delivery to the most vital organs, increasing blood flow to the brain, heart, and adrenal glands while decreasing the relative flow to the abdomen and kidneys. The decreased renal perfusion and increased antidiuretic hormone release from this hypoxemia result in decreased urine output and oligohydramnios [41]. In the presence of hypoxemia, fetal lung resorption of fluid increases, adding to the low amniotic fluid volume.

A variety of thresholds has been described to define an AFI diagnosis of oligohydramnios and polyhydramnios. In Manning's original work from 1980, oligohydramnios was defined as the absence of

a single pocket of amniotic fluid with a depth  $\leq 1.0$  cm [48]. When this definition was thought to be too restrictive, Manning redefined the MVP definition to be the absence of a 2.0 cm by 2.0 cm pocket of fluid [42]. Using amniotic fluid index, oligohydramnios was defined as an AFI  $\leq 5.0$  cm [36]. In 1990, Moore obtained AFIs in almost 800 normal pregnancies and defined oligohydramnios as an AFI below the fifth percentile for gestational age [37]. This value varies between 7.9 cm at 16 weeks' and 6.3 cm at 40 weeks' gestation. Although an AFI of  $\leq 5$  cm as Phelan suggested would include  $\leq 1\%$  of term gestations, this AFI definition of oligohydramnios is the one most commonly used by obstetricians.

Several studies have reported that intrapartum oligohydramnios is associated with a poor perinatal outcome [43–44]. These studies show increased rates of fetal heart rate abnormalities, a higher risk of low birthweight percentile, and an increased risk of cesarean delivery when oligohydramnios is present. A meta-analysis of the relationship between amniotic fluid volume and perinatal outcome found an association between oligohydramnios and an increased incidence of cesarean delivery for non-reassuring fetal heart rate patterns and low Apgar score [45].

Prior to ultrasound, *polyhydramnios* or excessive amniotic fluid, was diagnosed when the fundal height exceeded expectations for the patient's gestational age or there was difficulty palpating fetal parts. Ultrasonography allowed for a more quantitative assessment of polyhydramnios; the definition is either a deepest vertical pocket measurement of greater than 8 cm or an AFI greater than 24 cm. Numerous conditions, both maternal and fetal, are associated with polyhydramnios, including maternal diabetes mellitus, fetal structural and chromosomal abnormalities, erythroblastosis fetalis, twins, and certain medications. In a study by Carlson et al., an AFI of  $>24$  cm predicted 49/50 cases of true polyhydramnios confirmed at delivery and included 92% of all anomalies and 100% of all trisomies, fetal and neonatal deaths [46]. A recent publication assessed the relationship of amniotic fluid volume and perinatal outcome [47]. They found a significant relationship between the identification of polyhydramnios and large-for-gestational age fetuses as well as fetuses at risk for congenital anomalies and cesarean delivery.

### Biophysical Profile

The fetal *biophysical profile* (BPP) is a tool used by obstetric practitioners to evaluate fetal well-being both antepartum and on labor and delivery. More recently, BPP assessment of fetal status has even been attempted intrapartum and is discussed later in this chapter. The ultimate goal in establishing an investigation to assess the fetal condition is to distinguish between the healthy versus the hypoxic fetus. Furthermore, avoidance of a low rate of false-negative test results so that asphyxiated fetuses are not missed and a low rate of false-positive results to avoid unnecessary anxiety and operative procedures is imperative. With a non-stress test (NST) or a contraction stress test (CST), both of which are associated with high false-positive rates, only the fetal heart rate is evaluated, forcing the clinician to estimate fetal health based on simply one parameter.

With the addition of ultrasound appraisal of fetal activity and amniotic fluid volume to the evaluation of the fetal heart rate, it is feasible for the obstetrician to gain more insight into fetal welfare. Manning et al. were the first to describe the use of multiple biophysical parameters of the fetus on ultrasound examination in an attempt to predict perinatal outcome [48]. The authors observed 216 patients with high-risk pregnancies who were delivered within 1 week of the ultrasound assessment. They studied five different variables: fetal breathing, fetal movement, fetal tone, qualitative amniotic fluid volume, and the non-stress test. They found that using the five parameters in combination showed the greatest accuracy for predicting five-minute Apgar score, fetal distress in labor, and perinatal mortality versus using any of the parameters alone. Furthermore, these investigators introduced the use of a scoring system in which each activity was scored either as 0 if absent or abnormal, or 2 if present or normal. They continued the examination until all parameters were deemed present or until 30 minutes had elapsed. This assessment of fetal health continues to be widely used today, exploiting the same five parameters, time limitation, and scoring system first described over two decades ago.

How is the BPP a useful predictor of perinatal morbidity and asphyxia? The BPP, unlike the NST or CST, combines evaluation of both acute and chronic markers for fetal well-being. Amniotic fluid volume is a signal of chronic fetal health not

typically altered by acute changes in fetal acid–base status. The presence of a low amniotic fluid volume, or oligohydramnios, is considered to be the result of chronic fetal stress most likely reflecting the presence of long-term fetal hypoxia, resulting in the shunting of oxygenated blood to the fetal heart, brain, and adrenal glands, reducing perfusion of the fetal kidneys. This renal hypoperfusion results in decreased fetal urine output and oligohydramnios.

The other four parameters of the BPP, fetal breathing, movement, tone, and heart rate, are more acute markers of unbalanced fetal acid–base status. Individually, these markers are regulated by different regions of the fetal central nervous system and as such mature and respond to fetal hypoxemia and acidemia at different stages in fetal development. The earliest of the parameters established is fetal tone, which can be observed 8 weeks following the last menstrual period. Fetal body movements accelerate over the following gestational week, followed by fetal breathing, which typically commences at approximately 21 weeks' gestation. The fetal heart rate reactivity is the final biophysical portion to mature, typically doing so by the end of the second trimester. Vintzileos and others observed that the order in which the parameters characteristically disappear in response to acute fetal acidemia is the reverse order of when they emerge throughout gestation, implying that the first factor to regress is classically fetal heart rate reactivity, followed by fetal breathing, movement, and tone [49–51].

Although there are numerous confounders to consider when interpreting the value of a BPP, such as gestational age of the fetus, diurnal variation, specific disease states, and maternal drug administration, the overall benefit to using the BPP in the assessment of fetal status has been established. In their original study in 1980, Manning et al. found that when the BPP score was 10 out of 10, the perinatal and fetal death rates were zero [48]. Conversely, when the score was 0 out of 10, the perinatal loss rate rose to 60% and fetal death rate was 40%. In a prospective, blinded study of more than 700 patients comparing fetal BPP with NST, the same authors found that BPP had a significantly higher positive predictive value than NST with regard to low Apgar scores [52]. Furthermore, those authors pointed out that because BPP uses ultrasound examination to evaluate the fetus, the added benefit of detecting fetal congenital anomalies is included.

Baskett et al. managed 4,184 high-risk pregnancies with BPP and found that a normal BPP (score of 8 or 10 out of 10) was associated with a perinatal mortality rate of 0.1%, an intermediate score (6 out of 10) was associated with a perinatal mortality rate of 3.1%, and abnormal scores (0–4 out of 10) were associated with a perinatal mortality rate of 20% [53]. They also found an overall low false-negative rate of 0.7/1,000.

Intrapartum BPP has recently been studied in an effort to assess its role as an instrument for evaluating fetal status during labor as well as a method of assessing the effect of oxytocics, regional anesthesia, and ruptured membranes on fetal behavior [54]. Kim et al. prospectively performed BPPs on 100 non-anomalous, singleton pregnancies and blinded the managing physicians to the results. They observed that the ascertainment of the BPP was not influenced by the use of oxytocics, prostaglandins, and the presence of meconium or epidural anesthesia. Additionally, they found that fetal breathing and gross fetal movement decreased with rupture of amniotic membranes. Furthermore, they established that a BPP score of 6 or less was associated with a relative risk for cesarean delivery of 8.0. They also found that cessation of any component of the BPP significantly increased the risk of cesarean delivery and admission to the neonatal intensive care unit. With further evaluation, BPP could prove to be a clinically useful adjunctive tool in the assessment of fetal well-being not only during the antepartum period but also intrapartum.

### DOPPLER FLOW STUDIES

Doppler ultrasonography uses the principle described by Christian Doppler in 1842, which elucidates the physical properties associated with the changes in sound frequency emitted or reflected from a moving source. Sonographically, this property can be manipulated to observe the velocity of blood flow in both maternal and fetal blood vessels and translated to a frequency shift of the reflected sound waves. Because this section concerns sonographic fetal assessment of labor and delivery, the discussion of Doppler ultrasound is limited to the examination of relevant fetal vessels.

Clinically, the two vessels most often used to predict perinatal outcome are the fetal umbilical artery and the middle cerebral artery, although numerous

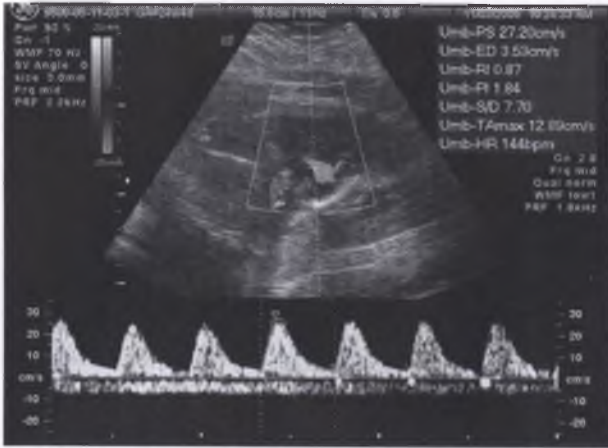


FIGURE 3.3.  
*Normal uterine artery Doppler.*

other vessels have been investigated for their role in evaluating the fetus in utero (see Figures 3.3 and 3.5). Doppler ultrasonography measurement of the umbilical arterial blood flow uses real-time Doppler velocimetry, providing information on perfusion of the fetoplacental unit. With advancing gestational age and trophoblastic invasion of the uterine decidual layer, the volume of flow in the umbilical arteries increases. Consequently, the high vascular impedance that can be detected early in the first and early second trimesters gradually declines until term. The relatively low vascular impedance in the placenta overall allows for continuous forward flow in the umbilical arteries throughout the cardiac cycle in most normal pregnancies.

How does measuring Doppler flow in the umbilical arteries help to assess fetal status? By using Doppler velocimetry, the obstetrician can measure and interpret vascular impedance in the umbilical arteries, thus determining the fetoplacental response to multiple obstetric and medical conditions that can adversely influence pregnancy. For example, with maternal hypertensive disorders, including preeclampsia, there is a substantial increase in the vascular resistance of the placenta, which leads to a decrease in the end-diastolic velocity of blood flow in the umbilical arteries that can be quantified by Doppler ultrasonography (see Figure 3.4). If this resistance continues to increase, the end-diastolic forward flow could eventually cease or even reverse and travel back toward the fetus, transporting deoxygenated blood away from the placenta. This change in flow pattern in the umbilical artery



FIGURE 3.4.  
*Abnormal uterine artery Doppler.*

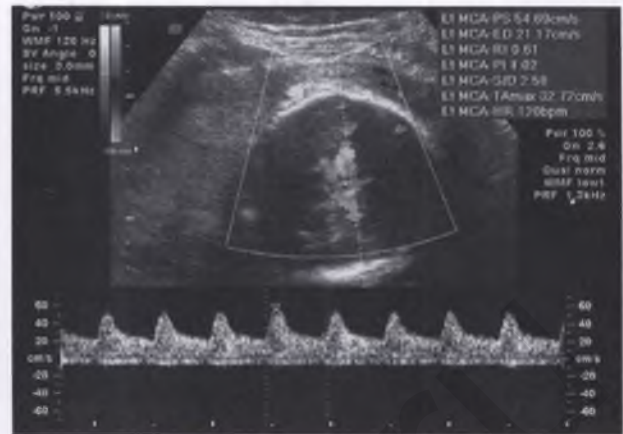
impairs transplacental oxygen transfer between the fetus and placenta and can lead to significant hypoxemia and growth restriction of the fetus.

Once the endpoint of reversed end-diastolic flow is obtained by Doppler interrogation of the umbilical artery, the perinatal mortality rate in a non-anomalous fetus is approximately 35% [55]. Meta-analysis of published randomized controlled studies indicates that antepartum umbilical artery Doppler assessment in high-risk patients reduces the perinatal mortality risk by more than 30% without increasing the rate of unnecessary obstetric interventions [56]. Conversely, there are studies that show no beneficial role of antepartum Doppler velocimetry as a screening test for low-risk pregnancies [57].

Intrapartum umbilical Doppler velocimetry assessment as a predictor of adverse perinatal outcome has been studied in a limited fashion. In 1999, Farrell et al. hypothesized that increased placental vascular resistance during late pregnancy would be expected to persist into the intrapartum period in both low- and high-risk patients [58]. They performed a meta-analysis to determine the clinical value of intrapartum umbilical artery Doppler analysis in identifying compromised infants at delivery. They determined that intrapartum umbilical artery Doppler velocimetry had minimal ability to predict low Apgar scores at 1 and 5 minutes, small for gestational age infants, fetal heart rate abnormalities during labor, umbilical arterial acidosis at delivery, or the resort to a cesarean for fetal distress. Unfortunately, the heterogeneity of the sample that these authors entered into the analysis, both



**FIGURE 3.5.**  
*Normal Doppler of the middle cerebral artery.*



**FIGURE 3.6.**  
*Abnormal Doppler of the middle cerebral artery.*

low- and high-risk patients, might have distorted the outcome. A more recent study correlated umbilical artery Doppler with fetal pulse oximetry and external cardiocography and observed alterations in the umbilical artery Doppler measurements in fetuses with labor-induced fetal hypoxia [59]. These authors thought that umbilical artery velocimetry indices correlated with perinatal outcome; their study, however, was limited by a small sample size (35 fetuses). With further study, umbilical artery Doppler velocimetry could prove to be a predictor of adverse perinatal outcome during the intrapartum period in certain high-risk pregnancies.

Blood flow through the umbilical arteries gives the practitioner an overview of the placenta and its ability to perfuse the fetus adequately. If one wishes to obtain information directly about the fetal response to decreased blood flow or oxygen content, however, the fetal middle cerebral artery can be evaluated (see Figure 3.5). The middle cerebral artery has a high sensitivity for the detection of fetal intrauterine growth restriction and related complications. In the normally developing fetus, the brain is an area of high vascular impedance with minimal forward flow at end diastole. Hypoxemia in the fetus causes a redistribution of fetal blood flow to the fetal brain by cerebral vasodilation at the expense of other fetal organs, such as kidneys, subcutaneous tissue, skeletal muscle, and liver. This response, evident by a decrease in cerebrovascular impedance, can be measured by Doppler flow studies (see Figure 3.6). The association between abnormal middle cerebral artery Doppler waveform and fetal hypoxemia has

been explored with the use of cordocentesis [60]. Rizzo et al. looked at growth-restricted fetuses and correlated Doppler findings with fetal blood parameters, concluding that hypoxemia and acid-base status in the fetus could be predicted by abnormalities in the flow pattern of the middle cerebral artery.

Furthermore, a Doppler cerebroplacental ratio, a ratio of the middle cerebral artery pulsatility index to the umbilical artery pulsatility index, has been developed and evaluated in its ability to identify the fetal redistribution of blood flow in the presence of hypoxemia. Bahado-Singh and coauthors found that Doppler identification of the fetal redistribution using the cerebroplacental ratio strongly predicted outcome in fetuses at risk for intrauterine growth restriction [61]. The clinical significance of fetal hypoxia in the middle cerebral artery as measured by Doppler during labor has also been studied. Siristatidis et al. found that middle cerebral artery Doppler showed significantly decreased vascular impedance in those fetuses with oxygen saturation values below 30% and abnormal cardiocographic patterns [62]. They concluded that middle cerebral artery Doppler investigation is an important predictor of adverse fetal outcome, especially fetal hypoxia, and could help the clinician to manage these patients intrapartum.

### **GESTATIONAL AGE/FETAL WEIGHT ASSESSMENT**

The management of a pregnant patient with little or no prenatal care who presents to labor and delivery

in the third trimester or in labor is frequently of concern. It is not only of the utmost consequence for the obstetrician to determine an accurate gestational age of the pregnancy to manage issues such as preterm labor, but it is imperative to the pediatricians who will assume care once the infant is born. Ultrasonography can facilitate obtaining an accurate gestational age and estimating fetal weight as well as diagnosing any gross abnormalities of the fetal anatomy. Studies have looked at less traditional ultrasound measurements and markers to help establish gestational age in the third trimester. The distal femoral and proximal tibial epiphyseal ossification centers in the fetus have been studied in the third trimester of pregnancy. It was determined that the distal femoral epiphysis was not identifiable before 28 weeks but was observed in 72% of fetuses at 33 weeks and in 94% of fetuses at 34 weeks [63]. The proximal tibial epiphysis was absent before 34 weeks and observed in 56% of fetuses at 36 weeks, 80% of fetuses at 37 weeks, and 100% of fetuses at 39 weeks gestation [63]. Other have looked at the proximal humeral ossification centers of the fetus and found that this center could be visualized after the 38th week with a specificity of 99%, sensitivity of 58%, and with a positive predictive value of 91% and negative predictive value of 93% [64].

Finally, an investigation was published that questioned the traditional dogma that third-trimester gestational age dating is relatively inaccurate with a 95% confidence interval of  $\pm 3$  weeks. Mongelli et al. derived third-trimester ultrasound dating algorithms from pregnancies conceived with artificial reproductive techniques [65]. They found that a formula using a combination of the measurements of the femur length and the head circumference had a 95% confidence interval of  $-13$  to  $+17$  days. Smulian et al. compared the accuracy of three different sonographic circumference measurement techniques to predict birthweight in term fetuses [66]. They found that measurement of the abdominal circumference within 24 hours of delivery showed a percent deviation from the actual birthweight of 1.9% (SD  $\pm 8.0\%$ ). This measurement was within 10% of actual birthweight in 80% of cases. These measurements along with identification of the fetal ossification centers can aid the clinician in making a relatively accurate assessment of gestational age in the third trimester.

## ULTRASONOGRAPHY OF THE PLACENTA

“Ultrasound is the most sensitive diagnostic tool for detecting abnormalities of the placenta, yet it may be unable to demonstrate the most clinically significant abnormalities (placenta accreta, abruption) even if one is highly skilled in placental sonography” [67].

### Placental Abruption

Placental abruption (abruptio placentae) is the premature separation of the normally implanted placenta. Most often a clinical suspicion and diagnosis, placental abruption can be catastrophic. The risk of preterm delivery is 20% to 40% with placental abruption [68]. Although it is one of the leading causes of perinatal mortality, accounting for 15% to 20% of perinatal deaths [69], the incidence of abruption is only 0.5% to 1% in the general population [70]. Abruption classically presents with the triad of vaginal bleeding, abdominal or pelvic pain, and uterine contractions and tenderness.

Ultrasound examination for placental abruption is helpful only if a retroplacental hematoma is seen, but the absence of this finding does not exclude abruption (see Figure 3.7). Historically, the sensitivity of ultrasonography for visualizing intrauterine hemorrhage has been reported as approximately 50% [67]. More recently, the sensitivity and specificity of sonography for identifying abruption have been reported as 24% and 96% respectively, and the



FIGURE 3.7.  
*Abruptio placentae.*

positive and negative predictive values were 88% and 53%, respectively [71].

Normally, there is a complex hypoechoic retroplacental collection that consists of uteroplacental vessels and myometrium that measures 1 cm to 2 cm in thickness. Increased thickening of this area can be caused by a uterine contraction, fibroid, or hematoma. Thickening secondary to contractions can be identified by the transient nature of its appearance or a hypervascular area of myometrium on color Doppler. Fibroids can appear more rounded, with a distinct border or capsule, and demonstrate greater vascularity than a hematoma but less than a contraction.

Sonographic appearance of hemorrhage varies depending on the age of the hematoma, location, and the transducer used. Acutely, hemorrhage appears hyperechoic at 0 to 48 hours, becoming isoechoic at 3 to 7 days, and then hypoechoic at 1 to 2 weeks [72]. The most common site of placental abruption is at the placental margin.

### PLACENTA PREVIA

The nomenclature of placenta previa describes its etiology: a placenta that is “previous” to or in front of the fetus relative to the birth canal. Placenta previa is the primary cause of third-trimester bleeding and is easily detectable on ultrasound examination, especially transvaginal or translabial ultrasound (see Figure 3.8). The only contraindication to transvagi-



FIGURE 3.8.  
*Placenta previa.*

nal ultrasound scan is bulging or arguably ruptured membranes.

The type of previa is defined by the actual distance between the placental edge and internal os. A *complete placenta previa* covers the entirety of the internal os. *Incomplete placenta previa*, a more inclusive term that includes both marginal and partial placenta previa, describes a placenta that impinges on some part of the internal os but does not completely cover it. *Low-lying placenta* denotes a placental edge that is within 2 cm of the internal os but does not cover a significant portion of it. Despite these definitions, the identification of the type of placenta previa is still somewhat subjective.

The incidence of abnormal placentation varies by gestational age. Placenta previa or low-lying placenta is usually physiologic and transient, with most placentas experiencing migration and resolution at term. In fact, the incidence of placenta previa in each trimester is approximately 42% at 11 to 14 weeks, 4% at 20 to 24 weeks, and 2% at term [73]. The clinical implications of any asymptomatic previa or low-lying placenta identified prior to 35 weeks should therefore be expectantly managed and followed for resolution. Conversely, given that abnormal placentation is the most common cause of second- and third-trimester bleeding, all patients presenting to the labor and delivery suite with this history should have a transvaginal or translabial ultrasound to identify placental location.

Transvaginal and translabial ultrasonography are superior to transabdominal ultrasonography in identifying and qualifying placenta previa. Transabdominal ultrasound examination will misdiagnose placenta previa in 25% of cases [74]. Transvaginal ultrasonography has a sensitivity and specificity of approximately 88% and 99%, respectively, and positive and negative predictive values of 93% and 98%, respectively [75]. The sensitivity and specificity of translabial ultrasonography is similar: 100% and 70% respectively when the distance between the placental edge and internal os is less than 2 cm, and 90% and 95% respectively when the distance is less than 1 cm [76].

### PLACENTA ACCRETA

Ultrasonography can be helpful in the detection and evaluation of abnormal placental adherence to the



uterus. Categorized by depth of invasion, *placenta accreta* denotes a placenta attached to but not yet invading the myometrium. *Placenta increta* occurs when the villi invade the myometrium. *Placenta percreta* is the penetration of the villi through the myometrium with possible attachment to adjacent structures, including the bladder or rectum. The overall prevalence is estimated to be 1 in 2,500 pregnancies. This risk increases in the presence of previa, when the prevalence is 10% and can be as high as 35% in women with a history of a previous cesarean delivery and subsequent pregnancy with previa.

The pathophysiology of placenta accreta is an absence or weakening of the decidua basalis and incomplete development of the fibrinoid layer. In addition to the site of the previous uterine scar from a cesarean delivery, any area of prior uterine surgery (i.e., myomectomy) is a risk for accreta if subsequent placental implantation occurs at that site.

There are three primary ultrasound findings that are used as markers for placenta accreta (see Figure 3.9). First, obliteration of the retroplacental clear space describes the loss of any portion of the echolucent area between the myometrium and placenta [77,78]. Second, presence of *lacunae*, defined as multiple, linear, irregular hypoechoic vascular spaces within the placenta giving it its euphemistic “Swiss-cheese” appearance [79]. Third, interruption of the posterior bladder wall and myometrial interface can distort the normal continuous echolucent line, making it appear as a series of dashes [80]. Comparing these findings at both 15 to 20 weeks and 15 to 40 weeks, the findings of placental lacunae have the highest sensitivity and positive predictive

value for placenta accreta. At 15 to 20 weeks, the presence of lacunae has a sensitivity of 79% and positive predictive value of 92%. At 15 to 20 weeks, the sensitivity and positive predictive value of placental lacunae increase to 93% each [80].

The use of color Doppler and magnetic resonance imaging (MRI) also have been proposed as adjuncts to aid in diagnosis. Although the use of color Doppler enhances the appearance of placental lacunae and perhaps fetoplacental blood vessels invading the myometrium, it has not been shown to increase the accuracy already exhibited by gray-scale ultrasonography [78]. Similarly, MRI has been shown to confirm ultrasound findings but has not been shown to be superior to gray-scale [81]. In fact, gray-scale ultrasonography has been shown to be superior to MRI in some studies [78].

Ultimately, the diagnosis of placenta accreta and its counterparts can be made only by pathologic examination in the laboratory after hysterectomy. Until then, ultrasound findings can raise suspicions, aid in identification and preliminary diagnosis, and thereby prepare physicians and patients for the possibility of the presence of accreta, so that appropriate surgical facilities are available.

## ASSESSMENT OF FETAL POSITION

### Fetal Presentation

*Presentation* denotes the fetal part that directly overlies the pelvic inlet. With a fetus in longitudinal lie, the presentation can be vertex, breech, or shoulder. Less common presentations include fundic and compound presentation. In a vertex fetus, the presentation is classified according to the leading bony landmark of the skull: occiput, mentum, or brow.

Leopold’s maneuvers and vaginal examination are the two most common means of identifying fetal presentation. Transabdominal ultrasonography is most often used for confirmation. Additionally, ultrasound scan can be used as the primary tool for assessing presentation in the patient with rupture of membranes not in labor, preterm or term, when vaginal examination could be potentially harmful.

### Position of the Fetal Occiput

Intrapartum assessment of the fetal occipital position is an essential part of managing labor. Correct



FIGURE 3.9.  
*Placenta accreta.*

determination influences induction, progress of labor, and mode of delivery. To date, most obstetricians rely on transvaginal digital examination to determine the position of the occiput. Numerous recent investigations comparing digital examination and transabdominal ultrasound scan, however, have shown that digital examination is accurate in only 24% to 61% of cases, depending on stage of labor and position [82–85].

Digital examination is least accurate in the first stage of labor. In active labor (with the cervix  $\geq 4$  cm dilated) with fetal head at ischial spine station  $-2$  or lower, 24% of assessments of position were correct when compared with transabdominal ultrasound examination (see Figures 3.10 and 3.11). This rate increased to 47% if digital examination assessments within 45 degrees of the ultrasound assessment were considered correct [83].



FIGURE 3.10.  
*Occiput anterior.*



FIGURE 3.11.  
*Occiput posterior.*

The accuracy of transvaginal digital examinations increases in the second stage of labor but remains suboptimal. The accuracy rate was 35% in 112 patients by digital examination versus transabdominal ultrasound scan. This rate increased to 61% when digital examination assessments within 45 degrees of the transabdominal ultrasound assessment were considered correct [84]. Transabdominal ultrasound and digital examination have also been compared with the actual occiput position at delivery after spontaneous resolution during a vaginal delivery or at the time of cesarean section. Vaginal examination was correct in 72% of cases, compared with 92% with ultrasound examination, when position assessments were within 45 degrees of the delivery position. (The 8% error rate for transabdominal ultrasonography occurred in fetuses that delivered in a variation of occiput anterior position and could be accounted for by unobserved spontaneous rotation occurring subsequent to the examination.) If the actual position was occiput posterior, the accuracy rates were only 31% of vaginal versus 100% of ultrasound examinations [85]. The improved accuracy of digital examination in the second stage of labor when compared with examination in the active phase of the first stage of labor is most likely due to the increased surface area of the fetal head and accompanying lower station that is palpable at 10 cm.

Management of the second stage of labor, particularly the safe and successful performance of an operative vaginal delivery, is contingent on correct assessment of fetal position. Incorrect placement of forceps or vacuum causes both fetal and maternal morbidity [86]. When transvaginal digital examination is compared with transabdominal ultrasonography prior to instrumental delivery, digital examination was correct in approximately 73% of cases [87]. Not surprisingly, accuracy varied according to position. For occiput anterior positions (see Figure 3.10), the accuracy was 83%. For occiput posterior (see Figure 3.11) or transverse, the accuracy was only 54%. In this study as in many others, a liberal definition of accurate was applied; the digital examination was considered correct if it was within 45 degrees of the transabdominal ultrasound assessment. Transabdominal ultrasound scan has also been shown to increase the accuracy of identifying engagement of the fetal head for both nulliparous and multiparous women [88]. Using

transabdominal ultrasonography to confirm fetal occiput position and station prior to instrumental delivery should be incorporated into the preprocedural evaluation of operative vaginal delivery.

While accumulated data clearly show the superiority of transabdominal ultrasonography over digital vaginal examination for the assessment of fetal occiput position prior to and during all stages of labor, transvaginal ultrasound scan provides even more information during the second stage of labor. Transvaginal ultrasound has been found to determine occiput position accurately in all cases of labor, whereas position could not be determined in 12% of digital exams ( $p \leq 0.03$ ) and 15% ( $p \leq 0.008$ ) of transabdominal ultrasound examinations. Transvaginal ultrasound examination also required the least time for performance, averaging less than 10 seconds per examination [89].

Determination of the fetal position by transabdominal ultrasonography could impact the ability to predict successful labor, either spontaneous or induced, by identifying fetuses in an occiput posterior position. Multiple studies have demonstrated the increased maternal and fetal morbidity of malposition in labor, including the increased risk for cesarean section. Occiput posterior position carries a statistically significant increased risk for prolonged first and second stage of labor, oxytocin augmentation, use of epidural analgesia, chorioamnionitis, assisted vaginal deliveries, third- and fourth-degree perineal lacerations, cesarean section, excessive blood loss, postpartum infection, and lower one-minute Apgar scores [90]. When combined with the parameters of cervical length and traditional maternal characteristics, ultrasonographically determined occiput position prior to induction can be predictive of the induction-to-delivery interval, and the likelihood of vaginal versus cesarean delivery [91]. Risk of cesarean delivery can be estimated in the early part of active labor (3 cm–5 cm) by sonographically determined occiput posterior position. In fact, fetuses that are occiput posterior at 3 cm to 5 cm of cervical dilatation have been found to have an odds ratio of 2.2 (CI 1.3–3.7,  $p = 0.004$ ) for cesarean section [92]. Although most occiput posterior positions at delivery are a consequence of persistence of this position and not malrotation, remember that most (53%–87%) of occiput posterior positions will rotate to occiput anterior during labor, even at 10 cm [93–95]. Future research on the

impact of alternative methods of induction or labor management for occiput posterior fetuses diagnosed by ultrasonography prior to labor could be useful in the prediction, diagnosis, and management of labor dystocia.

Most studies used the following landmarks to identify fetal occiput position: fetal orbits or cerebellum and posterior fossa for occiput-posterior position, midline cerebral echo for occiput-transverse positions, and cerebellum or occiput confirmed by tracing the spine for occiput-anterior positions. Additional views by a transperineal approach can be used to obtain landmarks when the vertex is below the level of the ischial spines. Assessment of the fetal occiput by transabdominal ultrasound examination is also easily reproducible. Interobserver agreement on sonographically determined fetal occipital position during labor (3 cm–10 cm) is within 15 degrees in 90% of cases and within 30 degrees in all cases [95].

Transvaginal sonographic examination is performed by inserting a sheath or glove-covered probe into the vagina until the resistance of the fetal head is felt. After applying the probe to the sagittal or coronal suture, a coronal or semicoronal section of the fetal brain is obtained. The most important landmark is a symmetric view of the midline and its structures, such as the pedunculi cerebri, or thalami and third ventricle. The exact position of the occiput is then calculated by determining the angle to which the transducer has to be turned clockwise to obtain the desired plane. The cerebellum and orbits can be used for confirmation [89].

### Twin Gestation

Because the presentation of a twin gestation prior to delivery often dictates the mode of delivery, all twin pregnancies must have presentation verified by ultrasound examination on admission to labor and delivery. Twin presentations can be classified as vertex/vertex, vertex/nonvertex, nonvertex/other, where the leading fetus' position (A) is described first. Cesarean deliveries are frequently performed when twin A is nonvertex.

If twin B is nonvertex, ultrasound examination is first used to evaluate eligibility for vaginal delivery; namely, ultrasound measurement for estimated fetal weight for both fetuses is performed. Vaginal delivery is relatively contraindicated when the

discordance between twins is greater than 500 g with twin B as the larger twin [96].

The ultrasound machine should be present in the delivery room of any twin pregnancy. After the delivery of twin A, ultrasound examination will identify the presentation and position of twin B immediately and accurately and also provides direct visual monitoring of twin B's heart rate as it settles into its possibly new presentation, thereby allowing accurate assessment of fetal well-being [97].

Depending on the presentation of twin B after delivery of twin A, ultrasound scan can aid in the management of twin B's delivery. External cephalic version (especially from a transverse or oblique lie) can be accomplished with ultrasound assistance by applying gentle pressure to the ultrasound transducer and using it to guide the fetal head physically toward the pelvic inlet and into the vertex presentation [98].

Internal podalic version can also be performed for a nonengaged vertex or transverse presentation under ultrasound guidance. With one hand held abdominally and one hand vaginally, the fetal head is displaced from the pelvic inlet. Ultrasound examination can then identify the fetal feet, which are grasped by the internal hand and guided caudally toward the lower birth canal. This eliminates the confusion of a blind grasp for fetal small parts, which could lead to grasping of one or both fetal hands. Amniotomy is then performed, and total breech extraction begun [97–98].

If a breech extraction is attempted for twin B, ultrasound examination can ensure that the fetal head is flexed [98]. The angle between the upward extension of the main axis of the thoracic vertebrae and a coronal slice through the skull parallel to its base is measured. If the angle is greater than 90 degrees, the head is extended [98]. Other potential complications of a twin delivery that benefit from ultrasound guidance are umbilical cord prolapse and premature placental separation prior to delivery of twin B.

## **ULTRASONOGRAPHY FOR PROCEDURE GUIDANCE**

### **Prenatal Diagnosis**

There are a variety of invasive procedures used to diagnose and treat different fetal genetic, infectious,

and hematologic pathologies. Several of these procedures are necessarily done by physicians on a labor and delivery unit, particularly if that procedure is being performed on a fetus at or beyond a viable gestational age (greater than 23 weeks). Performing these invasive procedures in the labor and delivery suite allows the physician to work in conjunction with the neonatologist, anesthesiologist, and the labor and delivery staff if expedited delivery is necessary. Achieving a positive outcome and reducing the procedure-related pregnancy loss rate for each of these procedures is the principal objective, and ultrasonography is of often an invaluable adjunct.

Prior to the performance of any invasive procedure during pregnancy, it is vital that the clinician obtain the greatest amount of information available about that gestation. Ultrasound examination allows the obstetrician to identify many characteristics, including gestational age, number of fetuses, gross anatomic abnormalities, abnormal amniotic fluid volume, fetal viability, and location of the placenta. Similarly, after delivery, ultrasonography is useful in the determination of retained pregnancy products within the uterine cavity and is invaluable to the surgeon performing a dilation and curettage for retained placental tissue.

Amniocentesis and chorionic villus sampling (CVS) are techniques in which a needle is inserted into the gestational sac to withdraw either a sample of amniotic fluid or a sample of placental tissue early in the pregnancy to determine genetic characteristics of the fetus, as well as later in the pregnancy to establish hematologic, infectious, and maturity characteristics. With amniocentesis, amniotic fluid from the uterine cavity is withdrawn using a needle inserted transabdominally. Although the most common indication for amniocentesis is for prenatal genetic studies, the assessment of fetal lung maturity, evaluation of the fetus for infection, degree of hemolytic anemia, blood or platelet type, and neural tube defects can be done using this procedure during pregnancy. Amniocentesis can also be executed as a therapeutic technique to remove excess amniotic fluid. CVS is a procedure in which a small sample of the placenta is obtained for genetic analysis. Whereas amniocentesis can safely be performed at any point in the gestation beyond 15 weeks, CVS is generally performed during the first trimester, between 10 and 13 weeks. In addition to a placental sample obtained transabdominally with a needle,

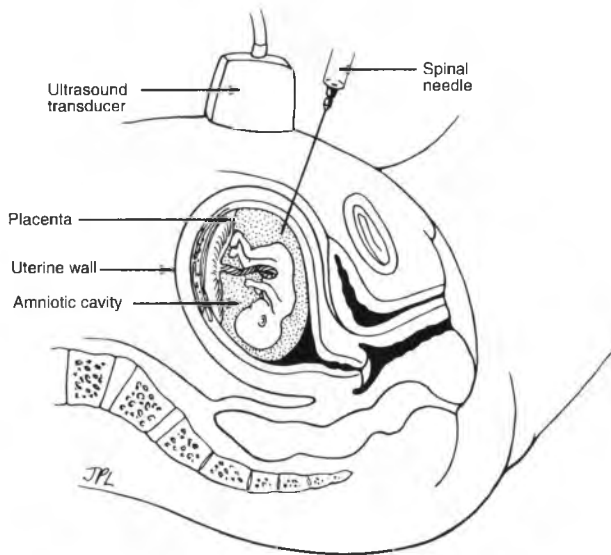


FIGURE 3.12.  
*Amniocentesis.*

CVS can be performed transcervically, using ultrasound scan to guide a specialized catheter into the placenta.

When an amniocentesis or a CVS is performed, continuous ultrasonographic visualization of the needle should be maintained throughout the procedure (see Figure 3.12). The ultrasound probe is typically covered with nonsterile gel and placed within a sterile probe cover, while sterile gel is placed on the outer surface of the cover in contact with the sterilized maternal skin. The optimal position for attainment of the sample is confirmed by ultrasound visualization prior to insertion of the needle. For abdominally approached CVS and amniocentesis, some practitioners prefer a free-hand technique because it allows adjustment in the path of needle insertion. Many ultrasound machines are also outfitted with a needle-guiding attachment that can be placed on the transducer to facilitate obtaining the optimal amount of fluid or tissue during difficult procedures. Those ultrasound machines are typically programmed to display an on-screen template of the needle tract that can be used to target the chosen sampling route and site. Although most proceduralists prefer a co-planar approach to guidance (i.e., aligning the long axis of the needle within the same plane as the ultrasound beam), a transverse (or cross-beam) alignment can sometimes offer more precision. Once the procedure is successfully completed, ultrasound examination should be used to assess and

document fetal viability and to rule out any gross tissue damage or hemorrhage.

The use of concurrent ultrasound guidance for amniocentesis rather than pre-procedure ultrasound evaluation has been studied and has not been shown to be associated with a reduced rate of fetal loss [99]. Ultrasonographic monitoring with continuous visualization of the needle throughout the procedure has become the standard of care in most regions of the United States, however, owing to the potential to reduce direct fetal injury, the number of punctures, and the incidence of bloody fluid. Furthermore, ultrasonography is important in identifying tenting of the membranes by the needle, fetal movement, or a uterine contraction during the procedure, allowing the operator to adjust the course of the needle to obtain a specimen. Finally, ultrasonography is a reliable means of ensuring that as little of the placenta is traversed as possible, the importance of which studies have suggested by demonstrating a greater risk of fetal complications with transplacental passage of the needle during invasive procedures [100–101].

### **PERCUTANEOUS UMBILICAL BLOOD SAMPLING**

Fetal blood sampling is an ultrasound-guided procedure that is classically performed in the labor and delivery unit, where rapid delivery of a viable fetus can occur if necessary. Fetal blood sampling is a practice used to gain access to the fetal blood for various indications; classically, obtaining a fetal blood sample can assist in the diagnosis of genetic disorders using a technique of rapid karyotyping, as well as to diagnose fetal infection and determine fetal blood type. Because amniocentesis and CVS are invasive techniques that have a lower procedure-related pregnancy loss rate, they are typically used for determination of fetal genetic disorders. Fetal blood sampling, however, is typically reserved for the diagnosis and treatment of fetal blood disorders such as anemia and thrombocytopenia. This procedure requires precise ultrasound visualization of the fetus; traditional ultrasound examination provides a two-dimensional image by which the clinician can identify the relative location of key components within the uterine cavity, including the fetus, placenta, and umbilical cord.

Fetal blood sampling is achieved by direct needle insertion into the fetal umbilical cord, also called *cordocentesis* or *percutaneous umbilical blood sampling* (PUBS), fetal heart, or fetal intrahepatic blood vessels. Before ultrasonography was used, fetal blood sampling was carried out by fetoscopic-guided puncture of the umbilical vessels, with a 5% procedure-related risk of pregnancy loss. The current approach of sampling fetal vessels under direct ultrasound guidance reduces the loss rate to approximately 1% per procedure [102]. The umbilical cord is the most frequently used site to obtain a fetal blood sample; the choice of whether to sample the umbilical artery or vein depends on the gestational age, presentation, and the indication for the procedure. The operator typically will identify and aim for a fixed segment of the umbilical cord 1 cm to 2 cm from the placental cord insertion, because the risk of maternal blood contamination is minimal and the cord is anchored there, offering the greatest stability for insertion of the needle, withdrawal of an adequate sample, and, if necessary, ease of transfusion of blood products. The Doppler color function of the ultrasound machine can be used to confirm the cord insertion site and flow of transfusion products through the fetal vessels.

Prenatal diagnosis using ultrasound-guided cordocentesis was studied by Daffos and coauthors, who performed more than 600 fetal blood sampling procedures from 17 to 38 weeks' gestation [102]. They established a procedure-related loss rate of 1.1% and a premature delivery rate of 5% for their cohort of patients. Similarly, Watts et al. published the outcomes of 77 fetal transfusions in 35 pregnancies managed with direct ultrasound guidance [103]. They reported no immediate transfusion-related deaths, and 5 transfusion-related complications, none of which required the immediate delivery of the fetus. The same group reported a 0% procedure-related mortality rate in nonhydropic fetuses.

### Three- and Four-dimensional Ultrasonography

Recently the techniques of three- and four-dimensional (3D, 4D) ultrasound examination have become an important addition to obstetric sonography, increasing its ability to identify fetal structures and guide invasive procedures. A two-dimensional ultrasound monitor display of three-dimensional

data is termed *3D ultrasonography*. Surface rendering of the fetus, placenta, or umbilical cord with 3D sonography can better demonstrate abnormalities that were previously undetectable with traditional two-dimensional sonography. The real-time imaging of three perpendicular planes of view simultaneously is termed *4D ultrasonography*. The theoretical benefit to using 3D or 4D visualization during invasive obstetric procedures is to increase the precision of needle placement when the target is relatively small. 2D ultrasound procedure guidance is prone to lateralization; this occurs when the width of the ultrasound beam is wider than the width of the needle tip, resulting in the needle image appearing to be within a tissue structure (e.g., umbilical cord) when it is actually adjacent to that structure.

In 2005, Dolkart et al. studied the feasibility of using 4D real-time, multiplanar ultrasonographic imaging to reduce lateralization during invasive procedures. They utilized 4D ultrasound examination in 99 patients undergoing amniocentesis, CVS, or cordocentesis procedures [104]. A historical control group of 99 patients whose procedure was carried out using 2D ultrasound were used for comparison. They found no difference in the number of needle insertions performed during amniocentesis, CVS, or cordocentesis in either the 2D or 4D groups; however, operator satisfaction with needle-tip visualization was improved in the 4D group. They concluded that it is indeed feasible and perhaps beneficial to use 4D ultrasonography for guiding these procedures more precisely. Similarly, Kim et al. published the results of 93 invasive procedures done under 4D ultrasound guidance and concluded that such imaging could significantly reduce the amount of time required to complete the procedure, thus reducing the associated pregnancy risks [105]. Although this could prove to become the standard of care, at this time, the role of 3D and 4D ultrasound technology for procedure guidance has not been optimally defined nor has the benefit been proved for widespread use.

### Retained Products/Dilation and Evacuation

A prolonged third stage of labor can be due to retained placental tissue, defined as a placenta that has not been fully expelled 30 minutes after delivery [106]. It occurs on labor and delivery units in 0.5% to 1% of all deliveries and is a common reason for

postpartum hemorrhage. Postpartum hemorrhage from retained uterine products occurs because the remaining tissue prohibits the uterus from contracting, thus inhibiting normal constriction of vascular beds that are subsequently left exposed and allowed to continue bleeding. The treatment of retained tissue requires removal of that tissue either manually or surgically to reduce the risk of severe bleeding and hypotensive shock that can occur with prolonged expectant management.

Ultrasound assessment of the uterus during the third stage of labor to verify the presence of retained placental tissue or membranes can assist the obstetrician in achieving the safest course of management while avoiding unnecessary and risky instrumentation of the postpartum uterus and minimizing bleeding. Transabdominal ultrasound examination immediately following delivery of the infant can demonstrate placental detachment, which allows the practitioner to comfortably pull on the umbilical cord without fear of uterine inversion or placental dismemberment. Separation of the placenta was studied with real-time ultrasonography during the third stage of labor in 100 patients [107]. In 97 of these patients, the authors found that separation of the placenta was multiphasic, beginning mostly in the lower pole of the placenta and then propagating upwards.

In addition to following the normal course of placental separation, several authors have used ultrasound examination to predict which patients might have difficulty with placental separation and to diagnose placental tissue retention. Krapp et al. used color Doppler to correlate the cessation of blood flow in placental basal plate vessels to the complete separation of the placenta from the myometrium [108]. They determined that continued blood flow in these vessels was associated with placenta adherence and the need for manual or instrumental removal. In a study of 39 women with suspected placental retention, Shen and coauthors performed ultrasound examination prior to exploration and found that sonography was an effective tool for identifying postpartum patients with retained placental fragments [109]. They found a sensitivity of 93.8% and specificity of 73.9% for ultrasound detection of this tissue. Determining the progression of placental separation and following it in real-time with ultrasound scan during the third stage of labor, as well as using Doppler techniques to monitor cessation

of blood flow to placental tissue, might allow the practitioner to predict which patients are destined for retained placental fragments. Furthermore, ultrasonography has proved to be helpful in the diagnosis of failed placental separation, allowing for expeditious surgical management prior to severe hemorrhage.

### Endoanal Ultrasound

Damage to the anal sphincter at the time of vaginal delivery predisposes women to fecal incontinence, especially when this damage goes undiagnosed and therefore is not repaired [110]. Disruption of the anal sphincter is clinically diagnosed in approximately 5% of all vaginal deliveries [111]. Endoanal ultrasound examinations in women without clinically recognized anal sphincter disruption after delivery have shown the prevalence to be as high as 44%, however [110]. *Anal sphincter rupture* is defined as a gap in the hyperechogenic ring of the internal or external anal sphincter [112]. Anal incontinence is subsequently reported in up to 50% of women with clinically unrecognized sphincter damage [110].

Recent studies have shown that performing routine endoanal ultrasound examination in women with second-degree perineal tears identifies clinically occult sphincter damage, allowing immediate surgical intervention. This intervention significantly decreases severe fecal incontinence from approximately 9% at 3 months and 7% at one year in women randomized to the control group, versus 3% at 3 months and one year ( $p = 0.002$ ,  $p = 0.03$  respectively) in women randomized to endoanal ultrasound and surgical repair when a defect was found [113]. Ultrasound examination of the perineum after childbirth improves the diagnosis of anal sphincter tears, and their immediate repair decreases the risk of severe fecal incontinence [113]. Endoanal ultrasonography needs to be performed in 29 women to prevent 1 case of severe fecal incontinence [113]. Adding routine endoanal ultrasound examination to the standard clinical examination after delivery has the potential to decrease occult sphincter damage and therefore fecal incontinence.

The aim of this chapter is to demonstrate the importance of ultrasonography in the proper assessment and management of the gravida in the labor and delivery suite. The proper use of this valuable

tool requires the same level of expertise, documentation, and state-of-the-art equipment (including transvaginal, pulsed and color Doppler, and 3D ultrasound capabilities) as is expected in the prenatal clinic. Use of these techniques in the labor and delivery suite will certainly lead to better management of the mother and fetus, reducing complications and leading to a healthier outcome for both.

## REFERENCES

- Iams JD, Goldenberg RL, Meis PJ, Mercer BM, Moawad A, Das A, et al. The length of the cervix and the risk of spontaneous premature delivery: National Institute of Child Health and Human Development Maternal Fetal Medicine Unit Network. *N Engl J Med*. 1996 Feb 29;334(9):567-72.
- Phelps JY, Higby K, Smyth MH, Ward JA, Arredondo F, Mayer AR. Accuracy and intraobserver variability of simulated cervical dilatation measurements. *Am J Obstet Gynecol*. 1995 Sep;173(3 Pt 1):942-5.
- Yang SH, Roh CR, Kim JH. Transvaginal ultrasonography for cervical assessment before induction of labor. *J Ultrasound Med*. 2004 Mar;23(3):375-82.
- Gabriel R, Darnaud T, Chalot F, Gonzalez N, Leymarie F, Quereux C. Transvaginal sonography of the uterine cervix prior to labor induction. *Ultrasound Obstet Gynecol*. 2002 Mar;19(3):254-7.
- Ware V, Raynor BD. Transvaginal ultrasonographic cervical measurement as a predictor of successful labor induction. *Am J Obstet Gynecol*. 2000 May;182(5):1030-2.
- Brown JE, Thieme GA, Shah DM, Fleischer AC, Boehm FH. Transabdominal and transvaginal endosonography: Evaluation of the cervix and lower uterine segment in pregnancy. *Am J Obstet Gynecol*. 1986 Oct;155(4):721-6.
- Leibman AJ, Kruse B, McSweeney MB. Transvaginal sonography: Comparison with transabdominal sonography in the diagnosis of pelvic masses. *AJR Am J Roentgenol*. 1988 Jul;151(1):89-92.
- Andersen HF. Transvaginal and transabdominal ultrasonography of the uterine cervix during pregnancy. *J Clin Ultrasound*. 1991 Feb;19(2):77-83.
- Timor-Tritsch IE, Yunis RA. Confirming the safety of transvaginal sonography in patients suspected of placenta previa. *Obstet Gynecol*. 1993 May;81(5 (Pt 1)):742-4.
- Carlan SJ, Richmond LB, O'Brien WF. Randomized trial of endovaginal ultrasound in preterm premature rupture of membranes. *Obstet Gynecol*. 1997 Mar;89(3):458-61.
- Burger M, Weber-Rossler T, Willmann M. Measurement of the pregnant cervix by transvaginal sonography: An interobserver study and new standards to improve the interobserver variability. *Ultrasound Obstet Gynecol*. 1997 Mar;9(3):188-93.
- Kurtzman JT, Goldsmith LJ, Gall SA, Spinnato JA. Transvaginal versus transperineal ultrasonography: A blinded comparison in the assessment of cervical length at midgestation. *Am J Obstet Gynecol*. 1998 Oct;179(4):852-7.
- Mahony BS, Nyberg DA, Luthy DA, Hirsch JH, Hickok DE, Petty CN. Translabial ultrasound of the third-trimester uterine cervix: Correlation with digital examination. *J Ultrasound Med*. 1990 Dec;9(12):717-23.
- Kushnir O, Vigil DA, Izquierdo L, Schiff M, Curet LB. Vaginal ultrasonographic assessment of cervical length changes during normal pregnancy. *Am J Obstet Gynecol*. 1990 Apr;162(4):991-3.
- Heath VC, Southall TR, Souka AP, Novakov A, Nicolaides KH. Cervical length at 23 weeks of gestation: Relation to demographic characteristics and previous obstetric history. *Ultrasound Obstet Gynecol*. 1998 Nov;12(5):304-11.
- Parulekar SG, Kiwi R. Dynamic incompetent cervix uteri. Sonographic observations. *J Ultrasound Med*. 1988 Sep;7(9):481-5.
- Gomez R, Galasso M, Romero R, Mazor M, Sorokin Y, Goncalves L, et al. Ultrasonographic examination of the uterine cervix is better than cervical digital examination as a predictor of the likelihood of premature delivery in patients with preterm labor and intact membranes. *Am J Obstet Gynecol*. 1994 Oct;171(4):956-64.
- Ziliani M, Azuaga A, Calderon F, Pages G, Mendoza G. Monitoring the effacement of the uterine cervix by transperineal sonography: A new perspective. *J Ultrasound Med*. 1995 Oct;14(10):719-24.
- Berghella V, Kuhlman K, Weiner S, Texeira L, Wapner RJ. Cervical funneling: Sonographic criteria predictive of preterm delivery. *Ultrasound Obstet Gynecol*. 1997 Sep;10(3):161-6.
- Iams JD, Johnson FF, Sonek J, Sachs L, Gebauer C, Samuels P. Cervical competence as a continuum: A study of ultrasonographic cervical length and



- obstetric performance. *Am J Obstet Gynecol.* 1995 Apr;172(4 Pt 1):1097-103.
21. Owen J, Yost N, Berghella V, MacPherson C, Swain M, Dildy GA 3rd, Miodovnik M, Langer O, Sibai B. Maternal-Fetal Medicine Units Network: Can shortened midtrimester cervical length predict very early spontaneous preterm birth? *Am J Obstet Gynecol.* 2004 Jul;191(1):298-303.
  22. Iams JD, Goldenberg RL, Mercer BM, Moawad AH, Meis PJ, Das AF, et al. The preterm prediction study: Can low-risk women destined for spontaneous preterm birth be identified? *Am J Obstet Gynecol.* 2001 Mar;184(4):652-5.
  23. Iams JD. Prediction and early detection of preterm labor. *Obstet Gynecol.* 2003 Feb;101(2):402-12.
  24. Tsoi E, Akmal S, Rane S, Otigbah C, Nicolaides KH. Ultrasound assessment of cervical length in threatened preterm labor. *Ultrasound Obstet Gynecol.* 2003 Jun;21(6):552-5.
  25. Tsoi E, Fuchs IB, Rane S, Geerts L, Nicolaides KH. Sonographic measurement of cervical length in threatened preterm labor in singleton pregnancies with intact membranes. *Ultrasound Obstet Gynecol.* 2005 Apr;25(4):353-6.
  26. Fuchs IB, Henrich W, Osthues K, Dudenhausen JW. Sonographic cervical length in singleton pregnancies with intact membranes presenting with threatened preterm labor. *Ultrasound Obstet Gynecol.* 2004 Oct;24(5):554-7.
  27. Rane SM, Guirgis RR, Higgins B, Nicolaides KH. The value of ultrasound in the prediction of successful induction of labor. *Ultrasound Obstet Gynecol.* 2004 Oct;24(5):538-49.
  28. Bartha JL, Romero-Carmona R, Martinez-Del-Fresno P, Comino-Delgado R. Bishop score and transvaginal ultrasound for preinduction cervical assessment: A randomized clinical trial. *Ultrasound Obstet Gynecol.* 2005 Feb;25(2):155-9.
  29. Roman H, Verspyck E, Vercoustre L, Degre S, Col JY, Firmin JM, et al. The role of ultrasound and fetal fibronectin in predicting the length of induced labor when the cervix is unfavorable. *Ultrasound Obstet Gynecol.* 2004 Jun;23(6):567-73.
  30. Reis FM, Gervasi MT, Florio P, Bracalente G, Fadalti M, Severi FM, et al. Prediction of successful induction of labor at term: Role of clinical history, digital examination, ultrasound assessment of the cervix, and fetal fibronectin assay. *Am J Obstet Gynecol.* 2003 Nov;189(5):1361-7.
  31. Chandra S, Crane JM, Hutchens D, Young DC. Transvaginal ultrasound and digital examination in predicting successful labor induction. *Obstet Gynecol.* 2001 Jul;98(1):2-6.
  32. Rozenberg P, Chevret S, Chastang C, Ville Y. Comparison of digital and ultrasonographic examination of the cervix in predicting time interval from induction to delivery in women with a low Bishop score. *BJOG.* 2005 Feb;112(2):192-6.
  33. Weissman A, Itskovitz-Eldor J, Jakobi P. Sonographic measurement of amniotic fluid volume in the first trimester of pregnancy. *J Ultrasound Med.* 1996 Nov;15(11):771-4.
  34. Magann EF, Doherty DA, Chauhan SP, Barrilleaux SP, Verity LA, Martin JN Jr. Effect of maternal hydration on amniotic fluid volume. *Obstet Gynecol.* 2003 Jun;101(6):1261-5.
  35. Manning FA, Hill LM, Platt LD. Qualitative amniotic fluid volume determination by ultrasound: Antepartum detection of intrauterine growth retardation. *Am J Obstet Gynecol.* 1981 Feb 1;139(3):254-8.
  36. Phelan JP, Ahn MO, Smith CV, Rutherford SE, Anderson E. Amniotic fluid index measurements during pregnancy. *J Reprod Med.* 1987 Aug;32(8):601-4.
  37. Moore TR, Cayle JE. The amniotic fluid index in normal human pregnancy. *Am J Obstet Gynecol.* 1990 May;162(5):1168-73.
  38. Miyamura T, Masuzaki H, Miyamoto M, Ishimaru T. Comparison between the single deepest pocket and amniotic fluid index in predicting fetal distress in small-for-gestational age fetuses. *Acta Obstet Gynecol Scand.* 1997 Feb;76(2):123-7.
  39. Chauhan SP, Doherty DD, Magann EF, Cahanding F, Moreno F, Klausen JH. Amniotic fluid index vs. single deepest pocket technique during modified biophysical profile: A randomized clinical trial. *Am J Obstet Gynecol.* 2004 Aug;191(2):661-7.
  40. Chauhan SP, Magann EF, Morrison JC, Whitworth NS, Hendrix NW, Devoe LD. Ultrasonographic assessment of amniotic fluid does not reflect actual amniotic fluid volume. *Am J Obstet Gynecol.* 1997 Aug;177(2):291-6.
  41. Brace RA. Physiology of amniotic fluid volume regulation. *Clin Obstet Gynecol.* 1997 Jun;40(2):280-9.
  42. Manning FA. Dynamic ultrasound-based fetal assessment: The fetal biophysical profile score. *Clin Obstet Gynecol.* 1995 Mar;38(1):26-44.

43. Voxman EG, Tran S, Wing DA. Low amniotic fluid index as a predictor of adverse perinatal outcome. *J Perinatol.* 2002 Jun;22(4):282–5.
44. Locatelli A, Vergani P, Toso L, Verderio M, Pezzullo JC, Ghidini A. Perinatal outcome associated with oligohydramnios in uncomplicated term pregnancies. *Arch Gynecol Obstet.* 2004 Jan;269(2):130–3.
45. Chauhan SP, Sanderson M, Hendrix NW, Magann EF, Devoe LD. Perinatal outcome and amniotic fluid index in the antepartum and intrapartum periods: A meta-analysis. *Am J Obstet Gynecol.* 1999 Dec;181(6):1473–8.
46. Carlson DE, Platt LD, Medearis AL, Horenstein J. Quantifiable polyhydramnios: diagnosis and management. *Obstet Gynecol.* 1990 Jun;75(6):989–93.
47. Ott WJ. Reevaluation of the relationship between amniotic fluid volume and perinatal outcome. *Am J Obstet Gynecol.* 2005 Jun;192(6):1803–9.
48. Manning FA, Platt LD, Sipos L. Antepartum fetal evaluation: development of a fetal biophysical profile. *Am J Obstet Gynecol.* 1980 Mar 15;136(6):787–95.
49. Vintzileos AM, Gaffney SE, Salinger LM, Campbell WA, Nochimson DJ. The relationship between fetal biophysical profile and cord pH in patients undergoing cesarean section before the onset of labor. *Obstet Gynecol.* 1987 Aug;70(2):196–201.
50. Vintzileos AM, Fleming AD, Scorza WE, Wolf EJ, Balducci J, Campbell WA, et al. Relationship between fetal biophysical activities and umbilical cord blood gas values. *Am J Obstet Gynecol.* 1991 Sep;165(3):707–13.
51. Ribbert LS, Snijders RJ, Nicolaides KH, Visser GH. Relationship of fetal biophysical profile and blood gas values at cordocentesis in severely growth-retarded fetuses. *Am J Obstet Gynecol.* 1990 Aug;163(2):569–71.
52. Manning FA, Lange IR, Morrison I, Harman CR. Fetal biophysical profile score and the nonstress test: A comparative trial. *Obstet Gynecol.* 1984 Sep;64(3):326–31.
53. Baskett TF, Allen AC, Gray JH, Young DC, Young LM. Fetal biophysical profile and perinatal death. *Obstet Gynecol.* 1987 Sep;70(3 Pt 1):357–60.
54. Kim SY, Khandelwal M, Gaughan JP, Agar MH, Reece EA. Is the intrapartum biophysical profile useful? *Obstet Gynecol.* 2003 Sep;102(3):471–6.
55. Kurkinen-Raty M, Kivela A, Jouppila P. The clinical significance of an absent end-diastolic velocity in the umbilical artery detected before the 34th week of pregnancy. *Acta Obstet Gynecol Scand.* 1997 May;76(5):398–404.
56. Alfirevic Z, Neilson JP. Doppler ultrasonography in high-risk pregnancies: Systematic review with meta-analysis. *Am J Obstet Gynecol.* 1995 May;172(5):1379–87.
57. Mason GC, Lilford RJ, Porter J, Nelson E, Tyrell S. Randomised comparison of routine versus highly selective use of Doppler ultrasound in low-risk pregnancies. *Br J Obstet Gynaecol.* 1993 Feb;100(2):130–3.
58. Farrell T, Chien PF, Gordon A. Intrapartum umbilical artery Doppler velocimetry as a predictor of adverse perinatal outcome: A systematic review. *Br J Obstet Gynaecol.* 1999 Aug;106(8):783–92.
59. Siristatidis C, Salamalekis E, Kassanos D, Creatas G. Alterations in Doppler velocimetry indices of the umbilical artery during fetal hypoxia in labor, in relation to cardiotocography and fetal pulse oximetry findings. *Arch Gynecol Obstet.* 2005 Sep;272(3):191–6.
60. Rizzo G, Capponi A, Arduini D, Romanini C. The value of fetal arterial, cardiac and venous flows in predicting pH and blood gases measured in umbilical blood at cordocentesis in growth retarded fetuses. *Br J Obstet Gynaecol.* 1995 Dec;102(12):963–9.
61. Bahado-Singh RO, Kovanci E, Jeffres A, Oz U, Deren O, Copel J, Mari G. The Doppler cerebroplacental ratio and perinatal outcome in intrauterine growth restriction. *Am J Obstet Gynecol.* 1999 Mar;180(3 Pt 1):750–6.
62. Siristatidis C, Salamalekis E, Kassanos D, Loghis C, Creatas G. Evaluation of fetal intrapartum hypoxia by middle cerebral and umbilical artery Doppler velocimetry with simultaneous cardiotocography and pulse oximetry. *Arch Gynecol Obstet.* 2004 Dec;270(4):265–70.
63. Goldstein I, Lockwood C, Belanger K, Hobbins J. Ultrasonographic assessment of gestational age with the distal femoral and proximal tibial ossification centers in the third trimester. *Am J Obstet Gynecol.* 1988 Jan;158(1):127–30.
64. Nazario AC, Tanaka CI, Novo NF. Proximal humeral ossification center of the fetus: Time of appearance and the sensitivity and specificity of

- this finding. *J Ultrasound Med.* 1993 Sep;12(9):513–5.
65. Mongelli M, Chew S, Yuxin NG, Biswas A. Third-trimester ultrasound dating algorithms derived from pregnancies conceived with artificial reproductive techniques. *Ultrasound Obstet Gynecol.* 2005 Aug;26(2):129–31.
  66. Smulian JC, Ranzini AC, Ananth CV, Rosenberg JC, Vintzileos AM. Comparison of three sonographic circumference measurement techniques to predict birth weight. *Obstet Gynecol.* 1999 May;93(5 Pt 1):692–6.
  67. Callen PW (ed.): *Ultrasonography in Obstetrics and Gynecology.* Philadelphia, W. B. Saunders Company; 4th edition: 20, p 597.
  68. Ananth CV, Berkowitz GS, Savitz DA, Lapinski RH. Placental abruption and adverse perinatal outcomes. *JAMA.* 1999 Nov 3;282(17):1646–51.
  69. Knab DR. Abruptio placentae. An assessment of the time and method of delivery. *Obstet Gynecol.* 1978 Nov;52(5):625–9.
  70. Abu-Heija A, al-Chalabi H, el-Iloubani N.: Abruptio placentae: Risk factors and perinatal outcome. *J Obstet Gynaecol Res.* 1998 Apr;24(2):141–4.
  71. Glantz C, Purnell L. Clinical utility of sonography in the diagnosis and treatment of placental abruption. *J Ultrasound Med.* 2002 Aug;21(8):837–40.
  72. Nyberg DA, Cyr DR, Mack LA, Wilson DA, Shuman WP. Sonographic spectrum of placental abruption. *AJR Am J Roentgenol.* 1987 Jan;148(1):161–4.
  73. Mustafa SA, Brizot ML, Carvalho MH, Watanabe L, Kahhale S, Zugaib M. Transvaginal ultrasonography in predicting placenta previa at delivery: A longitudinal study. *Ultrasound Obstet Gynecol.* 2002 Oct;20(4):356–9.
  74. Smith RS, Lauria MR, Comstock CH, Treadwell MC, Kirk JS, Lee W, Bottoms SF. Transvaginal ultrasonography for all placentas that appear to be low-lying or over the internal cervical os. *Ultrasound Obstet Gynecol.* 1997 Jan;9(1):22–4.
  75. Leerentveld RA, Gilberts EC, Arnold MJ, Wladimiroff JW. Accuracy and safety of transvaginal sonographic placental localization. *Obstet Gynecol.* 1990 Nov;76(5 Pt 1):759–62.
  76. Dawson WB, Dumas MD, Romano WM, Gagnon R, Gratton RJ, Mowbray RD. Translabial ultrasonography and placenta previa: Does measurement of the os–placenta distance predict outcome? *J Ultrasound Med.* 1996 Jun;15(6):441–6.
  77. Cox SM, Carpenter RJ, Cotton DB. Placenta percreta: Ultrasound diagnosis and conservative surgical management. *Obstet Gynecol.* 1988 Mar;71(3 Pt 2):454–6.
  78. Levine D, Hulka CA, Ludmir J, Li W, Edelman RR. Placenta accreta: Evaluation with color Doppler US, power Doppler US, and MR imaging. *Radiology.* 1997 Dec;205(3):773–6.
  79. Guy GP, Peisner DB, Timor-Tritsch IE. Ultrasonographic evaluation of uteroplacental blood flow patterns of abnormally located and adherent placentas. *Am J Obstet Gynecol.* 1990 Sep;163(3):723–7.
  80. Comstock CH, Love JJ Jr, Bronsteen RA, Lee W, Vetraino IM, Huang RR, Lorenz RP. Sonographic detection of placenta accreta in the second and third trimesters of pregnancy. *Am J Obstet Gynecol.* 2004 Apr;190(4):1135–40.
  81. Kirkinen P, Helin-Martikainen HL, Vanninen R, Partanen K. Placenta accreta: Imaging by gray-scale and contrast-enhanced color Doppler sonography and magnetic resonance imaging. *J Clin Ultrasound.* 1998 Feb;26(2):90–4.
  82. Rayburn WF, Siemers KH, Legino LJ, Nabity MR, Anderson JC, Patil KD. Dystocia in late labor: Determining fetal position by clinical and ultrasonic techniques. *Am J Perinatol.* 1989 Jul;6(3):316–9.
  83. Sherer DM, Miodovnik M, Bradley KS, Langer O. Intrapartum fetal head position I: Comparison between transvaginal digital examination and transabdominal ultrasound assessment during the active stage of labor. *Ultrasound Obstet Gynecol.* 2002 Mar;19(3):258–63.
  84. Sherer DM, Miodovnik M, Bradley KS, Langer O. Intrapartum fetal head position II: comparison between transvaginal digital examination and transabdominal ultrasound assessment during the second stage of labor. *Ultrasound Obstet Gynecol.* 2002 Mar;19(3):264–8.
  85. Chou MR, Kreiser D, Taslimi MM, Druzin ML, El-Sayed YY. Vaginal versus ultrasound examination of fetal occiput position during the second stage of labor. *Am J Obstet Gynecol.* 2004 Aug;191(2):521–4.
  86. Cunningham FG, Gant N, Leveno KJ, Gilstrap LC, Hauth JC, Wenstrom KD. *Forceps Delivery and Vacuum Extraction.* Williams Obstetrics, 21st Edition. New York, McGraw-Hill, 2001, 485–508.
  87. Akmal S, Kametas N, Tsoi E, Hargreaves C, Nicolaides KH. Comparison of transvaginal digital examination with intrapartum sonography to determine

- fetal head position before instrumental delivery. *Ultrasound Obstet Gynecol.* 2003 May;21(5):437–40.
88. Sherer DM, Abulafia O. Intrapartum assessment of fetal head engagement: Comparison between transvaginal digital and transabdominal ultrasound determinations. *Ultrasound Obstet Gynecol.* 2003 May;21(5):430–6.
  89. Zahalka N, Sadan O, Malinger G, Liberati M, Boaz M, Glezerman M, Rotmensch S. Comparison of transvaginal sonography with digital examination and transabdominal sonography for the determination of fetal head position in the second stage of labor. *Am J Obstet Gynecol.* 2005 Aug;193(2):381–6.
  90. Ponkey SE, Cohen AP, Heffner LJ, Lieberman E. Persistent fetal occiput posterior position: Obstetric outcomes. *Obstet Gynecol.* 2003 May;101(5 Pt 1):915–20.
  91. Rane SM, Guirgis RR, Higgins B, Nicolaidis KH. The value of ultrasound in the prediction of successful induction of labor. *Ultrasound Obstet Gynecol.* 2004 Oct;24(5):538–49.
  92. Akmal S, Kametas N, Tsoi E, Howard R, Nicolaidis KH. Ultrasonographic occiput position in early labour in the prediction of caesarean section. *BJOG.* 2004 Jun;111(6):532–6.
  93. Akmal S, Tsoi E, Howard R, Osei E, Nicolaidis KH. Investigation of occiput posterior delivery by intrapartum sonography. *Ultrasound Obstet Gynecol.* 2004 Sep;24(4):425–8.
  94. Gardberg M, Laakkonen E, Salevaara M. Intrapartum sonography and persistent occiput posterior position: A study of 408 deliveries. *Obstet Gynecol.* 1998 May;91(5 Pt 1):746–9.
  95. Akmal S, Tsoi E, Nicolaidis KH. Intrapartum sonography to determine fetal occipital position: Interobserver agreement. *Ultrasound Obstet Gynecol.* 2004 Sep;24(4):421–4.
  96. Ramsey PS, Repke JT. Intrapartum management of multifetal pregnancies. *Semin Perinatol.* 2003 Feb;27(1):54–72.
  97. Warenski JC, Kochenour NK. Intrapartum management of twin gestation. *Clin Perinatol.* 1989 Dec;16(4):889–97.
  98. Berkowitz RL, Hobbins JC. Delivering twins with the help of ultrasound. *Contemporary Ob-Gyn.* 1982 February; 19: 128–131.
  99. Seeds JW. Diagnostic mid-trimester amniocentesis: How safe? *Am J Obstet Gynecol.* 2004 Aug;191(2):607–15.
  100. Tabor A, Philip J, Madsen M, Bang J, Obel EB, Norgaard-Pedersen B. Randomised controlled trial of genetic amniocentesis in 4606 low-risk women. *Lancet.* 1986 Jun 7;1(8493):1287–93.
  101. Kappel B, Nielsen J, Brogaard Hansen K, Mikkelsen M, Therkelsen AJ. Spontaneous abortion following mid-trimester amniocentesis: Clinical significance of placental perforation and blood-stained amniotic fluid. *Br J Obstet Gynaecol.* 1987 Jan;94(1):50–4.
  102. Daffos F, Capella-Pavlovsky M, Forestier F. Fetal blood sampling during pregnancy with use of a needle guided by ultrasound: A study of 606 consecutive cases. *Am J Obstet Gynecol.* 1985 Nov 15;153(6):655–60.
  103. Watts DH, Luthy DA, Benedetti TJ, Cyr DR, Easterling TR, Hickok D. Intraoperative fetal transfusion under direct ultrasound guidance. *Obstet Gynecol.* 1988 Jan;71(1):84–8.
  104. Dolkart L, Harter M, Snyder M. Four-dimensional ultrasonographic guidance for invasive obstetric procedures. *J Ultrasound Med.* 2005 Sep;24(9):1261–6.
  105. Kim SR, Won HS, Lee PR, Kim A. Four-dimensional ultrasound guidance of prenatal invasive procedures. *Ultrasound Obstet Gynecol.* 2005 Nov;26(6):663–5.
  106. Combs CA, Laros RK. Prolonged third stage of labor: Morbidity and risk factors. *Obstet Gynecol.* 1991 Jun;77(6):863–7.
  107. Herman A, Zimerman A, Arieli S, Tovbin Y, Bezer M, Bukovsky I, Panski M. Down-up sequential separation of the placenta. *Ultrasound Obstet Gynecol.* 2002 Mar;19(3):278–81.
  108. Krapp M, Baschat AA, Hankeln M, Gembruch U. Gray-scale and color Doppler sonography in the third stage of labor for early detection of failed placental separation. *Ultrasound Obstet Gynecol.* 2000 Feb;15(2):138–42.
  109. Shen O, Rabinowitz R, Eisenberg VH, Samueloff A. Transabdominal sonography before uterine exploration as a predictor of retained placental fragments. *J Ultrasound Med.* 2003 Jun;22(6):561–4.
  110. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. *N Engl J Med.* 1993 Dec 23;329(26):1905–11.
  111. MacArthur C, Bick DE, Keighley MR. Faecal incontinence after childbirth. *Br J Obstet Gynaecol.* 1997 Jan;104(1):46–50.

112. Sultan AH, Nicholls RJ, Kamm MA, Hudson CN, Beynon J, Bartram CI. Anal endosonography and correlation with in vitro and in vivo anatomy. 1993 Br J Surg (79):104–106.
113. Faltin DL, Boulvain M, Floris LA, Irion O. Diagnosis of anal sphincter tears to prevent fecal incontinence: A randomized controlled trial. Obstet Gynecol. 2005 Jul;106(1):6–13.

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## Chapter 4 ECTOPIC PREGNANCY

Samantha F. Butts

David B. Seifer

*... one should regard sudden collapse associated with symptoms of abdominal hemorrhage in a woman during the childbearing period as prima facie evidence of a ruptured tubal pregnancy. By so doing, and operating promptly in suitable cases, a number of lives will be saved which otherwise would inevitably be lost.*

J. Whitridge Williams (1866–1931)

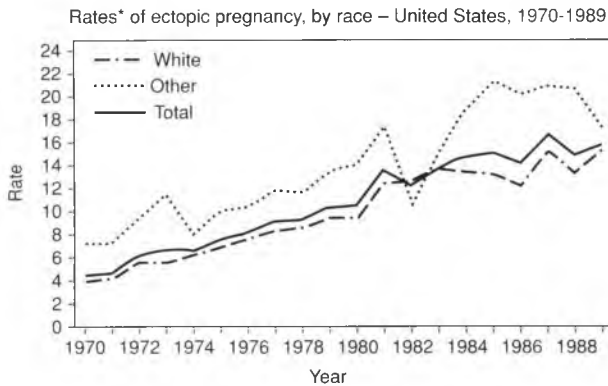
*Obstetrics: A Text-Book for the Use of Students and Practitioners,*

New York: D. Appleton and Company, 1903, p. 553.

The initiation of a normal pregnancy requires exquisitely timed coordination of several endocrine-sensitive tissues. After fertilization of the ovum in the fallopian tube, cleavage and embryonic development occur, followed by uterine implantation approximately six days later. Following fertilization and implantation, the syncytiotrophoblast begins to produce human chorionic gonadotropin (hCG), which eventually rescues and maintains the corpus luteum beyond its normal 14-day life span. When this course of physiologic events occurs normally, a pregnancy can progress, allowing the fetus to develop until birth. The development of an *ectopic pregnancy* is an aberration of this process, in which embryonic implantation occurs outside of the uterus, most commonly in the fallopian tube but also in extratubal locations. Ectopic pregnancy is an extremely serious threat to the general and reproductive health of a woman. The objective of this chapter is to provide a comprehensive discussion of the contemporary approach to ectopic pregnancy. Diagnosis and treatment options and the epidemiology and pathophysiology of the condition are also reviewed.

### EPIDEMIOLOGY

Ectopic pregnancies comprise approximately 2% of all pregnancies reported to the Centers for Disease Control and Prevention (CDC). Several important trends have emerged from data collected by the CDC with respect to ectopic incidence, and related morbidity and mortality. Notably, the incidence of ectopic pregnancy appears to have steadily and persistently risen since 1970, the first year that data on this subject were collected by the CDC (Figure 4.1). Between 1970 and 1992, the rate of ectopic pregnancy increased from 4.5 to 19.7 per 1,000 reported pregnancies (including live births, legal abortions, and ectopic pregnancies) [1,2]. This trend is likely due to the emergence of several key elements, including enhanced diagnostic capability to detect ectopic pregnancies early in gestation, the rising incidence of gonorrhea and chlamydial infections in reproductive-aged women, and the growing use



\*Per 1,000 reported pregnancies (live births, legal abortions, and ectopic pregnancies).

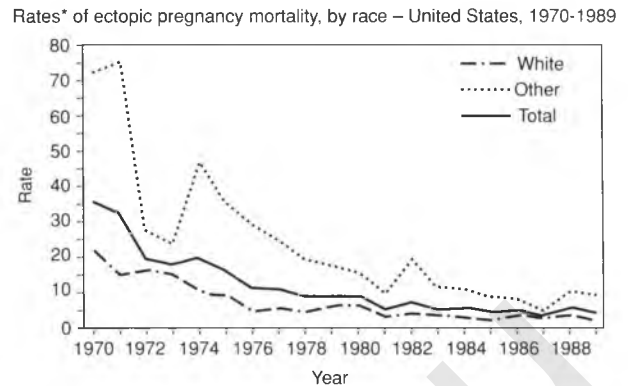
FIGURE 4.1.

*Incidence of ectopic pregnancy from 1970–1989 overall and stratified by race. (From Goldner TE, et al. Surveillance for Ectopic Pregnancy – United States, 1970–1989. MMWR 1993;73–78; with permission.)*

of treatments to circumvent infertility, including in vitro fertilization.

Determination of the overall incidence of ectopic pregnancy is not straightforward, because data on nonhospitalized cases are inconsistently recorded. After a reported increase in hospitalizations for ectopic pregnancy over a twenty-year period starting in 1970, there has been a steady decline from 1990 forward. The number of hospitalizations appears to have peaked at 88,400 in 1989, followed by a significant drop the following year to 64,400 admissions. This trend in reduced hospitalization is due to the increased use of conservative approaches to the treatment of ectopic pregnancy, including the use of laparoscopy and methotrexate. In addition, prompt diagnosis early in gestation makes the occurrence of tubal rupture less common, allowing many more ectopic pregnancies to be treated before rupture and hemodynamic instability ensue.

Despite these notable successes, ectopic pregnancy remains a source of serious maternal morbidity and mortality in the United States. Complications of ectopic pregnancy have made this condition the leading cause of maternal mortality in the first trimester of pregnancy. From 1991 to 1999, there were 237 ectopic-related deaths, which constituted 6% of all pregnancy-related deaths. In most cases, the proximate cause of death is hemorrhage (93.3%), and, less commonly, infection (2.5%) or embolism (2.1%) [3]. Fortunately, the risk of ectopic-related mortality appears to be declining



\*Per 10,000 ectopic pregnancies.

FIGURE 4.2.

*Ectopic pregnancy mortality overall and stratified by race 1970–1989. (From Goldner TE, et al. Surveillance for Ectopic Pregnancy – United States, 1970–1989. MMWR 1993;73–82; with permission.)*

despite the increase in incidence of this condition. From 1970 to 1989, the case fatality rate of ectopic pregnancy drastically declined from 35.5 deaths/10,000 ectopics to 3.8 deaths/10,000 (Figure 4.2) [2].

Another dominant theme in the demographics of ectopic pregnancy is the presence of disparities in incidence and mortality by race. The relative risk of ectopic pregnancy for African American women is up to 1.6 times that for white women [2]. This disparity is consistent across all age categories and extends to differences in mortality related to ectopic pregnancy. As concerns ectopic-related mortality, the health disparity by race is even more prominent. From 1970 to 1989, the risk of death caused by ectopic pregnancy was 3.4 times greater for African-American women and other minorities as it was for white women [2,3]. The sharp decline in ectopic-related mortality experienced by all women in recent years has been insufficient to eliminate this persistent racial gap.

## PATHOPHYSIOLOGY

Although the exact etiology of ectopic pregnancy is not completely understood, both maternal and embryonic factors are thought to contribute to its development. Abnormalities of tubal function and ovum quality or an altered hormonal milieu may each contribute to the development of an ectopic pregnancy [4]. Although this discussion focuses on tubal ectopic pregnancies, extrauterine pregnancies

can occasionally localize to the abdomen, cervix, ovary or uterine cornua. These less common presentations of ectopic pregnancy are discussed separately.

Normal embryo transport can be disrupted by damage to the structural integrity of the mucosal portion of the fallopian tube. It is easily understood that scarring secondary to infection or trauma could lead to trapping of a conceptus within intratubal adhesions or diverticulae. More subtle insults might not overtly disrupt normal anatomy but could cause ciliary dysfunction and compromise tubal transport. This type of insult could be most significant within the ampullary portion of the tube, where cilia are most concentrated and fertilization and early cleavage of the embryo take place.

Although defectively fertilized ova are logistically difficult to assess, the concept deserves further inquiry. It has been speculated that perhaps immature or postmature ova are more likely to implant prior to reaching the endometrial cavity [4]. This hypothesis requires further investigation, since the incidence of chromosomal abnormalities among ectopic pregnancies has not been found to be any greater than those noted in induced abortions [5].

Alteration of the hormonally mediated events leading to implantation offers another mechanism for consideration. A change in the estrogen-to-progesterone ratio could theoretically affect smooth muscle activity in the fallopian tube, immobilizing ciliary activity. The occurrence of this phenomenon would be particularly influential in the isthmic portion of the tube, which is suspected to contribute to the retention of the fertilized ovum for several days prior to implantation.

Any of these processes could be responsible for the detainment of the embryo and its developing trophoblast within the tube and subsequent mucosal invasion. Determination of whether tubal ectopic pregnancies are intraluminal or extraluminal in location has been studied. Initial evidence based on retrospective examination of tissue blocks directed attention to the extraluminal location between the muscularis and serosa [6]. Pauerstein and associates [7] examined this issue prospectively, however, and found most cases of unruptured ectopic pregnancies to be intraluminal. In contrast, ruptured ectopic pregnancies are located in both the intraluminal and extraluminal sites.

**TABLE 4.1 Risk Factors for Ectopic Pregnancy**

Risk Factors for Ectopic Pregnancy	Odds Ratio
Tubal surgery	4.7–21.0
Surgery for ectopic pregnancy	6.6–8.3
Documented tubal pathology	3.5–25
In utero DES exposure	5.6
Previous gonorrhea infection	2.9
Previous chlamydia infection	2.8–3.7
Previous PID infection	1.7–2.5
Infertility	2–2.5
Smoking	1.6–2.5

DES, diethylstilbestrol; PID, pelvic inflammatory disease.  
From Ankum WM et al. Risk factors for ectopic pregnancy: A meta-analysis. *Fertil Steril* 1996;65:1093; with permission.

Of note with regard to implantation of the trophoblast within the fallopian tube is that most tubal pregnancies do not consist of ongoing viable gestations but are in fact in the process of abortion within a confined area. Although some blood accumulates both medially and laterally to the implantation site, most luminal accumulation of blood is lateral, allowing collection in the most distensible portion of the tube and often leading to leakage of blood from the fimbria [8].

## **RISK FACTORS**

The decline in morbidity and mortality from ectopic pregnancy is related mostly to widespread awareness of important risk factors, facilitating early diagnosis. Conversely, changes in the prevalence of these risk factors are associated with the increased incidence of ectopic pregnancy in the United States. Some of the most significant risk factors for the development of ectopic pregnancy include history of pelvic inflammatory disease (PID), prior fallopian tube surgery, increasing age, and a history of infertility. These risk factors and others must be elicited from the patient to exclude alternative diagnoses and prevent a delay in diagnosis (Table 4.1).

### **Pelvic Infection**

PID is the most common cause of tubal abnormalities and can lead to deciliation, intratubal and extratubal adhesions, and fimbrial injury. The offending organisms are most likely Chlamydia,



gonorrhoea, or mixed anaerobic and aerobic organisms [9,10]. Westrom and associates [11] demonstrated the association of laparoscopically verified PID with tubal obstruction and ectopic pregnancy. In a study of 415 women with PID, the incidence of tubal occlusion after one, two, and three episodes was 13%, 35%, and 75% respectively. After one episode of PID, the ratio of ectopic-to-intrauterine pregnancies has been demonstrated to change from 1:147 to 1:24 by one group of investigators. This same group noted that women with laparoscopically proven salpingitis had a six- to sevenfold increase in the incidence of ectopic pregnancy after the episode of salpingitis [12].

### Prior Ectopic Pregnancy

A history of ectopic pregnancy is a powerful risk factor for women who have experienced an ectopic pregnancy; such women have a 7- to 13-fold increased risk of subsequent ectopic pregnancy compared with the general population. On average, after one ectopic pregnancy the odds of recurrence range from 9% to 27% [13,14]. After two ectopic pregnancies, a repeat ectopic pregnancy occurs in 36% to 40% of subsequent pregnancies [15,16]. High rates of infertility often follow single or recurrent ectopic pregnancies as well [14,17].

### Contraception and Surgical Sterilization

In general, the risk of ectopic pregnancy in women using any form of contraception is diminished compared with women using no contraception [17]. Nevertheless, different forms of birth control have very distinct degrees of risk of ectopic pregnancy when they fail. Contraceptive failure with the birth control pill is associated with a very low risk of ectopic pregnancy (0.005 ectopic pregnancies/1,000 woman-years) compared with much higher risks associated with the intrauterine device (IUD) and tubal sterilization (1.02 ectopics/1,000 woman-years and 0.3 ectopics/1,000 woman-years, respectively). Despite the fact that IUDs are highly effective at preventing pregnancy, when a pregnancy does occur, 6% to 50% are ectopic. This risk appears to be higher with the levonorgestrel IUD than the copper IUD [18].

Data from the U.S. Collaborative Review of Sterilization [19], which followed a cohort of greater

than 10,000 women, demonstrated that tubal ligation failure results in an ectopic pregnancy in one third of cases. The 10-year cumulative risk of ectopic pregnancy was 18.5/1,000 pregnancies. Variables that modify the risk of ectopic pregnancy after tubal sterilization include patient age at the time of procedure and length of time since surgery. The risk of ectopic pregnancy after tubal sterilization is inversely proportional to the age of the patient at the time of surgery. Moreover, ectopic pregnancies associated with failed tubal ligations are more likely to occur with the interval of time from the procedure, with most developing more than four years after the initial surgery [19].

The incidence of ectopic pregnancy also varies with the type (i.e., fulguration) of procedure that is performed [20–23]. As a result, interval laparoscopic tubal electrocautery poses the highest risk of all available methods, whereas postpartum tubal ligation is the least likely to result in development of an ectopic pregnancy. In a study of over 35,000 tubal sterilizations, 51% of pregnancies following laparoscopic tubal electrocautery were noted to be ectopic compared with 12% following nonlaparoscopic, nonfulgurative tubal ligations [20]. Coagulation sterilization failures are associated with a higher incidence of uteroperitoneal fistulas that can be large enough to allow sperm access to the oocyte but small enough to preclude the transport of the conceptus [20]. Corroborative evidence supporting this theory is the 75% of pregnancies following coagulation sterilization failures noted in the distal portion of the fulgurated tube [24]. It bears emphasizing that while these data demonstrate that a greater percentage of pregnancies following laparoscopic sterilization are ectopic, the absolute rate of ectopic pregnancies in this group is still much lower than in women using no contraception [23].

### PRIOR TUBAL SURGERY

Prior tubal surgery results in an increased risk of ectopic implantation. Risk for ectopic pregnancy varies depending on the type of reconstructive surgery and the extent of the underlying disease. Examples of reported rates of ectopic pregnancies following distal salpingostomies range between 12% and 18% [25], and approach 5% following a tubal anastomosis [26]. Ectopic rates following lysis of pelvic adhesions appear to depend on the

extent of peritubular adhesions [27]. Excluding a ruptured appendix, previous nontubal abdominal surgery does not appear to increase ectopic risk [28].

### **INFERTILITY AND INFERTILITY TREATMENT**

Infertility alone or in combination with treatment is a risk factor for ectopic pregnancy. Several studies have suggested an association between medications used for superovulation and ectopic pregnancy [29–30]. In one case–control study, investigators found a twofold increased risk of ectopic pregnancy associated with the administration of fertility drugs [31]. These studies were limited, however, by lack of detailed drug data (types and doses) and failed to control for a history of previous ectopic pregnancy or pelvic infection. Additional data to support an association with fertility medications came from a recent case–control study demonstrating a nearly fourfold risk of ectopic pregnancy in patients exposed to drugs for ovulation induction [32]. One possible explanation for this association could reside in the influence of higher-than-normal preovulatory levels of estradiol in these patients, which might adversely affect tubal peristalsis.

There has been concern regarding a possible association between in vitro fertilization (IVF) and ectopic pregnancy. Notably, the first pregnancy conceived as a result of IVF in 1976 was an ectopic pregnancy [33]. Several descriptive studies document the incidence of ectopic pregnancy to be 5% to 7% in IVF cases, two to three times the general population risk [34–35]. It has been postulated that reverse embryo migration toward an abnormal fallopian tube following embryo transfer is associated with the development of ectopic pregnancies after IVF [36]. In addition, heterotopic pregnancies, considered extremely rare in the general population, occur with greater frequency (0.3%–1% of pregnancies) in women who conceive with infertility treatments, especially IVF [37].

### **AGE**

As women delay childbearing beyond the age of 35 years, there appears to be a decrease in fertility accompanied by an increase in the rate of pregnancy complications, including spontaneous abortions and ectopic pregnancies [38–40]. It has been observed that women between the ages of 35 and 44 years

have a threefold increase in the incidence of ectopic pregnancy compared with women aged 15 to 24 years, when controlling for race [41]. Changes in tubal function resulting in impaired ovum transport could be a possible component in this age-related increase in risk [39,41]. Undoubtedly, the risk also represents additional acquired risks that are present in this age group compared with their younger counterparts.

### **SMOKING**

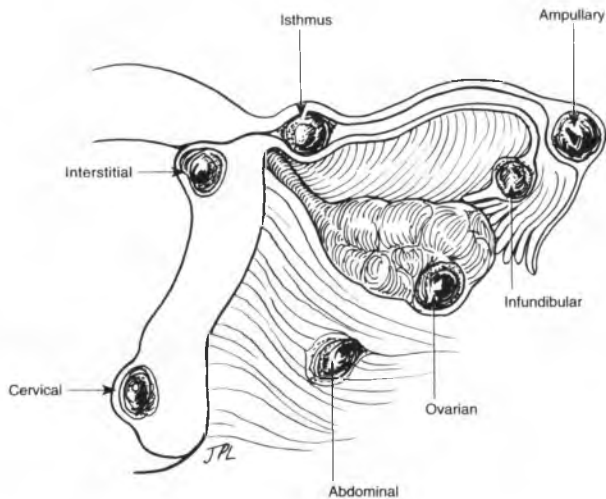
Smoking has emerged in recent years as an important risk factor for ectopic pregnancy, with an estimated relative risk of 2.5 [42]. Although the role of smoking in the etiology of ectopic pregnancy is less obvious than some of the other risk factors described, it has been theorized that nicotine or other additives in cigarettes might cause abnormal tubal motility and increase the odds of tubal implantation. Secondarily, nicotine could alter cellular and humoral immunity, diminishing the ability of the tubal epithelium to contain pathogens capable of causing inflammation and tubal scarring [43–45].

### **MATERNAL DIETHYLSTILBESTROL EXPOSURE**

Maternal diethylstilbestrol (DES) exposure has been described as having a potential role in increasing the odds of ectopic pregnancy in female offspring. Although maternal use of DES has been related to the development of numerous tubal abnormalities in daughters of exposed women, an association with ectopic pregnancy has not been well elucidated [43].

### **UNUSUAL ECTOPIC PREGNANCIES**

As they are far less common than tubal ectopic pregnancies, cervical, abdominal, ovarian, cornual, and heterotopic pregnancies often present significant diagnostic and therapeutic challenges. Overall, ectopic pregnancies in these locations compose less than 5% of all extrauterine pregnancies but are often associated with significant morbidity and mortality (Figure 4.3). Approaches to treatment of these special cases of ectopic pregnancy are discussed later in this chapter.



**FIGURE 4.3.**  
*Various potential locations of ectopic pregnancies.*

## **DIAGNOSIS**

The classically described triad of symptoms for ectopic pregnancy are pelvic pain, amenorrhea, and abnormal bleeding [14]; however, up to 50% of patients will not present with this constellation of symptoms. This makes clinical suspicion of paramount importance in the early detection of an ectopic pregnancy. Although some patients present acutely with a ruptured ectopic pregnancy and a hemoperitoneum [37,40], up to 80% of diagnoses are made in the outpatient setting [46]. Ultimately, transvaginal ultrasonography is the best noninvasive method to determine the location of a pregnancy. Ultrasound scans have limited diagnostic accuracy in some cases, however. Unfortunately, there are no reliable pathognomonic symptoms or signs to distinguish between a normal pregnancy with symptoms, an abnormal intrauterine pregnancy, and an ectopic pregnancy. Moreover, there are multiple gynecologic and nongynecologic diagnoses that can be confused with an ectopic pregnancy. Thus, diagnostic tests have gained increasing importance in allowing timely diagnosis of early abnormal pregnancies. The most important of these diagnostic tests are serial serum beta-human chorionic gonadotropin (B-hCG) and high-resolution transvaginal ultrasound scanning.

## **HORMONAL ASSAYS**

In the past 20 years, several clinical innovations have revolutionized the contemporary diagnosis and early

management of ectopic pregnancies. The development of serum pregnancy tests with increased sensitivity to B-hCG has contributed enormously to the prompt identification of ectopic pregnancies. The reference standard for B-hCG measurement discussed herein is the Third International Reference Preparation (IRP) established by the World Health Organization. The IRP is a highly purified preparation used in the assay to quantify B-hCG levels. The assay standard used by a particular laboratory must be known to interpret hCG results correctly and to make comparisons between values assayed using different standards. For instance, B-hCG values reported using the most recent reference preparation (Third International Standard), are twice as high as values calculated using the Second International Standard [47].

The development of accepted patterns for the rise of B-hCG values and “doubling rules” in early pregnancy has allowed clinicians to better identify abnormal pregnancies before ultrasound examination is required. Traditionally, serum B-hCG values have been described as doubling every 1.4 to 3.5 days in normal pregnancies early in the first trimester. Moreover, it has been observed that most abnormal pregnancies do not maintain this doubling rate. A B-hCG value that doubles less than 66% in 48 hours is associated with an abnormal pregnancy 80% of the time [48–51]. Despite this well-accepted clinical principal, up to 21% of women with ectopic pregnancies have normally rising B-hCG titers [51]. Therefore, patients in whom a high index of suspicion for ectopic pregnancy exists should still be closely followed even if the B-hCG titer is rising normally [52].

A recent study has revisited the traditional thinking concerning a normal B-hCG rise [53]. Novel data taken from women with symptoms (bleeding or pain), nondiagnostic ultrasound results, and ultimately normal pregnancies are now available. The median slope for a 48-hour rise in the B-hCG titer was 124%. A more conservative lower limit of a 53% B-hCG titer increase over the same time period was also described, however. This latter figure is below the accepted lower limit of a 66% rise in 2 days and supports a somewhat more conservative approach to interventions when following hCG values to prevent the interruption of normal pregnancies.

The use of progesterone measurements has limited diagnostic utility in discriminating normal

pregnancies from ectopic pregnancies. Although serum progesterone levels are often lower in ectopic pregnancies than in normal intrauterine pregnancies, there is significant overlap between values derived from normal and abnormal pregnancies [17,37]. Furthermore, values can be misleading in infertility patients receiving supplemental progesterone after ovulation induction.

### TRANSVAGINAL ULTRASONOGRAPHY

Transvaginal ultrasonography has essentially replaced transabdominal scanning in early pregnancy evaluation. Gestational and yolk sacs, as well as cardiac activity, are detected up to 1 week earlier, and free fluid in the cul-de-sac is more easily identified by transvaginal ultrasound scan than by the transabdominal approach [54–55]. In some cases, transvaginal ultrasound can detect a gestational sac as early as 1 week from a missed menstrual period [56–57]. A critical concept in the evaluation of early pregnancy by transvaginal ultrasound is that of the *B-hCG discriminatory zone*, or the level above which an examiner should see a normal intrauterine gestation, if present. In the setting of a B-hCG level above the discriminatory zone and no intrauterine pregnancy on ultrasound scan, an ectopic pregnancy or an abnormal intrauterine pregnancy is highly likely [58–62]. The exact hCG discriminatory zone for differentiating an ectopic pregnancy from an intrauterine pregnancy varies somewhat from institution to institution, depending on the experience of the ultrasonographer and the hCG standard used. The accepted value usually lies between 1,500 mIU/ml and 2,000 mIU/ml [17].

The earliest ultrasonographic finding of a normal intrauterine pregnancy is the gestation sac surrounded by a thick echogenic ring, located eccentrically within the endometrial cavity. On average, the gestational sac is seen on transvaginal ultrasound scan at 4 weeks' gestation. As the gestation sac grows, a yolk sac is seen within it, followed by an embryonic pole with cardiac activity. The appearance of a normal gestational sac can be simulated by a pseudogestational sac and intrauterine fluid collection, which occurs in 8% to 29% of patients with ectopic pregnancy. The pseudogestational sac likely represents bleeding into the endometrial cavity by the decidual cast.

Morphologically, the identification of the double decidual sac sign is a reliable method of discriminating true gestational sacs from pseudosacs. The double sac, believed to be the decidua capsularis adjacent to the decidua parietalis can be visualized ultrasonographically as two concentric echogenic rings separated by a hypoechogenic space. The sensitivity of this sign varies, however, ranging from 64% to 95%. The appearance of a yolk sac is superior to the double-sac sign at determining an intrauterine pregnancy [57–60].

The detection of color Doppler flow using transvaginal ultrasound scan can be of particular usefulness in the clinical context of a small intrauterine gestational sac that does not demonstrate a yolk sac or a double-sac sign. In such a situation it would be difficult to distinguish an early intrauterine pregnancy from a pseudosac of an ectopic pregnancy. Several studies using transvaginal ultrasound examination with color pulsed Doppler show improved diagnostic sensitivity, and thus this modality could lead to earlier treatment with associated reduced morbidity and mortality [63–64].

The demonstration of an adnexal gestational sac with a fetal pole and cardiac activity is the most specific but least sensitive sign of ectopic pregnancy, occurring in only 10% to 17% of cases. The recognition of other characteristics of ectopic pregnancy has improved ultrasonographic sensitivity. Adnexal rings (fluid sacs with thick echogenic rings) that have a yolk sac or nonliving embryo are accepted as specific signs of ectopic pregnancy. Adnexal rings are visualized in ectopic pregnancies 33% to 50% of the time but might not always be readily apparent owing to bleeding around them [14].

The diagnostic accuracy of transvaginal ultrasonography for ectopic pregnancy is not absolute and depends highly on the B-hCG level at the time of examination. In a recent report, the sensitivity of transvaginal ultrasound scan for ectopic pregnancy was significantly associated with B-hCG levels above or below a discriminatory zone of 1,500 mIU/ml [65]. Scans performed above this level had a sensitivity of 80% for ectopic pregnancy, a positive predictive value of 85.7%, and a negative predictive value of 98.8%. Conversely, transvaginal ultrasonography performed at B-hCG levels below 1500 mIU/ml had 25% sensitivity, 60% positive predictive value, and 84.7% negative predictive value for diagnosing ectopic pregnancy.

The overall sensitivity and negative and positive predictive values for transvaginal ultrasound scan regardless of B-hCG level were 55.6%, 96.2%, and 78.9%, respectively [65]. Endometrial thickness at the time of ultrasound examination has not been demonstrated to have predictive value for ectopic pregnancy. Finally, three-dimensional ultrasonography offers little diagnostic advantage over conventional two-dimensional ultrasonography for ectopic localization [37]. In sum, these data reflect the limitations of ultrasound examination to capture many cases of ectopic pregnancy – particularly early ectopic pregnancies – which often compels the use of invasive diagnostic procedures to confirm or rule them out.

### DIFFERENTIAL DIAGNOSIS

The differential diagnosis for ectopic pregnancy includes multiple gynecologic conditions, many of which can be easily distinguished by the serum B-hCG determination. One of the most difficult diagnoses to distinguish from an ectopic pregnancy is a hemorrhagic corpus luteum in a patient very early in gestation. The presence of pain, pelvic hemorrhage, and lack of intrauterine pregnancy can lead to confusion with ectopic pregnancy. Patients who have significant bleeding and a B-hCG below the discriminatory zone warrant a diagnostic laparoscopy to distinguish the two diagnoses definitively.

### **Surgical Diagnosis**

The patient with an abnormal rise in B-hCG or a value at or above the discriminatory zone with no detectable intrauterine pregnancy has an abnormal pregnancy in all but a few exceptional cases. The challenge for the clinician is to determine whether this pregnancy is an abnormal intrauterine pregnancy or an ectopic pregnancy. At this point, invasive measures are typically employed to differentiate the two possibilities. The most common approaches are listed below and each is discussed in turn. A full description of the therapeutic aspects of these decisions follows in the next section.

- Dilatation and curettage followed by frozen-section pathologic evaluation of the endometrial curettings. If no products of conception are detected, a laparoscopy is performed.

- Dilatation and curettage followed by frozen-section pathologic evaluation of endometrial curettings. If no products of conception are detected, medical management with methotrexate is implemented.
- Diagnostic laparoscopy to evaluate the fallopian tubes and pelvis for the presence of an ectopic pregnancy. If no ectopic pregnancy is found, a dilatation and curettage may be performed to evacuate the uterus.
- Empiric medical management without dilatation and curettage.

The first two options are similar except for the therapeutic approach taken once the ectopic pregnancy is diagnosed. The general principle behind these approaches is that frozen-section evaluation of endometrial curettings has sufficient diagnostic accuracy to capture ectopic pregnancies and prevent overtreatment of women with abnormal intrauterine pregnancies. There is evidence to suggest that this is the case. In a study published by Spandorfer and colleagues, the positive predictive value of frozen-section evaluation of endometrial curettings in a population of women suspected of ectopic pregnancy was 94.7%; the negative predictive value was 92.6% [66]. Although these values are reassuring, final pathologic diagnoses should always be evaluated to confirm the diagnosis. Furthermore, a B-hCG drawn up to 24 hours postprocedure can be extremely helpful in further discriminating patients if uncertainty about the frozen-section evaluation exists. A significant fall in B-hCG after a dilatation and curettage strongly favors the diagnosis of an abnormal intrauterine pregnancy, whereas a plateau or increase in the value suggests an ectopic pregnancy. This information is pertinent for stable patients desiring medical management, which could be administered, if necessary, after the results of the blood test.

An appealing alternative to dilatation and curettage to sample the uterus in cases of suspected ectopic pregnancy is the pipelle biopsy. Given the diagnostic accuracy of pipelle biopsy for endometrial carcinoma, it is reasonable to assume that it might be a useful means of tissue sampling in the evaluation of ectopic pregnancy. A recent study tested this hypothesis and determined that this was not the case, however. Pipelle biopsy in women with

suspected ectopic pregnancy had a sensitivity of 30% and a negative predictive value of 76%, suggesting that many cases of ectopic pregnancy would be missed using this method instead of a formal dilatation and curettage [67].

The third diagnostic option involving laparoscopy first represents a reasonable approach if the patient in question has pain or a significant pre-procedure probability of ectopic pregnancy (as determined by risk factors or ultrasound scan). Moreover, this approach can be used if the false-negative risk of frozen-section evaluation is unacceptable to the patient or clinician. The risk of this approach is the risk of a potentially unnecessary laparoscopy if the patient has an abnormal intrauterine pregnancy.

The last option of medical treatment without a dilatation and curettage is the least desirable owing to the risk of overtreatment of women without ectopic pregnancy. Empiric treatment of suspected ectopic pregnancy without the performance of a dilatation and curettage could result in inappropriate treatment of up to 40% of unaffected women [68]. In addition to lacking clinical utility, this treatment option is not cost effective [69].

### SPECIAL POPULATIONS

As discussed previously, patients with infertility who are undergoing ovulation induction or IVF are at increased risk for ectopic pregnancy. Moreover, these patients have an elevated risk of conceiving a multiple gestation if the intervention(s) are successful. Following conception, these patients are followed very closely, with early serial B-hCG levels and, once the value has crossed the discriminatory zone, transvaginal ultrasound scan. The problem for many of these patients is that the discriminatory zone was developed for singleton intrauterine pregnancies not for twins or higher order multiples. How are clinicians to reconcile this and appropriately manage these high-risk patients? The problem is compounded by the fact that the range of B-hCGs for normal singleton pregnancies is wide and overlaps to some degree with values for early twin intrauterine pregnancies. Clinicians need to follow patients very closely for symptoms and signs of ectopic pregnancy. If there is a high index of suspicion for multiple pregnancy, a more liberal discriminatory zone cutoff could be adopted.

A second high-risk population is patients who have had ectopic pregnancies before, many of whom are also monitored very closely in early pregnancy for recurrent ectopic pregnancies. A recent case-control study investigating risk factors and clinical signs of repeat ectopic pregnancy demonstrated that women with a repeat ectopic pregnancy, as compared with women experiencing their first ectopic pregnancy, were less like to develop bleeding prior to diagnosis [14]. Patients and perhaps physicians might be falsely reassured by the lack of bleeding early in gestation but must be vigilant about close laboratory and ultrasound surveillance of these patients.

### TREATMENT

Although surgery remains the mainstay of treatment for ectopic pregnancy, medical management is a widely used alternative. There has been a shift in recent years in the approach to treatment, emphasizing less invasive and more conservative treatments when appropriate. Safe and effective outcomes can be realized with these treatment options owing to the early diagnosis of many ectopic pregnancies. The choice of any treatment option depends on the presentation and particular risks of the patient. Factors that influence the decision include clinical presentation, status of the involved and contralateral fallopian tubes, and a history of previous ectopic pregnancy.

### SURGICAL MANAGEMENT

Patients presenting with hemodynamic instability caused by a ruptured ectopic pregnancy require laparotomy and salpingectomy of the involved fallopian tube owing to extensive tubal damage. Ruptured ectopic pregnancy as a clinical presentation is decreasing as gynecologists increase their vigilance, gain greater experience with transvaginal ultrasound scans, and use serial B-hCG assays. Thus, the unruptured and often very early ectopic pregnancy is an increasingly common presentation.

A woman with an unruptured ectopic pregnancy might or might not be symptomatic, depending on the stage of development of the ectopic pregnancy and its anatomic location. Once the diagnosis has been made, conservative surgery is the present standard of practice for treatment. Linear salpingostomy

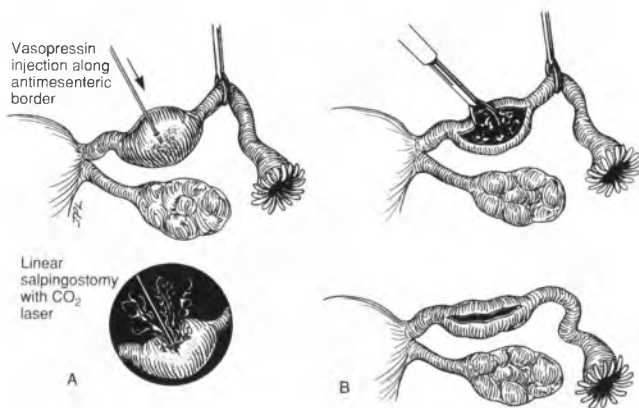


FIGURE 4.4.

*Surgical technique for linear salpingostomy.*

by laparoscopic approach is the favored approach in most cases, unless special circumstances such as limited access to the ectopic pregnancy necessitate the performance of a laparotomy. Controlled studies have demonstrated similar success rates and reproductive potential of salpingostomy in the treatment of ectopic pregnancies with either laparoscopy or laparotomy [70–72].

Linear salpingostomy is performed by making an incision in the antimesenteric aspect of the fallopian tube directly over the ectopic pregnancy. Any of several cutting instruments, including pinpoint cautery, laser, or cauterizing scissors, can be used for the incision. Injection of dilute vasopressin (Pitressin) adjacent to the ectopic pregnancy can improve hemostasis. The products of conception are then expressed through the incision, and hemostasis is achieved using cautery. The fallopian tube incision is allowed to heal by secondary intention (Figure 4.4).

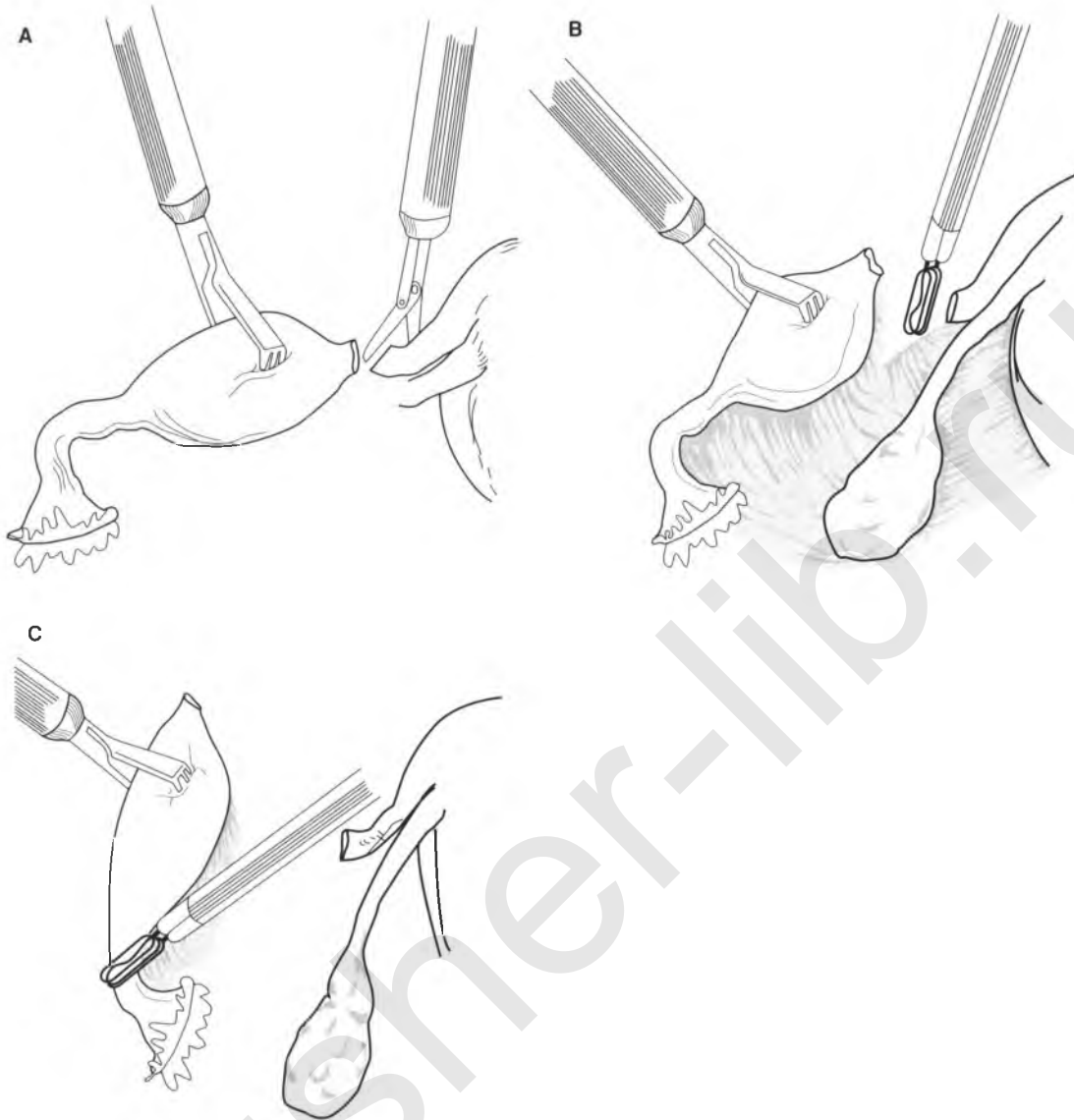
Candidates for linear salpingostomy include patients without tubal rupture and those who have an ectopic pregnancy in the ampulla or infundibulum of the fallopian tube. Linear salpingostomy is conservative surgery and as such is associated with some risk of failure. Persistent ectopic pregnancy after linear salpingostomy ranges in frequency from 3% to nearly 30% of procedures [73–77]. Few clinical predictors exist to determine which patients will be successfully treated by conservative surgery. Early ectopic pregnancies can prove more challenging to evacuate completely and therefore could present a slightly higher risk of persistence. Spandorfer and

colleagues demonstrated that postoperative day one B-hCG levels were predictive of persistent ectopic pregnancy after salpingostomy [78]. A drop in B-hCG of less than 50% was associated with a greater than threefold increased risk of a persistent ectopic pregnancy. Conversely, when levels declined by at least 77% on postoperative day one, no persistent ectopic pregnancies occurred. To ascertain whether salpingostomy has cured a patient, B-hCG levels must be checked regularly until complete resolution, a process that can take several weeks.

Prophylactic methotrexate has been proposed as a means of reducing the odds of a persistent ectopic pregnancy following conservative surgery [76,79]. The outcomes of a randomized controlled trial examining the efficacy of postoperative prophylaxis with methotrexate demonstrated a significantly lower incidence of persistence in patients who were treated compared with those who were not (1.9% vs. 14.5%) [79]. The decision to use prophylaxis must take into consideration the odds of persistence in the individual patient and risk of side effects of the medication. In a recent decision analysis examining this question, it was reported that prophylaxis was best used if the following conditions were met: rate of persistent ectopic with observation >9%, probability of tubal rupture with persistent ectopic pregnancy >7.3%, success of prophylaxis >95%, and complication rate associated with methotrexate ≤18% [76]. A possible approach to the risk of persistent ectopic after salpingostomy would be to incorporate a postoperative day one B-hCG into the monitoring strategy using a drop of less than 50% to help predict a persistent ectopic pregnancy or use prophylactic methotrexate.

Salpingectomy is reserved for patients with isthmic ectopic pregnancies, tubal rupture, or an ipsilateral recurrent ectopic pregnancy. Salpingectomy is more appropriate for isthmic ectopic pregnancies because the narrowness of the isthmic lumen of the fallopian tube can predispose to tubal obstruction and scarring after salpingostomy (Figure 4.5). Furthermore, women who have completed childbearing might be candidates for salpingectomy rather than salpingostomy.

With respect to future fertility, the preponderance of published data suggests similar odds of intrauterine conception following either salpingostomy or salpingectomy. Few studies indicate more



**FIGURE 4.5.**  
*Technique of laparoscopic salpingectomy for ectopic pregnancy. A, Coagulation and transection of the proximal aspect of the affected fallopian tube. B–C, Coagulation and transection of the mesosalpinx.*

favorable odds following conservative surgery [80–82]. The most important determinant of conception following surgical treatment is the condition of the contralateral tube. A healthy contralateral tube clearly confers a better prognosis. It has been reported that women with a healthy contralateral tube at the time of ectopic treatment are 2.3 times more likely to have a subsequent intrauterine conception than those who do not [83]. If the tube

is unhealthy, however, salpingostomy appears to be the superior surgical approach if future fertility is desired. Other important modifiers of the probability of conception after surgical treatment include parity (nulliparous women have lower odds of conception than multiparous women) and the presence of additional pertinent ectopic risk factors [37,80–82]. Of note, the odds of subsequent intrauterine pregnancy are not affected by treatment



of a prior persistent ectopic pregnancy [83]. The odds of recurrent ectopic pregnancy appear to be higher in women after conservative rather than radical surgical treatment [80–82,84]. The incidence of recurrent ectopic pregnancy with salpingostomy is 10% to 15%, while intrauterine pregnancy rates range from 55% to 75% [84].

### MEDICAL MANAGEMENT

Methotrexate therapy for ectopic pregnancy is a widely used medical alternative to surgery. Methotrexate is a folic acid antagonist administered intramuscularly that targets rapidly proliferating cells such as trophoblasts through inhibition of DNA synthesis. The contemporary use of methotrexate for ectopic pregnancy is a logical extension of its traditional use to treat gestational trophoblastic disease. Although medical treatment of ectopic pregnancy is an appealing option for many patients, certain contraindications exist to the use of the drug [85–86]. Absolute contraindications include

- Immunodeficiency
- Peptic ulcer disease
- Chronic liver disease or alcoholism
- Renal disease
- Hematologic abnormalities
- Known sensitivity to methotrexate
- Active pulmonary disease
- Hemodynamic instability or evidence of intraabdominal bleeding
- Inability to comply with follow-up

To ascertain whether a patient is eligible for methotrexate therapy, a comprehensive laboratory and medical evaluation should first be performed, including tests of renal and liver function as well as a complete blood count.

Relative contraindications to methotrexate treatment pertain to patient characteristics that reduce the odds of successful treatment. These include B-hCG levels of 10,000 mIU/ml or greater and ultrasonographic evidence of an ectopic pregnancy with fetal heart activity and an ectopic gestational mass measuring 4 cm or more in diameter. The strongest

predictor for the efficacy of methotrexate treatment is the B-hCG concentration. Values of 1,000 mIU/mL or less are associated with a 98% success rate, whereas values of 10,000 mIU/ml to 15,000 mIU/mL are associated with 82% treatment success [87].

Two methotrexate treatment regimens exist: single and multidose therapy. Multidose therapy is based on body mass index (1 mg/kg); up to four doses are given, alternating daily with leucovorin rescue. Once consecutive B-hCG levels decline by 15% or more, additional doses are held and the levels are followed until they become undetectable. If levels plateau during monitoring, additional doses can be administered; if the response to methotrexate is suboptimal after 4 doses, the physician should consider the treatment a failure. Most patients, however, require fewer than the maximal number of doses to be cured.

Alternatively, single-dose therapy is based on body surface area (50 mg/m<sup>2</sup>). Repeated doses are given if B-hCG levels do not drop by at least 15% between days four and seven after the initiation of therapy. At least 13% of women treated with the single-dose regimen will require an additional dose to be fully cured [17,37].

Although therapy with both regimens has demonstrated efficacy, there has never been a direct comparison between them in a randomized trial. A recent meta-analysis pooling data from 26 studies and examining the efficacy of both approaches shed some light on the comparison [88]. Single-dose treatment was found to be successful in 88.1% of cases, whereas multidose therapy was successful in 92.7%. The risk of failure was significantly higher for single-dose therapy than multidose methotrexate, with an odds ratio (after adjusting for multiple confounders) of 4.74. Notably, the meta-analysis demonstrated that patients designated to receive single-dose therapy often received more than one dose and patients getting multidose therapy often required fewer than four doses to be cured. It appears, therefore, that while neither option might be ideal, the optimal dose of methotrexate likely resides between two and four doses [88].

Side effects of methotrexate therapy occur in up to 30% of women; however, most of these resolve rapidly and are generally of minor consequence [88]. Abdominal pain is common early in treatment and is of concern as a possible indicator of tubal rupture.

A potential cause of this pain in nonacute patients can be tubal miscarriage. Additional potential side effects include nausea, vomiting, diarrhea, gastritis, stomatitis, and liver transaminitis. Serious side effects such as alopecia and neutropenia can occur but are extremely rare [89].

Reproductive success following successful methotrexate therapy appears similar to that following conservative surgery [90]. The most critical predictors of fertility after ectopic pregnancy treated by any conservative means are the condition of the contralateral fallopian tube and the presence of additional ectopic risk factors.

### EXPECTANT MANAGEMENT

Based on the fact that numerous ectopic pregnancies resolve spontaneously, there has been great interest in considering expectant management in selected patients. Expectant management includes close monitoring of symptoms, determination of B-hCG levels, and transvaginal ultrasound scanning. The likelihood of successful ectopic resolution are highest in the presence of a nondiagnostic ultrasound and B-hCG values less than 1,000 mIU/mL (Figure 4.6) [91–92].

### TREATMENT OF UNCOMMON ECTOPIC PREGNANCIES

Unusual ectopic pregnancies are less common, more morbid, and more difficult to diagnose and treat than tubal ectopics. Heterotopic, cervical, ovarian, interstitial, and abdominal pregnancies have unique characteristics and challenges.

#### **Heterotopic Pregnancy**

Heterotopic pregnancies have increased in incidence with recent increases in dizygotic twinning rates and the use of infertility treatments. The estimated incidence is between 1/4,000 to 1/7,000 pregnancies [18]. Because B-hCG levels associated with the intrauterine pregnancy in this condition rise normally, early detection is challenging in asymptomatic patients. As a result, most of these patients are diagnosed only after rupture of the ectopic component of the pregnancy.

The presence of a concurrent intrauterine pregnancy is the principal challenge to the treatment of

heterotopic pregnancies. Treatment of the ectopic pregnancy presents some degree of risk to the viability of the intrauterine pregnancy. Assessing treatment adequacy can be complicated by the inability to follow B-hCG levels as a marker of ectopic resolution. In most heterotopic pregnancies, the ectopic pregnancy is located in the fallopian tube, making salpingectomy or salpingostomy acceptable treatment options. Transvaginal ultrasound-guided salpingocentesis, followed by local injection of the ectopic pregnancy with potassium chloride or hyperosmolar glucose, has been reported as an effective means of treatment when the site of implantation is the fallopian tube or the uterine cornua (Figure 4.7) [93–94]. Although the procedure has attendant risks, it is less involved than alternative surgical methods and requires less anesthesia and operative time. Direct injection of the ectopic with methotrexate which has been described for treatment of solitary tubal ectopics [46], is contraindicated in the treatment of heterotopic pregnancies when a viable intrauterine gestation is present [17].

#### **Abdominal Pregnancy**

Despite the rarity of this condition (1/2,200–1/10,000 pregnancies), it is extremely dangerous and associated with the highest maternal mortality of any type of ectopic pregnancy [17]. Abdominal ectopic pregnancies can originate in the fallopian tube or in the abdomen. Once the diagnosis established, the treatment is surgical. Treatment of advanced abdominal pregnancies should involve complete removal of the placenta to prevent infectious and hemorrhagic sequelae. If removal of the placenta is incomplete, adjuvant methotrexate therapy can be used, but experience is limited and complications are common. This type of treatment remains controversial.

#### **Ovarian Pregnancy**

Preferred treatment of ovarian ectopic pregnancies is surgical resection. Ovarian ectopic pregnancies can be confused with hemorrhagic ovarian cysts and tubal ectopic pregnancies, given similar ultrasonic signs and clinical symptoms. As a result, many are diagnosed incidentally and treated with methotrexate therapy.

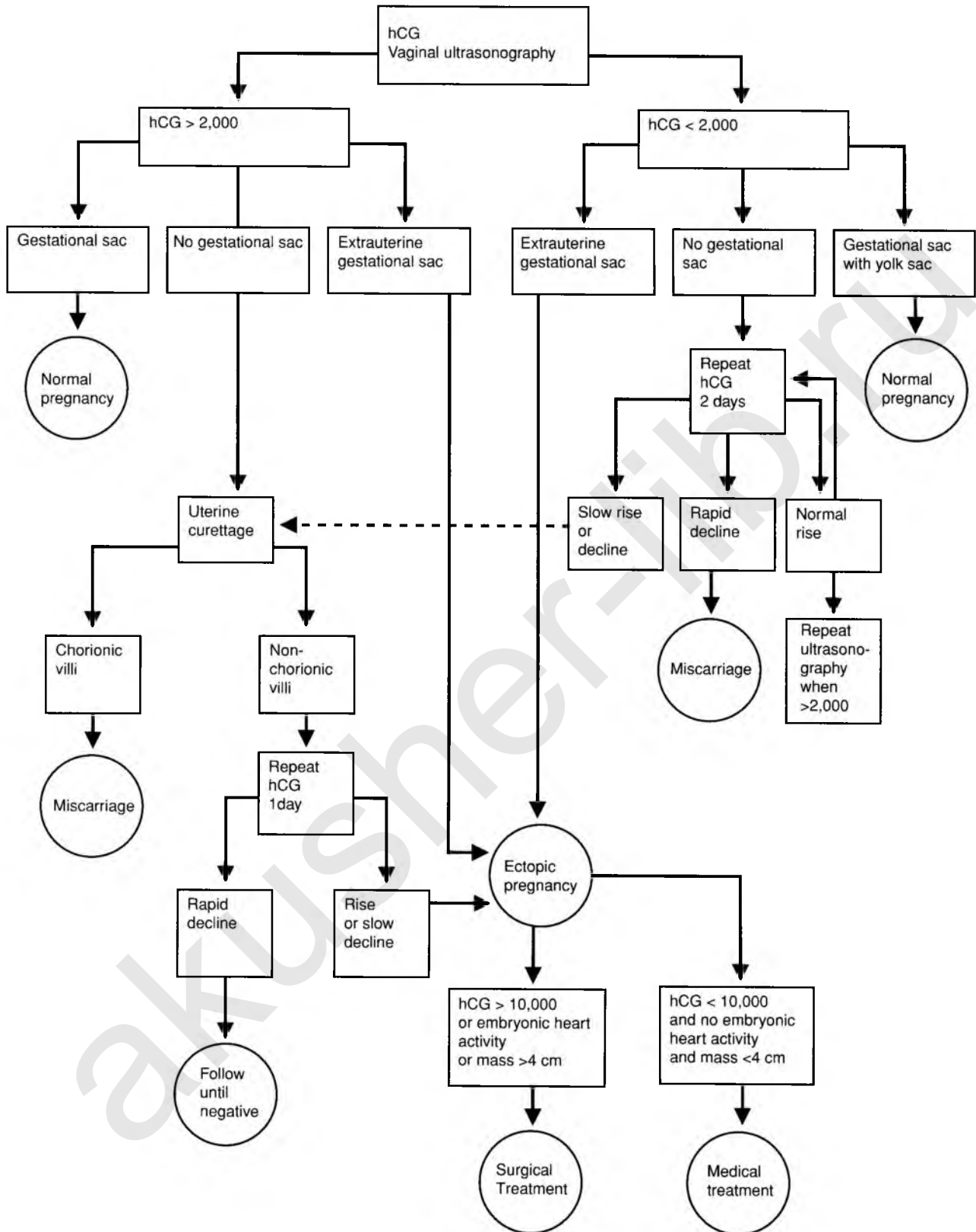
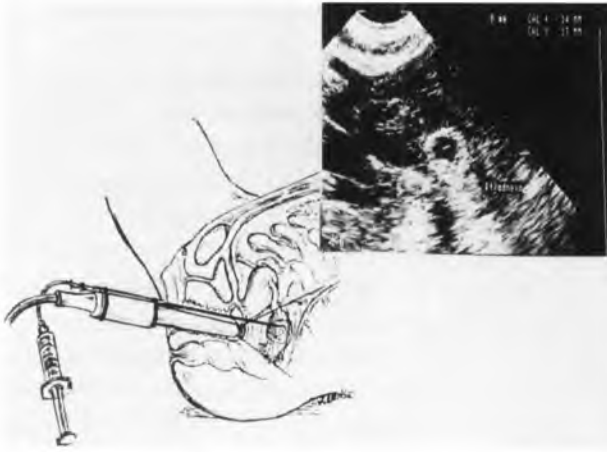


FIGURE 4.6.

Flow chart outline, diagnostic and management algorithm in patients suspected of having ectopic pregnancy. (From Speroff L, Fritz M eds: *Clinical Gynecologic Endocrinology and Infertility*, 7th edition. Baltimore, Lippincott Williams & Wilkins, 2004; with permission.)



**FIGURE 4.7.**  
*Technique of ultrasound-guided injection of tubal ectopic pregnancy. (From Natofsky JG, et al: Ultrasound-guided injection of ectopic pregnancy. Clin Obstet Gynecol 1999;42:29–34; with permission.)*

### Interstitial (Cornual) Pregnancy

Three groups of women are at particular risk for the development of an interstitial ectopic pregnancy: those who have had prior ectopic pregnancy, a salpingectomy, or have been treated with IVF for infertility, especially if they have had a salpingectomy prior to treatment [100]. Women who have had a previous ectopic pregnancy or salpingectomy followed by IVF are more likely to develop an ipsilateral interstitial pregnancy [18]. Careful ultrasound evaluation in early pregnancy should be undertaken, especially in high-risk groups, to distinguish interstitial from intrauterine gestations. The unique ultrasonographic signs of interstitial pregnancy include

- An eccentric gestational sac or heterogeneous mass
- A prominent interstitial tubal segment
- Abnormal thinning of the myometrial mantle

Treatment of interstitial ectopic pregnancies has traditionally been surgical, the procedure depending on the condition of the patient. Cornual resection and hysterectomy are the procedures of choice because many cases of interstitial pregnancy present with rupture and hemodynamic instability in affected patients. Conservative treatment has been reported with increasing frequency in early diagnosis of interstitial pregnancies, but data concerning efficacy are limited [95]. The options for

conservative treatment include administration of systemic methotrexate or the direct local injection of potassium chloride, hyperosmolar glucose, or methotrexate into the cornua. However, many patients who develop interstitial ectopic pregnancies have experienced prior tubal surgery or ipsilateral tubal ectopic pregnancies, and are thus poor candidates for conservative treatment. Overall, conservative treatment of interstitial pregnancy should be reserved for a few select patients, because the consequences of treatment failure in this setting are particularly grave. Those who might be best suited include patients who are poor surgical candidates (systemic methotrexate) and those with heterotopic pregnancies and an interstitial component (salpingocentesis).

### Cervical Pregnancy

Endocervical pregnancy implantation occurs in 1/2,500 to 1/10,000 pregnancies. The most commonly elicited risk factor of women with cervical ectopic pregnancy is previous dilatation and curettage. Those who have conceived with IVF or have had previous cesarean deliveries can also be at increased risk. Vaginal bleeding without pain is the most common symptom of cervical pregnancy, and this bleeding can rapidly evolve into substantial hemorrhage. As a result, therapeutic approaches are often directed both at eradication of the pregnancy and control of life-threatening bleeding. Local treatment options include injection with potassium chloride or methotrexate and uterine artery embolization. Systemic methotrexate has also been used for treatment, with reported efficacy in over 80% of patients [17]. Episodic hemorrhage can be controlled with the use of intracervical balloon tamponade or cerclage placement. In cervical pregnancies of advanced gestational age or in cases in which conservative approaches to treatment or bleeding are unsuccessful, hysterectomy may be required [96].

### Rh PROPHYLAXIS

Data to guide recommendations about the administration of Rh(D) immune globulin (RhoGAM) to Rh-negative women with ectopic pregnancies is extremely limited. It is theoretically possible that a first-trimester abortion in which a sufficient

quantity of fetal-maternal transfusion occurred with fetal RBCs that express the Rh antigen could result in maternal isoimmunization. This is thought to be most likely by approximately 8 weeks' gestation [97]. The American College of Obstetrics and Gynecologists Practice Bulletin, *Prevention of Rh(D) Alloimmunization* [98], states that all unsensitized Rh-negative women should be given prophylaxis within 72 hours of an abortion and does not provide a lower limit of gestational age for treatment. Based on the available data, and the minimal risk associated with Rh(D) immune globulin, prophylaxis of Rh-negative pregnant women is reasonable, regardless of the estimated period of gestation.

### FUTURE DIRECTIONS

Strategies in the area of improving ectopic pregnancy management have focused on discovering methods of highly accurate and minimally invasive early diagnosis. The use of proteomics to aid in the detection of early ectopic pregnancy is an active area of research [99]. Such an approach would permit the discovery of new serum markers that contain molecular profiles uniquely associated with ectopic pregnancy versus an abnormal intrauterine pregnancy or a normal early pregnancy. This could be a powerful and rapid adjunct to early ectopic detection.

### REFERENCES

1. Goldner TE, Lawson HW, Xia Z, Atrash HK. Surveillance for ectopic pregnancy – United States, 1970–1989. *MMWR* 1993;73–82.
2. Current Trends Ectopic Pregnancy – United States, 1990–1992. *MMWR Weekly* 1995;44: 46–48.
3. Chang J, Elam-Evans LD, Berg CJ, Herndon J, Flowers L, Seed KA, Syverson CJ. Pregnancy-Related Mortality Surveillance – United States, 1991–1999. *MMWR Surveillance Summaries* 2004;52:1–8.
4. Russell JB. The etiology of ectopic pregnancy. *Clin Obstet Gynecol* 1993; 81:287–292.
5. Elias S, LeBeau M, Simpson J, Martin AO. Chromosome analysis of ectopic human conceptuses. *Am J Obstet Gynecol* 1981 Nov; 141(6):698–703.
6. Budowick M, Johnson TR Jr, Genadry R, Parmley TH, Woodruff JD. The histopathology of the developing tubal ectopic pregnancy. *Fertil Steril* 1980 Aug;34(2):169–171.
7. Pauerstein CJ, Croxatto HB, Eddy CA, Ramzy I, Walters MD. Anatomy and pathology of tubal pregnancy. *Obstet Gynecol* 1986 Mar;67(3):301–308.
8. Stock RJ. Tubal pregnancy-associated histopathology. *Obstet Gynecol Clin North Am* 1991;18: 73–94.
9. Robertson J, Hogston P, Ward M. Gonococcal and chlamydial antibodies in ectopic and intrauterine pregnancy. *Br J Obstet Gynaecol* 1988;95:711–716.
10. Svensson L, Mardh PA, Ahlgren M, Nordenskjold F. Ectopic pregnancy and antibodies to *Chlamydia trachomatis*. *Fertil Steril* 1985 Sep;44(3): 313–317.
11. Westrom L, Bengtsson LPH, Mardh PA. Incidence, trends, and risks of ectopic pregnancy in a population of women. *Br Med J* 1981;282:15–18.
12. Westrom L. Effect of acute pelvic inflammatory disease on infertility. *Am J Obstet Gynecol* 1975; 121:707–713.
13. Berek JS, Adashi EY, Hillard PA, eds. *Novak's Gynecology*, 12th ed. Baltimore, Williams and Wilkins, 1996.
14. Butts S, Sammel M, Hummel A, Chittams J, Barnhart K. Risk factors and clinical features of recurrent ectopic pregnancy: A case-control study. *Fertil Steril* 2003;80:1340–1344.
15. Uotila J, Heinonen PK, Punnonen R. Reproductive outcome after multiple ectopic pregnancies. *Intl J Fertil* 1989;34:102–105.
16. Glock JL, Loy RA, Brumsted JR. Reproductive performance following a second ectopic gestation. *Intl J Gynaecol Obstet* 1993;43:191–194.
17. Speroff L, Fritz M, eds. *Clinical Gynecologic Endocrinology and Infertility*, 7th edition. Baltimore, Lippincott Williams and Wilkins, 2004.
18. Furlong LA. Ectopic pregnancy risk when contraception fails: A review. *J Reprod Med* 2002;47: 881–885.
19. Peterson HB, Xia Z, Hughes JM, Wilcox LS, Tylor LR, Trussell J. The risk of pregnancy after tubal sterilization: Findings from the U.S. Collaborative Review of Sterilization. *Am J Obstet Gynecol* 1996; 174:1161–1168.
20. Sheikh A, Yussman M. Ruptured ectopic pregnancy after bilateral laparoscopic tubal fulguration. *Am J Obstet Gynecol* 1976;125:569.
21. Chi IC, Laufe LE, Gardner SD, Tolbert MA. An epidemiologic study of risk factors associated with

- pregnancy following female sterilization. *Am J Obstet Gynecol* 1980 Mar;136(6):768-773.
22. McClausland A. High rate of ectopic pregnancy following laparoscopic tubal coagulation failures. *Am J Obstet Gynecol* 1980;136:97-101.
  23. Holt VL, Chu J, Daling JR, Stergachis AS, Weiss NS. Tubal sterilization and subsequent ectopic pregnancy: A case-control study. *JAMA* 1991 Jul;266(2):242-246.
  24. Wolf GC, Thompson JN. Female sterilization and subsequent ectopic pregnancy. *Obstet Gynecol* 1980;55:17-19.
  25. Russell JB, DeCherney AH, Laufer N, Polan ML, Naftolin F. Neosalpingostomy: Comparison of 24- and 72-month follow-up time shows increased pregnancy rate. *Fertil Steril* 1986 Feb;45(2):296-298.
  26. Henderson S. The reversibility of female sterilization with the use of microsurgery: A report on 102 patients with more than one year of follow-up. *Am J Obstet Gynecol* 1984;149:57-61.
  27. Caspi E, Halperin Y. Surgical management for periadnexal adhesions. *Intl J Fertil* 1981;26:49-52.
  28. Ni HY, Daling JR, Chu J, Stergachis A, Voigt LF, Weiss NS. Previous abdominal surgery and tubal pregnancy. *Obstet Gynecol* 1990 Jun;75(6):919-922.
  29. McBain JC, Evan JH, Pepperell RJ, Robinson HP, Smith MA, Brown JB. An unexpectedly high rate of ectopic pregnancy following the induction of ovulation with human pituitary and chorionic gonadotropin. *Br J Obstet Gynaecol* 1980 Jan;87(1):5-9.
  30. Gemzell C, Buillome J, Wang CF. Ectopic pregnancy following treatment with human gonadotropins. *Am J Obstet Gynecol* 1982;143:761-765.
  31. Yang CP, Chow WH, Daling JR, Weiss NS, Moore DE. Does prior infertility increase the risk of tubal pregnancy? *Fertil Steril* 1987 Jul;48(1):62-66.
  32. Fernandez H, Coste J, Job-Spira N. Controlled ovarian hyperstimulation as a risk factor for ectopic pregnancy. *Obstet Gynecol* 1991;78:656-659.
  33. Steptoe PC, Edwards RG. Reimplantation of a human embryo with subsequent tubal pregnancy. *Lancet* 1976;1:880-882.
  34. Cohen J, Mayaux MJ, Guihard-Moscato ML, Schwartz D. In-vitro fertilization and embryo transfer: A collaborative study of 1163 pregnancies on the incidence and risk factors of ectopic pregnancies. *Hum Reprod* 1986 Jun;1(4):255-258.
  35. In vitro fertilization-embryo transfer (IVF-ET) in the United States: 1990 results from the IVF-ET Registry. Medical Research International and the Society for Assisted Reproductive Technology, The American Fertility Society. *Fertil Steril* 1992 Jan;57(1):15-24. Erratum in: *Fertil Steril* 1993 Jan;59(1):250.
  36. Herman A, Ron-El R, Golan A, Weinraub Z, Bukovsky I, Caspi E. The role of tubal pathology and other parameters in ectopic pregnancies occurring in in vitro fertilization and embryo transfer. *Fertil Steril* 1990 Nov;54(5):864-868. PMID: 2226919.
  37. Farquhar CM. Ectopic pregnancy. *Lancet* 2005;366:583-591.
  38. Makinen JI, Erkkola RU, Laippala PJ. Causes of the increase in the incidence of ectopic pregnancy: A study on 1017 patients from 1966 to 1985 in Turku, Finland. *Am J Obstet Gynecol* 1989;160:642-646.
  39. Pulkkiinen M, Talo A. Tubal physiologic consideration in ectopic pregnancy. *Clin Obstet Gynecol* 1987;30:164-172.
  40. Dorfman SF. Epidemiology of ectopic pregnancy. *Clin Obstet Gynecol* 1987;30:173-180.
  41. Yeko TR, Handler A. The effect of aging on tubal function and ectopic pregnancy. *Semin Reprod Endocrinol* 1991;9:215-220.
  42. Ankum WM, Mol BWJ, Van der Veen F, Bossuyt PM. Risk factors for ectopic pregnancy: A meta-analysis. *Fertil Steril* 1996;65:1093-1099.
  43. Campbell OM, Gray RH. Smoking and ectopic pregnancy: Multinational case-control study. In: Rosenberg MJ, ed. *Smoking and Reproductive Health*. Littleton, MA: PSG Publishing Company, 1987; pp. 70-75.
  44. Chow WH, Daling FJ, Weiss NS, Voigt LF. Maternal cigarette smoking and tubal pregnancy. *Obstet Gynecol* 1988;71:167-70.
  45. Handler A, Davis F, Ferre C, Yeko T. The relationship of smoking and ectopic pregnancy. *Am J Public Health* 1989;79:1239-1242.
  46. Natofsky JG, Lense J, Mayer JC, Yeko TR. Ultrasound-guided injection of ectopic pregnancy. *Clin Obstet Gynecol* 1999;42:39-47.
  47. Storing PL, Gaines-Das RE, Bangham DR. International Reference Preparation of Human Chorionic Gonadotropin for Immunoassay: Potency estimates in various bioassays and protein binding assay systems, and International Reference Preparations of the alpha and beta subunits of human chorionic

- gonadotropin for immunoassay. *J Endocrinol* 1980 Feb;84(2):295–310.
48. Pittaway DE, Reisch RL, Wentz AC. Doubling times of human chorionic gonadotropin increase in early viable intrauterine pregnancies. *Am J Obstet Gynecol* 1985;152:299–302.
  49. Batzer FR, Schlaff S, Goldfarb A. Serial B subunit: hCG doubling times as a prognostication of pregnancy outcome in an infertile population. *Fertil Steril* 1981;35:307–312.
  50. Kadar N, Romero R. Serial human chorionic gonadotropin measurements in ectopic pregnancy. *Am J Obstet Gynecol* 1988;158:1239–1240.
  51. Kadar N, Romero R. Observations on the log human chorionic gonadotropin-time relationship in early pregnancy and its practical implications. *Am J Obstet Gynecol* 1987;157:73–78.
  52. Kader N, Caldwell BU, Romero R. A method of screening for ectopic pregnancy and its indications. *Obstet Gynecol* 1981;58:162–166.
  53. Barnhart KT, Sammel MD, Rinaudo PF, Zhou L, Hummel AC, Guo W. Symptomatic patients with an early viable intrauterine pregnancy: hCG curves redefined. *Obstet and Gynecol* 2004;104:50–55.
  54. Jain KA, Hamper UM, Saunders RC. Comparison of transvaginal and transabdominal sonography in the detection of early pregnancy and its complications. *Am J Radiol* 1988;151:1139–43.
  55. Pennell RG, Baltarowich OH, Kurtz AB et al. Complicated first-trimester pregnancies: Evaluation with endovaginal ultrasound versus transabdominal technique. *Radiology* 1987;165:79–83.
  56. Timor-Tritsch IE, Yen MN, Peisner DB, Lester KB, Slavik BS. The use of transvaginal ultrasound in the diagnosis of ectopic pregnancy. *Am J Obstet Gynecol* 1989;161:157–61.
  57. Goldstein SR, Snyder JR, Welson C, Danon M. Very early pregnancy detection with endovaginal ultrasound. *Obstet Gynecol* 1988;72:200–204.
  58. Barnhart KT, Mennuti MT, Benjamin I, Jacobson S, Goodman D, Coutifaris C. Prompt diagnosis of ectopic pregnancy in an emergency department setting. *Obstet Gynecol* 1994;84:1010–1015.
  59. Aleem FA, DeFazio M, Gintautas J. Endovaginal sonography for the early diagnosis of intrauterine and ectopic pregnancies. *Hum Reprod* 1990;5:755–758.
  60. Rottem S, Thaler I, Levron J, Peretz BA, Itskovitz J, Brandes JM. Criteria for transvaginal sonographic diagnosis of ectopic pregnancy. *J Clin Ultrasound* 1990;18:274–279.
  61. Mateer JR, Valley VT, Aiman EJ, Phelan MB, Thoma ME, Kefer MP. Outcome analysis of a protocol including bedside endovaginal sonography in patients at risk for ectopic pregnancy. *Ann Emerg Med* 1996;27:283–289.
  62. Cacciatore G, Stenman UH, Ylostalo P. Diagnosis of ectopic pregnancy by vaginal ultrasonography in combination with a discriminatory serum B-hCG level of 1000 IU/L. *Br J Obstet Gynaecol* 1990;97:904–908.
  63. Emerson DS, Cartier MS, Altieri LA et al. Diagnostic efficacy of endovaginal color Doppler flow imaging in an ectopic pregnancy screening program. *Radiology* 1992;183:413–420.
  64. Pellerito JS, Taylor KJW, Quedens-Case C, et al. Ectopic pregnancy: Evaluation with endovaginal color flow imaging. *Radiology* 1992;183:407–411.
  65. Barnhart KT, Simhan H, Kamelle SA. Diagnostic accuracy of ultrasound above and below the beta hCG discriminatory zone. *Obstet Gynecol* 1999;94:583–587.
  66. Spandorfer SD, Menzin AW, Barnhart KT, Livolsi VA, Pfeifer SM. Gynecology: Efficacy of frozen-section evaluation of uterine curettings in the diagnosis of ectopic pregnancy. *Am J Obstet Gynecol* 1996;175:603–605.
  67. Barnhart KT, Gracia CG, Reindl B, Wheeler JE. Usefulness of Pipelle endometrial biopsy in the diagnosis of women at risk for ectopic pregnancy. *Am J Obstet Gynecol* 2003;188:906–909.
  68. Barnhart KT, Katz I, Hummel A, Gracia CR. Presumed diagnosis of ectopic pregnancy. *Obstet Gynecol* 2002;100:505–510.
  69. Ailawadi M., Lorch SA, Barnhart KT. Cost-effectiveness of presumptively medically treating women at risk for ectopic pregnancy compared with first performing a dilatation and curettage. *Fertil Steril* 2005;83:376–382.
  70. Brumstead J, Kessler C, Gibson D, et al. A comparison of laparoscopy and laparotomy for the treatment of ectopic pregnancy. *Obstet Gynecol* 1988;71:889–892.
  71. Vermesh M, Silva PD, Rosen GF, et al. Management of unruptured ectopic gestation by linear salpingostomy: A prospective randomized clinical trial of laparoscopy versus laparotomy. *Obstet Gynecol* 1989;73:400–404.

72. Lundorff P, Hahlin M, Kallfelt B, et al. Laparoscopic surgery in tubal pregnancy: A randomized trial versus laparotomy. *Acta Obstet Gynecol Scand* 1992;70:343–348.
73. Seifer DB, Gutmann JN, Doyle MB, Jones EE, Diamond MP, DeCherney AH. Persistent ectopic pregnancy following laparoscopic linear salpingostomy. *Obstet Gynecol* 1990;76:1121–1125.
74. Seifer DB, Diamond MP, DeCherney AH. Persistent ectopic pregnancy. *Obstet Gynecol Clin North Am* 1991;18:153–159.
75. Seifer DB, Gutmann JN, Grant WD, Kamps CA, DeCherney AH. Comparison of persistent ectopic after laparoscopic salpingostomy versus salpingostomy at laparotomy for ectopic pregnancy. *Obstet Gynecol* 1993;81:378–382.
76. Gracia CG, Brown MD, Barnhart KT. Prophylactic methotrexate after linear salpingostomy: A decision analysis. *Fertil Steril* 2001;76:1191–1195.
77. Hajenius PJ, Mol BW, Ankum WM, van der Veen F, Bossuyt PM, Lammes FB. Clearance curves of serum human chorionic gonadotropin for the diagnosis of persistent trophoblast. *Hum Reprod* 1995;10:683–687.
78. Spandorfer SD, Sawin SW, Benjamin I, Barnhart KT. Postoperative day 1 serum human chorionic gonadotropin level as a predictor of persistent ectopic pregnancy after conservative surgical management. *Fertil Steril* 1997;68:430–434.
79. Graczykowski JW, Mishelle DR. Methotrexate prophylaxis for persistent ectopic pregnancy after conservative treatment by salpingostomy. *Obstet Gynecol* 1997;89:118–122.
80. Tulandi T, Saleh A. Surgical management of ectopic pregnancy. *Clin Obstet Gynecol* 1999;42:31–38.
81. Ory SJ, Nnandi E, Herrmann R, O'Brien PS, Melton LJ. Fertility after ectopic pregnancy. *Fertil Steril* 1993;60:231–235.
82. Tuomivaara L, Kauppila A. Radical or conservative surgery for ectopic pregnancy? A follow-up study of fertility of 323 patients. *Fertil Steril* 1988;50:580–583.
83. Seifer DB, Silva PD, Grainger DA, Barber SR, Grant WD, Gutmann JN. Reproductive potential after treatment for persistent ectopic pregnancy. *Fertil Steril* 1994;62:194–196.
84. Hajenius PJ, Mol BW, Bossuyt PM, Ankum WM, Van Der Veen F. Interventions for tubal ectopic pregnancy. *Cochrane Database Syst Rev*:CD00324, 2000.
85. Lipscomb GH, Stovall TG, Ling FW. Nonsurgical treatment of ectopic pregnancy. *N Engl J Med* 2000;343:1325–1329.
86. Practice Bulletin No 3.: Medical Management of Tubal Pregnancy. Washington, DC, American College of Obstetricians and Gynecologists, 1998.
87. Libscomb GH, et al. Predictors of success of methotrexate treatment in women with tubal ectopic pregnancy. *N Engl J Med* 1999;341:1974–1978.
88. Barnhart KT, Gosman G, Ashby R, Sammel M. The medical management of ectopic pregnancy: A meta-analysis comparing “single dose” and “multidose” regimens. *Obstet Gynecol* 2003;101:778–784.
89. Isaacs JD, McGehee RP, Cowan BD. Life-threatening neutropenia following methotrexate treatment of ectopic pregnancy: A report of two cases. *Obstet Gynecol* 1996;88:694–696.
90. Gervaise A, Masson L, de Tayrac R, Frydman R, Fernandez H. Reproductive outcome after methotrexate treatment of tubal pregnancies. *Fertil Steril* 2004;82:304–308.
91. Oloffson JI, Poromaa IS, Ottander U, Kjellberg L, Damber MG. Clinical and pregnancy outcome following ectopic pregnancy: A prospective study comparing expectancy, surgery and systemic methotrexate treatment. *Acta Obstet Gynecol Scand* 2001;80:744–749.
92. Shalev E, Peleg D, Tsabari A, Romano S, Bustan M. Spontaneous resolution of ectopic pregnancy tubal pregnancy: Natural history. *Fertil Steril* 1995;63:15–19.
93. Doubilet PM, Benson CB, Frates MC, Ginsberg E. Sonographically guided minimally invasive treatment of unusual ectopic pregnancies. *J Ultrasound Med* 2004;23:359–370.
94. Strohmmer H, Obruca A, Lehner R, Egarter C, Huslein P, Feichtinger W. Successful treatment of a heterotopic pregnancy by sonographically guided instillation of hyperosmolar glucose. *Fertil Steril* 1998;69:149–151.
95. Lau S, Tulandi T. Conservative medical and surgical management of interstitial ectopic pregnancy. *Fertil Steril* 1999;72:207–215.
96. Boyko TR, O'Brien JF. Cervical pregnancy: A case report. *Ann Emerg Med* 2001;38:177–180.
97. Jabara S, Barnhart KT. Is Rh immune globulin needed in early first-trimester abortion? A review. *Am J Obstet Gynecol* 2003;188:623–627.



98. Practice Bulletin No. 4: Prevention of Rh(D) Alloimmunization. Washington, DC, American College of Obstetricians and Gynecologists, 1999.
99. Gerton GL, Fan XJ, Sammel M, Hummel A, Strauss JF, Barnhart K. A serum proteomics approach to the diagnosis of ectopic pregnancy. *Ann NY Acad Sci* 2004;1022:306–316.
100. Agarwal SK, Wisot AL, Garzo G, Meldrum DR. Cornual pregnancies in patients with prior salpingectomy undergoing in vitro fertilization and embryo transfer. *Fertil Steril* 1996 Mar;65(3): 659–60.

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### Munir A. Nazir

*When women are with child, the mouth of the womb is closed.*

Hippocrates (c. 460–377 B.C.E.)

F. Adams (Frans)

*Hippocratic Writings*

Aphorisms

Chicago: William Benton, 1952, I, 51, P9 139

The cervix, or the most distal portion of the uterus, serves as its functional sphincter. The cervix must be permeable to allow conception, and, when the gestation is complete, allow for dilatation and extrusion of the uterine contents. In some women the cervix shortens and dilates prematurely, unable to retain the intrauterine pregnancy, causing early pregnancy loss or midtrimester delivery. Cervical insufficiency can exist from either congenital or acquired cervical weakness allowing recurrent midtrimester losses. This condition was in the past termed *cervical incompetence*. Owing to the unfortunate associations with this terminology, the term *cervical insufficiency* is preferred and is used in this text [93].

The difficulty is the many borderline or complicated cases in which the conditions of cervical dysfunction versus the early and inappropriate onset of labor cannot easily be differentiated, especially by retrospective record review. In fact, almost uniformly, a woman has to experience pregnancies with early losses for which good records are available before she can be clearly identified as having either cervical insufficiency or premature labor. Placement of a cervical cerclage is the standard surgical therapy for cervical structural weakness. For recurrent preterm labor and for short cervix other modalities are being investigated, such as the use of prophylactic progesterone either in suppository form or intramuscular injection.

The measurement of cervical length and other features of cervical anatomy by transvaginal ultrasound scanning is now common in the assessment of the risk of preterm delivery and for the diagnosis of cervical insufficiency. There are a number of clinical indications for such testing, including women suspected of cervical insufficiency, those carrying a multiple gestation, or women with a history of preterm labor/delivery [20,25–26,28,70–73,128]. Unfortunately, there are varying criteria for the identification and screening of at-risk pregnancies. There are also different measures of success for medical and surgical interventions when cervical weakness or insufficiency is suspected [80]. This complicates literature reviews. Since ultrasound

scanning is often a major factor in the performance of cervical cerclage, and as the criteria for normality/abnormality are not well established, it is not surprising that the available clinical trials reveal contradictory results concerning the efficacy of prophylactic and emergency cerclage procedures, except for select subgroups of high-risk women [48–50, 81–85].

The association between objectively measured cervical length and an increased risk for preterm delivery has been extensively studied in recent years [8,20,24–38,46–53,56,69,72,84,86,117,125–130]. These studies have investigated ultrasonically demonstrated cervical anatomy as risk factors for early delivery. Further, after the identification of pregnancies at risk for cervical insufficiency, both cerclage and other means of treatment have been tested in randomized trials with varying degree of success.

The problem has long been proper identification of a true at-risk population and understanding the relationship between any observed cervical changes and pregnancy loss or preterm delivery. In theory, a congenital or acquired cervical weakness (incompetence/insufficiency) and premature labor are separate clinical conditions; however, they do overlap considerably in presentation and perhaps in etiology. Differentiating between these conditions at the time a specific case is first encountered clinically can be difficult or even impossible.

This chapter reviews the problem of cervical change and cervical insufficiency as related to preterm delivery. Recommendations for surveillance and best practice are made, and the principal surgical procedures for cervical reinforcement (cerclage) are discussed and critiqued.

## HISTORICAL BACKGROUND

Cervical weakness as a condition leading to pregnancy loss was first noted by Rivi re in 1658 when he described “Slackness of the orifice of the womb,” a condition suspected to contribute to barrenness in women [1]. Further observations on the “weak cervix” were made more than 200 years later, in 1865, by Gream [2]. He suggested that dilatation or division of the cervix might result in the physical inability of the uterus to retain the products of conception until term. Emmet first performed tra-

chelorrhaphy in 1862, but his experience was not published until a dozen years later [3]. In 1902, Herman [4] presented results of trachelorrhaphy procedures performed on three women with recurrent losses, two of whom later had successful pregnancies. In 1948, Palmer and Lacomme [5] introduced the phrase *la beance de l'orifice interne* (“the gaping internal os”), and described an operation for the surgical repair of this condition. In 1950, Lash and Lash [6] described “the incompetent internal os of the cervix” and proposed a type of surgical repair now known as the Lash procedure. All of these initial operations were performed on nonpregnant women.

The first successful cervical suturing procedure intended for performance during pregnancy was introduced by Shirodkar in 1955 [7]. A simplified purse-string suturing technique that later became extremely popular was described by McDonald in 1957 [116]. Abdominal cerclage was introduced in the 1960s as an additional technique, reserved for unusual or selected cases, especially when there had been marked cervical injury or a prior excision [53]. Many variations on the surgical techniques described in these original articles have since been reported. Many clinicians, the authors included, have incorporated elements of both the classic Shirodkar and McDonald operations into unique cerclage operations.

## PATHOPHYSIOLOGY

The cervix principally consists of fibrous tissues including collagen and elastin [10,134]. The smooth muscle content is approximately 10% to 15%. The cervical matrix is composed of dermatan sulfate-glycosaminoglycans and hyaluronic acid. Complex hormonally mediated changes in the biochemistry of the fibrous tissue are believed to be responsible for the process of cervical softening and both preterm and term cervical dilatation. As pregnancy progresses, under the influence of proteinases, hyaluronidases, prostaglandins, and various cytokines, the hyaluronic acid content of the cervical matrix increases while the level of dermatan declines. Prostaglandin E<sub>2</sub> and F<sub>2</sub> assist cervical change by activating collagenases. Release of these local hormones is a final pathway leading to cervical shortening and dilatation. Under their effect,

collagen fibrils progressively denature, absorbing fluid. The net result of these processes is a softening of the cervix prior to eventual labor. A variety of causes can initiate this process of cervical change, including hormonal, inflammatory, or infectious factors. Localized hemorrhage and chronic bleeding can also initiate this cascade [9].

The anatomy of the cervix, especially the fibrous-to-muscular-tissue transition at the cervico-isthmic junction, is an area of particular interest. The percentage of smooth muscle content in the lower third of uterus is 29%, but this rapidly declines to approximately 10% by the upper portion of the cervix. The length of the transition from uterus to cervix varies between 1 mm and 10 mm (the isthmus forms the lower uterine segment in pregnancy).

It is unclear which proportion of the cervical closure mechanism is due to the muscle tone in the isthmic and upper cervical tissues, and which results from the resistance of the predominantly fibrous structures of the lower cervix. Because the isthmus is formed by the fifth month of pregnancy, and labor is associated with both a decline in collagen concentration and a dissociation of collagen fibrils in the cervix, cervical resistance and not isthmic tone is the best explanation for the mechanism that holds the cervix closed.

More than one factor is probably involved in the cervical changes that result in a structurally weak cervix, predisposing to premature cervical thinning or dilatation. Cervical insufficiency might reflect anatomic variation with an increased muscle or a reduced collagen content of the cervix. Alternatively, early and inappropriate activation of the endogenous mechanism for cervical ripening could be the trigger for cervical weakening. It seems likely that both factors are present in many cases, with a wide range of individual variation.

### **Congenital and Acquired Cervical Insufficiency**

Congenital causes for cervical shortening/dilation include Müllerian anomalies and in-utero exposure to diethylstilbestrol (DES). In DES-exposed patients, clinically observed cervical change is common and there is an increased rate of second-trimester losses [11,12]. In a prospective study involving a group of 63 DES-exposed women reported by Ludmir and coworkers [13], 44%

required emergency cerclage. Prophylactic cerclage in *unselected* DES patients has heretofore failed to show a clear improvement in outcome, however. The problem in DES cases is that the drug-induced abnormalities involve much more than a simple change in cervical anatomy or resistance. Hormonally induced abnormalities in uterine shape and function are also common and predispose to early delivery. These conditions are not remedied by simple cervical reinforcement by a cerclage.

Acquired cervical lesions leading to insufficiency or preterm deliveries are due to trauma from various obstetric maloccurrences or gynecologic interventions. Cervical lacerations from difficult or instrumental deliveries occur mainly in the lateral part of the cervix and occasionally extend into the internal os. In many cases involving laceration, if the injuries are recognized and adequately repaired, subsequent cervical insufficiency is uncommon. However, some lacerations avoid detection and some repairs prove ineffectual. This emphasizes the importance of routine postdelivery examinations of the birth canal, with close attention to the repair of significant lacerations. Despite past concerns, first-trimester pregnancy terminations performed by modern methods of vacuum aspiration result in no consistently demonstrated increased risk for cervical insufficiency or preterm delivery [17,87].

There are conflicting data regarding risks of pregnancy complications as a result of indicated surgical procedures on the cervix [14–16,18,67,68], Carbon dioxide laser or cold-knife conization has been reported to increase the risk of preterm delivery in some studies, but the data on recurrent second-trimester losses are mixed [14–16,18]. Large-loop excision of the cervical transformation zone probably increases the risk of preterm delivery [68]. A history of such procedures is inconclusive as a risk factor when second-trimester losses are considered, however [136]. Nonetheless, the literature suggests that the greater the mass of excised tissue during the conization, the greater the risk for cervical dysfunction. Cervical cone-biopsy specimens longer than 20 mm or more than 4 cm<sup>3</sup> in total bulk, or carbon dioxide conizations longer than 10 mm are associated with an increased risk of preterm delivery [18].

In sum, there probably is an increased risk of preterm delivery following significant cervical trauma or the excision of cervical tissue. There is

continued controversy over whether such injuries result in an increased risk of second-trimester losses from cervical weakness, and if there is a benefit to cervical cerclage in these women. As noted, a major problem in evaluating published data is the uncertainty concerning which portion(s) of the cervix have been either excised or injured.

## INCIDENCE

The incidence of cerclage operations is difficult to estimate accurately. Composite data from both the United States and Europe suggest that cerclage operations are performed in approximately 5 per 1,000 births [86]. The criteria to establish the diagnosis of cervical insufficiency and to choose cases for surgery are neither consistent nor well established. Thus, there are wide differences between institutions and practitioners in the frequency with which such surgeries are performed. With the common use of ultrasound today, it is estimated that many more cerclages are performed based on the length of the cervix alone. Although there are little data to support this practice, it is a commonplace occurrence.

## EPIDEMIOLOGY

Despite major advances in medical knowledge, and the common use of vaginal sonography, prophylactic cerclage, and several intense remedial programs for identification of at-risk pregnancies, neither the rate of preterm births nor the incidence of first fetal losses from cervical insufficiency have appreciably declined in recent decades. The number of preterm births resulting from cervical weakness is unknown; however, it is quite clear that preterm birth is not prevented by cerclage alone and there are many other important factors.

Early delivery remains a major clinical problem. The incidence of preterm birth (before 37 weeks) is between 7% and 11%; however, preterm birth contributes up to 75% of the perinatal mortality rate. The risk is especially high for births before 28 weeks. Preterm delivery is also frequently repeated. In otherwise unselected cases of women with a history of preterm delivery, the recurrence risk is 10% to 35% [86].

Earlier studies of cerclage (prior to ultrasound measurement of the cervix) using women as their

own historical controls yielded reported success rates of 75% to 90% when apparently high-risk cases were studied [21]. What makes the role of cerclage difficult to evaluate is that over 70% of unselected women with prior losses will have a successful pregnancy without cerclage [33]. The question, therefore, is proper case selection.

Studies of the efficacy of cerclage versus no cerclage have yielded conflicting results. This is partly because of the variation in the definition of the cervical abnormality used as the entry criterion in various investigations, the different parity of the women studied, and the inclusion or exclusion of women with various high-risk factors for preterm birth in the specific study groups. The 2003 Cochrane review included seven randomized studies and found no conclusive evidence for a reduction in second-trimester loss, preterm delivery, or any improvement in morbidity associated with preterm births in women undergoing cerclage [37]. The Fetal Medicine Foundation Second-Trimester Screening Group [38] also undertook a multicenter randomized controlled trial of 47,123 women identified as having a short cervix. The cervix was found to be  $\leq 15$  mm short in 470 women on routine vaginal sonography performed between 22 and 24 weeks. A group of 253 of these women participated in the study and were subsequently randomized to cerclage versus expectant management [34,38]. The percentage of preterm delivery before 33 weeks was similar in both groups, 22% in the cerclage group versus 26% in the control group (relative risk 0.84, 95% CI 0.54–1.31). There were also no differences in perinatal or maternal morbidity or mortality between the two groups.

As an additional example of the current literature, the Medical Research Council/Royal College of Obstetricians and Gynaecologists multicenter trial (MRC trial) reported no improvement in perinatal survival in a cohort of women randomized to cerclage [34]. To interpret the MRC trial, as with the many others concerning cerclage/cervical insufficiency, one must be aware of the details of how the investigation was conducted. In this trial, the cerclage group included both prophylactic and therapeutic cerclages performed at a wide range of gestational ages. Furthermore, in some cases the original diagnosis of cervical insufficiency was considered uncertain. For these reasons, the significance of this

randomized trial, in terms of whether to perform a cerclage in more carefully selected women, is questionable [28].

Recent randomized clinical trials that reached different conclusions emphasize the importance of case choice. Althuisius and coworkers [35,130] enrolled women with a previous history of preterm delivery before 34 weeks. Some women with other risk factors for preterm delivery were also included. A total of 35 patients were initially randomized either to primary cerclage or no cerclage. Serial cervical length measurements by vaginal sonography were continued in the remaining test population. A second randomization to cerclage or no cerclage was performed if the cervical length was observed to shorten to  $\leq 25$  mm. The mean gestational age at delivery in women undergoing cerclage was  $37.9 \pm 1.2$  weeks. Furthermore, there were no deliveries before 34 weeks in this group. In contrast, the mean gestational at delivery in the noncerclage group was  $33.1 \pm 6.4$  weeks, with 44% these women delivering before the 34th week. Although the sample size was small, these results were statistically significant and demonstrate the potential benefits of cerclage in a properly selected group of very-high-risk patients defined by both prior early delivery and demonstrated cervical shortening.

A recent meta-analysis of randomized clinical trials by Berghella and coworkers evaluated the role of cerclage in the prevention of preterm birth in women with a short cervix by using individual patient data [38]. Four randomized trials were included. In the total population, preterm birth at  $<35$  weeks of gestation occurred in 29.2% (89/305) of the cerclage group, as compared with 34.8% (105/302) of the no-cerclage group (RR 0.84, 95% CI 0.67–1.06). There was, however, a significant reduction in preterm birth at  $<35$  weeks in the cerclage group in the following categories: singleton gestations (RR 0.74, 95% CI 0.57–0.96), singleton gestations with a history of prior preterm birth (RR 0.61, 95% CI 0.40–0.92), and singleton gestations and a history of prior second-trimester loss (RR 0.57, 95% CI 0.33–0.99). There was a significant increase in preterm births at  $<35$  weeks with twin gestation in the cerclage-treated group (RR 2.15, 95% CI 1.15–4.01). The authors reasonably concluded that cerclage does not prevent preterm birth

in all patients with a sonographically measured short cervix.

These studies have one clinical point in common; that is, the placement of the cerclage was intended for prevention of premature delivery, not specifically for correction of presumed, isolated cervical insufficiency. Most of these cerclages were placed in the late second or early third trimester (20–22 weeks). Based on these data, it seems probable that when there is a history of prior preterm births, cerclage *could* reduce the incidence of a recurrence in women who are observed to have a short cervix. Cerclage has also been recommended as a treatment for placenta previa, a therapy that has failed to gain popularity as a method of extending the period of gestation [135].

## DIAGNOSIS

The diagnosis of cervical insufficiency is difficult, because there is no specific or scientifically validated confirmatory test [37,38,80,86,128]. The diagnosis could be suggested initially by the obstetric history if there has been a prior preterm delivery. Unfortunately, nulliparous women who are incidentally found to have a short cervix or who present with advanced cervical dilatation and bulging membranes will lack such a prior obstetric history. When their clinical history is reviewed, women with insufficiency frequently deny perceiving strong contractions prior to their diagnosis/delivery and often complain only of mild cramping, spotting, or heavy vaginal discharge, or there may have been increased urinary frequency and urgency. They are often seen in the late or early third trimester with both advanced cervical dilatation and bulging of the membranes, either at the external os or prolapsing into the vagina. Second-trimester premature membrane rupture is also common. At the time of the initial presentation, the fetus is usually alive and these women are commonly afebrile. Subsequently, the fetus is either born alive only to succumb to prematurity or dies during labor and delivery. On review of the antenatal record and the necropsy data there is no evidence of an in-utero growth disturbance, and the pathologist identifies no specific findings, with the exception of histologic evidence of chorioamnionitis or occasionally funisitis.

Several techniques to diagnose cervical weakness in either nonpregnant or pregnant women are described in the literature. As an example, if there is easy passage of No. 8 Hegar or No. 15 Pratt dilators through the internal os, or there is absence of "snapping closure" of the internal os while the physician is removing the dilator, cervical insufficiency has been reported to be suspect [76]. The pressure/volume characteristics of an inflated endocervical balloon have also been investigated [75]. Hysterosalpingography can reveal unusual widening of the internal os, also suggesting the correct diagnosis [74]. These radiographic or ultrasound data are often difficult to interpret except when the defect is marked, however. Such studies can have other benefits, however. Both hysterosalpingography and sonohysteroscopy provide information regarding abnormalities of the uterine cavity. Structural uterine abnormalities of potential obstetric consequence might include Müllerian anomalies (septate, arcuate, or bicornuate uteri), intrauterine adhesions, polyps, or submucous myomas, among other findings. Despite the efforts of several investigations, there is no established preoperative test or evaluation for cervical insufficiency that is scientifically reliable in confirming the diagnosis of cervical insufficiency.

### **ROLE OF SONOGRAPHY**

Endovaginal sonography is the best method for the evaluation of women at risk for preterm delivery or cervical insufficiency during pregnancy. Such cervical measurements have been shown to be both reliable and reproducible when compared with digital examinations and traditional abdominal sonography [8,24,30,131]. Older clinical studies conflict to some extent with more recent ultrasound findings. Specifically, there is often a poor relationship between the findings on clinical examination of a short cervix and the concomitant ultrasound measurements. These data emphasize the importance of objective study when evaluations of the cervix are performed to determine whether to place a cerclage or not.

### **Ultrasonic Measurement of Cervical Length**

Transperineal and transvaginal ultrasound examinations are the principal methods for objective mea-

surement of cervical length. Although less ideal, transperineal ultrasonography can be used for measurement of cervical length if there are ruptured amniotic membranes or if the patient wishes to avoid a vaginal study [131]. Image clarity is much superior with an endovaginal probe; however, the transperineal technique might not result in useful measurements due to difficulties in adequate visualizations.

When transvaginal studies are performed good technique is important to obtain proper images and avoid patient discomfort. The maternal urinary bladder should be empty. A distended urinary bladder can artificially close or lengthen the observed or measured cervical canal.

The endovaginal probe best images the cervix from the anterior fornix, with the patient positioned in dorsal lithotomy. The operator should avoid pressing too firmly on the anterior fornix with the transducer because this can artificially lengthen the cervix and can be perceived as uncomfortable. The best technique is to first gently advance the probe until it abuts against the cervix. The transducer is then pulled slightly back until the image blurs; then it is again advanced a very short distance. This should result in a clear image; the measurements are then made [8,24–26,79]. The image is frozen at a level depicting the internal os, cervical canal, and the external os. The authors routinely obtain a number of images. The shortest length recorded on the best image is reported as the final value.

To evaluate for funneling or dynamic changes, fundal pressure is applied or the woman is requested to perform a Valsalva maneuver [27]. If funneling is observed, the cervical length measurement is repeated from the apex of the funnel to the external os. Best management of cases where the initial cervical length is normal but dynamic changes occur either spontaneously or following Valsalva is not clear.

### **SIGNIFICANCE OF VAGINAL SONOGRAPHY**

There is an inverse relationship between the ultrasound-determined length of the closed cervix and the risk of preterm birth. In general, the shorter the closed cervical length the higher the risk is for preterm delivery [20,25,26,29,31,32,38,39,78,92,129]. Cervical length serves as an independent

marker for the prediction of preterm birth in both low- and high-risk patients. Between 18 and 28 weeks, the mean cervical length in pregnancy delivery at term is approximately 40 mm. This measurement is independent of parity [77,78]. Thereafter, the cervical length slowly decreases after 28 weeks, to an average of approximately 33 mm. At the critical juncture of 20 to 28 weeks, the 25th percentile for cervical length is approximately 30 mm; the 10th percentile is 25 mm [23,77,86].

As previously discussed, several studies randomizing "short-cervix" pregnancies to bed rest or to cerclage have shown no improvement in the gestational age at delivery; however, other studies with more restrictive inclusion criteria, and excluding women with other established high-risk factors for premature delivery, showed definite gains in the number of days to delivery in the cerclage group. Case choice is critical.

The risk of preterm birth when a short cervix is present is well established. The proportion of preterm births due to true cervical insufficiency and those women likely to benefit from cerclage in women found to have a short cervix; however, is unknown. Because the discriminating power of current tests is limited and the available treatments are at best imperfect, routine vaginal sonography for measurement of cervical length in unselected otherwise low-risk women is of uncertain value.

## **POTENTIAL COMPLICATIONS AND GENERAL MANAGEMENT**

Placement of a cerclage is not without risk. The dangers of cerclage fall into two major categories [86]. First, there are the risks inherent in surgery, discussed in greater detail later. Second, there is the risk inherent in an erroneous diagnosis of cervical insufficiency leading to the performance of a procedure when none is indicated. Experience, proper training, and meticulous attention to detail reduce the surgical risks. The errors in the choice of cases for surgery are much more difficult to remedy, however, due to the limitations of current methods of diagnosis.

The major postoperative complications of cerclage include infection; delayed membrane rupture; and tearing, loosening, or displacement of the suture. Hemorrhage is a more serious risk in trans-

abdominal cerclages, where control is potentially more difficult. Among the standard transvaginal techniques, bleeding is more common with the Shirodkar than with the McDonald operations owing to the greater dissection required with the former procedure. In surgeries performed in non-pregnant women when vaginal/cervical incisions are performed, blood loss is reduced by the initial injection of dilute solutions of epinephrine or phenylephrine. The intraoperative use of such agents during pregnancy is, however, not recommended. Cervical manipulation is associated both with uterine irritability and premature rupture of the membranes when advanced cervical dilatation is present and the membranes are exposed. The likelihood of spontaneous cerclage displacement is reported in 3% to 13% of elective procedures [21]. Suture displacement or development of a cervical laceration or fenestration can require a repeat operation, although the efficacy of repeat suturing is unclear. If the suture has slipped, cut through, or is observed hanging loosely, the management options principally include prompt placement of another McDonald or Shirodkar suture or conservative, nonsurgical treatment with restricted activity and observation. The results of resuturing are often far from satisfactory, and treatment failure with subsequent premature delivery is a common event. Rescue with pessary placement (Smith-Hodges) to support the cervix is an old technique that is occasionally effective when there is a loosened or ineffective suture. After placing the pessary, an endovaginal ultrasound is performed. If the pessary is effective, the membrane prolapse resolves and the cervix appears closed. The pessary can then be left in place until delivery, with a weekly program of removal, cleaning, and replacement. A properly sized pessary should not cause irritation or be uncomfortable (if so a smaller size should be inserted), and should also allow for digital or ultrasound examinations. The likelihood for serious complications following cerclage is approximately 2%. The principal intraoperative problems include inability to perform the procedure, the induction of additional cervical trauma, bleeding, the occurrence of uterine contractions, and rupture of membranes [21]. Cervical manipulations are associated both with uterine irritability and a heightened risk of premature rupture of the membranes when advanced cervical dilatation is present and the membranes exposed.



Bleeding or uterine contractions can be due to potentially serious obstetric complications such as placental separation or chorioamnionitis or labor. Active premature labor is a potential indication for tocolysis and might require removal of the suture. Chorioamnionitis is most common in cerclages performed after the 19th week, perhaps because of the greater area of exposure of the membranes. The diagnosis of intrauterine infection is often difficult and requires a high index of suspicion since chorioamnionitis can be delayed, developing up to 4 weeks postoperatively. Especially in later operations, occult infection might have preceded the cerclage placement, emphasizing the importance of case selection. Serial white blood counts, careful palpation for increasing uterine tenderness, close attention to patterns of uterine irritability, and observation of maternal vital signs are among the clinical findings suggesting the diagnosis. In selected cases, amniocentesis is performed for special testing.

The small study by Mays and coworkers [96] emphasizes the importance of occult intrauterine infections as a cause of prematurity and cerclage failure. In Mays' report, amniocentesis was performed in a series of pregnant woman who were candidates for rescue cerclage (cervix,  $\leq 2$  cm, with 50% effacement and visible membranes). The markers used in this study for infection were amniotic fluid glucose levels of 14 mg/dl or less or a lactic dehydrogenase (LDH) level of more than 400 IU, or both [94,95]. Cerclage was withheld if such markers were present. When these markers were present all the women either aborted or delivered prematurely within three days. All placentas from these pregnancies revealed histologic evidence of infection. Of interest, in only a minority of cases were amniotic fluid Gram stains or bacterial cultures positive, indicating the limitations of these traditional tests. Finally, all of the placentas from these preterm deliveries were also culture positive for *Candida* species.

Although this was not a randomized study, it does support other work suggesting intrauterine infection as an important risk factor for both preterm delivery and neonatal or fetal loss [94–97]. These data have led some to recommend routine amniocentesis for cases under consideration for late urgent or emergency cerclage.

Delayed rupture of the membranes is suggestive of an occult amnionitis. How best to manage these cases when the cerclage remains in place in the absence of overt signs and symptoms of infection is controversial [110–115]. In the authors' opinion on rupture of the membranes, patients with a cerclage should be managed in the same manner as others with preterm premature rupture of the membranes who lack a cerclage. Literature support for either retention or removal of the suture post-membrane rupture can be found. Our recommended treatment is the administration of wide-spectrum antibiotics, covering aerobic and anaerobic bacteria as well as *Chlamydia*, followed by close observation. We do not routinely remove the cerclage. Amniocentesis for fluid analysis and culture, if technically possible, can aid management. If clear signs and symptoms consistent with chorioamnionitis are identified, however, the cerclage should be removed and delivery accomplished without delay, regardless of the period of gestation.

Late and uncommon complications of cerclage include fistula formation and, rarely, cervical stenosis. Cicatrix formation can result in cervical dystocia in labor (conglutination of the cervical os) or eventuate in deep cervical lacerations at delivery, which can extend into the broad ligament. Potential fetal complications associated with cerclage include prematurity, sepsis, and intrauterine demise. Other rare problems associated with cerclage include uterine rupture or endotoxic shock. Most rarely, maternal death has been reported [28].

How best to treat women postoperatively who have had a cerclage has never been systematically studied. Maternal activity, the administration of tocolytic agents, and prophylactic antibiotics are the principal issues. Intraoperatively, broad-spectrum antibiotics are commonly administered, especially for procedures performed at  $\geq 16$  weeks or when the membranes are exposed. Although there are virtually no data on this point, such treatment is ubiquitous and has not been shown to be detrimental [47,88]. Whether it is helpful, however, has not been established. Immediate postoperative bed rest or restricted activity lasting up to 1 week is a common recommendation. Many clinicians also advise some restriction of maternal activity for the remainder of the pregnancy, following the initial

postoperative rest period. It is to be emphasized that these recommendations are made refreshingly free of any data.

In our management protocol, tocolytic agents usually are not routinely administered but remain reserved for cases involving emergency cerclage procedures in which a membrane bulge is observed, or in perioperative period when uterine irritability is either documented or is perceived by the mother. There are no good data on long-term tocolytic use. As noted, we favor the use of tocolytic drugs only for documented episodes of uterine activity or for very-high-risk cases. Usually the episodes of uterine irritability cluster around the time of surgery or soon thereafter. Subsequent episodes of uterine activity are managed expectantly with tocolytics administered only for short courses at the discretion of the attending physician. In the authors' practice, post-cerclage women are seen on a 1- to 2-week basis for the remainder of the pregnancy, with the scheduling depending primarily on the perceived precariousness of the cerclage. For follow-up, both the location of the cerclage and any changes in cervical length after surgery can be easily demonstrated by postoperative transvaginal sonography [89–91]. Although the initial increase in cervical length resulting from a cerclage is not predictive of pregnancy outcome, overall progressive postoperative shortening apparently is [92]. For this reason, a program of serial transvaginal studies with cervical length measurements can help to predict pregnancies likely to deliver early despite the cerclage. Thus alerted, the clinician could potentially modify maternal activity, perform fetal fibronectin studies, or choose to administer steroids or, presumably, parenteral 17-OH progesterone.

### SURGICAL PROCEDURES

Cervical cerclages are best classified based on their timing and the anatomic approach taken for the repair. In terms of timing, these procedures are considered as *elective*, *urgent*, or *emergent*. The current approach to the placement of cerclage is most often transvaginal, and most procedures are performed during pregnancy. For select indications, a transabdominal cerclage is placed either at laparotomy in the first trimester or, occasionally, by laparoscopy in the nonpregnant woman.

### ELECTIVE CERCLAGE

An *elective cerclage* is a cervical suture that is usually inserted in late first or early second trimester, before the 16th week of gestation. Such procedures are sometimes categorized as *prophylactic cerclages*. Classically, the cerclage is placed before the gestational age at which the patient's prior loss occurred. The procedure is performed only after confirmation of fetal viability and after the exclusion of fetal anomalies, by ultrasonic evaluation and other testing. Women who are identified early in pregnancy as strong cerclage candidates should be offered first-trimester nuchal lucency and serum biochemical testing (i.e., HCG, PAPP-A).

In an individual case, if a firm diagnosis of cervical insufficiency has not been established, elective cerclage as a primary procedure is best replaced by a program of serial cervical measurements by vaginal sonography. In this plan, a cerclage is performed only if there is demonstrated cervical shortening (see Urgent Cerclage section). Several longitudinal studies indicate that a management protocol of serial sonographic cervical assessment, followed by cervical cerclage restricted to women found to have a short cervix, does not compromise efficacy and could reduce the incidence of surgical intervention [43,99–101]. Primary elective cerclage *without* ultrasonic demonstration of cervical change can be considered in women with a history of three or more second-trimester losses, known anatomic defects that involve or are close to the internal os, or the rare patient who for any reason cannot or will not undergo serial cervical measurements.

### URGENT CERCLAGE

An *urgent cerclage* is a therapeutic cerclage procedure performed when the cervix is observed to have shortened to a critical level. Depending on the surgeon, this is usually  $\leq 15$  mm to 25 mm in the interval of 16 to 23 weeks. In such cases, the membranes are not exposed to the vagina. There are no significant differences in pregnancy outcome between elective and urgent cerclages [45,46]. This more conservative use of cerclage with initial documentation of cervical shortening is now considered the preferred management for women who do not have a clear and strong history of cervical insufficiency.

## EMERGENCY CERCLAGE

When the cervix dilates and the membranes funnel through the internal and external os, the risk of pregnancy loss from spontaneous membrane rupture is high. A “rescue” cerclage can then be attempted if there is no clinical evidence of infection, the membranes are intact, and the fetus is alive and ultrasonically normal. Surgical procedures to reinforce and close the cervix under these circumstances are termed *emergency cerclages*.

### Techniques Unique to Emergency Cerclage

Several methods have been described to reduce bulging membranes to permit safe placement of the cervical suture [58–60,106–108]. Use of a Foley catheter with inflation of the balloon, following the removal of the distal end flush with the bulb, has been described [57,105]. This technique can be sufficient to hold back the membranes when the cervical dilatation is not advanced. The catheter is passed through the cervix and then inflated. The bulb holds the membranes upward and away from the internal os while the cerclage is performed. The bulb is subsequently deflated and then removed as the suture is tied (Figure 5.1). Tsatsaris and coworkers [106] have also described the use of a modified balloon originally designed for endoscopic surgery to reduce membranes. In their trials, this technique was successful in 24 of 25 cases. Olatunbosun and Dyck [58] developed an unusual technique in which six to ten stay sutures were applied at the edges of the cervix. Traction on the sutures and patient repositioning permitted the membranes to slip back into the uterine cavity, permitting a cerclage to be performed. Goodlin [59] and Locatelli [107] suggested

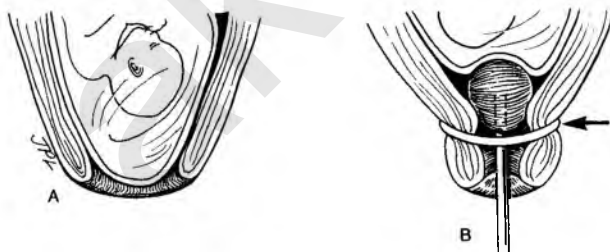


FIGURE 5.1. *A, cervical insufficiency with advanced cervical dilation and membrane prolapse; B, Membrane displacement by use of a Foley catheter (Orr technique) during cervical cerclage. (See text for details.)*

transabdominal amniocentesis as a method to temporarily reduce amniotic fluid volume to assist in replacement of the membranes. This is not necessarily an easy technique. If amniocentesis is performed, real-time ultrasound guidance is required, and the removal of volumes of fluid in excess of 150 ml to 200 ml can be required to cause the membranes to recede [108]. This process may initiate uterine contractions and should not be performed without tocolytics. Scheerer and coworkers [60] described the technique of urinary bladder distension. Up to 1,000 ml of 0.45% saline was installed by catheter as a noninvasive method to displace the membranes upward. This technique has long been in clinical practice. Finally, Olatunbosun has also described the simple and common technique of using a surgical sponge on a forceps to simply displace the membranes upward [58].

When cerclage operations are attempted in the face of advanced cervical dilatation, most clinicians employ one or more of these techniques. The maneuvers chosen are based on the unique features of the case, the maternal anatomy, and the preference and experience of the surgeon. Placing the patient, with a full urinary bladder, in steep Trendelenburg’s position, using a Foley balloon to reduce membranes, and applying gentle traction on the cervix with ring forceps or Allis clamps (being careful not to grasp the membranes) are among the authors’ recommended techniques.

### RECOMMENDED PRACTICE

Although all choices are controversial and the literature recommendations are inconsistent, there are data to suggest that in selected cases, performance of a cerclage improve perinatal outcomes. The following outline indicates the authors’ current clinical practice, attempting to offer surgery when it could prove helpful while avoiding intervention where success is unlikely.

Emergency cerclage cases require immediate evaluation. The clinician must restrict surgery to cases likely to benefit (Figure 5.2). The therapeutic aim is to gain sufficient time so that the pregnancy can extend into the period of potential viability ( $\geq 24$ –25 weeks). Many of the acutely presenting cases are not candidates for suturing, however, either owing to an advanced gestational age, the presence of recurrent contractions, or a suspicion of infection

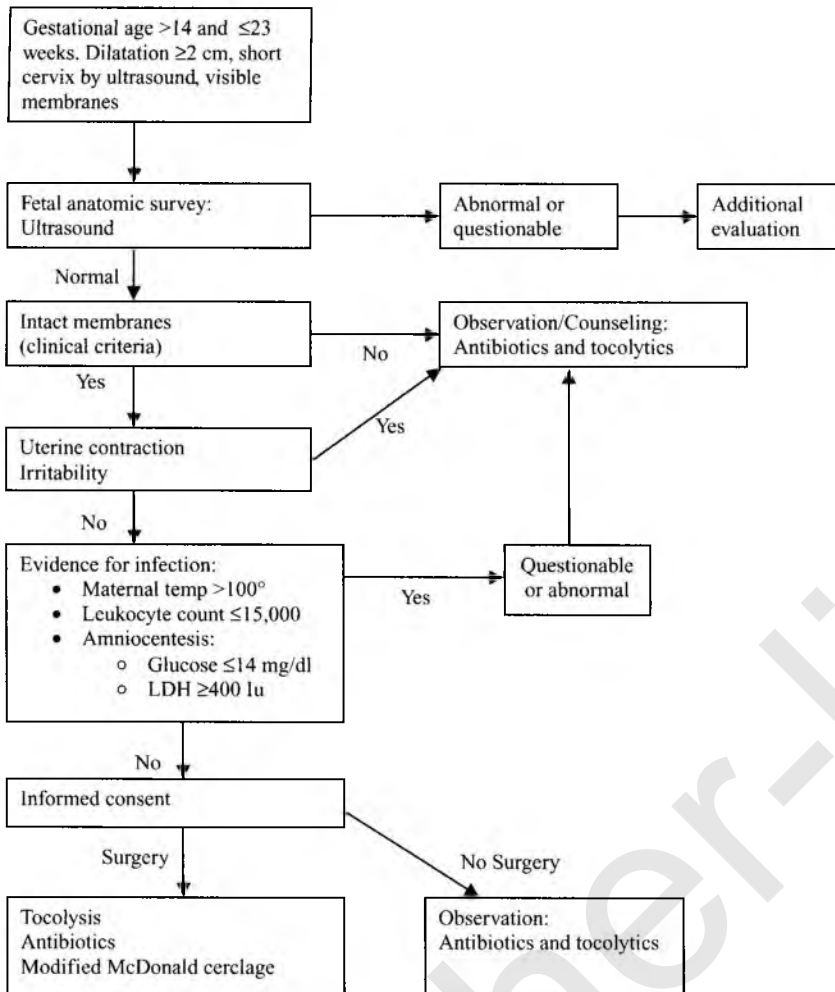


FIGURE 5.2.  
*Evaluation for emergency cerclage.*

or abruptio placentae. These cases are best followed without surgery and treated with antibiotics, tocolytic agents, or delivery, whichever is clinically appropriate.

Elective cerclages have become increasingly uncommon in the authors' practice (Figure 5.3). The clearest cases have a history of two or more suspicious prior losses and evidence of a cervical abnormality (i.e., anatomic distortion, progressive shortening). These cases require an initial screening evaluation of the fetus by both ultrasound scan and the new biochemical methods, to avoid surgery on abnormal pregnancies. For these procedures, insertion of the cerclage occurs no later than the 16th week. Retrospective care analysis fails to demonstrate an advantage to either the Shirodkar or McDonald technique [22,23]. Thus the authors' preference is a modified version of the

latter because it is faster and requires less surgical intervention.

The largest number of cases in the authors' practice involves women with either incomplete data concerning their prior pregnancy losses or with an atypical presentation for insufficiency (Figure 5.4). These cases are followed serially, with intervention occurring only if cervical change has been documented.

A relatively common problem is the management of the situation in which the woman has had prior successful pregnancy with a cerclage and requests another in her current gestation. In a good number of these cases, when critically reviewed, the original diagnosis is shaky. The authors do vary their approach in the management of these cases. While most go on to repeat surgery, others are followed serially with the intention of later urgent cerclage

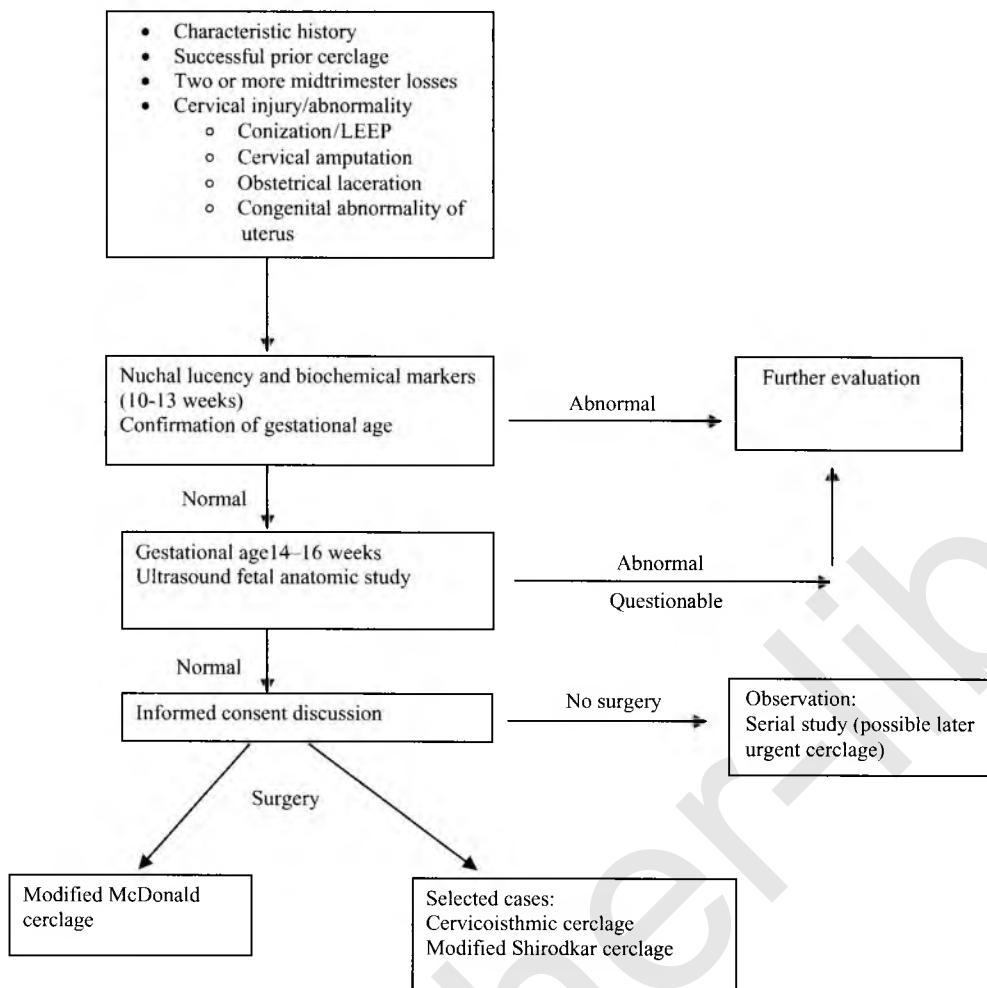


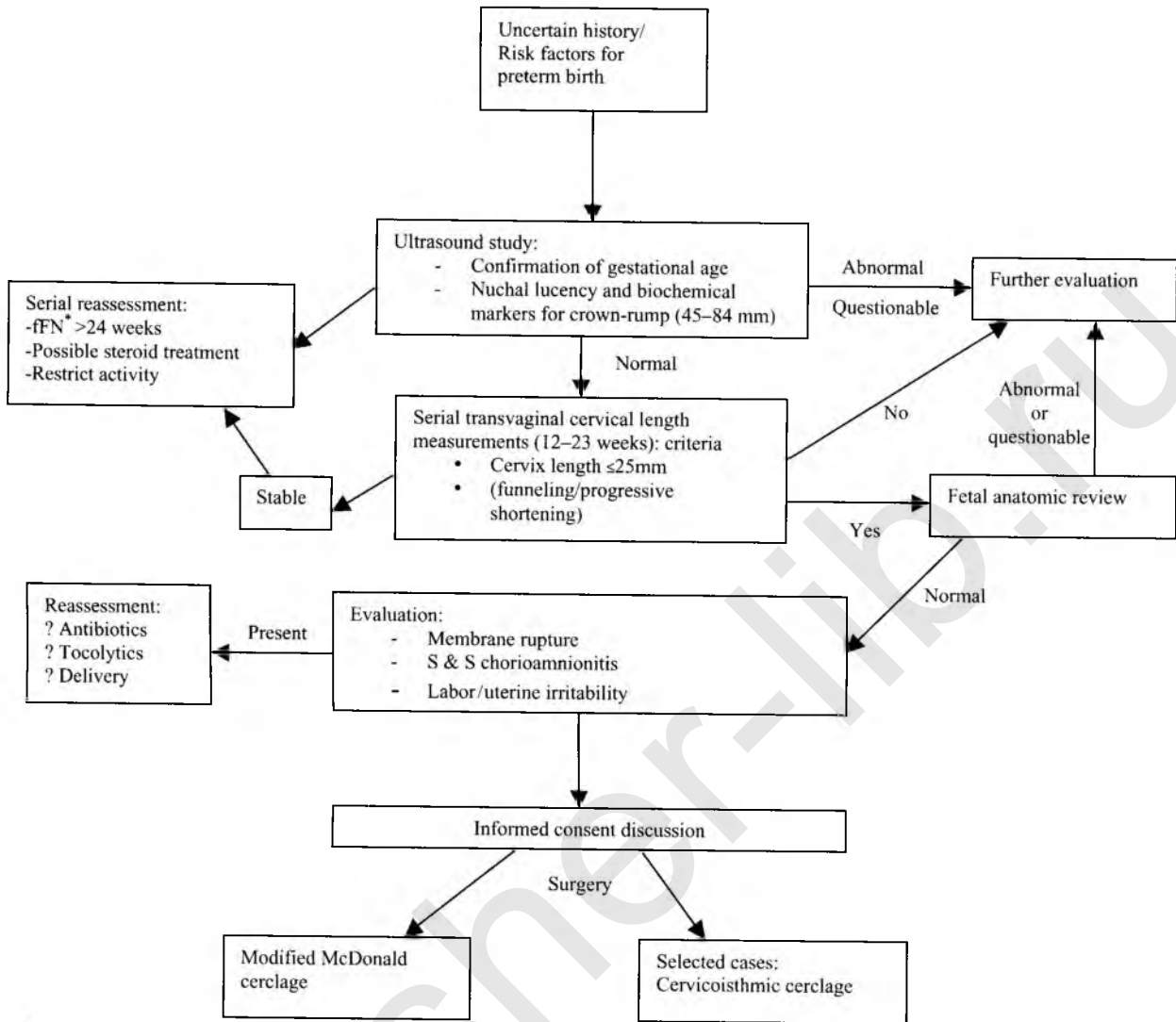
FIGURE 5.3.  
*Evaluation for elective  
cerclage.*

placement should it become necessary. In recent years, the authors also have inserted several cervicoisthmic sutures by laparoscopy in nonpregnant patients, attempting to reduce overall morbidity. There is no simple formula for proper management in these complex situations, and ultimately, care must be individualized.

The possible benefit of an emergency cerclage has been studied in both retrospective studies and prospective trials comparing a program of cerclage and bed rest in women with advanced cervical dilatation and exposed membranes versus bed rest alone [47-50,102-104]. As an example, Olatunbosun and coworkers [49] performed a prospective study involving 45 patients with a mean cervical dilation of 6 cm. This was not a randomized trial, and management was at the discretion of patient's physician. Nonetheless, the mean gestational age at eventual delivery in the cerclage group was  $33.0 \pm$

$4.4$  weeks versus  $28.8 \pm$  weeks in the group managed by bed rest alone. This difference is statistically significant ( $p = 0.001$ ). Althuisius and coworkers [50] randomized 23 patients with advanced cervical dilatation and what was considered a risk of imminent delivery to either cerclage or bedrest. Randomization was performed on average at 23 to 24 weeks' gestation. All patients remained in the hospital until 30 weeks and all received initial antibiotic treatment. The mean interval between the gestational age at randomization and delivery was 54 days for the emergent-cerclage group versus a mean of 24 days in the bedrest-alone group. This was again a statistically significant finding ( $p = 0.046$ ). It should be noted that one patient in the emergent cerclage group ruptured membranes during the procedure, leading to its abandonment.

What is best management? The upper limit for a rescue cerclage is considered to be 23 to 24 weeks.



\*fFN = fetal fibronectin

FIGURE 5.4.  
*Evaluation for urgent cerclage.*

If the gestational age is >24 weeks, the risk of the procedure in terms of risking membrane integrity and predisposing to early delivery or inciting labor probably exceeds the benefit to the surgery. Further, a fair number of these late-appearing cases involve women in premature labor and some have established or suspected infection, both contraindications to surgery. Based on available studies, however, there remains a limited role for emergent cerclage. The specific setting is advanced cervical dilation with exposed membranes but in the absence of labor, and when clinical evidence of infection is not present. The reported series are small, however, and the likelihood of prematurity is extremely high. Given

the limitations in the data and the difficulties in diagnosis, the majority of such late presenting cases are still best managed by bed rest, steroids, occasional use of tocolytics, and observation. Again, given the difficulty in establishing best practice and in recognition of the high risk for early delivery and subsequent neonatal complications, individualization of case management is suggested.

### General Aspects of Surgical Procedures

The Shirodkar and McDonald operations are the most frequently performed transvaginal surgical techniques for cervical insufficiency. They are

generally considered equal in their success rates, and most clinicians consider them essentially equivalent in efficacy [21,22]. There are no randomized clinical trials directly comparing the efficacy and safety of these two procedures, however. The Shirodkar procedure requires more extensive surgery because it requires dissection of the vaginal epithelium with advancing of the bladder base, and thus is performed less frequently. Proponents for the Shirodkar procedure, often argue that the supportive suture is positioned closer to the anatomic internal os than is possible for the McDonald. Although this might be true, no difference in outcome between the procedures has been demonstrated. Thus, this potential benefit is of uncertain importance unless there is a laceration that extends to or through the internal os. Conversely, the McDonald cerclage technique is easy to master and is suitable for most cases when there is an anatomically identified and structurally intact cervix.

Ultimately, the choice of the procedure to be performed and the suture material employed is by physician's preference. For routine cerclage procedures, the authors favor the McDonald operation because of its simplicity and speed. The type of suture used is largely inconsequential as long as permanent synthetic sutures are employed. In terms of management, a successful cerclage is usually removed around 37 weeks or earlier if active labor commences. Any cerclage procedure is contraindicated if labor bleeding, known or suspected chorioamnionitis, or ruptured membranes are present.

The Trendelenburg position before and during vaginal procedures is recommended. If the membranes are hour-glassing in the vagina and there is little fluid remaining around the fetus, success is unlikely and the procedure should not be attempted. If there is a membrane bulge, one of the standard techniques for membrane displacement is employed. It is best to first attempt bladder overfilling, combined with Trendelenburg's positioning, because this is the easiest and least invasive procedure. If these simple maneuvers are to no avail, balloon use, direct membrane displacement, or amniocentesis with fluid aspiration are potential additional procedures to consider in order of invasiveness or difficulty.

The efficacy of tocolytic therapy, duration of hospitalization, and the use and choice of antibiotic

therapy have not been prospectively studied. These aspects of management are left to the physician's discretion. For procedures after 14 to 16 weeks or if the membranes are exposed, the authors favor the administration of a first-generation broad-spectrum antibiotic with the addition of azythromycin to cover possible infection with chlamydial/ureaplasma.

Tocolytic agents are not recommended for routine use in cerclage operations unless uterine irritability is present, the membranes bulge beyond the external os, or therapeutic amniocentesis is performed to facilitate the cerclage placement.

The degree of how aggressive to be in the exclusion of intrauterine infection has not been established. An accurate diagnosis of amnionitis, especially in emergency cerclage procedures, is difficult. This condition is commonly occult, and the mother can show few, if any, clinical signs or symptoms of infection, beyond the observed cervical shortening [96,97]. Physical examination, determination of vital signs, and a maternal white blood cell and differential count are minimal requirements. If uterine activity is present, initial tocolysis and a period of observation are best while options are being considered. Amniocentesis for a Gram stain, determination of the glucose level, and other tests such as an amniotic fluid lactate dehydrogenase (LDH), in addition to the evaluation of clinical signs of infection can be performed and are prudent if an emergency cerclage beyond 22 or 23 weeks is considered [94–95,97]. Best management in this setting is not established. In the authors' experience and extrapolating from the extant literature, the greater the suspicion of infection, the more atypical the presentation, and the more advanced the dilatation and effacement, the stronger the case is for invasive testing, and the poorer the likelihood for cerclage success, and the less likely to care is appropriate for intervention.

Management of cases if spontaneous rupture of membranes occurs while a cerclage is in place is unsettled. As previously reviewed, the available studies provide no consistent findings to aid the clinician [110,112–115]. As is too often true in complex clinical situations, the available studies include small numbers and the results are diametrically opposed. These data preclude rational analysis and probably represent features unique to the specific patient population studied. As a practical matter, the authors administer antibiotics

when early membrane rupture occurs, regardless of the presence of a cerclage. We do not routinely remove a cerclage following spontaneous rupture unless sufficient uterine activity is present so as to threaten cervical injury, there are signs and symptoms of chorioamnionitis, or significant bleeding is observed.

## STANDARD CERCLAGE OPERATIONS

### **Shirodkar Cerclage**

Shirodkar first published a description of his operation in 1955 [7]. The *Shirodkar operation* was the first procedure intended to be performed on pregnant rather than nonpregnant women with cervical insufficiency. Shirodkar subsequently described several modifications of this procedure, including the use of various suture materials, and several different methods for tying, burying, or exposing the knot anteriorly or posteriorly. The procedure was initially proposed as a permanent method of cervical repair with subsequent cesarean delivery.

Shirodkar's operation is an open technique, requiring incisions in the cervix, as well as mucosal dissection. This procedure, which is technically more complex and invasive than the McDonald operation, is most often reserved for patients with a short cervix. A prior Shirodkar cerclage, a high cervical laceration involving the internal os, or previous failed McDonald cerclage are additional potential indications for either a primary or repeat Shirodkar procedure.

There are numerous modifications to the Shirodkar operation. Placement of a band or strip of fascia lata (autograft) with an aneurysm needle, as originally described by Shirodkar, is no longer performed. A synthetic (Mersilene) 5-mm tape with needles wedged on each end to facilitate placement is now the most popular suture material. Vaginal incisions can involve substantial blood loss. For this reason, many surgeons either limit or entirely avoid the posterior cervical incision that was part of the original operation.

If permanently epithelialized, a properly placed Shirodkar suture can be left in place and elective cesarean delivery performed without impairing future fertility. If the knot is exposed, however, most clinicians remove the suture at or near term, regardless of the final mode of delivery.

### **Procedure**

Currently, a modified Shirodkar technique is favored. Either a large polyester fiber (Tevdek 11, #9) or a 5-mm woven polyester fiber tape (Mersilene) band suture mounted on an atraumatic needle is employed. As traditionally conducted, the procedure requires an initial anterior transverse incision at the cervicovaginal junction (Figure 5.5). The bladder flap is then advanced above the level of the internal cervical os by blunt and sharp dissection. A second vertical (or horizontal) incision is sometimes made in the posterior cervix at the same level. The suture is inserted as close to the internal os as possible and then tied with multiple square knots. The knot is usually left exposed posteriorly to facilitate later removal; however, some clinicians prefer to position the knot anteriorly. Electively, the knot is buried under the cervical epithelium. If this is the surgeon's intention, after tying, the suture is cut short. The ends are then sutured down with a fine permanent synthetic suture, either to the band itself or to adjacent cervical tissues. Any cervical incisions are closed with a simple running suture, the type determined by the clinician's discretion.

### **McDonald Cerclage**

The *McDonald procedure* was first described in 1957 [116]. In this simple operation, a simple purse-string suture is placed around the cervix as high as is technically possible (Figure 5.6). The basics of the McDonald procedure have been unchanged for many years, and multiple variations of the original technique exist. Whereas the McDonald procedure is generally simple to perform, extreme care is warranted if the cervix is markedly effaced or dilated at the time of surgery, because of the risk of membrane rupture.

Because no incisions are made in the cervix, the McDonald procedure is simple and usually rapid. At the surgeon's discretion, the suture can be placed entirely submucosally by using the same points for exit and reentry of the needle at each successive bite. The suture is removed at the onset of labor, or electively before labor, and is reinserted for subsequent pregnancies as required. McDonald originally performed the procedure between 20 and 24 weeks' gestation but later revised the timing to 14 weeks.



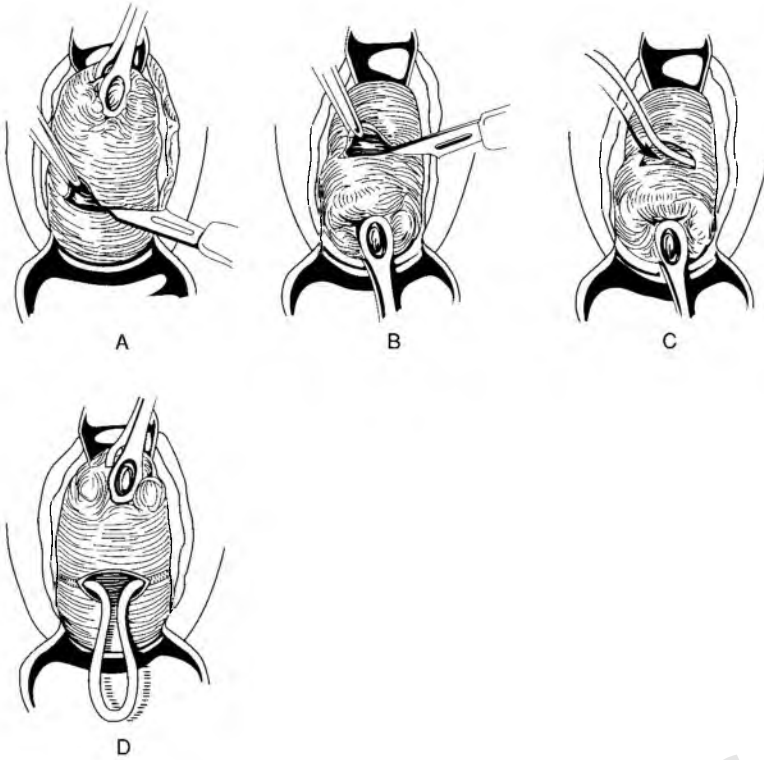


FIGURE 5.5.

*Shirodkar cervical cerclage procedure. A and B, Incisions are made in the cervix. C and D, A suture is placed and tied. E. Note that the cut ends are subsequently sutured down to the cervical band. Closure of the cervical incisions follows. (See text for details.)*

Double sutures are preferred by some surgeons for greater security or when a short or malshaped cervix precludes easy insertion of a high stitch. In this technique, an initial suture is inserted and tied. Traction is then applied to this suture, drawing the cervix firmly toward the perineum, permitting the insertion of a higher, second cerclage (see Figure 5.6E). There are no data supporting an advantage to single or double suturing or to any specific or suture material over another.

### Procedure

In the usual technique, four to six circumferential bites are taken in the substance of the cervix, usually beginning at the 12 o'clock position. If the bites are taken counterclockwise after the initial entry, subsequent needle insertions occur at the 10–11, 7–8, 4–6, and 1–2 o'clock positions. The knot is usually tied anteriorly, with the ends left long for ease in later removal. Several different suture materials can be used, including one of the nonabsorbable monofilaments, a thick braided polyester fiber suture (e.g., Tevdek #9) or a 5-mm polyester fiber band (Mersilene). Because monofilament sutures are difficult to

tie and can cut the cervix (especially if over tightened), they are less popular than other alternatives. Silk has been replaced by the modern hyporeactive synthetic suture materials and is specifically *not* recommended for use.

### Cervicoisthmus Cerclage

A *cervicoisthmus cerclage* (CIC) is placed higher than the usual McDonald or Shirodkar suture, at the level of the isthmus. This procedure can be performed abdominally (by laparotomy), laparoscopically, or infrequently vaginally. Most of the published experience is with the transabdominal technique (TACIC) [52–56,117–123]. These procedures are more challenging than either the McDonald or Shirodkar operations and thus are reserved for cases in which the cervix is extremely short, lacerated, or amputated. A previously failed transvaginal failed cerclage is another potential indication. These operations should not be attempted by a neophyte surgeon unless they operate under the direct supervision of a surgeon experienced in such procedures. Case selection is critical. This procedure is generally performed during pregnancy; the best timing is

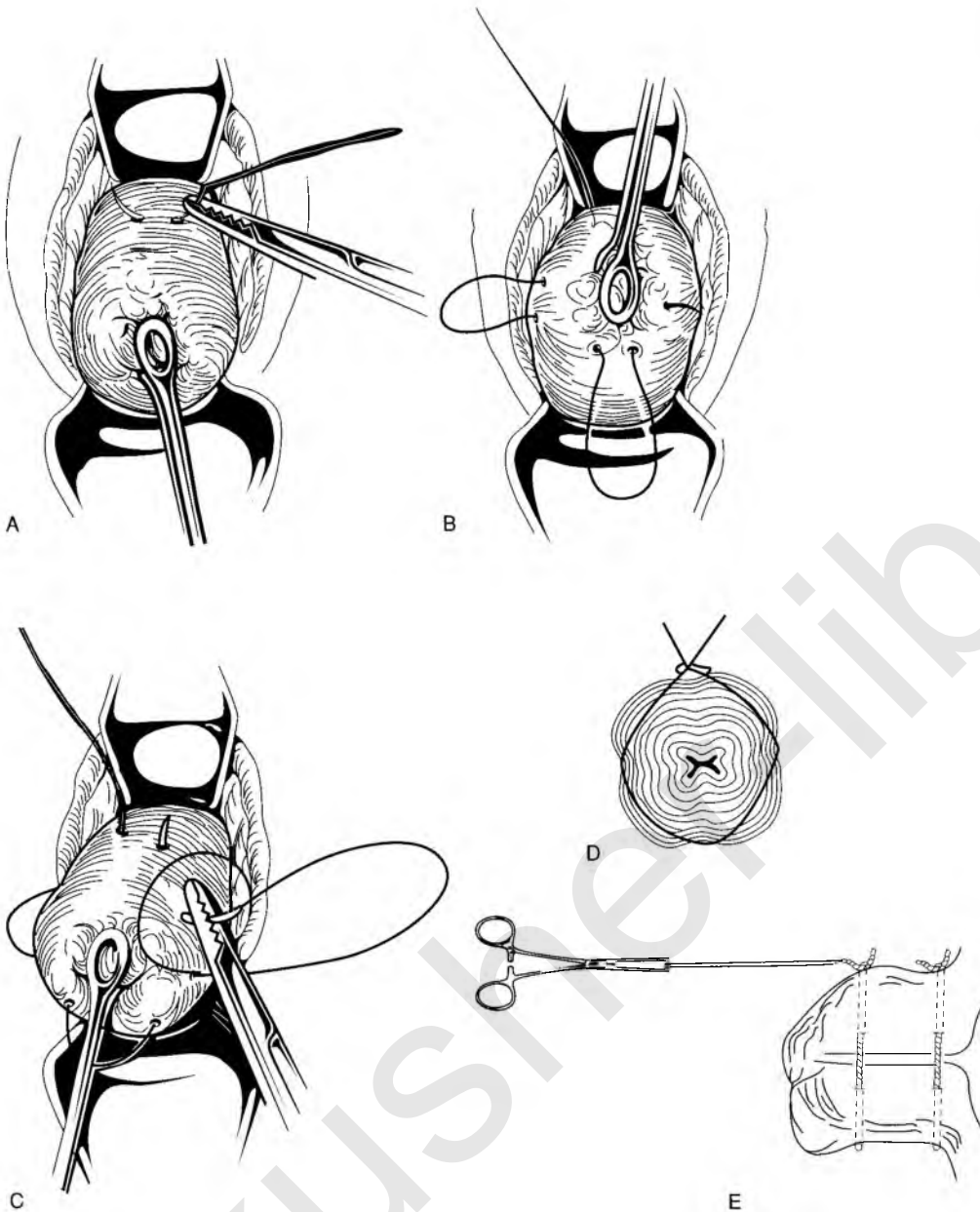


Figure 5.6.  
 McDonald cervical  
 cerclage. Note the  
 recurrent circumferential  
 suture bites (A–D). The  
 knot can be placed  
 anteriorly or posteriorly.  
 (E) Indicates technique  
 if a secured suture is  
 placed. (See text for  
 details.)

before 12 to 14 completed weeks to facilitate exposure. Occasionally, the surgery is performed laparoscopically in non-pregnant women.

### Procedure

For the transabdominal approach, for gestations before 14 weeks either a Pfannenstiel or Maylard skin incision is made. Vertical skin incisions can improve visualization for gestations over 14 weeks and are preferred by some surgeons for their more advanced cases [52]. The abdomen is entered in the

usual manner, exposing the uterus. With appropriate retraction, the vesicouterine fold is developed with bunt and sharp dissection, and the bladder is advanced. The uterine fundus is then elevated and deviated laterally by an assistant. If the uterus is irritable, tocolysis is advisable.

The surgical procedure as originally described by Benson and Durfee [53] involved the dissection and creation of a “tunnel” between the ascending and descending uterine vessels and the uterine isthmus as a site to insert the cerclage suture. This can prove difficult (Figure 5.7A). Major vessels are proximal

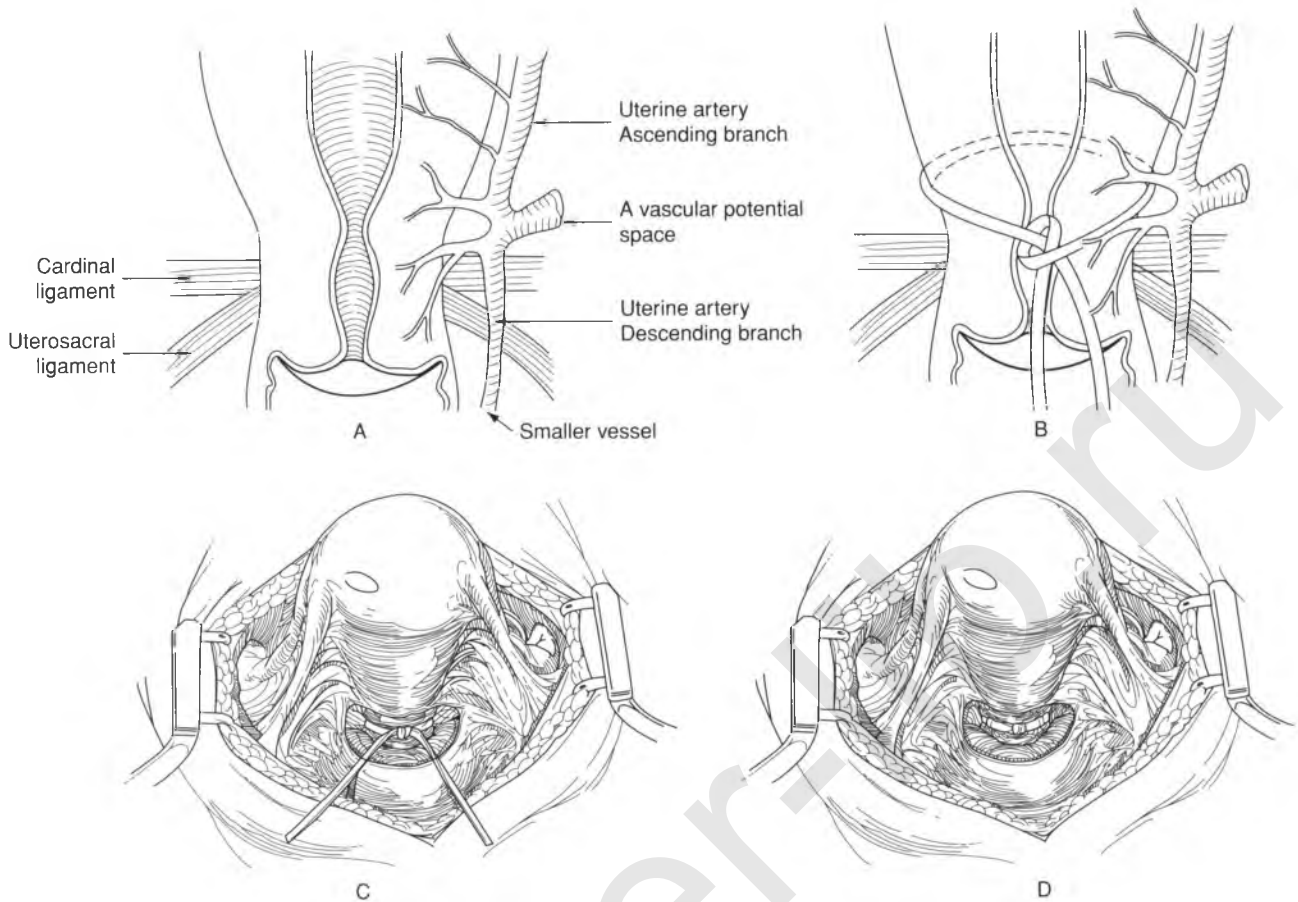


Figure 5.7.

*Cerclage suture is inserted through the potential space between the uterine artery and the myometrium. (A and B). The knot is placed anteriorly (occasionally posteriorly), and if a band is used, the cut edges are sutured down as indicated (C and D; see text for details.)*

to the potential space, and working room is limited. Other techniques help to avoid difficulty, if surgical exposure is difficult or a clear space is not easily demonstrated, the authors favor simply puncturing the wall of the myometrium just medial to the identified vessels to insert the suture. This modification does not seem to reduce the effectiveness of the repair but better avoids contact with the major vessels. Transillumination can also assist in locating an avascular area before needle insertion [123].

Once the correct site has been correctly identified, a lubricated 5-mm tape doubly loaded with CT-21 needles or another large-diameter suture (e.g., Tevdek #9) is inserted on each side between the uterine isthmus and the uterine vessels, from anterior to posterior (Figure 5.7B). If a tape is

used, it should be laid flat before tying the knot posteriorly (or anteriorly, if posterior exposure is limited).

In a late or difficult case, vaginal ultrasound scan can be used to confirm that neither the fetal parts nor the amniotic membrane are trapped below the cerclage. If they are at risk for entrapment, the uterine contents and fetal parts are gently “milked” superiorly before the suture is knotted (Figure 5.7C and D). Thereafter, the cut ends of the cerclage are usually sutured down using 00 or 000 Prolene or a similar permanent suture material. After the knot is fixed, the bladder flap is reapproximated anteriorly. The position of the cerclage is easily verified postoperatively by transvaginal ultrasound scanning. The stitch is usually left in place permanently for future

childbearing, as long as it is well epithelized and properly sited.

The TACIC procedure has also been performed by a laparoscopic approach [54,55]. Mingione and coworkers [55] reported 11 cases, employing a disposable laparoscopic suturing device to insert the suture (EndoClose, Tyco Health Care, Gasport, UK). The results included ten term live births, with one elective delivery at 34.5 weeks. This success rate is similar to that reported for the usual transabdominal approach. When considering laparoscopic cerclage, physicians should recall that the total number of reported cases is limited. If subsequent experience proves favorable, the laparoscopic approach could become the procedure of choice for TACIC operations performed on nonpregnant women. This approach avoids the morbidity and increased expense of the usual laparotomy and seems to result in equally effective anatomic repair as the traditional transabdominal approach.

Transvaginal placement has also been reported [56,138,139,140]. Katz and Abrahams [56] recently reviewed the pregnancy course and outcome in 56 pregnancies after transvaginal placement of a TACIC, using similar indications as for abdominal cerclage. There was 100% fetal survival. Preterm birth rate was 32%, with births  $\leq 30$  weeks occurring in 21% of the cases. In six gravidas, the suture was not removed, and three had subsequent pregnancies using the original suture. There were reported complications, however; these included an intraoperative bladder laceration and an intrapartum cervical tear.

#### **TRANSCERVICAL CERCLAGE: OTHER PROCEDURES**

*Trachelorrhaphy* was first described by Emmet as a specific treatment for high cervical lacerations [3]. The original procedure was intended to be performed while the patient was not pregnant and specified denudation of the cervical lesion – the tear – and the use of silver-wire sutures to close the deficit. The technique consisted of making a V-shaped incision and excising the scarred portion of the cervix. This resulted in two raw surfaces of full cervical thickness. These edges were then closed with interrupted chromic or polyglycolic acid suture. A 6-mm

cervical dilator was placed during the closure of the cervix to judge the degree of cervical tightening. Currently, this procedure is of historical interest only.

The *Lash operation*, published in 1950, is another cervical reinforcement technique intended for the nonpregnant state (Figure 5.8) [7]. The procedure is intended for an obviously traumatized cervix with an isolated defect or laceration, which can be demonstrated in the nonpregnant state. The Lash procedure is a permanent technique, and subsequent cesarean delivery is required. In the Lash procedure, a transverse incision is made through the anterior vaginal mucosa about 2 cm above the external os, and the bladder base is reflected. Scar tissue is excised, as required, and the edges are then freshened and reapproximated. The cervical defect is reapproximated with interrupted sutures, and the vaginal incision is closed. This operation has been replaced by the Shirodkar and McDonald procedures.

The *Mann cerclage* is another transvaginal cervicoisthmic technique also performed in the nonpregnant state [19]. In this procedure, an abnormally shortened or scarred cervix is dissected to enable suture placement at the level of internal os. A nonabsorbable suture is inserted, as in the Shirodkar technique. Unique to this procedure, the uterosacral ligaments and additional cervical tissue anteriorly and posteriorly are incorporated into the suture. A second suture is then placed 1 to 2 cm distal to the first.

The *Page "wrapping" technique* is also intended for the preconception period [62]. Sutures are placed deeply at the level of the internal os at 12, 4, and 8 o'clock, and a strip of gauze sprinkled with talc is positioned around them. This is meant to stimulate granulomatous fibroblastic proliferation and constrict the cervix at this level. This procedure is rarely attempted and is no longer performed.

The *Wurm technique* was developed in 1959 but not reported until 1961 [61]. Following the original description, a mattress suture of No. 3 heavy braided silk is inserted at the level of the internal os from 12 to 6 o'clock. A second similar suture is placed from 3 to 9 o'clock (Figure 5.9). This is a very quick and simple operation but is uncommonly performed, save in emergency cases where a previous McDonald suture has failed, the cervix

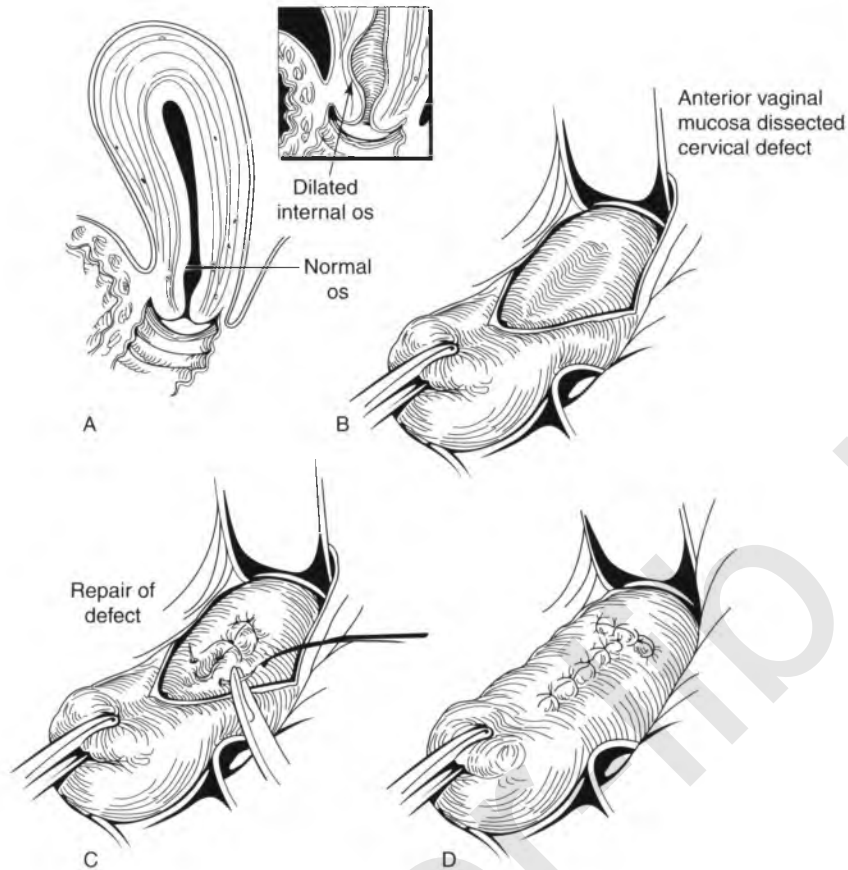


Figure 5.8.  
Lash procedure for repair of cervical insufficiency. (See text for details.)

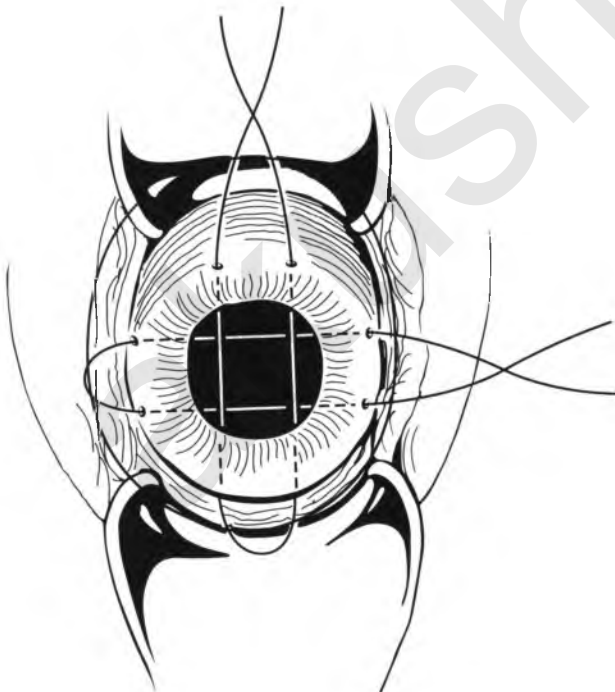


Figure 5.9.  
Wurm cervical cerclage procedure.

is effaced, and the membranes are at the external os. In the usual and less extreme cases, the Wurm technique has no specific advantage over the Shirodkar or McDonald procedures. This operation is prone to failure, especially when performed emergently.

#### PREFERRED CERCLAGE PROCEDURE

The technique for cerclage that the authors teach and normally employ is an amalgam of both the Shirodkar and McDonald procedures (Figure 5.10). This composite procedure is suited for cases in which significant cervical tissue remains and a substantial membrane bulge into the lower cervix is not present. No cervical incisions are routinely made, and the critical positioning of the cerclage suture in the cervix is directed by the initial placement of long curved Allis clamps. The authors do not use the traditional 5-mm band as we find this suture difficult to flatten and tie. Our preference instead is to insert



Figure 5.10.

*A, The proper site for insertion of the cerclage suture is chosen by the placement of a long, curved Allis clamp while the cervix is drawn laterally and down by an assistant. The position of the clamp tip determines the site of the suture placement. B, The cerclage suture is passed from anterior to posterior at the tip of the long, curved Allis clamp while assistants help with exposure, deviating the cervix laterally while also drawing it downward. Note that individual retractors are depicted rather than a self-retaining Guttman type. C, On the other side a similar procedure is performed, passing the cerclage suture from posterior to anterior at a site again determined by Allis clamp application. A small bite of posterior cervical tissue can be taken electively. D, A surgeon's knot is made anteriorly and tensioned until the operator's finger tip can just enter the reinforced endocervix. Three to four firmly applied square knots follow to secure the cerclage. (See text for details.)*

a large nonabsorbable suture (Tevdek #9) swedged to a blunt, curved needle. The specifics of the procedure are explained in detail here:

- Spinal or epidural anesthesia is employed unless precluded by the anesthesiologist.
- The surgeons operate standing with the patient positioned in the dorsal lithotomy position. Steep Trendelenburg's position is employed, as required.
- Both a dedicated surgical assistant and a scrub nurse are identified for the procedure because retraction for suture placement is critical to safety and success.
- Initially, a bimanual pelvic examination is performed to evaluate the cervical anatomy, membranes, and fetal position. A real-time ultrasound examination follows.
- After induction of anesthesia and correct patient positioning, a three-bladed Guttman-type self-retaining vaginal retractor is inserted. The surgeon should also have available selected narrow retractors because they might be required for adequate exposure if the self-retainer proves inadequate.
- At the beginning of the operation, the cervix is visualized and the retractor(s) correctly positioned. The anterior lip of the cervix is then grasped with a ring forceps, an Allis clamp, or a similar atraumatic instrument. The cervix is drawn outward and to one side, exposing its lateral aspect (Figure 5.10A). The corner of the cervix is next grasped with a long, straight Allis clamp, and the forceps holding the anterior lip is removed.
- As the assistants provide lateral and downward cervical traction, the surgeon positions a long, curved Allis clamp across the lateral side of the cervix, angling the handle of the clamp to the side, positioning the tip of the clamp to include a substantial amount of cervical tissue (see Figure 5.10B). Usually the bite into the cervix includes approximately one third or more of the overall width of the cervical tissue. Because the tip of the clamp marks the area where the cerclage suture will be placed, the positioning of the curved Allis clamp is the most important part of the operation.
- With site for suture placement chosen, #9 Tevdek (or other preferred suture) is positioned on a heavy Heaney needle holder and then passed straight down from anterior to posterior, just at the tip of the Allis clamp (Figure 5.10B).
- The suture is drawn through the tissue, and a small midline bite is made in the posterior cervix as high as is reasonably possible.
- The Allis clamps are then removed and attention directed to the other side. The other cervical angle is grasped with a straight Allis clamp in the same manner as the first and again drawn down and laterally by an assistant. A curved Allis clamp is applied in a similar fashion as was performed on the contralateral side. The suture is then driven through the cervix at the tip of the clamp, posterior to anterior. A small midline bite of anterior cervical tissue is usually made before the knot is tied (Figure 5.10C).
- A surgeon's knot is then placed and the knot snugged down until the operator's fingertip can just begin to enter the cervix. Several well-tensioned square knots are then added, securing the suture (Figure 5.10D).
- The suture ends are then grasped with a Kelly or similar clamp, and the cervix is drawn firmly outward. The surgeon palpates the cervix to judge the adequacy of the cerclage and considers if it is prudent or necessary to place an additional suture above the first (Figure 5.10E).
- The suture ends are cut long to facilitate eventual removal, and the instruments are removed from the vagina.
- Before the patient is removed from the table, a final real-time ultrasound examination verifies suture position and fetal cardiac motion and notes the amniotic fluid volume. This completes the surgical procedure.

#### ADDITIONAL COMMENTS

One of the most important and often difficult parts of vaginal cerclage procedures is adequate exposure. Two surgical assistants are often necessary even if self-retaining retractors are used. The authors' standard cerclage operating kit contains a large collection of retractors of various types, shapes, and

sizes. The type most useful for the specific case cannot be confidently identified in advance and is chosen intraoperatively. Furthermore, the authors tailor the cerclage procedure actually performed to the specifics of the maternal anatomy. Thus, when marked cervical effacement is present, a classic McDonald cerclage, with multiple small bites in the cervix, is generally best. In the unusual setting of an unrepaired cervical tear or an unusually short cervix, a modified Shirodkar procedure is sometimes indicated. Because of its substantial morbidity, abdominal cerclage is best reserved for women with little residual cervical tissue, a permanent injury including the internal os, or a history of prior failed cerclage procedures in previous pregnancies.

### NONSURGICAL TREATMENT

A pessary is sometimes an appropriate choice in a patient who refuses surgery or in women awaiting surgery while a cervical/vaginal infection is being treated [63,64,124]. Vitsky proposed the use of the Smith-Hodge pessary to alter the axis of the cervical canal [63]. In theory, this works by shifting the hydrostatic force of the amniotic sac posteriorly to the cul de sac. Oster and Javert later suggested that the pessary might act as a sling, preventing direct pressure from the fetal presenting part on the region of the internal os [64].

If employed, a pessary should be inserted at 12 to 14 weeks of gestation. The device is removed weekly for cleaning and clinical reassessment. It is left in place until about the 37th week. The device originally fitted might need to be replaced by a larger size as the pregnancy advances. The pessary technique has never gained great popularity and is not without complications. Because the pessary can induce a vaginal or bladder infection or become silently displaced, close clinical observation is necessary. Despite some favorable clinical experience in small uncontrolled studies, the efficacy of pessary use for cervical insufficiency has not been conclusively proven [124].

### CONCLUSION

The literature concerning cerclage and its indications is complex, contentious, and contradictory [44,128]. Cases for surgery should be carefully

chosen, and a conservative use of cerclage is best practice. Because of these inherent limitations, as part of the consent process, women believed to be cerclage candidates should understand the uncertainties and limitations of current methods of case identification and the potential risks of surgery versus no surgery in terms of pregnancy loss and preterm delivery.

There clearly is an association between early cervical shortening as identified by mid-trimester ultrasound scanning and preterm delivery. Unfortunately, in most cases the ultimate cause for early pregnancy cervical shortening is unknown. Possible contributing factors include prior cervical injury, occult infection, anatomic variations in uterine shape, and subclinical uterine contractions, or some combination of these events.

*The central problem for cerclage is properly identifying the population for whom the procedure is likely to provide benefit.* Although still controversial, the extant data can be fairly read to indicate a benefit to cerclage in selected high-risk pregnancies in which both a classic history is obtained and cervical shortening is documented [23,37,38,46,80,81,128]. Appropriate practice in the situation of the chance discovery of advanced cervical shortening in asymptomatic women, especially nulliparas, remains unclear. These cases require individualization of management.

Routine cerclage is avoided in twin gestations because it appears to increase rather than diminish the risk of prematurity. Emergency cerclages are problematic and are rarely attempted after the 24th week of gestation. Further, potential candidates for late emergent procedures should be screened for occult infection by amniocentesis, physical examination, and laboratory analysis.

Abdominal cerclage is reserved to experienced surgeons operating principally for documented cervical anatomic abnormalities under circumstances in which there has been a prior failed procedure of another type [137].

### REFERENCES

1. Rivière L, Culpeper N, Cole A, Rowland W. On barrenness. In *The Practice of Physick*, Book 15. London: Peter Cole, 1658;131, p. 916.
2. Gream GT. Dilatation or division of the cervix uteri. *Lancet*. 1865;1:381.



3. Emmet TA. Laceration of the cervix uteri as a frequent and unrecognized cause of disease. *Am J Obstet.* 1874;7:442.
4. Herman GE. Notes on Emmet operation as a prevention of abortion. *J Obstet Gynaecol Br Commonw.* 1902;2:256–257.
5. Palmer R, Lacomme M. La béance de l'orifice interne, cause d'avortements à répétition? Une observation de déchirure cervico-isthmique réparée chirurgicalement, avec gestation terme consécutive. [Is gaping of the internal cervical orifice a cause of reported abortion? An observation of surgical repair of cervico-isthmic gaping with subsequent term gestation] *Gynecol Obstet.* 1948;47:905–909.
6. Lash AF, Lash SR. Habitual abortion: The incompetent internal os of the cervix. *Am J Obstet Gynecol.* 1950 Jan; 59(1):68–76.
7. Shirodkar VN: A new method of operative treatment for habitual abortion in the second trimester of pregnancy. *Antisep.* 1955;52:299–300.
8. Iams JD, Johnson FF, Sonek JD, Sachs L, Gebauer C, Samuels P. Cervical competence as a continuum: A study of ultrasonographic cervical length and obstetric performance. *Am J Obstet Gynecol.* 1995 Apr; 172(4 Pt. 1):1097–1103, discussion 1104–1106.
9. Gilstrap LC 3rd, Cunningham FG, VanDorsten JP, editors. *Operative Obstetrics.* 2nd ed. New York: McGraw-Hill; 2002, pp. 503–522.
10. Danforth DN, Buckingham JC, Roddick JW Jr. Connective tissue changes incident to cervical effacement. *Am J Obstet Gynecol.* 1960 Nov;80:939–945.
11. Goldstein DP. Incompetent cervix in offspring exposed to diethylstilbestrol in utero. *Obstet Gynecol.* 1978 Jul;52(1 Suppl):73S–75S.
12. Cousins L, Karp W, Lacey C, Lucas WE. Reproductive outcome of women exposed to diethylstilbestrol in utero. *Obstet Gynecol.* 1980 Jul;56(1):70–76.
13. Ludmir J, Landon MB, Gabbe SG, Samuels P, Mennuti MT. Management of the diethylstilbestrol-exposed pregnant patient: A prospective study. *Am J Obstet Gynecol.* 1987 Sept;157(3):665–669.
14. Kristensen J, Langhoff-Roos J, Kristensen FB. Increased risk of preterm birth in women with cervical conization. *Obstet Gynecol.* 1993 Jun;81(6):1005–1008.
15. Moinian M, Andersch B. Does cervix conization increase the risk of complications in subsequent pregnancies? *Acta Obstet Gynecol Scand.* 1982;61(2):101–103.
16. Buller RE, Jones HW 3rd. Pregnancy following cervical conization. *Am J Obstet Gynecol.* 1982 Mar 1;142(1):506–512.
17. Daling JR, Emanuel I. Induced abortions and subsequent outcome of pregnancy in a series of American women. *N Engl J Med.* 1977 Dec 8;297(23):1241–1245.
18. Raio L, Ghezzi F, Diaro E, Gomez R, Luscher KP. Duration of pregnancy after carbon dioxide laser conization of the cervix: Influence of cone height. *Obstet Gynecol.* 1997;90(6):978–982.
19. Mann EC, McLarn WD, Hayt DB. The physiology and clinical significance of the uterine isthmus. I. The two-stage intrauterine balloon in the diagnosis and treatment of cervical incompetence. *Am J Obstet Gynecol.* 1961 Feb;81:209–222.
20. Iams JD, Goldenberg RL, Meis PJ, Mercer BM, Moawad A, Das A, Thom E, McNellis D, Copper RL, Johnson F, Roberts JM. The length of the cervix and the risk of spontaneous premature delivery. National Institute of Child Health and Human Development Maternal Fetal Medicine Unit Network. *N Engl J Med.* 1996 Feb;334(9):567–572.
21. Harger JH: Comparison of success and morbidity in cervical cerclage procedures. *Obstet Gynecol* 1980 Nov;56(5):543–548.
22. Odibo AO, Berghella V, To MS, Rust OA, Althuisius SM, Nicolaidis KH. Shirodkar versus McDonald cerclage for the prevention of preterm birth in women with short cervical length. *Am J Perinatol.* 2007 Jan;24(1): 55–60.
23. ACOG Practice Bulletin, Number 48. Cervical Insufficiency. *Obstet Gynecol.* 2003 Nov;102(5 Pt 1):1091–1099.
24. Sonek JD, Iams JD, Blumenfeld M, Johnson F, Landon M, Gabbe S. Measurement of cervical length in pregnancy: Comparison between vaginal ultrasonography and digital examination. *Obstet Gynecol.* 1990 Aug;76(2):172–175.
25. Heath VC, Southall TR, Souka AP, Elisseou A, Nicolaidis KH. Cervical length at 23 weeks of gestation: Prediction of spontaneous preterm delivery. *Ultrasound Obstet Gynecol.* 1998 Nov;12(5):312–317.
26. Oven J, Yost N, Berghella V, Thom E, Swain M, Dildy GA. Midtrimester endovaginal sonography in

- women at high risk for spontaneous preterm birth. *JAMA*. 2001 Sept 19;286(11):1340–1348.
27. Guzman ER, Rosenberg JC, Houlihan C, Ivan J, Waldron R, Knuppel R. A new method using vaginal ultrasound and transfundal pressure to evaluate the asymptomatic incompetent cervix. *Obstet Gynecol*. 1994 Jul;83(2):248–252.
  28. Althuisias S, Dekker G: Controversies regarding cervical incompetence, short cervix, and the need for cerclage. *Clin Perinatol*. 2004 Dec;31(4):695–720, v–vi.
  29. Hibbard JU, Tart M, Moawad AH. Cervical length at 16–22 weeks' gestation and risk for preterm delivery. *Obstet Gynecol*. 2000 Dec;96(6):972–978.
  30. Berghella V, Tolosa JE, Kuhlman K, Weiner S, Bolognese RJ, Wapner RJ. Cervical ultrasonography compared with manual examination as a predictor of preterm delivery. *Am J Obstet Gynecol*. 1997 Oct;177(4):723–730.
  31. Hassan SS, Romero R, Berry SM, Dang K, Blackwell SC, Treadwell MC, Wolfe HM. Patients with an ultrasonographic cervical length  $\leq 15$  mm have nearly a 50% risk of early spontaneous preterm delivery. *Am J Obstet Gynecol*. 2000 Jun;182(6):1458–1467.
  32. Andrews WW, Copper R, Hauth JC, Goldenberg RL, Neely C, Dubard M. Second-trimester cervical ultrasound: Association with increased risk for recurrent early spontaneous delivery. *Obstet Gynecol*. 2000 Feb;95(2):222–226.
  33. Harger JH. Cervical cerclage: Patient selection, morbidity, and success rates. *Clin Perinatol*. 1983 Jun;10(2):321–341.
  34. Interim report of the Medical Research Council/Royal College of Obstetricians and Gynaecologists multicentre randomized trial of cervical cerclage. *Br J Obstet Gynecol*. 1988 May;95(5):437–445.
  35. Althuisius SM, Dekker GA, Hummel P, Bekedam DJ, van Geijn HP. Final results of the Cervical Incompetence Prevention Randomized Cerclage Trial (CIPRACT): Therapeutic cerclage with bed rest versus bed rest alone. *Am J Obstet Gynecol*. 2001 Nov;185(5):1106–1112.
  36. Rust OA, Atlas RO, Reed J, van Gaalen J, Balducci J. Revisiting the short cervix detected by transvaginal ultrasound in the second trimester: Why cerclage therapy may not help. *Am J Obstet Gynecol*. 2001 Nov;185(5):1098–1105.
  37. Drakeley AJ, Roberts D, Alfirevic Z. Cervical stitch (cerclage) for preventing pregnancy loss in women. *Cochrane Database Syst Rev*. 2003;(1).
  38. Berghella V, Odibo AO, To MS, Rust OA, Althuisius SM. Cerclage for short cervix on ultrasonography: Meta-analysis of trials using individual patient-level data. *Obstet Gynecol*. 2005 Jul;106(1):181–189.
  39. Iams JD, Paraskos J, Landon MB, Teteris JN, Johnson FF: Cervical sonography in preterm labor. *Obstet Gynecol*. 1994 Jul;84(1):40–46.
  40. Schaffner F, Schanzer SN: Cervical dilatation in the early third trimester. *Obstet Gynecol*. 1966 Jan;27(1):130–133.
  41. Pereira L, Levy C, Lewis D, Berghella V. Effect of suture material on the outcome of emergent cerclage. *Obstet Gynecol*. 2004;103(4):35s.
  42. Rust OA, Atlas RO, Meyn J, Wells M, Kimmel S. Does cerclage location influence perinatal outcome? *Am J Obstet Gynecol*. 2003 Dec;189(6):1688–1691.
  43. To MS, Palaniappan V, Skentou C, Gibb D, Nicolaides KH. Elective cerclage vs. ultrasound-indicated cerclage in high-risk pregnancies. *Ultrasound Obstet Gynecol*. 2002 May;19(5):475–477.
  44. Rust OA, Roberts WE. Does cerclage prevent preterm birth? *Obstet Gynecol Clin North Am*. 2005 Sep;32(3):441–456.
  45. Kelly S, Pollock M, Maas B, Lefebvre C, Manley J, Sciscione A. Early transvaginal ultrasonography versus early cerclage in women with an unclear history of incompetent cervix. *Am J Obstet Gynecol*. 2001 May;184(6):1097–1099.
  46. Berghella V, Haas S, Chervoneva I, Hyslop T. Patients with prior second-trimester loss: Prophylactic cerclage or serial transvaginal sonogram. *Am J Obstet Gynecol*. 2002 Sept.187(3):747–751.
  47. Novy MJ, Gupta A, Wothe DD, Gupta S, Kennedy KA, Gravett MG. Cervical cerclage in the second trimester of pregnancy: A historical cohort study. *Am J Obstet Gynecol*. 2001 Jun;184(7):1447–1456, discussion 1454–1456.
  48. Chasen ST, Silverman NS. Mid-trimester emergent cerclage: A ten-year single institution review. *J Perinatol*. 1998 Sep–Oct;18(5):338–342.
  49. Olatunbosun OA, al-Nuaim L, Turnell RW. Emergency cerclage compared with bed rest for advanced cervical dilatation in pregnancy. *Int Surg*. 1995 Apr–Jun;80(2):170–174.

50. Althuisius SM, Dekker GA, Hummel P, van Geijn HP. Cervical incompetence prevention randomized cerclage trial: Emergency cerclage with bed rest versus bed rest alone. *Am J Obstet Gynecol.* 2003 Oct;189(4):907–910.
51. Novy MJ: Transabdominal cervicoisthmic cerclage: A reappraisal 25 years after its introduction. *Am J Obstet Gynecol.* 1991 Jun;164(6 Pt 1):1635–1641; discussion 1641–1642.
52. Hole J, Tressler T, Martinez F. Elective and emergency transabdominal cervicoisthmus cerclage for cervical incompetence. *J Reprod Med.* 2003 Aug;48(8):596–600.
53. Bensen RC, Durfee RB. Transabdominal cervicouterine cerclage during pregnancy for the treatment of cervical incompetency. *Obstet Gynecol.* 1965 Feb;25:145–155.
54. Al-Fadhli R, Tolandi T. Laparoscopic abdominal cerclage. *Obstet Gynecol Clin North Am.* 2004 Sep; 31(3):497–504, viii.
55. Mingione MJ, Scibetta J, Sanko SR, Phipps WR. Clinical outcomes following interval laparoscopic transabdominal cervicoisthmic cerclage placement: Case series. *Hum Reprod.* 2003 Aug;18(8):1716–1719.
56. Katz M, Abrahams C. Transvaginal placement of cervicoisthmic cerclage: Report on pregnancy outcome. *Am J Obstet Gynecol.* 2005 Jun;192(6): 1989–1992; discussion 1992–1994.
57. Orr C. An aid to cervical cerclage. *Aust NZ J Obstet Gynecol.* 1973 May;13(2):114.
58. Olatunbosun OA, Dyck F. Cervical cerclage operation for a dilated cervix. *Obstet Gynecol.* 1981 Feb;57(2):166–170.
59. Goodlin RC. Cervical incompetence, hourglass membranes, and amniocentesis. *Obstet Gynecol.* 1979 Dec;54(6):748–750.
60. Scheerer LJ, Lam F, Bartolucci L, Katz M. A new technique for reduction of prolapsed fetal membranes for emergency cervical cerclage. *Obstet Gynecol.* 1989 Sep;74(3 Pt 1):408–410.
61. Hefner JD, Patow WE, Ludwig JM. A new surgical procedure for the correction of the incompetent cervix during pregnancy: The Wurm Procedure. *Obstet Gynecol.* 1961 Nov;18:616–620.
62. Page EW. Incompetent internal os of the cervix causing late abortion and premature labor. Technique for surgical repair. *Obstet Gynecol.* 1958 Nov;12(5):509–515.
63. Vitsky M. Simple treatment of the incompetent cervical os. *Am J Obstet Gynecol.* 1961 Jun;81:1194–1197.
64. Oster S, Javert CT. Treatment of the incompetent cervix with the Hodge pessary. *Obstet Gynecol.* 1966 Aug;28(2):206–208.
65. Hartmann K, Thorpe JM Jr, McDonald TL, Savitz DA, Granados JL. Cervical dimensions and risk of preterm birth: A prospective cohort study. *Obstet Gynecol.* 1999 Apr;93(4):504–509.
66. Lockwood CJ: The diagnosis of preterm labor and the prediction of preterm delivery. *Clin Obstet Gynecol.* 1995 Dec;38(4):675–687.
67. Leiman G, Harrison NA, Rubin A. Pregnancy following conization of the cervix: Complications related to cone size. *Am J Obstet Gynecol.* 1980 Jan 1;136(1):14–18.
68. Cruickshank ME, Flannelly G, Campbell DM, Kitchener HC. Fertility and pregnancy outcome following large-loop excision of cervical transformation zone. *Br J Obstet Gynaecol.* 1995 Jun;102(6):467–470.
69. To MS, Alfirevic Z, Heath VC, Cicero S, Cacho AM, Williamson PR, Nicholaides KH, Fetal Medicine Foundation Second Trimester Screening Group. Cervical cerclage for prevention of preterm delivery in women with short cervix: Randomised controlled trial. *Lancet.* 2004 June 5;363 (9424):1849–1853.
70. Imseis HM, Albert TA, Iams JD. Identifying twin gestations at low risk for preterm birth with a transvaginal ultrasonographic cervical measurement at 24 to 26 weeks' gestation. *Am J Obstet Gynecol.* 1997 Nov;177(5):1149–1155.
71. Durnwald CP, Walker H, Lundy JC, Iams JD. Rates of recurrent preterm birth by obstetrical history and cervical length. *Am J Obstet Gynecol.* 2005 Sep;193(3 Pt 2):1170–1174.
72. Iams JD, Goldenberg RL, Mercer BM, Moawad A, Thom E, Meis PJ, McNellis D, Caritis SN, Miodovnik M, Menard MK, Thurnau GR, Bottoms SE, Roberts JM. The Preterm Prediction Study: Recurrence risk of spontaneous preterm birth. National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. *Am J Obstet Gynecol.* 1998 May;178(5):1035–1040.
73. Roman AS, Rebarber A, Pereira L, Sfakianaki AK, Mulholland J, Berghella V. The efficacy of

- sonographically indicated cerclage in multiple gestations. *J Ultrasound Med.* 2005 Jun;24(6):763–768.
74. Rubovits FE, Cooperman NR, Lash AF. Habitual abortion: A radiographic technique to demonstrate the incompetent internal os of the cervix. *Am J Obstet Gynecol.* 1953;66:269–280.
  75. Kiwi R, Neuman MR, Merkatz IR, Selim MA, Lysikiewicz A. Determination of the elastic properties of the cervix. *Obstet Gynecol.* 1988 Apr;71(4):568–574.
  76. Anthony GS, Calder AA, MacNaughton MC. Cervical resistance in patients with previous spontaneous mid-trimester abortion. *Br J Obstet Gynaecol.* 1982 Dec;89(12):1046–1049.
  77. Kushnir O, Vigil DA, Izquierdo L, Schiff M, Curet LB. Vaginal ultrasonographic assessment of cervical length changes during normal pregnancy. *Am J Obstet Gynecol.* 1990 Apr;162(4):991–993.
  78. Okitsu O, Mimura T, Nakayama T, Aono T. Early prediction of preterm delivery by transvaginal ultrasonography. *Ultrasound Obstet Gynecol.* 1992 Nov;2(6):402–409.
  79. Yost NP, Owen J, Berghella V, MacPherson C, Swain M, Dildy GA 3rd, Miodovnik M, Langer O, Sibai B. National Institute of Child Health and Human Development, Maternal-Fetal Medicine Units Network. Second-trimester cervical sonography: Features other than cervical length to predict spontaneous preterm birth. *Obstet Gynecol.* 2004 Mar;103(3):457–462.
  80. Drakeley AJ, Roberts D, Alfirevic Z. Cervical cerclage for prevention of preterm delivery: Meta-analysis of randomized trials. *Obstet Gynecol.* 2003 Sep;102(3):621–627. Review. Erratum in: *Obstet Gynecol.* 2004 Jan;103(1):201.
  81. Honest H, Coomarasamy A, Bachmann LM, Khan KS. Cervical cerclage for prevention of preterm delivery: Meta-analysis of randomized trials. *Obstet Gynecol.* 2004 Mar;103(3):584–586.
  82. Daskalakis G, Papantoniou N, Mesogitis S, Antsaklis A. Management of cervical insufficiency and bulging fetal membranes. *Obstet Gynecol.* 2006 Feb;107(2 Pt 1):221–226.
  83. Rush RW, Isaacs S, McPherson K, Jones L, Chalmers I, Grant A. A randomized controlled trial of cervical cerclage in women at high risk of spontaneous preterm delivery. *Br J Obstet Gynaecol.* 1984 Aug;91(8):724–730.
  84. Lazar P, Gueguen S, Dreyfus J, Renaud R, Pontonnier G, Papiernik E. Multicentred controlled trial of cervical cerclage in women at moderate risk of preterm delivery. *Br J Obstet Gynaecol.* 1984 Aug;91(8):731–735.
  85. Dor J, Shalev J, Mashiach S, Blankstein J, Serr DM. Elective cervical suture of twin pregnancies diagnosed ultrasonically in the first trimester following induced ovulation. *Gynecol Obstet Invest.* 1982;13(1):55–60.
  86. Harger JH. Cerclage and cervical insufficiency: An evidence-based analysis. *Obstet Gynecol.* 2002 Dec;100(6):1313–1327. Review. Erratum in: *Obstet Gynecol.* 2003 Jan;101(1):205.
  87. Atrash HK, Hogue CJ. The effect of pregnancy termination on future reproduction. *Baillieres Clin Obstet Gynaecol.* 1990 Jun;4(2):391–405. Review.
  88. Kessler I, Shoham Z, Lancet M, Blickstein I, Yemini M, Miskin A. Complications associated with genital colonization in pregnancies with and without cerclage. *Int J Gynaecol Obstet.* 1988 Dec;27(3):359–363.
  89. Funai EF, Paidas MJ, Rebarber A, O'Neill L, Rosen TJ, Young BK. Change in cervical length after prophylactic cerclage. *Obstet Gynecol.* 1999 Jul;94(1):117–119.
  90. Guzman ER, Houlihan C, Vintzileos A, Ivan J, Benito C, Kappy K. The significance of transvaginal ultrasonographic evaluation of the cervix in women treated with emergency cerclage. *Am J Obstet Gynecol.* 1996 Aug;175(2):471–476.
  91. Andersen FH, Karimi A, Sakala EP, Kalugdan R. Prediction of cervical cerclage outcome by endovaginal ultrasonography. *Am J Obstet Gynecol.* 1994 Oct;171(4):1102–1106.
  92. Dijkstra K, Funai EF, O'Neill L, Rebarber A, Paidas MJ, Young BK. Change in cervical length after cerclage as a predictor of preterm delivery. *Obstet Gynecol.* 2000 Sep;96(3):346–350.
  93. Fox HA. The incompetent cervix: Words that can hurt. *Am J Obstet Gynecol.* 1983 Oct;147(4):462.
  94. Bobitt JR, Ledger WJ. Amniotic fluid analysis: Its role in maternal neonatal infection. *Obstet Gynecol.* 1978 Jan;51(1):56–62.
  95. Romero R, Jimenez C, Lohda AK, Nores J, Hanaoka S, Avila C, Callahan R, Mazor M, Hobbins JC, Diamond MP. Amniotic fluid glucose concentration: A rapid and simple method for the detection of

- intraamniotic infection in preterm labor. *Am J Obstet Gynecol.* 1990 Sep;163(3):968-74.
96. Mays JK, Figueroa R, Shah J, Khakoo H, Kaminisky S, Tejani N. Amniocentesis for selection before rescue cerclage. *Obstet Gynecol.* 2000 May;95(5 Pt 1):652-655.
  97. Romero R, Gonzalez R, Sepulveda W, Brandt F, Ramirez M, Sorokin Y, Mazor M, Treadwell MC, Cotton DB. Infection and labor. : Microbial invasion of the amniotic cavity in patients with suspected cervical incompetence: Prevalence and clinical significance. *Am J Obstet Gynecol.* 1992 Oct;167(4 Pt 1):1086-1091.
  98. MacDougall J, Siddle N. Emergency cervical cerclage. *Br J Obstet Gynaecol.* 1991 Dec;98(12):1234-1238.
  99. Higgins SP, Kornman LH, Bell RJ, Brennecke SP. Cervical surveillance as an alternative to elective cervical cerclage for pregnancy management of suspected cervical incompetence. *Aust N Z Obstet Gynecol.* 2004 Jun;44(3):228-232.
  100. Groom KM, Bennett PR, Golará M, Thalon A, Shennan AH. Elective cervical cerclage versus serial ultrasound surveillance of cervical length in a population at high risk for preterm delivery. *Eur J Obstet Gynecol Reprod Biol.* 2004 Feb;112(2):158-161.
  101. Althuisius SM, Dekker GA, van Geijn HP, Bekedam DJ, Hummel P. Cervical incompetence prevention randomized cerclage trial (CIPRACT): Study design and preliminary results. *Am J Obstet Gynecol.* 2000 Oct;183(4):823-829.
  102. Barth WH Jr, Yeomans ER, Hankins GD. Emergent cerclage. *Surg Gynecol Obstet.* 1990 Apr;170(4):323-326.
  103. Latta RA, McKenna B. Emergent cervical cerclage: Predicators of success or failure. *J Matern Fetal Med.* 1996 Jan-Feb;5(1):22-27.
  104. Lipitz S, Libshitz A, Oelsner G, Kokia E, Goldenberg M, Mashiach S, Schiff E. Outcome of second-trimester, emergency cervical cerclage in patients with no history of cervical incompetence. *Am J Perinatol.* 1996 Oct;13(7):419-422.
  105. Holman M. An aid for cervical cerclage. *Obstet Gynecol.* 1973 Sep;42(3):468-469.
  106. Tsatsaris V, Senat MV, Gervasis A, Fernandez H. Balloon replacement of fetal membranes to facilitate emergency cervical cerclage. *Obstet Gynecol.* 2001 Aug;98(2):243-246.
  107. Locatelli A, Vergani P, Bellini P, Strobelt N, Arreghini A, Ghidini A. Amnioreduction in emergency cerclage with prolapsed membranes: Comparison of two methods for reducing the membranes. *Am J Perinatol.* 1999;16(2):73-77.
  108. Ayromlooi J. Balloon replacement of fetal membranes to facilitate emergency cervical cerclage. *Obstet Gynecol.* 2002 Feb;99(2):345.
  109. Romero R, Mazor M, Morrotti R, Avila C, Oyarzun E, Insunza A, Parra M, Behnke Montiel F, Cassell GH. Infection and labor. VII. Microbial invasion of the amniotic cavity in spontaneous rupture of membranes at term. *Am J Obstet Gynecol.* 1992 Jan;166(1 Pt 1):129-133.
  110. Blickstein I, Katz Z, Lancet M, Molgilner BM. The outcome of pregnancies complicated by preterm rupture of the membranes with and without cerclage. *Int J Gynaecol Obstet.* 1989 Mar;28(3):237-242.
  111. Ludmir J, Bader T, Chen L, Lindenbaum C, Wong G. Poor perinatal outcome associated with cerclage in patients with premature rupture of membranes. 1994 Nov;84(5):823-826.
  112. Kominiarek MA, Kemp A. Perinatal outcome in preterm premature rupture of membranes at  $\leq 32$  weeks with retained cerclage. *J Reprod Med.* 2006 Jul;51(7):533-538.
  113. McElrath TF, Norwitz ER, Lieberman ES, Heffner LJ. Perinatal outcome after preterm premature rupture of membranes with in situ cervical cerclage. *Am J Obstet Gynecol.* 2002 Nov;187(5):1147-1152.
  114. Jenkins TM, Berghella V, Shlossman PA, McIntyre CJ, Maas BD, Pollock MA, Wapner RJ. Timing of cerclage removal after preterm premature rupture of membranes: Maternal and neonatal outcomes. *Am J Obstet Gynecol.* 2000;183:847-852.
  115. McElrath TF, Norwitz ER, Lieberman ES, Heffner LJ. Management of cervical cerclage and preterm premature rupture of the membranes: Should the stitch be removed? *Am J Obstet Gynecol.* 2000;183:840-846.
  116. McDonald IA. Suture of the cervix for inevitable miscarriage. *J Obstet Gynaecol Br Emp.* 1957 Jun;64(3):346-350.
  117. Novy MJ. Transabdominal cervicoisthmic cerclage: A reappraisal 25 years after its introduction. *Am J Obstet Gynecol.* 1991 Jun;164(6 Pt 1):1635-1641; discussion 1641-1642.

118. Craig S, Fliegner JR. Treatment of cervical incompetence by transabdominal cervicoisthmic cerclage. *Aust NZ J Obstet Gynaecol.* 1997 Nov;37(4):407–411.
119. Lesser KB, Childers JM, Surwit EA. Transabdominal cerclage: A laparoscopic approach. *Obstet Gynecol.* 1998 May;91(5 Pt 2):855–856.
120. Cho CH, Kim TH, Kwon SH, Kim JI, Yoon SD, Cha SD. Laparoscopic transabdominal cervicoisthmic cerclage during pregnancy. *J Am Assoc Gynecol Laparosc.* 2003 Aug;10(3):363–366.
121. Anthony GS, Walker RG, Cameron AD, Price JL, Walker JJ, Calder AA. Transabdominal cervicoisthmic cerclage in the management of cervical incompetence. *Eur J Obstet Gynecol Reprod Biol.* 1997 Apr;72(2):127–130.
122. Lotgering FK, Gaugler-Senden IP, Lotgering SF, Wallenburg HC. Outcome after transabdominal cervicoisthmic cerclage. *Obstet Gynecol.* 2006 Apr;107(4):779–784.
123. Olatunbosun O, Turnell R, Pierson R. Transvaginal sonography and fiberoptic illumination of uterine vessels for abdominal cervicoisthmic cerclage. *Obstet Gynecol.* 2003 Nov;102(5 Pt 2):1130–1133.
124. Newcomer J. Pessaries for the treatment of incompetent cervix and premature delivery. *Obstet Gynecol Surv.* 2000 Jul;55(7):443–448.
125. Owen J, Yost N, Berghella V, MacPherson C, Swain M, Dildy GA 3rd, Miodovnik M, Langer O, Sibai B, Maternal-Fetal Medicine Units Network. Can shortened midtrimester cervical length predict very early spontaneous preterm birth? *Am J Obstet Gynecol.* 2004 Jul;191(1):298–303.
126. Guzman ER, Mellon C, Vintzileos AM, Ananth CV, Walkters C, Gipson K. Longitudinal assessment of endocervical canal length between 15 and 24 weeks' gestation in women at risk for pregnancy loss or preterm birth. *Obstet Gynecol.* 1998 Jul;92(1):31–37.
127. Guzman ER, Walters C, Ananth CV, O'Reilly-Green C, Benito CW, Palermo A, Vintzileos AM. A comparison of sonographic cervical parameters in predicting spontaneous preterm birth in high-risk singleton gestations. *Ultrasound Obstet Gynecol.* 2001 Sep;18(3):195–199.
128. Alfrevic Z. Cerclage: We all know how to do it but can't agree when to do it. *Obstet Gynecol.* 2006 Feb;107(2 Pt 1):219–220.
129. Vayssiere C, Favre R, Audibert F, Chauvet MP, Gaucherand P, Tardif D, Grange G, Novoa A, Descamps P, Perdu M, Andrini E, Janse-Marec J, Maillard F, Nisand I. Cervical length and funneling at 22 and 27 weeks to predict spontaneous birth before 32 weeks in twin pregnancies: A French prospective multicenter study. *Am J Obstet Gynecol.* 2002 Dec;187(6):1596–604.
130. Althuisius S, Dekker G, Hummel P, Bekedam D, Kuik D, van Geijn H. Cervical Incompetence Prevention Randomized Cerclage Trial (CIPRACT): Effect of therapeutic cerclage with bed rest vs. bed rest only on cervical length. *Ultrasound Obstet Gynecol.* 2002 Aug;20(2):163–167.
131. Carr DB, Smith K, Parsons L, Chansky K, Shields LE. Ultrasonography for cervical length measurement: Agreement between transvaginal and translabial techniques. *Obstet Gynecol.* 2000 Oct;96(4):554–558.
132. Terkildsen MFC, Parilla BV, Jumar P, Grobman WA. Factors associated with success of emergent second-trimester cerclage. *Obstet Gynecol.* 2003 Mar;101(3):565–569.
133. Airoidi J, Berghella V, Sehdev H, Ludmir J. Transvaginal ultrasonography of the cervix to predict preterm birth in women with uterine anomalies. *Obstet Gynecol.* 2005 Sep;106(3):553–556.
134. Rechberger T, Ulbjerg N, Oxlund H. Connective tissue changes in the cervix during normal pregnancy and pregnancy complicated by cervical incompetence. *Obstet Gynecol.* 1988 Apr;71(4):563–567.
135. Arias F. Cervical cerclage for the temporary treatment of patients with placenta previa. *Obstet Gynecol.* 1988 Apr;71(4):545–548.
136. Cruickshank ME, Flannelly G, Campbell DM, Kitchener HC. Fertility and pregnancy outcome following large-loop excision of the cervical transformation zone. *Br J Obstet Gynaecol.* 1995 Jun;102(6):467–470.
137. Davis G, Berghella V, Talucci M, Wapner RJ. Patients with a prior failed transvaginal cerclage: A comparison of obstetric outcomes with either transabdominal or transvaginal cerclage. *Am J Obstet Gynecol.* 2000 Oct;183(4):836–839.
138. Deffieux X, De Tayrac R, Louafi N, Gervaise A, Bonnet K, Frydman R, Fernandez H. Novel application of polypropylene sling: Transvaginal

- cervicoisthmio-cerclage in women with high risk of preterm delivery. *J Minim Invasive Gynecol*. 2006 May-Jun;13(3):216–212.
139. Golfier F, Bassai J, Paparel P, Cassagnol A, Vaudoyer F, Raudrant D. Transvaginal cervicoisthmio-cerclage as an alternative to the transabdominal technique. *Eur J Obstet Gynecol Reprod Biol*. 2001 Dec 10;100(1):16–21.
140. Deffieux X, de Tayrac R, Louafi N, Gervaise A, Senat MV, Chauveaud-Lambling A, Picone O, Faivre E, Bonnet K, Frydman R, Fernandez H. Transvaginal cervico-isthmio-cerclage using polypropylene tape: Surgical procedure and pregnancy outcome: Fernandez's procedure. *J Gynecol Obstet Biol Reprod (Paris)*. 2006 Sep;35(5 Pt 1):465–471.

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## Chapter 6 PREGNANCY TERMINATION

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Heather Z. Sankey

*Grammatici certant et adhuc subjudice lis est.  
(Scholars dispute, and the case is still before the  
courts.)*

Horace (Quintus Horatius Flaccus) (65–8 B.C.E.)  
*Ars Poetica* 18 B.C.E., III, 7.

### HISTORY OF ABORTION

This chapter reviews the history and epidemiology of modern pregnancy termination. In this review, the surgical and medical techniques appropriate for various gestational ages are presented, potential complications are considered, and the psychological issues surrounding abortion are discussed.

Controversy concerning human fertility contraception or spontaneous or induced abortion dates to ancient times. Issues involving the ethics of pregnancy termination and techniques for abortion have long been a part of medical practice. Society continues to struggle both with the problems of abortion ethics as well as access to procedures. Recent decades have seen various legal efforts by individual states to limit or entirely proscribe pregnancy termination. A historical review of abortion practices provides some perspective to modern practitioners on these contentious modern debates and indicates how long these issues have been debated without societal resolution.

Greek, Roman, and Hebrew laws generally did not protect the fetus before recognizable features were formed during development. Before that point, abortion could be performed without official reprisal [1]. The Old Testament refers to accidental miscarriage but does not refer specifically to induced abortion. The Talmud, however, states that the fetus can be sacrificed to save the life of the mother [2]. The issue of pregnancy termination was discussed in some detail by the classical philosophers. Both Plato and Aristotle favored controls on conception to ensure population stability in their theorized, ideal city-states. Plato advocated abortion for all pregnancies resulting from “nonoptimal matings.” Aristotle favored abortion for pregnancies occurring in women who were either over 40 years of age or who had already delivered a prescribed number of children.

Among other injunctions, the Hippocratic Oath formulated in the fifth century B.C.E. required initiates to medical practice to swear not to administer an abortive suppository. This admonition has been widely interpreted as forbidding physicians to



perform abortions by *any* technique. According to Riddle [1], this interpretation arose from a misreading of Hippocratic writings by Scribonius Largus, a Roman physician of the 1st century C.E. Scribonius was among the first to interpret Hippocrates' injunction against abortive suppositories as a general condemnation of all abortion. Rather than a presumption against a specific technique of pregnancy termination, Scribonius wrote that ". . . Hippocrates, who founded our profession, laid the foundation for our discipline by an oath in which it was proscribed to give a pregnant woman a kind of medicine that expels the embryo or fetus." Although the oath proscribes abortive suppositories and pessaries, Riddle argues that it was clearly implied that the physician was free to use contraceptives, provide oral abortifacients, and use the various surgical and manipulative procedures then available and widely used for pregnancy termination. It is safe to predict that this interpretation of the oath will remain controversial.

Early Christians believed that anything that interrupted human life was morally equivalent to murder; however, the church fathers were divided about the ethics of abortion. Between the fifth and the twelfth centuries, the concept of a distinction between a "formed" or ensouled and an "unformed" (and thus unensouled) fetus gradually became established in Catholic doctrine. This distinction followed the commentaries of Gregory, Bishop of Nyssa (530–395), and Augustine of Hippo (354–430), and it was supported by Pope Gregory IX in 1234 [1,2].

These various ideas formed the basis for theoretical discussions concerning the beginning of human life. In these debates, it was argued initially that abortion was justified if necessary to save the woman's life when the fetus was in an "unformed" state. In 1869, Pope Pius XI eliminated this philosophical distinction between a formed and an unformed fetus, declaring that the human soul was created at the moment of conception. Thereafter in Catholic doctrine, abortion was prohibited even in situations where it might save the mother's life. Until the second quarter of the 20th century, all Protestant denominations generally opposed both contraception and abortion. After the 1930s, however, contraception was progressively approved by many Protestant churches and slowly became widely accepted in the general population. Despite many conditions,

abortion was generally approved in situations of undeniable medical necessity, although a wide diversity of opinion persisted [2].

Legal constraints on abortion were initially imposed in the nineteenth century. Prior to 1840, abortion was a commonly performed procedure before quickening – the maternal perception of fetal activity. In fact, vendors of abortifacients and abortion practitioners advertised openly in newspapers and even in religious journals. At the time, under common law, abortion was a criminal act only if performed after quickening had occurred [3]. The open practice of abortion stopped soon after the midcentury, when statutory laws prohibiting pregnancy termination replaced the common law doctrine of quickening. Physician groups, including the newly organized American Medical Association (1847), launched antiabortion campaigns at midcentury; this movement was eventually joined by antiobscenity crusaders and feminists. The latter group opposed abortion because they associated it with female suppression. At this time in the history of abortion practice, religious leaders were not at the forefront of the discussions concerning pregnancy termination. The eventual result of this political activity was that by 1900 both the performance of abortion and advertising for abortion were illegal throughout the United States. The consequence of criminalization was not to prevent pregnancy termination but simply to force the practice underground. Abortion fell to inexperienced, disreputable, or even totally untrained practitioners who could not or would not avail themselves of safe methods [3]. As its practitioners were stigmatized by social disapprobation and illegality, pregnancy termination progressively became isolated from the general advances of medicine. The complex legacy of these social and legal restrictions persists into the 20th century despite the reversal of many statutory rules by legal review and legislative actions in the 1960s and 1970s, as well as the introduction of oral medications (mifepristone and misoprostol) as an effective method for first-trimester abortion. This provided women with another, potentially more private, option for pregnancy termination but also helped revive political opposition. Abortion remains a major, contentious issue in the political and social life of much of the Western world, including the United States, and doubtless will continue to remain so.

## EPIDEMIOLOGY

In 2003, 848,163 legal abortions were reported to the Centers for Disease Control, a decrease of 0.1% over the number reported for 2002 [4]. Rather than consider absolute numbers, however, it is best to analyze the number of terminations per 1,000 live births as a measure of abortion frequency. This *national abortion ratio* (NBR) increased gradually from 196 terminations per 1,000 live births in 1973, to 358 per 1,000 live births in 1979. After remaining stable for several years, the ratio peaked at 364 per 1,000 in 1984 and since then has demonstrated a decline. In 2003, NBR was 241 legal abortions per 1,000 live births. The *national abortion rate*, defined as the number of legal abortions per 1,000 women aged 15 to 44 years, increased from 14 in 1973 to 23 to 24 during the 1980s. It decreased to 20 during 1994 to 1997 and then remained stable at 16 in the interval from 2000 to 2003.

When the demographic data are reviewed, women who obtain legal abortions are predominantly younger than 25 years old (51%), white (55%), and unmarried (82%) [5]. More than one half of legal abortions are performed during the first 8 weeks of gestation and approximately 88% during the first 12 weeks. Only 5.6% of legal abortions are performed at gestational ages greater than 16 weeks. For women whose type of procedure is adequately reported, 91% of abortions are performed by curettage. These numbers include procedures performed by dilatation and evacuation (D and E). Only 0.4% of terminations are performed by techniques involving intrauterine instillation. Approximately 8% of all procedures reported from the 45 areas of the United States with adequate procedure recording are medical abortions performed by the administration of mifepristone or another cytotoxic drug combined with an uterotonic.

## SURGICAL PROCEDURES

### Preoperative Evaluation

Most women requesting termination of pregnancy are self-referred. Physicians who care for pregnant patients should assess the patient's attitudes toward the gestation at the time of the first prenatal visit. A simple, nondirective question such as "How do you feel about being pregnant?" or "What are your

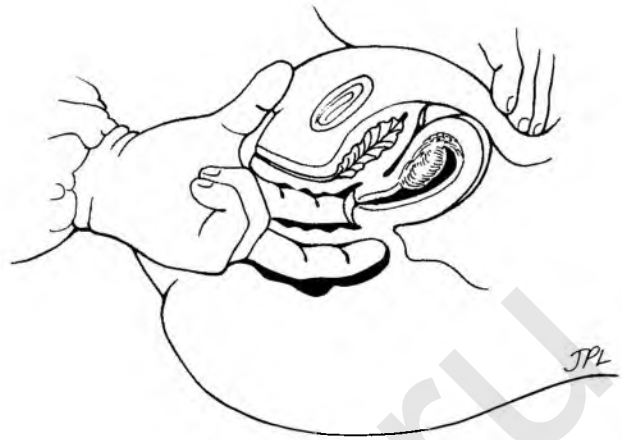


FIGURE 6.1.  
*Uterine size is evaluated by preoperative bimanual examination.*

concerns about this pregnancy?" can elicit a marked degree of ambivalence. Such patients must be counseled concerning their options. If termination is the patient's choice, the preoperative evaluation should include 1) an estimate of gestational age; 2) a review of the woman's physical state, including assessment of any important preexisting medical conditions, such as cardiac disease, asthma, diabetes, among others; and 3) a consideration of the woman's mental state, including the psychological factors that are influencing her decision.

The initial assessment of gestational age is based on the last reported menstrual period and the physical examination. If the diagnosis of pregnancy is uncertain, serum or urinary pregnancy testing should be performed and, in selected cases, real-time ultrasound scanning ordered to confirm and date the pregnancy. During the physical examination, the gestational age is estimated by uterine palpation (Figure 6.1). In the authors' practice, the evaluation of patients who initially request pregnancy termination does not routinely include auscultation for fetal heart tones. For gestations suspected to be beyond 12 weeks based on physical examination, regardless of the menstrual history provided, or if there is uncertainty concerning the true gestational age for any other reason, or if there is possibility of an ectopic pregnancy, a real-time ultrasound examination is mandatory. There must be accurate data concerning the period of gestation before any recommendations are made concerning specific termination techniques.

As part of the total evaluation, the authors obtain a standard medical and surgical history, including a review of possible allergies, current medications, and habits (e.g., smoking). The obstetric and gynecologic history is also closely reviewed. The authors routinely perform a Pap smear, obtain cultures for gonorrhea and Chlamydia, and draw a serologic test for syphilis. Some services omit these studies based on their own protocols. A preoperative complete blood count and blood typing is also performed. With the increasing emphasis on preconceptual care, a Rubella titer is recommended. Patients with no demonstrable Rubella titer are candidates for subsequent immunization. If the history suggests multiple partners or unsafe sexual activity, both HIV and hepatitis C testing should be frankly discussed.

The most important part of the preoperative evaluation is patient counseling. This can most easily be divided into the general categories of *surgical counseling* and *motivation counseling*, both of which are essential to obtain a prior informed consent. During counseling for either a surgical procedure or an oral medication regimen, the specifics of the contemplated procedure are carefully reviewed. Surgical risks such as infection, bleeding, and perforation of the uterus are fully explained, as are potential complications of oral treatment. The possible effects of a termination of pregnancy on future childbearing are discussed. For women with a history of multiple abortions, it is appropriate to review the risk of future cervical insufficiency. Additionally, a method of contraception intended for use after the procedure should be agreed on. Deciding on which contraceptive method to recommend requires an assessment of the patient's previous contraceptive history, her level of understanding, and her reliability. Women who express an interest in sterilization are counseled and, when appropriate, the necessary permission forms are completed. Although pregnancy termination and sterilization can be performed jointly, it is normally prudent not to do so [6].

Exploring the patient's motivation for terminating her pregnancy is time-consuming and requires both experience and skill in intrapersonal interactions. In the beginning, it is helpful to explore with the woman how she usually arrives at decisions and the problems associated with her decision-making style [7]. Possible options for her pregnancy, such as adoption or raising the child, can be reviewed. A

technique the authors often practice is to have her imagine and discuss her vision of each of her possible options. This technique, called *decision counseling* or *nondirective counseling*, outlines the benefits and consequences of abortion, the costs and the potential advantages of giving the baby up for adoption, or of keeping the baby, without indicating a preference for any option [8]. The woman's social supports, including her partner, family members, and friends, should also be assessed. Her previous psychiatric history and the strength of her coping mechanisms also require evaluation. The counselor must be able to distinguish between "normal" feelings of ambivalence about pregnancy termination versus genuine confusion. Confusion is not necessarily expressed in a straightforward manner but can hide behind such outward behavior as taciturnity, arrogance, extreme impatience, or hostility [7]. In the patient who is markedly ambivalent, more than one counseling session might be needed until she establishes a plan acceptable to her. In the unusual instance when a serious psychological disturbance is suspected, the assistance of a social worker, psychologist, or psychiatrist might be required.

Given the multilingual nature of modern society, a word must be said about counseling the patient whose native language differs from that of the counselor. In such a situation, it is strongly recommended that the services of an *independent* and experienced translator be obtained. Translating through family members or sexual partners is fraught with problems. Although input from intimates of the patient can be useful, there is a risk of addressing the translator's concerns rather than those of the patient. There is also a risk that the person translating could effectively be making the decision for the women by modifying the information provided.

Counseling patients with an unintended or unwanted pregnancy requires considerable skill, experience, and empathy. There is a risk of the patient's answering questions "appropriately" to please the physician who will be performing the termination procedure. If possible, counseling therefore should be conducted by someone (e.g., a social worker or specially trained nurse) whose approval is not perceived by the woman as being important to her acceptance as an abortion candidate. Long-term follow-up studies of women undergoing pregnancy termination document few psychological sequelae [9,10]. Nonetheless, as Rosenthal poignantly notes,

**TABLE 6.1 Techniques for Pregnancy Termination**

First-trimester
Vacuum or sharp curettage/menstrual extraction
Antiprogesterones (RU-486, mifepristone)
Antimetabolites (methotrexate)
Mid-trimester
Cervical dilatation and instrumental uterine evacuation (D and E)
Hypertonic solution injection (NaCl, urea)*
Prostaglandin administration (15-methyl-F2 $\alpha$ intramuscularly or intrauterine; E2 vaginally)*
Hysterotomy/hysterectomy <sup>†</sup>

\*Combined protocols are common. See text for details.

<sup>†</sup>Rarely utilized or performed.

there is no painless way to go through an unplanned pregnancy [9].

## PREGNANCY TERMINATION PROCEDURES

The method chosen for pregnancy termination depends on the period of gestation, the experience and preference of the operator, and the extent to which safe options are available that fit the patient's desires. For example, in a specific case the desire to examine fetal anatomy for genetic analysis might weigh heavily in the decision concerning the method of mid-trimester termination, leading to an instillation procedure rather than dilatation and evacuation. Certain procedures, such as hysterotomy, are rarely performed except in the most unusual of circumstances owing to the unacceptably high rate of morbidity with these operations and the comparative safety of other methods (Table 6.1) [11,12].

### **Technique: First-trimester Termination**

#### ***Vacuum Curettage***

Vacuum curettage is the most common procedure for first-trimester termination. Advantages include speed, safety, and the ability to perform outpatient procedures with the use of local anesthesia. Performance of first-trimester abortion with the patient under local anesthesia with intravenous or conscious sedation, however, does require considerable interpersonal skill and deft technique. The practitioner must convey to the patient a sense of calm and compassion. A physician who is cold, abrupt, or

unsympathetic is more likely to encounter problems. Initially, it is helpful to explain, in general terms, what is being done during the procedure and what to expect. "I'm now going to put in a speculum; it will feel like an examination for a Pap smear." "You're going to hear a noise as I turn on the suction; don't let that alarm you." This type of ongoing verbal instruction is especially important in adolescent patients, who are frightened and whose knowledge and understanding of medical procedures is often limited, at best.

Before beginning the surgical procedure, it is the surgeon's responsibility to review the operating equipment to ensure that all the appropriate instruments are at hand, and to mentally review the contemplated procedure. As the operation is considered, the rules for instruments and the pregnant uterus are simple: 1) the largest instrument that will pass through the cervix should always be employed; 2) when there is a choice of instruments the one to be used is always that with the dullest point; and 3) the clinician must be prepared to stop the procedure immediately and reassess if difficulty is encountered or the patient complains of sudden, severe discomfort.

For first-trimester curettage, a pelvic examination is performed immediately prior to the operation and after the woman has voided. The uterus is palpated for size and position (Figure 6.1). The operator next decides on the size of the suction cannula appropriate for the case. If there is *any* question about the gestational age, transvaginal or transabdominal ultrasound scanning should be performed before the procedure is started. Routine use of ultrasound scan during the actual curettage procedure does reduce complications in the first trimester, but equipment for this use might not be available in all settings [13].

The American College of Obstetrics and Gynecology (ACOG) recommends antibiotic prophylaxis prior to surgical abortion [14]. This recommendation is supported by several studies where antibiotic pretreatment of patients undergoing first-trimester abortion significantly reduced the rate of postabortion pelvic infection [15,16]. Doxycycline is an inexpensive broad-spectrum drug that is efficacious in reducing complications and is favored as a prophylactic agent, unless there is a history of allergy. Antibiotic regimens vary but among those commonly recommended are 1) doxycycline 100 mg PO 1 hour prior to the procedure, followed by

200 mg PO after the procedure; 2) doxycycline 100 mg PO twice daily for 7 days, and 3) metronidazole 500 mg twice daily PO for 5 days. Any of these is acceptable. The use of Laminaria is discussed in detail later.

Commonly, ibuprofen (600 mg–800 mg) or another nonsteroidal anti-inflammatory medication is administered at least one hour prior to the procedure. This moderately decreases the severity of pain during and after the procedure. It should be noted that the efficacy of such pretreatment not been extensively studied, however [17].

In young nulliparous women or any woman with a small or firm cervix, placement of one or more Laminaria 12 or more hours before the procedure facilitates dilatation. The use of Laminaria tents in such patients reduces the risk of cervical laceration or trauma [11,12,18].

An alternative to Laminaria tents is the preoperative administration of oral or vaginal misoprostol (15-methyl-prostaglandin E1). Whether misoprostol is superior to Laminaria for cervical ripening remains a matter of debate [19,37]. There is also inconsistency regarding the recommended dosing intervals. As an example, Stubblefield [20] recommends 40 mg of misoprostol placed vaginally 3 to 4 hours before the procedure. In performing operative hysteroscopy, however, Sharma and coworkers [21] demonstrated that for both oral and vaginal misoprostol, a one-hour timing interval was insufficient to provide detectable cervical change.

After the explanations and an examination, the vulva, vagina, and cervix are cleansed with a standard antiseptic solution such as povidone-iodine, following institutional protocols. Perineal and leg drapes are not necessary for first-trimester procedures, but sterile gloves and instruments are. The surgeon should use the “no-touch technique.” This simply means that the part of the instrument that enters the uterus or cervix is not touched by the operator’s hand at any time. Additionally, the physician must follow the dictates of the hospital or clinic regarding eye wear, the downing of gowns and gloves, or the use of a mask during the procedure.

Next, a speculum is passed, and the cervix is visualized. If a paracervical block is chosen, the local anesthetic is injected into the cervix using a 20- or 22-gauge spinal needle. The choice of local anesthetic drug is important. The ester 2-chloroprocaine is substantially less toxic than the amide lidocaine,

although lidocaine is less expensive and lasts longer. If lidocaine is used, the total dose should not exceed 2 mg/kg or 300 mg, whichever is less. In general, the physician should use the smallest volume and the lowest concentration required. For reasons of cost, effectiveness, and convenience, the authors prefer lidocaine. We routinely administer 10 ml to 20 ml of the 0.5% to 1.0% solution without epinephrine mixed with 0.5 ml to 1.0 ml of NaHCO<sub>3</sub> to reduce stinging.

These are several techniques for paracervical blockade, and the sites of injection are of little consequence. The authors favor injection at 4 o’clock and 8 o’clock at the cervicovaginal junction. A useful technique is to place the needle adjacent to the mucosa and then have the patient cough. This “pops” the mucosa over the needle tip, making the injection less painful. It also distracts the patient, and she might then be entirely unaware that the injections are performed. Some practitioners also inject 1 ml to 2 ml of local anesthetic in the anterior lip of the cervix, to decrease the discomfort from the subsequent placement of the tenaculum or counter-traction clamp.

The key to a good paracervical block is time. Once the injections have been made, the practitioner must wait at least 3 to 5 minutes (by the clock) for the block to take effect. During this time, the physician may talk to the patient, in soothing tones, explaining “we’re just waiting for the anesthetic to take effect.” This is a good time to judge the effect of any intravenously injected analgesic or relaxation agents and to be certain that all instruments are positioned on the operating table to suit the surgeon. The use of intravenous drugs for relaxation or additional analgesia is elective. Their use depends on the preference of the patient, whether she has someone to drive her home, when she last ate, the availability of trained personnel for monitoring, and the protocols of the hospital or clinic.

The authors usually administer a combination of rapid-acting medications, because most patients are quite anxious (Table 6.2). Fentanyl (0.025 mg–0.100 mg) with midazolam (0.5 mg–1.0 mg) intravenously, titrated to patient response, is the authors’ usual preference. Whenever such drugs are administered, clinic or institutional requirements for such conscious sensation in terms of patient evaluation and monitoring must be followed. For *all* patients receiving intravenous analgesics, the authors apply

TABLE 6.2 Agents for Intravenous Analgesia and Sedation\*

Medication	IV Doses and Interval	Peak Effect and Duration	Suggested Total Doses and Comments
<b>Sedative/hypnotic Agents</b>			
Midazolam (Versed)*	0.5–2.0 mg IV over 1–2 min Dosing interval: q5min, PRN to desired effect	Peak: 3 min Max effect: 5 min gradual declining effects: 30–40 min. Gross recovery within 2 hr but effects may last to 6 hr Onset: 1–5 min Duration: 2–8 hr	Total recommended: <5 mg Never administer as rapid IV bolus Extended half-life makes this a second-line agent
Lorazepam (Ativan)	0.03–0.05 mg/kg	Onset: 1–5 min Duration: 2–8 hr	
<b>OPIOIDS</b>			
Fentanyl (Sublimaze)	0.025 mg–0.100 mg IV (0.05 mg/kg–3 mg/kg) over 1–2 min Dosing interval: q3–5 min	Peak 2–3 min Duration: 30–60 min	Total recommended: .025 mg–.150 mg. This is a very potent agent and must be titrated carefully to avoid overdosing and excess sedation.
Meperidine (Demerol)	15 mg–35 mg IV over 1 min Dosing interval: q5–10 min	Peak: 3–5 min. Duration: 2–3 hr	Total recommended: 25–125 mg (0.5 mg/kg–1 mg/kg)
Morphine Sulfate	2 mg–5 mg IV over 2 min or 0.03–0.1 mg/kg Dosing interval: q5–10 min, PRN	Peak: 3–10 min. Duration: 4 hr	Total recommended: 10 mg (0.1 mg/kg–0.5 mg/kg) Never administer as a rapid IV bolus. Due to the relatively slow onset and long duration, this drug is uncommonly used.
<b>ANTIEMETIC/SEDATIVE</b>			
Doperidol (Inapsine)	0.625 mg IV Dosing interval: q15–30 min		Total recommended: 2.5 mg
<b>EMERGENCY AND REVERSAL AGENTS</b>			
Atropine	0.5 mg–1.0 mg IV Dosing interval: as clinically required		Total recommended: 2 mg Treatment limited to symptomatic bradycardias. It is uncommon to require more than 1 mg.
Naloxone (Narcan): (narcotic reversal)	For minor opioid side effects (e.g., itching, nausea, somnia): 0.04 mg–0.100 mg For severe opioid side effects (e.g., significant marked respiratory depression, inability to arouse): 0.120 mg	Apparent within 1–3 min Dosing interval: As clinically required	Total dose: 1.0 mg Requires titration to effect: brief duration of action. Doses >0.120 mg are to be avoided; Warning: has been associated with acute MI, AF, HTN, pulmonary edema, VT, and sudden death.

(Continued)

TABLE 6.2 (Continued)

Medication	IV Doses and Interval	Peak Effect and Duration	Suggested Total Doses and Comments
Flumazenil (Romazicon) (benzodiazepine reversal)	IV only: 0.1 mg (1 ml) to 0.2 mg (2 ml) administered IV over 15 sec; Additional doses of 0.2 mg (2 ml) repeated at 1 min intervals, as required.	Effects within 1–2 min postinjection. Peak: 6–10 min Duration: 30–60 min.	Total dose of 1.0 mg (10 ml). For benzodiazepine reversal only. Drug is a partial antagonist only. In the event of re-sedation, repeated doses can be administered at 20-min intervals, as required. For repeat treatment, no more than 1.0 mg (10 ml) administered as 0.2 mg/min (2 ml/min) should be given in any 1 hr.

\*Midazolam is not an analgesic. Pain requires treatment with an analgesic.

<sup>†</sup>Individualization and titration of doses is essential for safe and effective sedation: IM and SC dosing may be used but schedules for treatment may be different. These protocols are not intended to produce either loss of consciousness or respiratory reflexes in unintubated patients. Dosing for these effects must only be administered by an anesthesiologist or other especially trained personnel. Drugs and specific doses should only be administered based on institutional protocol with the immediate availability of special equipment and oxygen.

MI = myocardial infarction; AF = atrial fibrillation; HTN = acute hypertension; VT = ventricular tachycardia.

an oxygen saturation monitor and standard electrocardiographic leads and monitor these tracings continuously during the procedure. Reversal agents (including naloxone and flumazenil) and atropine, as well as oxygen, suction, and a resuscitation bag, are immediately available in the surgical suite. The surgical attendants must be trained to identify undue sedation and to treat respiratory distress if it occurs. The medical record must reflect the drugs given, the timing of administration, and the patient's response.

These guidelines are intended only as general suggestions for appropriate drug dosing. Because patient sensitivity to these agents varies widely, these drugs must be administered with close clinical attention to effect and titrated to the unique requirement of each case. In all instances, appropriately credentialed staff must follow institutional protocol for conscious sedation. The total dose given is the recommended maximum for any one operative procedure. Subcutaneous or intramuscular dosing results in delay of onset and longer duration of action.

After the paracervical block has been established, the anterior lip of the cervix is grasped with a single-

toothed tenaculum. Some practitioners prefer to use a long Allis clamp, a Bierer, or another atraumatic clamp because they are less likely to lacerate the cervix or result in pesky bleeding from a puncture site.

The cervix is next gradually dilated using cervical dilators (either Pratt, Hanks, or Hegars, at the operator's discretion). There is a 3-to-1 relationship between the diameter of the largest dilator (French) and the suction curette (millimeters) to be used; that is, one dilates to 24 Fr to use an 8-mm suction curette. *The uterine cavity should never be probed with a sound or similar instrument.* Sounding risks a perforation, can initiate bleeding, and provides no useful information. If there is uncertainty of uterine size or orientation, the surgery should be conducted under real-time ultrasound guidance.

The dilator is best used when the surgeon holds it like a pencil (Figure 6.2). To stabilize the dilator and to decrease the risk of perforation, the practitioner's outer three fingers rest gently against the patient's inner thigh. As the cervix is dilated, there is normally a characteristic "snap" of the endocervix around the dilator. This sensation can be missing

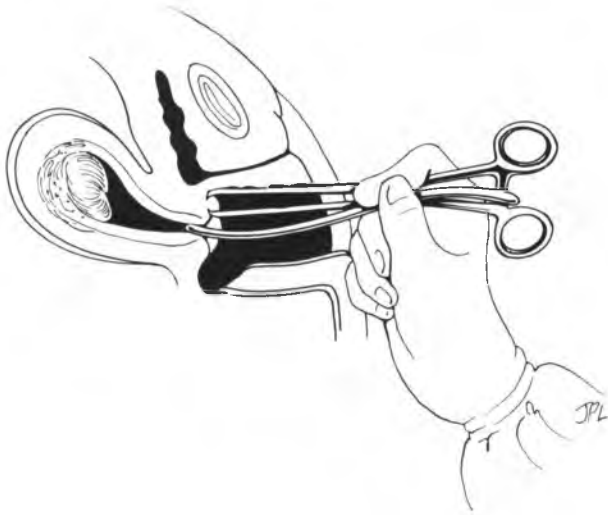


FIGURE 6.2.  
*Suggested technique for cervical dilatation. (See text for details.)*

in women receiving chronic steroid therapy. If the practitioner does not feel this “snap,” a false passage might have been created, and reassessment is in order. Particular care is necessary in patients with a markedly anteverted or retroverted uterus because perforation is more likely in these settings. If there is any question of a false passage, or if the case proves difficult, dilatation is best performed under direct ultrasonic guidance.

Once the cervix is adequately dilated, the designated suction curette is lubricated with a sterile gel and advanced through the cervix with light finger pressure and a slight twist. The diameter of the curette in millimeters equals the gestational age of the pregnancy in weeks. The choice between a curved curette and a straight curette is at the discretion of the practitioner. Some providers advance the suction curette with the tubing attached, whereas others think that for maximal control and safety, the suction tubing should not be attached until after the curette has been successfully advanced into the uterine cavity. The authors favor the latter technique.

Traditionally, suction curettage has been performed by attaching the curette to an electrical vacuum pump. Over the past decade, there has been a steady increase in use of a manual vacuum aspirator. This device is equally efficacious as the freestanding electric pump up to a gestational age of 10 weeks. It has the added benefit of being much quieter and is easily portable. This makes it possible to perform

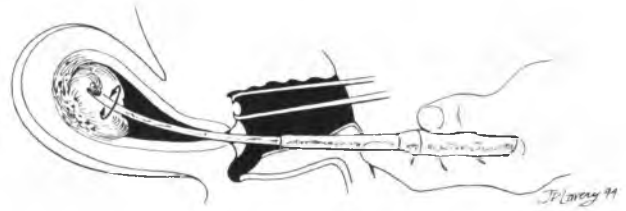


FIGURE 6.3.  
*Technique of suction curettage; first-trimester procedure.*

abortions in an office setting without a great deal of extra equipment [22].

Vacuum tubing or the manual aspirator is next attached to the curette and the vacuum initiated. When a standard vacuum pump is used, once the vacuum reaches 50 mm Hg to 60 mm Hg the curettage can begin. For the initial suction, the curette is positioned in the lower uterine segment. On subsequent passes with the curette, the suction is released, and the curette is gently advanced into the fundus first (Figure 6.3). To decrease the risk of perforation, the cannula is never actively advanced with the suction applied. With the curette positioned at the fundus, the best technique is for the surgeon to pull the curette slowly and gently backward while turning it in the palm of the hand through 360°. The suction is then discontinued when the level of the internal os is reached. Active suctioning of the endocervix unnecessarily traumatizes tissues and increases the blood loss. Only two or three passes should be required to empty the products of conception from a first-trimester uterus. Completeness of the curettage is ascertained by subsequently passing a sharp curette and performing a brief sharp curettage of the entire uterus. If good uterine *cri* is felt and heard, the procedure is complete (Figure 6.4). Some clinicians omit this part of the procedure, believing it to be unnecessary.

With the curettage complete, the instruments are removed from the vagina. Careful attention should be paid to the tenaculum puncture site on the cervix because it is a common site of bleeding. Bleeding usually responds easily to direct pressure. Occasionally Monsel's solution, silver nitrate, or even a suture is needed to control the ooze. A postoperative bimanual examination is always performed to determine uterine size and firmness and to palpate laterally for possible expanding hematomas.

The physician performing the abortion should always conduct a tissue examination at the conclusion of the procedure. Felding and coworkers [23]



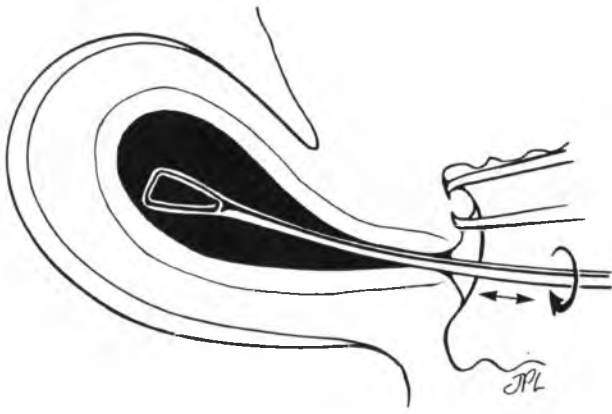


FIGURE 6.4.  
*Technique for sharp curettage; first-trimester procedure.*  
(See text for details.)

suggest that retained tissue (secundines) should be suspected when the volume of tissue recovered after the aspiration is less than 15 ml in the seventh to eighth week, less than 25 ml in the ninth to tenth week, and less than 35 ml in the eleventh to twelfth week of gestation.

Failure to note villi or fetal tissue in the curettings of what was believed to be a first-trimester pregnancy – especially when there is minimal tissue – suggests occult perforation, an ectopic pregnancy, or retained products (secundines) [4,12,18]. In such cases, immediate reassessment is necessary (see Complications). For more advanced gestations, careful study of the products of conception might note absence of major body parts, particularly the cranium, prompting an ultrasound study and re-exploration of the uterus. Whenever the clinician is uncertain if products of conception are retained, or if the products seem complete but bleeding persists, an immediate real-time ultrasound examination is mandatory. When this is performed, retained products, an expanding lateral mass, a hematometrium, or fluid in the cul de sac is easily and immediately identified.

In uncomplicated cases, the patient is observed for at least 20 minutes if the procedure was performed under local anesthesia only, and usually 1 to 2 hours if conscious sedation were used. Once stable, she is discharged to home with a prescription for a nonsteroidal anti-inflammatory/analgesic agent (e.g., ibuprofen or naproxen), an antibiotic, and the birth control method that was agreed on during the initial evaluation. Verbal and written warnings about

signs of infection, hemorrhage, and other complications are given, and emergency contact numbers are reviewed. A follow-up visit is scheduled in 2 to 4 weeks. The administration of postoperative uterotonics is at the clinician's discretion. A common recommendation is for methylergonovine maleate (Methergine) 0.2 mg PO every six hours for four total doses. The regimen for antibiotic treatment follows the protocol of the hospital or clinic.

### Menstrual Extraction

Menstrual extraction (i.e., aspiration, induction, regulation) is the induction of uterine bleeding by minivacuum extraction, usually performed when the menses are delayed up to 14 days [24]. This is an outpatient procedure, and anesthesia is normally not required. If necessary, a paracervical block can be placed as described previously. The equipment required for an aspiration consists of a modified 50-ml syringe and a soft plastic cannula (e.g., Karman cannula). This instrument is 4 mm to 6 mm in diameter and is scored beginning 6 mm to 8 mm from the tip, allowing the practitioner to gauge the depth of insertion.

As with a first-trimester termination, a bimanual pelvic examination is performed prior to beginning the procedure, to ascertain the size and position of the uterus. A speculum is next inserted into the vagina and the cervix is cleansed with an antiseptic solution. The cervix is grasped with a single-tooth tenaculum, or an Allis or Bierer clamp. The Karman cannula is inserted into the cervix in the same way as a uterine sound. Insertion should not exceed 8 cm. If the cannula passes to a greater depth, reevaluation is necessary since perforation is possible or the uterine size has been incorrectly estimated. An aspirating syringe is then attached to the cannula, the plunger withdrawn, and the pinch valve released. A flow of blood and tissue should begin almost immediately. The operator then rotates the cannula through 360° to bring it into contact with the entire uterine cavity. When the active flow diminishes, a back-and-forth scraping motion is begun, resulting in a curette-like effect. When good *cri* is perceived, the procedure is completed. At this point, bubbles usually appear in the cannula, a marker that the uterus has been evacuated. The cannula is not withdrawn through the cervix while vacuum is retained in the syringe; instead, the pinch valve is closed or the

cannula is detached from the syringe. The instruments are then removed, completing the procedure. While currently little discussed among practitioners or in the literature, menstrual extraction remains a controversial procedure. In the past, several arguments were raised against the operation, including the following:

- It is not possible to justify a surgical procedure when the diagnosis of pregnancy is unverified.
- An intrauterine pregnancy is liable to be missed during an extraction.
- Movement of instruments within the uterus is limited, making retained tissue more likely.
- Extraction is more painful than a later abortion.

Many of these concerns derive from studies conducted in the 1970s, before the availability of sensitive pregnancy tests and transvaginal ultrasound scanning. More recent protocols using preoperative and postoperative ultrasound, sensitive HCG assays, and meticulous tissue inspection have demonstrated failure rates of 0.13% to 2.3% and an overall complication rate of 4% with extraction procedures [25,26]. A limited role persists for these operations.

#### Technique: Second-trimester Termination

There are four basic methods for second-trimester termination pregnancy: dilatation and evacuation (D and E), intrauterine instillation of abortifacients, administration of systemic abortifacients, and hysterectomy/hysterotomy.

#### *Dilatation and Evacuation*

There is a common misperception that D and E procedures are simply "big" curettages. Although aspects of the two procedures are similar, a D and E is a more involved surgical procedure with a higher complication rate than either first-trimester curettage or a routine gynecologic D and E. D and E operations should never be attempted by an inexperienced surgeon without immediate expert assistance [27–29]. It should be noted that D and E has the lowest mortality rate of any of the second-trimester termination procedures and is associated with a morbidity rate comparable to the other techniques. Because of its difficulties and risk of serious

**TABLE 6.3** Complication Rates for Mid-trimester D and E\* Procedures and Use of Intraoperative Sonography†

Complication	Without Sonography (n = 353)	With Sonography (n = 457)
Infection <sup>a</sup>	5 (1.42%)	4 (0.88%)
Transfusion <sup>a</sup>	3 (0.85%)	2 (0.44%)
Uterine perforation <sup>b</sup>	5 (1.42%)	1 (0.22%)
Other <sup>a</sup>	7 (1.98%)	6 (1.31%)
All complications <sup>b</sup>	20 (5.67%)	13 (2.84%)

\*D and E = instrumental uterine evacuation.

†Procedures performed 16–24 weeks gestation, same clinic, standard technique.

<sup>a</sup>Difference in rate is not significant ( $p < 0.05$ ).

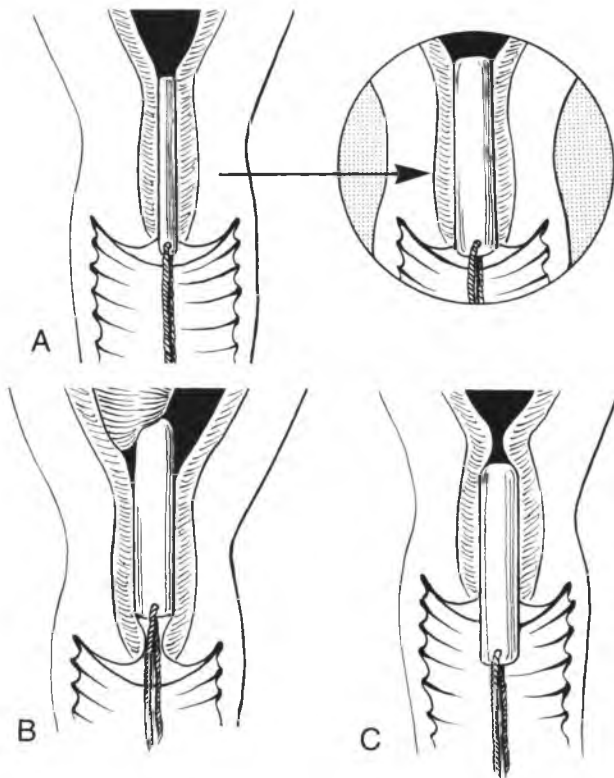
<sup>b</sup>Difference in rate is significant ( $p < 0.002$ ).

From Darney PD, Sweet RL. Routine intraoperative ultrasonography for second-trimester abortion reduces incidence of uterine perforation. *J Ultrasound Med.* 1989 Feb;8(2):71–5; with permission.

complications, the D and E is restricted to practitioners with established experience in the performance of this procedure or to those operating under immediate, expert supervision.

As with first-trimester procedures, a thorough history and physical is mandatory. As it has with so many other aspects of healthcare, obesity recently has been identified as a risk factor for the D and E procedure [30]. Accurate dating is crucial to success and safety in the performance of a D and E. To verify the gestational age, the authors require that patients who are potential D and E candidates have a preoperative real-time ultrasound examination performed within 1 week of the scheduled surgery. The use of real-time ultrasound during the procedure is also important as it shortens the operative time and decreases the risk of complications. In a review of 353 D and E procedures at 16 to 24 weeks performed without ultrasound scan compared with 457 in which sonography was routinely used, Darney and Sweet [33] reported a reduction in perforation rate from 1.4% to 0.2% (Table 6.3). D and E procedures should *never* be attempted without the immediate availability of an ultrasound scanner and someone experienced in the interpretation of mid-trimester ultrasound images [27,31–33].

When a D and E is performed in pregnancies of greater than 12 weeks' gestation, preliminary



**FIGURE 6.5.**  
**Technique for insertion of Laminaria. A, Correct placement; B and C, incorrect placement. (See text for discussion.)**

cervical ripening by either Laminaria insertion or misoprostol administration is advisable (Figure 6.5) [34–37]. In pregnancies of greater than 15 weeks' gestation, such preoperative cervical preparation is mandatory. Pretreatment reduces the operating time, restricts blood loss, and results in fewer complications. Laminaria function by hydroscopic action to slowly dilate the cervix. At present, three types are available: naturally occurring Laminaria tents (*L. japonicum* or *L. digitata*), the magnesium sulfate sponge (Lamicel), and hydrophilic polymer rods (Dilapan). If natural Laminaria are chosen, they should be placed in the cervix at least 12 hours before the procedure is scheduled. The authors generally perform their Laminaria insertions the afternoon of the day preceding the operation. This provides a convenient venue for review of the proposed operation and obtaining of informed consent. The authors routinely administer 600 mg to 800 mg of ibuprofen PO 1 hour before the insertion is performed. Patients should be advised that they must return to have the Laminaria removed and

that failure to do so could result in serious infection or other complications. In some institutions, the patient is required to sign a specific form. The authors employ a standard surgical consent form for Laminaria insertion but also always include a note in the medical record indicating that the woman has been instructed to return the next day and that possible complications of both the contemplated procedure (D and E) and her failure to return for Laminaria removal have been discussed. The magnitude of risk for retained Laminaria is probably not great but we prefer to err on the side of conservative management.

Some practitioners favor the use of synthetic Laminaria because they might have fewer complications and require less time for comparable cervical dilatation [35]. In terms of effectiveness of the synthetic Laminaria, the hydrophilic polymer rods are apparently best. A randomized, prospective, double-blind study of 51 patients undergoing second-trimester abortion, dilatation with magnesium sulfate sponges resulted in a mean French size of  $38.5 \pm 6.4$ , whereas dilatation with the polymer rods resulted in a mean dilatation of  $50.4 \pm 9.6$  [36]. Based on the authors' experience with all types, we favor the continued use of the traditional Laminaria tents finding them to be easy of insertion and removal and clinically effective.

On occasion, the removal of the Laminaria is a problem. Problems with retrieval are quite uncommon with naturally occurring Laminaria tents as long as a false passage has not been made and the tent advanced into the retroperitoneum or other sites, beyond easy retrieval. Artificial Laminaria, however, do swell irregularly, resulting in a variable caliber to the tent. On occasion, this blocks easy removal especially if multiple tents have been inserted. If moderate traction fails to remove a Laminaria, gentle cervical dilatation by passing a small dilator adjacent to the tent will sometimes release it. Care should be taken not to fracture the tip of the tent, avulse the string, or develop a false passage in the cervix. If the effort at removal from the cervix fails, it is best to simply grasp the Laminaria firmly with either a ring forceps or long Allis clamp and advance it into the uterus. The tent is then subsequently retrieved after the termination procedure is completed. Laminaria remaining in utero are easily identified by ultrasound scanning and are usually easily retrieved with a ring forceps. If a Laminaria becomes "lost,"

real time scanning should be performed. If the tent is not within the uterus, laparoscopy may be necessary to remove it. Management of these cases needs to be individualized.

Prior to placing Laminaria, the cervix is cleansed with an antiseptic solution of povidone-iodine or its equivalent. Some practitioners prefer to place a paracervical block prior to the Laminaria insertion. This can be prudent in a particularly anxious woman or if the cervix is tightly closed. Using the “no-touch technique,” Laminaria are introduced into the cervix until only the strings are visible at the external os (Figure 6.5A). When natural Laminaria are inserted, better dilatation is obtained by using several small instead of one large tent, because of the amount of surface area exposed. When working with natural Laminaria they are first lubricated and then grasped with a ring forceps and inserted through the external os using a rotary motion. Tents are placed into the endocervical canal until it is full and additional tents cannot be easily inserted. After 20 weeks' gestation, as many as ten or more natural Laminaria tents can be required [20]. If the cervix is firmly closed, a paracervical block is performed and a small dilator used to open the cervix for the insertion of the tent. Uncommonly, adequate dilatation can require sequential Laminaria treatment with removal of the initial tents and their replacement with new ones. After the chosen number of Laminaria are inserted, and after the removal of the instruments, one or two gauze sponges are placed in the vagina. The physician should document in the medical record the number of Laminaria inserted and the number of sponges placed. The woman is instructed that if any sponges or Laminaria are passed spontaneously, they should be kept and brought to the hospital for examination by a clinician. The patient is then discharged to home. A sleeping medication is prescribed at the clinician's discretion. The woman is instructed to take acetaminophen or ibuprofen for discomfort. If pain is great and is not relieved by oral drugs, if cramping is severe and does not rapidly abate, or if vaginal bleeding occurs beyond that usually seen during a normal period, the woman is instructed to return immediately for examination.

A potential alternative to Laminaria for early second-trimester surgical abortion (13.0–16.0 weeks) is to administer vaginal misoprostol (400 µg 3–4 hours preoperatively). A recent randomized, double-blinded and controlled study has shown

that cervical ripening with same-day misoprostol is substantially inferior to the use of overnight Laminaria, however [37]. The significant differences in efficacy were essentially limited to nulliparous women, however. Importantly, many of the second-trimester terminations performed in this series, regardless of pretreatment, were not challenging for the study's experienced practitioners. Of interest, women in the study generally preferred the misoprostol technique as opposed to the discomforts of overnight Laminaria. Based on these data, there is a role for preoperative vaginal misoprostol especially in multiparous women when an experienced surgeon perceives that the procedure will not be difficult.

On the day of surgery, the patient should arrive for the procedure after at least 6 hours of fasting. Preoperative antibiotic prophylaxis is recommended. Some practitioners begin the prophylactic regimen at the same time as the insertion of Laminaria; others will administer the first dose just prior to the actual surgical procedure. The types of antibiotics and doses recommended are the same as those for first-trimester procedures: either doxycycline 100 mg PO one hour prior to surgery followed by 200 mg PO following the procedure, or doxycycline 100 mg BID for three days or metronidazole 500 mg PO twice daily for 5 days [38]. An intravenous access line is inserted. During the procedure, drugs for intravenous analgesia and relaxation are administered, as required (see Table 6.2). The woman is then positioned in the dorsal lithotomy position, and the Laminaria and sponges are removed. As part of the preoperative evaluation, a pelvic examination is always performed before beginning the surgery, to assess the uterine size and orientation. The cervical dilatation is assessed by direct observation and bimanual examination. A paracervical block is next placed in the manner described previously. The addition of 2 IU to 4 IU of vasopressin to the local anesthetic is a safe method of decreasing postoperative blood loss [39].

The dilatation is then completed using Pratt or Hawkins-Ambler dilators. The extent of required dilatation depends on the size of the suction cannula needed for the estimated gestational age of the pregnancy. At 13 to 15 weeks, evacuation can be readily performed with vacuum cannula of 12-mm to 14-mm diameter. For gestation of 16 weeks or beyond, it is best to dilate sufficiently to insert the

largest cannula available (16 Fr in the authors' institution).

The cannula selected is then lubricated and passed through the cervix with a slight twist and then slowly advanced into the lower uterine segment. During a D and E it is best not to routinely introduce the suction cannula into the upper uterus without the concomitant use of ultrasonic guidance because the risk of perforation is too great. When the suction tubing is attached to the cannula and the suction started, there is initially a gush of fluid, reflecting membrane rupture. This is often followed by the cord prolapse into the cannula and then by the aspiration of placental tissue. The suction cannula is subsequently rotated in the lower uterine segment to remove as much of the easily removed products of conception as possible.

Once the membranes are ruptured, if misoprostol was not used preoperatively, 0.2 mg of methylergonovine maleate (Methergine) is administered IM. Alternatively, an infusion of IV oxytocin (50 units–100 units/L at  $\geq 150$  ml/hr) is begun at the time the cannula is inserted or immediately before to ensure uterine tonus. Firm contractions of the uterus induced by the uterotonic advances tissue from the upper uterus toward the lower uterine segment, where the cannula is positioned to remove it. The use of uterotonics limits blood loss, provides a firmer uterine wall, and reduces the need to advance instruments high into the uterus to retrieve products of conception. Collectively, these effects reduce the morbidity of the operation, specifically the risk of uterine perforation, and probably also limit blood loss.

In more advanced gestations, the calvarium and the spinal column are too large for the suction cannula to extract. The Sopher forceps, which have serrated jaws but no lock, should be employed for this stage of the procedure. This instrument is inserted into the lower uterine segment in a closed position and then opened, grasping fetal or placental fragments that the suction cannula has drawn into the lower segment. The extraction is completed in this fashion, preferably under real-time ultrasound guidance. If after examination of the extracted tissue there is any question whether the calvarium or other products of conception have been removed, immediate re-scanning guides the surgeon to any residual intrauterine material. If ultrasonography has not been used routinely during the case, if at any time

the extraction proves difficult or if the blood loss becomes excessive, it is prudent to complete the D and E procedure under direct ultrasonic guidance.

When the procedure is believed to be complete, some practitioners follow the suction curettage with a sharp curettage, using the largest possible curette. Other experienced practitioners believe that suction curettage is sufficient [35]. When the operation is over, the patient is then observed for several hours and, once stable, discharged to home. Most practitioners subsequently administer methylergonovine (Methergine) 0.2 mg PO every 6 hours for two to four doses to patients undergoing mid-trimester procedures unless there is a history of hypertension or another contraindication. As noted, the authors routinely administer antibiotics such as doxycycline to D and E patients unless there is a specific history of allergy. A birth control method and a prostaglandin-inhibiting analgesic are also routinely offered. Before discharge, all patients are carefully counseled concerning possible complications, including hemorrhage, fever, or unusual discomfort. Repeat clinical examination in 10 to 14 days is prudent. The patient's blood type is carefully rechecked prior to her discharge, and Rh immune globulin is administered intramuscularly, as indicated.

### *Instillation Methods*

Instillation procedures are now infrequently performed and are usually restricted to cases in which evaluation of the intact fetus is desired. The preprocedure evaluation is the same as described previously. Laminaria are inserted on the day before the procedure to shorten the length of time from instillation to delivery, and an antibiotic such as doxycycline is administered. Instillation methods are usually combined methods of pregnancy termination. Several protocols exist, with none clearly preferable. Serious complications are possible whenever hypertonic substances are injected into the uterus, and these techniques must be performed by only experienced personnel or under the direct supervision of experienced personnel.

After pretreatment with Laminaria, an amniocentesis is performed. Then 100 ml to 200 ml of 30% saline or a similar volume of hypertonic urea is instilled into the uterus. After the intrauterine instillation, graded and increasing doses of oxytocin are subsequently infused intravenously. Prostaglandin

**TABLE 6.4 Mid-trimester Pregnancy Termination Installation Protocol\*****Initial Procedures**

- Pack the cervix with Laminaria the afternoon/evening before admission; administer oral antibiotics.
- After admission, perform amniocentesis, aspirating as much amniotic fluid as possible.
- Inject a test dose ( $\leq 20$  ml) and then a total of 100 ml 23.4% NaCl (or hypertonic urea)

**Subsequent Dosing postinstallation**

- At 2 hr: After the amniocentesis, begin intravenous oxytocin 50 IU, 500 ml NS or RL at 170 ml/hr (i.e., to run over).
- At 5 hr: discontinue oxytocin and insert a 20-mg PGE2 suppository, removing the Laminaria unless previously expelled or removed.
- At 6 hr: resume intravenous oxytocin with 100 IU/500 mL of NS or RL at 170 ml/hr.
- At 9 hr: again discontinue oxytocin and repeat the E2 vaginal suppository.
- At 10 hr: resume oxytocin with 150 IU / 500 mL of NS or RL at 170 ml/hr. Thereafter add an additional 50 IU oxytocin in each subsequent IV bag to a maximum of 200 IU until abortion occurs.
- If abortion does not occur after a total of 12–14 hours of treatment, stop and reevaluate for possible D and E or other management.

\* This is only one of several possible installation protocols; see text for additional discussion.

NS = normal saline; RL = Ringer's lactate.

E2 suppositories are administered in a set protocol (Table 6.4). Patients who do not deliver after 12 to 14 hours of treatment are reevaluated and can be taken to the operating room for a D and E under real-time ultrasound guidance. Whereas instillation methods were once a common method of second-trimester pregnancy termination, they have largely been abandoned because of development of safe and effective systemic agents and the newer D and E operative techniques [40].

**Hysterotomy**

Pregnancy termination by hysterotomy or hysterectomy is rarely performed [41]. Indications for this operation are rare. These procedures have a substantially increased morbidity when compared with either the installation or the D and E techniques described previously and must, whenever possible, be avoided. Potential indications might include

failed induced abortion when a D and E cannot be safely performed, suspected uterine rupture, unusual cases combining sterilization with the need to treat other intraperitoneal disease, or surgery to protect an abdominal or high Shirodkar cerclage. Elective hysterectomy during pregnancy is rare and usually reserved for cases of coexisting trophoblastic disease or cervical cancer when fertility is not an issue.

**Medical Methods**

Knowledge of nonsurgical methods of pregnancy termination dates to ancient times, and such techniques were well established by the time of the Renaissance. One such example is the seeds of Queen Anne's lace, or wild carrot. Hippocrates, among others, declared that this botanical would both prevent and terminate pregnancy when taken orally [1]. Even today, a small number of women in Watauga County, North Carolina, drink a glass of water containing a teaspoonful of Queen Anne's lace seeds immediately after intercourse to prevent pregnancy. In 1976, seeds from Queen Anne's lace given to mice very early in pregnancy were reported to prevent fetal growth, and in 1986 chemical compounds in the seeds were noted to block the production of progesterone [1].

In modern times, the development of orally effective antiprogesterones, specifically mifepristone (RU-486), has contributed to the polarization of the two sides of the abortion debate [42–52]. The antiabortion movement's view is that this type of medical abortion sets a new threshold in trivializing abortion, whereas those who would prescribe this drug view the safety, proven efficacy, and limited side effects as significant benefits of this method.

Mifepristone is a synthetic 19-norsteroid with potent antiprogesterone activity that, when administered early in pregnancy, functions as an abortifacient. Its competitive inhibition of endometrial progesterone receptors leads to sloughing of the endometrium. Although the decidua undergoes necrosis, the trophoblast is unaffected. RU-486 also stimulates prostaglandin production by the myometrium, initiating contractions.

In September 2000, the FDA approved a regimen of mifepristone and misoprostol for medication abortion in the United States. Since then, almost 500,000 women have chosen this option, according

TABLE 6.5 Contraindications to Use of Mifepristone\*

- Confirmed or suspected ectopic pregnancy or undiagnosed adnexal mass<sup>†</sup>
- Medical complications
  - Porphyria
  - Chronic adrenal insufficiency
  - Hereditary coagulation disorders
  - Treatment with anticoagulants
  - Treatment with corticosteroids
- IUD in utero
- History of allergy to mifepristone, misoprostol, or other prostaglandins

\* See text for discussion.

<sup>†</sup> RU-486 treatment alone is insufficient to safely terminate an ectopic pregnancy.

to the American distributor of mifepristone (Danco Laboratories). The FDA approved regimen consists of 600 mg (three 200-mg tablets) of mifepristone PO on day 1, followed by 400  $\mu$ g of misoprostol PO on day 3. There is substantial evidence published since the data were submitted to the FDA showing equal (or improved) effectiveness with changes to the regimen. A commonly utilized treatment protocol consists of a single dose of 200 mg mifepristone on day 1, followed by 800  $\mu$ g of misoprostol taken vaginally on day 2, 3, or 4 up to 63 days from the last menstrual period [44,45]. This regimen has been reported to be between 96% and 98% effective (Table 6.5).

In recent years a number of investigators have studied alternative methods of treatment with mifepristone/misoprostol in the effort to extend the applicable gestational age range and simplify the dosing. Such regimens have used varying doses of mifepristone or misoprostol and studied decreased dosing intervals between the two drugs [46–52].

Medical contraindications to the other agent in the oral abortion regimen, misoprostol, include allergy to the prostaglandins or an uncontrolled seizure disorder. In addition to the strictly medical issues, one must also consider the woman's comfort level. With oral treatment, cramping and bleeding occur at home. The woman must be aware of the discomforts and have access to a health facility in case of a hemorrhage or another acute problem. Patients choosing this method should be provided with pain medication such as ibuprofen or acetaminophen, or a mild narcotic such as acetaminophen with codeine before her discharge to home. Common side effects of oral treatment include cramping, bleeding, and

nausea. Detailed training on medical abortion is readily available and strongly recommended for all providers who wish to prescribe mifepristone.

In the years since the approval of RU-486 in the United States, there have been at least four deaths from *Clostridium sordellii* sepsis following abortion with the combination of mifepristone and misoprostol [53]. The FDA tests of the medications for contaminants have been negative. Of significance is the fact that women with *Clostridium sordellii* endometritis often have pain, nausea, vomiting, and foul-smelling discharge but do not have fever or elevated white blood cell counts. How these recently reported cases should affect current treatment protocols is unclear. The vaginal administration of any of the abortifacients will likely be suspended, although the relationship of mode of treatment to this rare complication is unknown. The most common response to these reports has been to add prophylactic antibiotics to the abortion regimen. Unfortunately, the signs and symptoms for these rare fatal infections are essentially indistinguishable from those routinely reported with the use of the medications alone. Furthermore, it is currently unknown whether changing the mode of drug administration or administering antibiotics will prevent this completion. Presumably, certain women harbor this clostridial bacteria in the birth canal, and the abortion process opens an opportunity for ascending infection.

Mid-trimester pregnancy termination by medical as opposed to surgical methods is also possible. All of the existing regimens include a prostaglandin. There are three different prostaglandin compounds readily available in the United States: dinoprostone (prostaglandin E<sub>2</sub>), carboprost tromethamine (Hemabate), and misoprostol (Cytotec). Dinoprost 10 mg every 6 hours combined with high-dose oxytocin is efficacious for mid-trimester abortion and has fewer of the gastrointestinal side effects seen in higher-dose prostaglandin E<sub>2</sub> regimens [54]. Carboprost tromethamine 250  $\mu$ g IM every 2 hours leads to mean induction to abortion times of 15 to 17 hours, with most patients aborting within 24 hours.

The first study of misoprostol for second-trimester abortion was published in 1994 [55]. In that study, a dose of 200  $\mu$ g of misoprostol administered vaginally every 12 hours was compared with dinoprostone 20 mg every 3 hours. These drugs were equally efficacious, but misoprostol was associated with fewer side effects. Although higher doses

at 12-hour intervals result in shorter induction to abortion times, the reports of nausea, vomiting, and fever increased proportionally [56]. Su and coworkers reported that vaginal misoprostol (400 mg every 3 hours) is more effective than intraamniotic carboprost. In this study, fever and shivering were the only adverse effects that were more common with misoprostol than with the carboprost [57]. They also noted that beside the 30-fold higher cost of carboprost versus misoprostol, the use of intraamniotic carboprost also mandated especially trained medical personnel and the use of ultrasound equipment, leading to additional cost.

In an excellent review of 1,002 consecutive cases, published before the issue of infection was apparent, Ashok and coworkers demonstrated that mifepristone with misoprostol proved to a safe, effective method of second-trimester pregnancy termination, with a mean induction to abortion interval of 6.25 hours, with 98.3% of patients having aborted within 24 hours [58]

### OTHER AGENTS

As methotrexate is cytotoxic to trophoblast, it has been successfully used for several years in the medical treatment of ectopic pregnancy [75]. Methotrexate is also effective in the termination of intrauterine pregnancy but is uncommonly used for this indication. Its use as an abortifacient was described as early as 1952 [59].

In a recent study of 10 women at  $\leq 42$  days' gestation who were treated with methotrexate 50 mg/mm<sup>2</sup> IM, this drug alone was sufficient to abort very early intrauterine pregnancies [59]. Owing to the potential toxicity of methotrexate and the current availability of other, safer drugs, this compound is rarely administered for intrauterine pregnancies. Currently, methotrexate is essentially restricted to the treatment of ectopic pregnancies, including the rare cervical pregnancy [75]. Another potential use for methotrexate is when placental tissue is retained, which occurs in circumstances when the placenta is not entirely removed, such as abdominal pregnancy, or in placenta accreta, increta, or percreta, when large segments of the placenta may be left behind.

### COMPLICATIONS

Abortion-related morbidity is difficult to measure because there are no systematically collected

**TABLE 6.6 Potential Complications of Surgical Pregnancy Termination Procedures\***

- 
- Uterine perforation
  - Injury to bowel/bladder/vessels
  - Cervical laceration
  - Pulmonary embolism
  - Sepsis
  - Hemorrhage/anemia
  - Hematometrium
  - Requirement for repeat curettage, D and E, or laparotomy
  - Secundines/endometritis
  - Coagulopathy
    - amniotic fluid embolism
    - DIC, excessive blood loss
- 

\*See text for details.

DIC = disseminated intravascular coagulopathy.

national surveillance data. Hospital admission is the most commonly used marker for complications [60]. The Centers for Disease Control and Prevention define major complications from induced abortion as those that result in a major unintended surgery, a hemorrhage requiring a blood transfusion, a hospitalization of 11 days or more, or a temperature of at least 38.0°C (100.4°F) that lasts for 3 or more days. This section reviews complications, their management, and their sequelae (Table 6.6).

#### **Uterine Perforation**

The most common operative complication of pregnancy termination is uterine perforation (Figure 6.6). Perforation increases the risk of abortion-related death from infection more than 100-fold, and that from hemorrhage more than 1,000-fold [61,62]. There is a greater likelihood of perforation if the physician is inexperienced, if the uterus is retroverted, or if the gestational age is advanced. The use of general anesthesia has also been associated with a slight increase in the risk of perforation. Perforation is suspected when a curette or forceps easily passes through the cervix without apparent resistance, passes farther than it did given the patient's preoperative examination (based on a previous uterine entry), or a sudden vaginal hemorrhage occurs during instrumentation. Less commonly, perforation is diagnosed when an awake or light sedated patient reports the sudden onset of generalized abdominal pain during a procedure. In some cases, omental fat or even bowel is identified



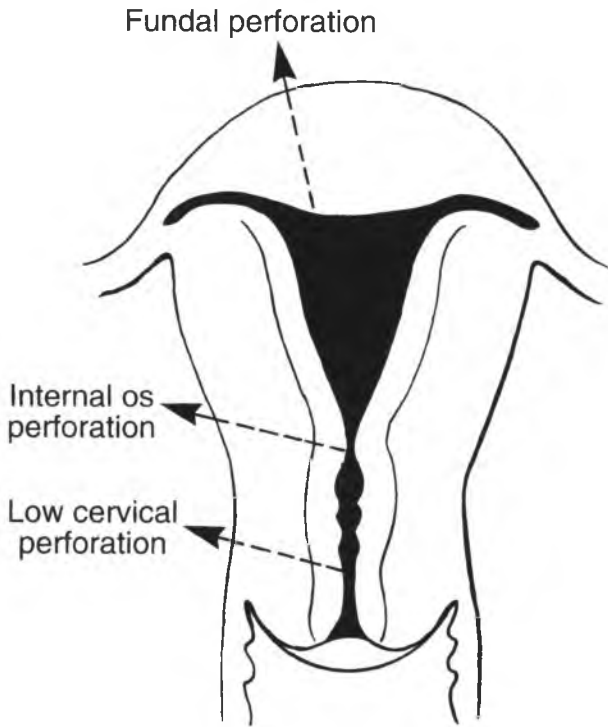


FIGURE 6.6.  
*Potential sites for uterine perforation during pregnancy termination. (See also Figs 6.7 and 6.8.)*

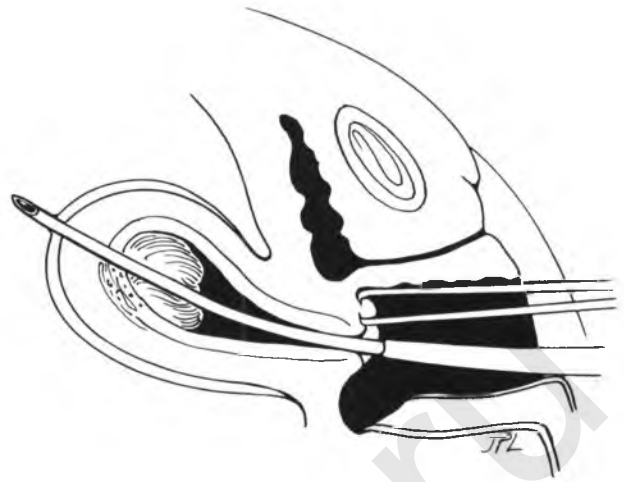


FIGURE 6.8.  
*Uterine fundal perforation with suction curette without bowel injury. (See text for discussion.)*

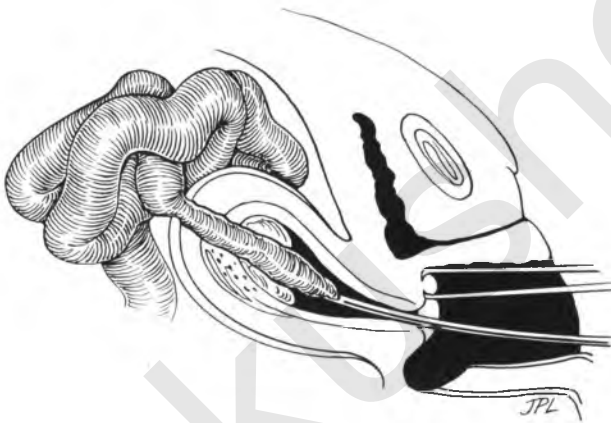


FIGURE 6.7.  
*Uterine fundal perforation with suction curette with small bowel drawn into the uterine cavity.*

in the curetted material, indicating that intraperitoneal structures have been injured (Figures 6.7 and 6.8). The authors recall a patient sent to the emergency department from a local abortion clinic with the appendix drawn through the cervix and protruding into the vagina!

The incidence of uterine perforation during first-trimester abortion remains a matter of some debate. Kaali and coworkers reported that the incidence varies between 0.8 and 6.4 per 1,000 procedures [62]. In a 1990 review of 170,000 first-trimester abortions, Hakim-Elaki [63] documented 16 cases (0.009%), with only one case resulting in laparotomy for a suspected bowel injury. These latter data were obtained from a clinic that employs experienced personnel and not from a teaching program, however.

Management of a perforation in which bowel is drawn into the uterus or cervix is straightforward. The loop is left in place and a laparotomy is performed. At surgery the pelvic structures, peritoneal contents, and bowel are carefully examined, and all injuries are repaired. Postoperatively, transfusions are administered if required, and broad-spectrum antibiotics are given. The authors have not favored laparoscopy for these explorations owing to the limitations in examining the bowel and omentum closely.

If a perforation is recognized and the curettage is incomplete, the operation should be completed under direct laparoscopic or continuous real-time ultrasonic guidance. If the perforation is recognized at the end of a completed procedure, and there is minimal bleeding, and the perforation is fundal, not lateral, and no bowel was drawn into the vagina, the patient can simply be observed for several hours. Serial postoperative pelvic examinations

and real-time ultrasonic study are recommended to help in the early recognition of a broad ligament hematoma. Serial determination of the hematocrit is also prudent. If the termination was completed and the patient is stable, she may be discharged on an oral methylergonovine series and broad-spectrum antibiotics, with careful instruction. These patients should be contacted in 24 to 48 hours to review their conditions. Reexamination should take place within a week or earlier if problems ensue.

When hemorrhage accompanies uterine perforation, the site of injury is usually the lateral uterine wall (see Figure 6.6). Perforations at the junction of the cervix and lower uterine segment can lacerate the ascending branch of the uterine artery, resulting in severe pain, the rapid development of a broad ligament hematoma, or sudden and potentially severe vaginal, intraperitoneal, or retroperitoneal bleeding. Low cervical perforations can injure the descending branch of the uterine artery, resulting in bleeding through the cervical false passage. A vascular injury is normally managed successfully with laparotomy and vessel ligation. Rarely, hysterectomy is necessary for control. A potential alternative to hysterectomy is arteriography with selected embolization of the injured vessel. Such serious complications are all rare, however.

### Infection

The most common complication following induced abortion is pelvic inflammatory disease (PID). The basic cause is operative contamination of the endometrium. In a randomized, prospective study of 68 women having first-trimester abortions, Jonasson and coworkers [15] showed that microflora from the lower genital tract are transferred into the uterine cavity in two thirds of patients who were instrumented despite thorough cleansing of the vagina and external os with a standard antibacterial solution. Although the rate of infection is low, it is of concern. Pelvic infection can lead to serious sequelae including sepsis, abscess formation, and permanent infertility because of tubal injury [64,65]. A fever (38°C or higher) within 72 hours of an abortion is usually the marker for infection. Vaginal bleeding is also a common complaint and occasionally can be marked. In cases of severe infection, there can be a purulent discharge and accom-

panying abdominal/pelvic pain. Once a fever is noted, a complete physical examination should be performed to evaluate the pelvis and to exclude other sources of infection. When endometritis is present, the uterus is usually enlarged, boggy, and tender to cervical motion and direct palpation on pelvic examination. Cultures of the blood, urine, and cervix are obtained prior to beginning antibiotics. In these cases, retained products of conception should always be suspected and sought by ultrasound examination. If present, uterotonics and broad-spectrum antibiotics are immediately administered, and a repeat curettage should be performed under real-time ultrasonic guidance. The antibiotics are continued intravenously until the patient has been afebrile for 24 hours.

The women who presents with fever and a small, firm, or slightly tender uterus can have cultures taken and can be treated with intravenous broad-spectrum antibiotics without curettage if a real-time ultrasound scan fails to identify secundines. In this situation, the volume of intrauterine material is small to nil, and a curettage might not speed resolution but simply further traumatize the endometrial cavity. If such a patient does not respond within 12 to 24 hours of antibiotic therapy with prompt disappearance of pain, fever, and bleeding, curettage is indicated, however.

In postabortion infection, the inoculum is usually polymicrobial and includes endogenous perineal or vulvovaginal flora. Common genital pathogens such as *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, or *Mycoplasma hominis*; *Ureaplasma urealyticum* can also be present. In terms of treatment for commonly encountered genital tract pathogens, some authors suggest the use of multiple antibiotic regimens (e.g., combined ampicillin, gentamicin, and clindamycin), whereas others advocate the use of the newer broad-spectrum cephalosporins, or a combination of penicillin/clavulanic acid (Augmentin) and ampicillin/sulbactam (Unasyn). The choice is best based on local experience with pelvic infection.

### Failure to Empty the Uterus

The postabortion patient who presents within several days of the procedure with fever and a boggy uterus is considered to have infected retained products of conception. They are managed surgically

in the manner previously discussed. In contrast, women who are not grossly infected but do have retained products of conception remain afebrile and usually present at an interval of several days complaining of cramping and some degree of bleeding. On examination, the uterus is usually slightly and often only moderately tender. Real-time ultrasonic scanning will identify irregular, strongly echogenic material within the uterine cavity, at times interspersed with small fluid collections. In these cases, if bleeding has been a persistent problem, the volume of secundines is estimated to be large, infection is suspected for any reason, or symptoms did not improve under prior medical management, a repeat curettage is indicated. In contrast, if the bleeding is not severe, the amount of intrauterine material is small, and if infection is not believed to be established, outpatient medical management can be attempted in selected cases. This includes treatment with a potent uterotonic (e.g., 0.2 mg of methylergonovine (Methergine) PO every 6 hours for four to six doses) combined with broad-spectrum antibiotics. These women should be reevaluated within 48 to 72 hours for symptoms and signs suggestive of nonresolution such as bleeding, fever, and uterine tenderness. If these findings are present, curettage should be promptly performed. The authors generally do *not* recommend this approach to treatment, but there are circumstances when an outpatient regimen is necessary or appropriate, such as when surgical treatment is refused.

More problematic is the woman who presents a week or more after an abortion complaining of persistent spotting but without other symptoms. In these cases, real-time ultrasonic scanning might identify retained products. If there are secundines present, the best treatment is usually repeat curettage. If secundines are not identified, other causes of spotting should be considered, for example, breakthrough bleeding on the birth control pill or following administration of medroxyprogesterone acetate (Depo-Provera). For these patients, as secundines are not noted and the usual signs and symptoms of infection are not present, the best therapy is unclear. The most common initial therapy is to repeat the oral methylergonovine/doxycycline regimen and reevaluate in 7 to 10 days. Alternatively, an endometrial biopsy can be performed to guide management. Either approach is acceptable.

## Hemorrhage

Hemorrhage is uncommon during or following first-trimester abortion. Fewer than 1% of first-trimester patients experience a blood loss of more than 25 ml (64). In women undergoing a second-trimester termination, hemorrhage requiring transfusions occurs in 0.3% of D and E procedures and in 3% of saline/prostaglandin instillations [34]. Rarely, hemorrhage during an abortion is dramatic and even life-threatening. In such instances, rapid and systematic evaluation is required, because prompt intervention is mandatory. The differential diagnosis includes uterine atony, perforation with laceration of the uterine artery, a low-lying placental implantation site, a previously unsuspected coagulation disorder, and most rarely, an amniotic fluid embolism.

For a large majority of cases the initial and usually successful management of sudden hemorrhage consists of prompt completion of the procedure followed by removal of the speculum from the vagina and vigorous bimanual massage. Additionally, a liter of normal saline or Ringer's lactate with 60 to 100 units of oxytocin IV is infused briskly, by infusion pump if necessary. If there are no contraindications, the patient is also administered 0.2 mg methylergonovine maleate (Methergine) or 0.250 mg 15-methyl-prostaglandin F<sub>2α</sub> (Hemabate) intramuscularly or intracervically. If an initially boggy uterus firms quickly, the problem is probably atony and the prognosis for control is good. If the uterus was firm to begin with, however, or if after firming, brisk bleeding persists, the most likely diagnosis is either retained products, a low-lying placental implantation site, or a perforation. In this setting, a repeat curettage is performed as quickly as possible under direct real-time ultrasonic guidance. Bleeding that continues briskly in the face of a well-contracted and empty uterus must be suspected to be due to a perforation or laceration of either the uterine artery or its cervical branch. If a perforation is present, compression, uterotonics, or recurettage will not stop the bleeding. Real-time ultrasonic scanning might document the rapid development of a hematoma immediately adjacent to the uterus. When such unremitting hemorrhage occurs, the patient must be promptly transported to a fully equipped operating room for surgical exploration or other definitive treatment.

While the initial management steps including emptying the uterus, slowing the blood loss with uterotonics, replacing circulating volume, and restoring coagulation competence with blood or blood products are usually successful, rarely there are other problems requiring special treatment. If a woman experiences profuse bleeding during an abortion that is difficult to control or subsequently begins to bleed from mucous membranes or needle sites, a previously undiagnosed hereditary coagulation defect or an acquired coagulopathy must be considered in the differential diagnosis. A tube of whole blood for clot observation should be drawn, along with the standard studies for prothrombin, an activated partial thromboplastin time, platelet count, fibrinogen, and fibrin degradation products along with a CBC. If the tube of whole blood retained by the clinician fails to clot within 5 to 7 minutes or a clot forms that dissolves, it is highly likely that a coagulation defect has developed. Additional baseline studies include hemoglobin and hematocrit levels. Depending on the severity of the situation, therapy with fresh-frozen plasma, platelets, cryoprecipitate, or packed red blood cells might be required. Except in most unusual circumstances, coagulation deficits following abortion are due to the excessive loss of blood with coagulation factors. In this situation, the rapid replacement of appropriate fluids (crystalloids, blood and blood products) suffices for control. If severe or combined coagulation deficits are present, especially if maternal cardiovascular collapse occurs or seems imminent, additional specialized treatment is required and rare complications such as anaphylaxis, amniotic fluid or pulmonary embolism or sepsis need to be considered. In serious cases, immediate consultation with a hematologist or intensivist is prudent.

### ***Postabortal Syndrome (Hematometrium)***

A hematometrium occur after approximately 1% of surgical abortions, producing characteristic acute symptoms. This condition is due to the rapid collection of blood within the uterine cavity at a time when cervical closure blocks its exodus. As the blood trapped within the uterine cavity cannot escape, the uterus rapidly becomes distended and acute symptoms ensue. The patient usually presents within the first 24 hours after the procedure, often within an

hour of the abortion. The primary symptoms are severe and usually constant pelvic and lower abdominal pain accompanied by little or no vaginal bleeding. Occasionally the complaint may be that of sudden severe pelvic cramping. As these women are evaluated by the clinician, the differential diagnosis must include occult uterine perforation and early infection.

Pelvic examination reveals an enlarged, globular, and acutely tender uterus. Depending upon the interval of time since the original surgery, the cervix might be open, with palpable clot in the endocervix or lower uterine segment. A real time ultrasound examination will document a distended uterus, usually filled with densely echogenic material (clotted and partially clotted blood). Immediate surgical evacuation of the uterus, perhaps involving as simple an intervention as passing a small dilator or a ring forceps through the cervix, provides relief. Probing or limited dilatation usually releases a gush of retained clot and liquid blood (hematometrium). If a surgical evacuation is not immediately available or is delayed, misoprostol 400 µg to 800 µg combined with a potent analgesic can be administered. This treatment is occasionally successful in emptying the uterus and arresting the acute symptoms. To avoid masking another problem, nonsurgical treatment should be used with care, however, and only after the exclusion of other potentially serious postoperative complications. In very unusual cases the problem might recur, requiring reinstrumentation.

### **Psychological Complications**

There is a large body of literature about pregnancy termination and its psychological sequelae. Unfortunately, many of these studies are flawed by small sample size, poor study design with lack of appropriate controls, or poor follow-up. Other reports are unduly biased by the author's political or moral beliefs [66,67]. In 1989, after reviewing more than 250 studies of the emotional aftermath of abortion, former Surgeon General C. Everett Koop testified before the United States Congress that the data were "... insufficient ... to support the premise that abortion does or does not produce a postabortion syndrome" [60]. In related testimony at these same hearings, Nancy E. Adler, PhD, stated that "... if severe reactions were common, there would be an

epidemic of women seeking [mental health] treatment. There is no evidence of such an epidemic" [60]. The weight of evidence from several scientifically valid studies indicates that although there are sensations of regret, sadness, or guilt after a termination, legal abortion of an unwanted pregnancy in the first-trimester does not pose a psychological hazard for most women [9,65–67]. After abortion, the major reaction for most women is relief, with an accompanying decrease of stress, despite their sense of loss. Factors that correlate with higher rates of psychological distress after abortion include medical or genetic indication for the abortion, previous psychiatric contact, perceived lack of social support, second-trimester abortion, and ambivalence about the abortion decision. Most women who obtain second-trimester abortions do not do so out of a lack of awareness of their pregnancy but because they are markedly ambivalent and procrastinate in seeking help. Late presenters are more likely to have initially accepted the fetus and regard it as a potential child, thus adding to the psychological stress of the abortion. These patients should be closely monitored postoperatively and should be offered postabortion counseling when needed. In cases in which abortion was initially denied by one clinic or provider, up to 40% of the women eventually obtain abortions elsewhere. Relatively few women who are denied abortion put their babies up for adoption. About one third of these women report negative feelings toward the child and difficulty adjusting [66]. Children in this setting may exhibit long-lasting, broad-based negative effects, including a more insecure childhood, greater need for psychiatric care, more childhood delinquency, criminality, and lower emotional maturity.

### Continued Pregnancy

Failure to interrupt an intrauterine pregnancy occurs in fewer than 0.5% of suction-curettage patients. This complication is most likely to occur in abortions performed before 6 weeks' gestation [12,64]. During the period 1971 through 1985, the incidence of ectopic pregnancy concurrent with induced abortion was 1.35/1,000 induced abortions, and 24 women died as a result of this complication [68]. Such problems are best avoided by careful tissue examination post curettage, histologic examination when needed, and careful follow-up of suspected

cases with serial pelvic examinations and quantitative hCG levels. The risk of a spontaneous combined intra- and extrauterine (heterotopic) gestation is estimated as approximately 1/30,000 pregnancies [69]. The use of techniques of induced ovulation and embryo replacement increases the heterotopic risk.

### Postcoital Contraception

The failure of physicians to counsel their patients as to the availability and efficacy of emergency postcoital contraception is tragic, in consideration of the consequences. All sexually active women should be informed of these methods, and a simple protocol should be explained to the patient in the event that an episode of unprotected intercourse occurs. Such education should be a part of abortion counseling as well.

To recall normal reproductive physiology: unprotected intercourse 3 days before ovulation results in pregnancy about 20% of the time. The pregnancy risk is about 25% 1 day before ovulation, and about 15% on the day of ovulation. More than 2 days after ovulation, the probability of pregnancy drops to near zero [68]. Interruption of implantation by steroid administration is the method used in currently available postcoital contraceptives. It has been known for many years that high-dose estrogen prevents implantation of the fertilized ovum in experimental animals, but the exact mechanism of action remains unknown.

Medical emergency contraception consists of the prompt oral administration of potent hormonal substances to block implantation. This regimen is now packaged and sold under the trade name "Plan B." The FDA-approved course of treatment consists of two doses of levonorgestrel (0.75 mg) taken 12 hours apart, commencing within 72 hours of unprotected intercourse. Postcoital treatment with this dosing protocol has been shown to be more effective (85%) than the original Yuzpe regimen (57%), which used levonorgestrel with ethinyl estradiol [70–72]. New studies have demonstrated the equivalent effectiveness of taking both pills at once within 120 hours of intercourse [73,74]. This simplified regimen allows more women the chance to prevent an unintended pregnancy. Careful counseling is necessary, and a follow-up pregnancy test is prudent to ensure that implantation has not occurred if

characteristic pregnancy symptoms or amenorrhea develop despite treatment.

## CONCLUSION

The subject of pregnancy termination engenders major legal, social, and religious turmoil. Those opposed to abortion might well view any medical interference with early conception as equivalent to murder. This position includes a moral condemnation of those who participate in pregnancy termination procedures. What is frightening for physicians is the fact that some zealous antiabortion advocates have been willing to commit felonies to impress their views on others.

As a licensed professional, the physician is legally delegated the responsibility for helping women make decisions regarding both their fertility and the termination of pregnancy. In this role, the doctor becomes a facilitator who helps a woman to understand how these decisions will influence her reproductive health.

Abortion procedures have heretofore been cloaked with constitutional protection that has permitted procedures performed prior to viability to continue with limited state interference. Heretofore the public has been consistently ambivalent concerning abortion but has not favored eliminating this option; however, the political support for termination practices is progressively unfavorable, and some additional restrictions on the performance of abortion is probably inevitable. In our opinion, physicians must continue to be able to provide safe termination procedures to women or be willing to refer them to those who will.

## REFERENCES

- Riddle JM, Estes JW, Russell JC. Ever since Eve ... birth control in the ancient world. *Archaeology*. 1993;47:29–35.
- Shain RN. A cross-cultural history of abortion. *Clin Obstet Gynecol*. 1986;13:1–17.
- Kunis H, Rosenfield A. Abortion: A legal and public health perspective. *Annu Rev Publ Health*. 1991; 12:361–382.
- Statistics and Computer Resources Branch, Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control. Abortion surveillance: Preliminary data – United States, 2003. *MMWR*. 2005;54:1–31.
- Henshaw SK. The accessibility of abortion services in the United States. *Fam Plann Perspect*. 1991; 23:246–252.
- Kaali SG, Szigetvari I, Bartfai A, Feinman M. Laparoscopic sterilization combined with dilation and evacuation up to 18 weeks' gestation. *J Reprod Med*. 1989; 34:463–464.
- Landy U. Abortion counseling: A new component of medical care. *Clin Obstet Gynecol*. 1986;13:33–41.
- Steinberg TN. Abortion counseling: To benefit maternal health. *Am J Law Med* 1989;15:483–517.
- Rosenthal M. Elective abortion. In: O'Grady JP, Rosenthal M (eds): *Obstetrics: Psychological and Psychiatric Syndromes*. New York: Elsevier, 1992, pp. 307–318.
- Rosenfeld JA. Emotional responses to therapeutic abortion. *Am Fam Physician*. 1992;45:137–140.
- Brown H. Pregnancy termination. In: Plauché WC, Morrison JC, O'Sullivan MJ (eds): *Surgical Obstetrics*. Philadelphia: Harcourt Brace Jovanovich, 1992; pp. 119–131.
- Castadot RG. Pregnancy termination: Techniques, risks, and complications and their management. *Fertil Steril*. 1986;45:5–17.
- Acharya G, Morgan H, Paramanatham L, Fernando R. A randomized controlled trial comparing surgical termination of pregnancy with and without continuous ultrasound guidance. *Eur J Obstet Gynecol Reprod Biol*. 2004;114:69–74.
- American College of Obstetricians and Gynecologists. Antibiotic Prophylaxis for Gynecologic Procedures Practice Bulletin, Number 74, July, 2006.
- Jonasson A, Larsson B, Bygdeman S, Forsum U. The influence of cervical dilatation by Laminaria tent and with Hegar dilators on the intrauterine microflora and the rate of postabortal pelvic inflammatory disease. *Acta Obstet Gynecol Scand*. 1989; 68(5):405–410.
- Lichtenberg ES, Shott S. A randomized clinical trial of prophylaxis for vacuum abortion: 3 versus 7 days of doxycycline. *Obstet Gynecol*. 2003 Apr; 101(4):726–731.
- Suprpto K, Reed S. Naproxen sodium for pain relief in first-trimester abortion. *Am J Obstet Gynecol*. 1984 Dec;150(8) 1000–1001.
- Stubblefield PG. Surgical techniques for first-trimester abortion. In: Gabbe SG, Niebyl JR,

- Simpson JL (eds): *Obstetrics: Normal and Problem Pregnancies*, 2nd ed. New York: Churchill Livingstone, 1991, pp 1303–1332.
19. Burnett M, Corbett C, Gertenstein R. A randomized trial of Laminaria tents versus vaginal misoprostol for cervical ripening in first-trimester surgical abortion. *J Obstet Gynaecol Can.* 2005 Jan; 27(1):38–42.
  20. Stubblefield PG, Carr-Ellis S, Borgatta L. Methods for induced abortion *Obstet Gynecol.* 2004 Jul; 104(1):174–185.
  21. Sharma S, El-Refacy H. The use of misoprostol as a cervical ripening agent in operative hysteroscopy. *Am J Obstet Gynecol.* 2003 Jan;188(1):297–298.
  22. Goldenburg AB, Dean G, Kang M, Youssof S, Darney PD. Manual versus electric vacuum aspiration for early first-trimester abortion: A controlled study of complication rates. *Obstet Gynecol.* 2004 Jan;103(1):101–107.
  23. Felding C, Mikkelsen AL, Villerslev L. First-trimester legal induced abortions: The amount of aspirated tissue in relation to gestational age. *Arch Gynecol Obstet.* 1991;249:149–151.
  24. Laufe LE. Menstrual regulation. In: Zatuchni GI, Laferla JJ, Sciara JJ (eds): *Gynecology and Obstetrics*, vol 6. Philadelphia: JB Lippincott, 1990. 56; 1–6.
  25. Edward J, Creinin MD. Surgical abortion for gestations of less than 6 weeks. *Current Problems in Obstetrics, Gynecology, and Fertility.* 1997;20:11–19.
  26. Paul M, Mitchell C, Rogers AJ, Fox MC, Lackie EG. Early surgical abortion: Efficacy and safety. *Am J Obstet Gynecol.* 2002 Aug;187(2):407–411.
  27. Darney PD, Atkinson E, Hirabayashi K. Uterine perforation during second-trimester abortion by cervical dilation and instrumental extraction: A review of 15 cases. *Obstet Gynecol.* 1990;75:441–444.
  28. Cates W, Schulz KF, Grimes DA, Horowitz AJ, Lyon FA, Kravitz FH, Frisch MJ. Dilatation and evacuation procedures and second-trimester abortions: The role of physician skill and hospital setting. *JAMA.* 1982 Aug 6;248(5):559–563.
  29. Peterson WF, Berry FN, Grace MR, Gulbranson CL. Second-trimester abortion by dilatation and evacuation: An analysis of 11,747 cases. *Obstet Gynecol.* 1983 Aug;62(2):185–190.
  30. Dark AC, Miller L, Kathenbeutel RL, Mandel L. Obesity and second-trimester abortion by dilation and evacuation. *J Reprod Med.* 2002;47:226–230.
  31. Goldenberg RL, Davis RO, Hill D. The use of real-time ultrasound as an aid during difficult therapeutic abortion procedures. *Am J Obstet Gynecol.* 1984;148:826–827.
  32. Romero R, Copel JA, Jeanty P, Reece EA, Reiss R, Hobbins JC. Sonographic monitoring to guide the performance of postabortal uterine curettage. *Am J Obstet Gynecol.* 1985 Jan;151(1): 51–53.
  33. Darney PD, Sweet RL. Routine intraoperative ultrasonography for second-trimester abortion reduces incidence of uterine perforation. *J Ultrasound Med.* 1989 Feb;8(2):71–75.
  34. Schulman H. Second-trimester abortion: Techniques and complications. In: Zatuchni GI, Laferla JJ, Sciara JJ (eds): *Gynecology and Obstetrics*, vol 6. Philadelphia: JB Lippincott, 1990; pp. 1–11; 64.
  35. Grimes DA, Ray IG, Middleton CJ. Lamicel versus laminaria for cervical dilation before second-trimester abortion: A randomized clinical trial. *Obstet Gynecol.* 1987 Jun;69(6):887–889.
  36. Wells EC, Hulka JF. Cervical dilation: A comparison of Lamicel and Dilapan. *Am J Obstet Gynecol.* 1989;161:1124–1126.
  37. Goldberg AB, Drey EA, Witaker AK, Kang M, Meckstroth KR, Darney PD. Misoprostol compared with Laminaria before early second-trimester surgical abortion: A randomized trial. *Obstet Gynecol.* 2005 Aug;106(2):234–241.
  38. Lichtenberg S, Scott S. Clinical trial of prophylaxis for vacuum abortion: 3 versus 7 days of doxycycline. *Obstet Gynecol.* 2003;101:726–731.
  39. Schulz KF, Grimes DA, Christensen DD. Vasopressin reduces blood loss from second-trimester dilation and evacuation. *Lancet.* 1985 Aug 17;2 (8451):353–356.
  40. Schulman L, Lig F. Termination of pregnancy: Second trimester. <http://www.UptoDate.com>.
  41. Krutner AK. Hysterotomy and hysterectomy for abortion: In: Zatuchni GI, Laferla JJ, Sciara JJ (eds): *Gynecology and Obstetrics*, vol 6. Philadelphia: JB Lippincott, 1990;61: 1–9.
  42. Kovács L. Future direction of abortion technology. *Baillières Clin Obstet Gynecol.* 1990 Jun;4(2):407–415.
  43. Grimes D, Mishell D, Shoupe D, Lacarra M: Early abortion with a single dose of antiprogesterin RU-486. *Am J Obstet Gynecol.* 1988; Jun;158(6 Pt 1):1307–1312.

44. Schaff EA, Eisinger SH, Stadalius LS, Franks P, Gore BZ, Poppema S. Low-dose mifepristone 200 mg and vaginal misoprostol for abortion. *Contraception*. 1999 Jan;59(1):1–6.
45. Schaff EA, Fielding SL, Eisinger SH, Stadalius LS, Fuller L. Low-dose mifepristone followed by vaginal misoprostol at 48 hours for abortion up to 63 days. *Contraception*. 2000 Jan;61(1):41–46.
46. Schaff EA, Fielding SL, Westhoff C. Randomized trial of oral versus vaginal misoprostol at one day after mifepristone for early medical abortion. *Contraception*. 2001 Aug;64(2):81–85.
47. Guest J, Chien PF, Thomson MA, Kosseim ML. Randomised controlled trial comparing the efficacy of same-day administration of mifepristone and misoprostol for termination of pregnancy with the standard 36- to 48-hour protocol. *BJOG*. 2007 Feb;114(2):207–215.
48. van Bogaert LJ, Sedibe TM. Efficacy of a single misoprostol regimen in the first- and second-trimester termination of pregnancy. *J Obstet Gynaecol*. 2007 Jul;27(5):510–512.
49. Khazardoost S, Hantouschzadeh S, Madani MM. A randomised trial of two regimens of vaginal misoprostol to manage termination of pregnancy of up to 16 weeks. *Aust N Z J Obstet Gynaecol*. 2007 Jun;47(3):226–229.
50. Nilas L, Glavind-Kristensen M, Vejborg T, Knudsen UB. One- or two-day mifepristone-misoprostol interval for second-trimester abortion. *Acta Obstet Gynecol Scand*. 2007;86(9):1117–1121.
51. Pymar HC, Creinin MD, Schwartz JL. Mifepristone followed on the same day by vaginal misoprostol for early abortion between 50 and 63 days' gestation. *Contraception*. 2001 Aug;64(2):87–92.
52. Fox MC, Creinin MD, Harweed B. Mifepristone followed on the same day by vaginal misoprostol for early abortion between 50 and 63 days gestation. *Contraception*. 2002 Oct;66(4):225–229.
53. FDA Public Health Advisory. Sepsis and Medical Abortion Update, March 17, 2006.
54. Owen J, Hauth JC. Concentrated oxytocin plus low-dose prostaglandin E2 compared with prostaglandin E2 vaginal suppositories for second-trimester pregnancy termination. *Obstet Gynecol*. 1996 Jul; 88(1):110–113.
55. Jain JK, Mishell DR. A comparison of intravaginal misoprostol with prostaglandin E2 for termination of second-trimester pregnancy. *N Engl J Med*. 1994 Aug;331(5):290–293.
56. Herabutya Y, O-Prasertsawat P. Second-trimester abortion using intravaginal misoprostol. *Int J Gynecol Obstet*. 1998 Feb;60(2):161–165.
57. Su L, Biswas A, Choolani M, Kalaichelvan V, Singh K. A prospective randomized comparison of vaginal misoprostol versus intra-amniotic prostaglandins for midtrimester termination of pregnancy. *Am J Obstet Gynecol*. 2005 Oct;193(4):1410–1414.
58. Ashok P, Templeton A, Wagaarachchi P, Flett G. Midtrimester medical termination of pregnancy: A review of 1002 consecutive cases. *Contraception*. 2004 Jan;69(1):51–58.
59. Creinin M. Methotrexate for abortion at 42 days' gestation. *Contraception*. 1993;48:519–525.
60. Council Report: Induced termination of pregnancy before and after Roe v. Wade. *JAMA*. 1992;268:3231–3239.
61. Grimes DA, Schulz KF, Cates WJ. Prevention of uterine perforation during curettage abortion. *JAMA*. 1984;251:2108–2111.
62. Kaali SG, Szigetvari IA, Bartfai GS. The frequency and management of uterine perforations during first-trimester abortions. *Am J Obstet Gynecol*. 1989;161:406–408.
63. Hakim-Elahi E, Tovell HM, Burnhill MS. Complications of first-trimester abortion: A report of 170,000 cases. *Obstet Gynecol*. 1990;76:129–135.
64. Hern WM. First-trimester abortion complications and their management. In: Zatuchni GI, Laferla JJ, Sciara JJ (eds): *Gynecology and Obstetrics*, vol 6. Philadelphia: JB Lippincott, 1990;23: 1–9.
65. Hern WM. Long-term risks of induced abortion. In: Zatuchni GI, Laferla JJ, Sciara JJ (eds): *Gynecology and Obstetrics*, vol 6. Philadelphia: JB Lippincott, 1990;52: 1–8.
66. Lazarus A, Stern R. Psychiatric aspects of pregnancy termination. *Clin Obstet Gynecol*. 1986;13:125–134.
67. Dagg PKB. The psychological sequelae of therapeutic abortion – derived and computed. *Am J Psychiatry*. 1991;148:578–585.
68. Atrash KK, MacKay HT, Hogue CJR. Ectopic pregnancy concurrent with induced abortion: Incidence and mortality. *Am J Obstet Gynecol*. 1990;162: 726–730.
69. Reece EA, Petrie RH, Sirmans MF, Finster M, Todd WD. Combined intrauterine and extrauterine gestations: A review. *Am J Obstet Gynecol*. 1983; 146:323–330.



70. Yuzpe AA, Smith RP, Rademaker AW. A multi-center clinical investigation employing ethinyl estradiol combined with dl-norgestrel as a postcoital contraceptive agent. *Fertil Steril.* 1982 Apr;37(4):508-513.
71. Cheng L, Gulmezoglu AM, Oel CJ, Piaggio G, Ezcurra E, Look PF. Interventions for emergency contraception. *Cochrane Database Syst Rev.* 2004; (3).
72. Allen RH, Goldberg AB. Emergency contraception: A clinical review. *Clin Obstet Gynecol.* 2007 Dec; 50(4):927-936.
73. Johanson E, Brache V, Alvarez F, Faundes A, Cochon L, Ranta S, Lovern M, Kumar N. Pharmacokinetic study of different dosing regimens of levonorgestrel for emergency contraception in healthy women. *Hum Reprod.* 2002 Jun;17(6):1472-1476.
74. Ngai SW, Fan S, Li S, Cheng L, Ding J, Jing X, Ng EH, Ho PC. A randomized trial to compare 24 h versus 12 h double-dose regimen of levonorgestrel for emergency contraception. *Hum Reprod.* 2005 Jan; 20(1):307-311.
75. Lipsomb GH. Medical Therapy for ectopic pregnancy. *Semin Reprod Med.* 2007;25(2):93-98.

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*I am very far from wishing to be understood, that I advocate the indiscriminate interference of art, during healthy labour – it is the very reverse of my opinion, . . . ; I wish merely to insist, that nature is not competent to all exigencies; For in very many instances, the sufferings of the patient might have been most probably very much abridged, by the judicious interposition of skill. Of this, from long experience, I am entirely convinced.*

William Potts Dewees (1768–1841)  
*Compendious System of Midwifery*  
Philadelphia: Carey and Lea  
Second Edition, 1826, xii.

Placental evaluation is important in assessing perinatal outcome [1,11]. Precise descriptions of antepartum findings, gross appearance of the placenta at delivery, postpartum microscopic examination, and other special testing allow for a retrospective assessment of the influence of various factors on fetal development. Study of the placenta helps the clinician in the evaluation of the influence of various maternal diseases on fetal life as well as the potential effects of smoking, drug use, and various other factors.

Obstetric complications secondary to placental dysfunction can occur at any point in gestation. Improvements in perinatal diagnostic techniques such as focused ultrasound studies of fetal growth, placental blood flow, and fetal/placental anatomy now permit the identification of certain complications related to poor implantation or abnormal early development that can be linked to placental function. In some instances, this process has led to intervention and treatment with improvement in maternal health and/or perinatal outcome. Because of the importance of the placenta to fetal life and development and due to its increasing accessibility to antepartum investigation, physicians seeking information and explanations for disordered fetal growth or poor obstetric outcomes are advised to study the placenta. Even with a thorough placental examination by an experienced perinatal pathologist, some questions will remain unanswered, because our understanding of the pathologic processes that result in fetal disease are still incomplete.

#### PLACENTAL DEVELOPMENT

Extensive discussion of placental development and physiology is beyond the scope of this chapter and is the subject of numerous reviews [1–6]. A condensed version of this complex process is presented to assist the clinician in a general understanding of the obstetric complications attributable to placental dysfunction.

The placenta provides the interface between the fetus and the mother. Throughout nature in all

species that have placentas, the organ serves to maintain separation between maternal and fetal circulations with the specific type of placental anatomy varying by species. Similarly, independent of exact anatomy, the placenta serves numerous identical functions in different species, including certain endocrine functions, gas exchange, acid-base balance, nutritional support, and secretory and excretory roles. To accomplish these functions, the placenta must have the capacity to adapt both its structure and function as the pregnancy advances and the demands of the ever enlarging fetus change. To maintain this flexibility, the placenta retains a functional reserve. The degree of reserve varies by species, the integrity of the maternal-fetal circulation, and the type of challenge [1,2,3].

An important feature of placentation is the number and thickness of the layers separating maternal and fetal circulations. Among humans, the placental anatomy is hemochorial [1]. This type of placenta, characteristic of rodents, bats, and humans, develops because of destruction of intervening maternal vessels by invasive trophoblast. In a hemochorial placenta there is direct contact between fetal trophoblasts and maternal blood cells. Presumably, this facilitates the secretory and transport functions of the placenta by reducing the number of cellular layers between the fetal and maternal blood stream.

The unique features of human placentation have their origin early in development. In normal embryonic development, trophoblast invasion begins by day twelve after conception (primary invasion). The cytotrophoblast migrates to form anchoring sites to establish placental adherence. By the 5th week, the fetoplacental circulation develops and vascular connections with the maternal circulation provide for fetal nutrient and oxygen delivery [4,7]. Any major abnormality during this initial phase of placentation leads to major disruptions in embryonic development, usually resulting in a spontaneous abortion.

As pregnancy progresses, the physical distance between the maternal and fetal circulations decreases from approximately 50  $\mu\text{m}$  in the first trimester to 4  $\mu\text{m}$  to 5  $\mu\text{m}$  by the end of the mid-trimester. Initially, this separation between circulations is due to trophoblastic invasion into the decidual layer of the maternal spiral arteries of the myometrium. By the fourth month a second phase of trophoblastic invasion into the spiral arteries begins (secondary invasion). This latter phase

depends on the continued presence of a developing fetus [8]. In this process, fetal cells progressively replace maternal cells, at the same time altering the physical anatomy of maternal spiral arterioles in the decidua/myometrium. The normally thick-walled muscular spiral arteries progressively become thin walled and enlarged tube- or cone-shaped vascular conduits with decreased sensitivity to vasoconstrictors [9]. This process changes the maternal spiral arteries into high-capacitance, low-resistance vessels [10]. At the same time, the fetal portion of the placenta continues to grow by formation of new villi and the continued branching of existing villi. This process rapidly expands the villous surface area, permitting increasing placental exchange. Overall, a more efficient placenta is the result. Failure of these physiologic changes to occur or to occur only incompletely is a common finding in pregnancies complicated by preeclampsia and intrauterine growth restriction [7,10,26,27]. The mechanism of these changes and the etiology of abnormal placentation is not well understood but is believed to be heavily influenced by complex immunologic interactions at the cellular or membrane level.

As pregnancy progresses further, the weight and size of the placenta change in proportion to fetal weight [1]. At the beginning of the third trimester, the ratio of placental weight to fetal weight is approximately 1:3, which then decreases to 1:6 at term. By term, the normal placenta weighs approximately 450 g to 550 g. The mean length of the umbilical cord is 50 cm. Cord coiling is another potentially important finding that is readily evaluated during gross examination of the placenta. The usual number of spirals or coils is up to 40. When placentas are examined, counterclockwise spiraling exceeds clockwise spiraling by a ratio of 7:1. The reason is unknown. The number of cord coils is established early in gestation with little apparent change in the third trimester. Both spiraling and cord length are believed to reflect fetal activity. Lack of normal spiraling may reflect relative fetal inactivity, possibly due to abnormalities in the central nervous system. Of interest, both poorly spiraled cords as well as hypercoiled ones are associated with an increased risk of intrauterine fetal demise. Other features of gross placental examination are also of clinical consequence. Such findings include the presence and adequacy of Wharton's jelly, the number of vessels, presence or absence of abnormal cord attachment

(e.g., velamentous insertion), and the color of the membranes and cord. Easily obtained and important histologic findings during microscopic placental examination include, among others, the presence or absence of pigment-laden macrophages (meconium), and evidence of inflammation of the umbilical cord (funisitis) or fetal membranes (chorioamnionitis).

## PLACENTAL PATHOPHYSIOLOGY

Various intrinsic structural abnormalities of the placenta or membranes such as the presence of a chorioangioma, circumvallate placentation, amniotic bands, or a velamentous insertion of the umbilical cord can result directly in adverse perinatal outcomes. In most cases, however, placental problems that result in problems are developmental and due to disordered fetal-maternal interactions arising in the first and second trimester. Common problems involve abnormal placental implantation predisposing to eventual placental insufficiency or placental separation. These various abnormalities in placentation are associated with spontaneous abortion, varying degrees of fetal growth restriction, placental previa, abruptio placentae (antepartum hemorrhage), and the syndromes of abnormal placental adherence (accreta/increta/percreta).

### **Intrinsic Placental Abnormalities**

*Chorioangioma*, a placental mass typically arising from an abnormal proliferation of primitive chorionic vessels, is a common benign tumor with small lesions seen in approximately 1% of all pregnancies [11]. Larger lesions, especially those larger than 4 cm, occasionally have clinical consequences, with significant arteriovenous shunting or fetal red blood cell (RBC) sequestration. This can lead to hydramnios, fetal anemia, hydrops fetalis, or fetal cardiomyopathy. Sonographically, these tumors appear as well-circumscribed solid or complex masses on the fetal side of the placenta, frequently located close to the cord insertion. Doppler flow imaging demonstrates the hypervascular nature of these tumors, noting turbulent high-velocity flow. Antenatal management includes close antenatal surveillance with evaluation of peak systolic velocity flows in the central nervous system to indirectly evaluate fetal anemia. The effort is to delay delivery until the latter

portion of the third trimester unless prompted by evidence of fetal compromise. Invasive intrauterine management has also been described with obliteration of these placental lesions by embolization or laser coagulation of feeding vessels [12,13].

In continuing pregnancies, the placenta can assume various shapes, most of little clinical importance. An exception is the *circumvallate* placenta, in which the chorionic plate is smaller than the basal plate and the amniotic membranes insert medial to the placental edge. On gross inspection this results in a thick yellow-white rolled placental edge. This abnormality is associated with chronic antepartum bleeding, low birthweight infants, and possibly, congenital malformations [14,15]. This placental variant is not commonly detected by ultrasonic scanning.

An unusual placental abnormality resulting in both major and minor congenital malformations is the *amniotic band syndrome*. These bands can entangle various fetal parts and lead to amputations and other major injuries. Grossly distorting abnormalities involving craniofacial anatomy, visceral organs, and fetal extremities can result in complex limb-body wall anomalies [16]. The pathophysiology underlying this placenta-based syndrome remains unexplained. An exogenous theory proposes the formation of fibrous bands, resulting from first-trimester amnion rupture. An endogenous theory proposes that the occurrence of both fetal malformations and amniotic bands are secondary to underlying fetal vascular complications. Prognosis for the fetus in an amniotic band syndrome varies depending on the associated findings. This condition is to be differentiated from minor "tenting" of the amnion that occurs before the amnion attaches to the chorion in the 12th to 15th week [17,18].

A potentially serious anomaly of cord and membranes is a *velamentous insertion* of the cord. In a velamentous insertion the umbilical vessels separate in the membranes before the cord reaches the chorionic plate. In this situation, the vessels are unprotected by Wharton's jelly and are easily compressed or ruptured. This condition is associated with increased perinatal risk, primarily from fetal hemorrhage caused by vessel laceration associated with membrane rupture. If these vessels course in front of the endocervix, the condition becomes a vasa previa. This is a precarious state, and the vessels are subject to tearing when rupture of

membranes occurs. The observation is made of increased vaginal bleeding following either spontaneously ruptured membranes or during induction of labor when AROM (artificial rupture of membranes) is performed [6]. Velamentous insertion is also associated with fetal growth disturbances and is common in multiple gestations. Approximately 6% of twins and up to 30% of triplet pregnancies will have one or more velamentous insertions. The diagnosis is made by tracing the umbilical vessel from the placental plate and through the membranes by color flow Doppler ultrasound.

If the diagnosis is suspected either owing to low placentation, multiple gestation or other factors, ultrasonic confirmation is usually easy.

In approximately 1% of singleton pregnancies and 5% of twins, one umbilical artery is absent. Such *single umbilical arteries* (SUA) can result from the failed development of one vessel early in pregnancy or from atrophy later in gestation. A SUA has an association with congenital malformations, especially those involving the renal and cardiac structures. When an isolated SUA is present, however, the fetus is usually chromosomally normal, and is usually normal.

### Implantation and Early Development

Disruptions in placentation occurring in the first few weeks of gestation lead to abnormal placental anchoring and disruption of early angiogenesis. This combination of events frequently results in spontaneous abortion. In vitro fertilization studies indicate that a maximum of 30% to 40% of embryos returned to the uterus are able to implant [8,24]. Of recognized pregnancies, at least 20% to 25% abort spontaneously by or before 12 weeks of gestation [25]. (Additional developmental abnormalities that occur early in gestation, including ectopic implantation, are discussed in detail in Chapter 4, Ectopic Pregnancy.)

Chromosomal disorders can be the etiology of some cases of failed implantation. A complete *hydatidiform mole* is usually composed of 46 chromosomes (XX) of paternal origin, whereas an incomplete or partial mole, commonly associated with a growth-restricted fetus, is characterized by placental and fetal triploidy. With molar degeneration, a misnomer, the placenta undergoes cystic changes

after the embryonic or fetal demise. At times it can be confused with a partial mole or even a complete mole. This is more common in early losses (first trimester). There is little evidence of malignant changes or invasion unless the finding is true trophoblastic disease.

### Abnormal Cytotrophoblastic Invasion

After implantation, an important occurrence in placental development is the formation of a fully mature intervillous circulation. The failure to complete this normal physiologic maturation adequately has been observed in the placentas of pregnancies affected by preeclampsia and fetal growth restriction. Most commonly, this involves the absence of a secondary invasion of the endovascular trophoblast into the maternal spiral arteries of the myometrial layer, which normally occurs during weeks 15 to 22. The mechanisms underlying impaired trophoblast migration that interferes with this process are not well known. These normal physiologic changes can be altered when there is a disturbance in maternal blood flow and maternal oxygenation [2,6]. The link to various abnormal placental growth factors (PGIF, sFlt1) is clear, but the etiology of these abnormalities or the association of other co-morbidities, such as chronic hypertension and diabetes, in the appearance of preeclampsia is still poorly understood [26–30].

A common lesion that can arise in the setting of preeclampsia, chronic hypertension, diabetes or other co-morbid situations is acute atherosclerosis in the spiral arterioles or maternal vessels. Such changes can obstruct/obliterate the lumen of these arteries with an aggregation of foam cells. The resulting thrombosis further reduces placental perfusion within the affected villi and can lead to placental infarction. If the placenta is unable to compensate fully for syncytial damage, fibrin and platelet deposition increase, and further infarction of the placental villi occurs leading to an inhibition of fetal growth [2]. Poor placenta perfusion and abnormal maternal spiral arterioles as well as the circulating anti-angiogenic factors such as tyrosine kinase 1 (SFLT1) are the probable precursors needed for the appearance of preeclampsia [29]. More recent studies have suggested that these hypoxic placentas are the basis for abnormal angiogenic factor secretions including low

levels of placental growth factor (PGIF) and vascular endothelial growth factor A (VEGF-A). Apparently, an abnormal balance in pro-angiogenic and anti-angiogenic growth factors are related to the development of abnormal placentation characteristic of preeclampsia [28].

In addition to preeclampsia and fetal growth restriction, similar placental abnormalities are seen in women with chronic hypertension, suggesting that the common etiology is reduced maternal blood flow. Associated clinical factors include extremes of maternal age, underlying maternal diseases, such as long-standing diabetes mellitus, collagen vascular or renal disease, and nulliparity [3,27]. In addition to these conditions the use of illicit drugs such as cocaine and poor maternal nutrition can also adversely affect placental structure and function [3]. Maternal smoking is also an issue [4,11,31–32]. With both active and passive cigarette smoking there is increased fibrosis and decreased vascularization of the placental villi; however, there is a surprising protective effect of cigarette smoking for preeclampsia. Smoking is, however, an important risk factor for circumvallate placenta, chronic abruptio placentae, and placental infarction. All of these conditions reduce placental surface area, limiting effective fetal-maternal exchange. The observed beneficial effect of smoking on preeclampsia is not understood but could be related to the effects of nicotine as a protective intermediary.

### Abnormal Placental Separation

*Placental abruptio* (abruptio placentae) is defined as the complete or partial early separation of a normally implanted placenta [33,34]. This condition is the most frequently encountered placental abnormality of the third trimester. The pathophysiologic processes underlying placental abruptio remain unclear. Physiologically, bleeding initially occurs from vessels in the decidua or the spiral arteries and subsequently spreads along the path of least resistance behind the fetal membranes. There is probably more than one mechanism resulting in this clinical entity. A wide range of clinical conditions are associated with placental abruptio, including accidental trauma, hypertension, diabetes, renal disease, autoimmune disease, smoking, cocaine use, and a history of prior separation. Traumatic separation

of an intact placenta as a result of falls or automobile accidents is a recognized etiology for abruptio; however, it accounts for few cases. In the large majority of instances an abruptio is the consequence of a poorly understood underlying abnormality in the intervillous circulation.

The degree of separation in placental abruptio varies greatly, as can the clinical signs and symptoms. Fully one third of abruptio cases are concealed (i.e., vaginal bleeding is not observed). As such, a high index of suspicion is the key to early diagnosis and appropriate management. In an acute event, subsequent pathologic examination may observe few if any pathologic findings, other than a superficial or attached clot. Abnormal trophoblastic invasion into the spiral arteries has been identified in at least some pregnancies complicated by abruptio.

### Placenta Previa

In *placenta previa*, the placental implantation is either over or adjacent to the internal cervical os. Ultrasonic study permits the classification of placenta previa into the categories of complete and marginal types. In a complete placenta previa, the implantation covers the internal cervical os. A marginal placenta previa is diagnosed when the proximal edge of the placenta is implanted within less than 2 cm of the internal cervical os. The vulnerable placement of these abnormally situated placentas predisposes to separation and antepartum hemorrhage prior to delivery, usually associated with uterine contractions. Cervical trauma and physiologic cervical changes that occur with advancing gestational age are other precipitating factors leading to episodes of bleeding. The cause of low implantation is unclear, but it is theorized that it is secondary to the lack of a suitable implantation site in the uterine fundus and corpus [24,33]. Numerous predisposing factors have been identified and include multi-gravidity, prior cesarean delivery, prior placenta previa, a history of in vitro fertilization, uterine malformations, and the presence of leiomyomas. Placenta previa is often complicated further by various degrees of abnormal placental adherence (placenta accreta/increta/percreta). This is thought to occur because the lower uterine segment and endocervix do not decidualize to the same degree as the uterine fundus [32].

### Placenta Accreta

*Placenta accreta* and its variations, *percreta* and *incretata*, are consequences of an abnormal maternal-fetal tissue interaction early in gestation. In these conditions of abnormal placental adherence, villi penetrate or perforate the uterine muscle without an intervening endometrial layer. Superficial invasion defines an accreta. When the invasion passes deeper into the myometrium the condition is termed a placenta increta. Invasion through and beyond the uterine serosa is placenta percreta. In the rare event of placenta percreta, trophoblast tissue can invade adjacent pelvic structures, most often the bladder. These types of placenta adherence are believed to occur secondary to uterine abnormalities (i.e., absent or damaged uterine decidua) and not as a result of an overly aggressive trophoblast [32].

The clinical consequence of this type of placental implantation is incomplete placental separation at delivery, with resultant hemorrhage. Although uncommon, the accreta, increta, and percreta syndrome can lead to unanticipated and life-threatening hemorrhage, mostly at the time of delivery, with the need for rapid surgical intervention. Antepartum bleeding from an anterior placenta percreta rarely occurs due to either gross hematuria or uterine rupture. (For a more extensive discussion, see Chapter 11, Third Stage.)

### Placental Function

Abnormalities of placentation directly contribute to observed perinatal complications. Maternal illnesses (e.g., chronic hypertension or autoimmune disease) and certain maternal behaviors (e.g., tobacco or substance abuse) adversely influence placental function. The availability of ultrasound scan to assess placental morphology, placental location, and markers for placental function (e.g., fetal growth, Doppler) provides further insight to this relationship. Currently such antepartum testing essentially directs clinical management.

## EPIDEMIOLOGY

Vaginal bleeding remains the most common presenting symptom of placental dysfunction. Approximately 4% of pregnancies that continue beyond the first trimester are complicated by vaginal bleeding.

Placental abruption is the cause for approximately 30% of these cases, with placental previa accounting for another 20%. The remaining 50% arise from various causes, including preterm labor, genital tract lesions, the poorly defined condition of marginal placental separation, or from unknown causes [33]. As the cause-and-effect relationships among these conditions often overlap, so do the epidemiologic risk factors for each.

### Marginal Placental Separation

Bleeding of unknown etiology is the diagnosis of exclusion that follows a thorough evaluation including both an ultrasound evaluation and a physical examination. When visible sites such as the vagina or cervix are eliminated as a source of bleeding, and an ultrasound examination is grossly normal, the diagnosis of bleeding of unknown etiology is made. Bleeding without a specific diagnosis is common in the first and early second trimesters and is attributed to an uncertain entity termed *marginal placental separation* or *marginal sinus bleeding*. Some of these episodes result from punctate decidual bleeding at sites separate from the placental implantation site or probably from small bleeds from the endocervix which are not accurately diagnosed. The overall risk to mother and fetus from bleeding of unknown etiology is low, assuming the exclusion of more significant perinatal pathologies (e.g., placental abruption, preterm labor, and placental previa). When antepartum bleeding begins prior to 37 weeks, however, approximately 15% of fetuses will be delivered prematurely, and the rate of stillbirth doubles. When bleeding occurs in the earlier weeks of gestation, it can herald miscarriage, premature rupture of membranes, chorioamnionitis, preterm delivery, or an intrauterine fetal demise [33].

### Placenta Previa

*Placenta previa* occurs in 0.3% to 0.5% of all pregnancies. The incidence is probably increasing [33]. Variations in the incidence arise because of the gestational age at diagnosis as well as the accuracy of the diagnosis. In recent years the large number of women with prior cesarean deliveries has also increased the prevalence of this condition. In the second trimester, the incidence of previa by transabdominal ultrasound scanning is reported as

TABLE 7.1 Risk Estimates for Antecedents to the Development of Placenta Previa: Collaborative Perinatal Study, 1959–1966\*

Risk Factors	No. of Cases of Placenta Previa per 1,000 Cases with Risk Factor <sup>†</sup>	Relative Risks (95% CI)	Attributable Risks (95% CI)
Frequency of previa/total sample	7 (362)		
Maternal factors:			
Leiomyoma or prior gynecologic surgery	12 (153), <i>P</i> ≤ 0.001	1.9 (1.5–2.4)	0.13 (0.12–0.15)
Parity ≥ 5	14 (123), <i>P</i> ≤ 0.001	2.1 (1.6–2.6)	0.12 (0.10–0.14)
Race: White	7 (211), <i>P</i> ≤ 0.02	1.3 (1.0–1.6)	0.07 (0.06–0.09)
Smoked during early pregnancy	10 (107), <i>P</i> ≤ 0.001	1.5 (1.2–1.9)	0.06 (0.05–0.07)
Age ≥ 35 yr	16 (52), <i>P</i> ≤ 0.001	1.7 (1.2–2.3)	0.03 (0.02–0.04)
Fetal factor: Twins	14 (16), <i>P</i> ≤ 0.005	1.8 (1.2–2.5)	0.02 (0.00–0.04)
Population attributable risk			<b>0.36 (0.34–0.39)</b>
Pregnancy outcomes			
Preterm birth	17 (201), <i>P</i> ≤ 0.001	4.7 (3.8–5.8)	
Fetal growth retardation	7 (33), <i>P</i> ≤ 0.1	1.1 (0.8–1.4)	
Stillbirth	26 (25), <i>P</i> ≤ 0.001	1.0 (0.6–1.5)	
Neonatal death	46 (45), <i>P</i> ≤ 0.001	3.0 (2.2–4.0)	
Neurologic abnormalities at 7 yr	10 (17), <i>P</i> ≤ 0.01	1.6 (1.0–2.5)	
Motor abnormalities + severe mental retardation		4.0 (2.2–6.0)	
Cerebral palsy		3.2 (1.3–5.2)	

\*Significant values are in boldface.

<sup>†</sup>Cases in CPS sample are in parentheses.

From Naeye RL: Disorders of the Placenta, Fetus, and Neonate: Diagnosis and Clinical Significance. St. Louis: Mosby Year Book, 1992; p. 218; with permission.

5% (50/1000) of all pregnancies and is sometimes higher [35,42–45]. Second-trimester studies that incorporate transvaginal ultrasound visualization of the placenta demonstrate a much lower rate of placenta previa. The gestational age at which placenta previa is diagnosed and the degree of cervical overlap influence the likelihood of persistence at term [42]. The large majority of mid-trimester partial placenta previas will resolve (“migrate”) prior to delivery. When the leading edge of the placenta overlaps the cervix by more than 2.5 cm at 20 to 23 weeks’ gestation, however, subsequent vaginal delivery is unlikely as placentas remaining as previas at this time are unlikely to resolve [43].

A prior cesarean delivery increases 1.5- to 5-fold the likelihood of placenta previa in the 3rd trimester [44,46]. Placenta previa is also more common among multiparous women regardless of the mode of prior deliveries. The incidence can be as high as 1 in 200 in grand multiparas, compared with

1 in 1,500 nulliparas [47]. The incidence of previa is also associated with advanced maternal age, multiple gestations, and maternal tobacco or cocaine use (Table 7.1 and Figure 7.1). The recurrence risk for placenta previa is 4% to 8%. Unfortunately, the serious complications of accreta, increta, and percreta are also strongly associated with low implantations and prior cesarean deliveries [48].

These associations suggest that damage to the endometrium is the important factor in the etiology of placenta previa, although the exact pathophysiology remains unknown [50]. Presumably, each subsequent pregnancy provides fewer available “good” implantation sites and therefore a higher likelihood of implantation within the lower uterine segment. These data and the well-established associations between abnormal placental adherence and uterine malformations or previous uterine surgery such as myomectomy or curettage suggest decidual damage as the primary cause for abnormal placental



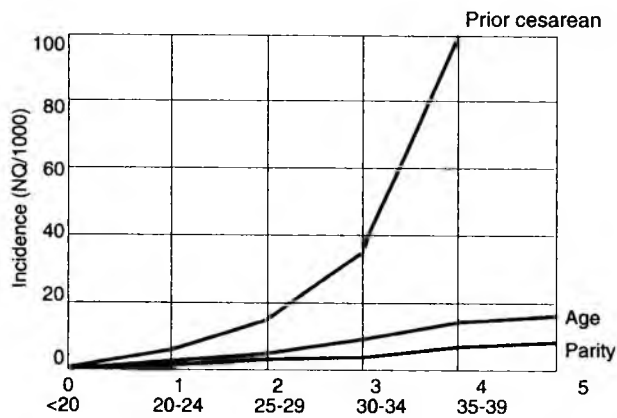


FIGURE 7.1.

*The incidence of placenta previa according to risk factors of prior cesarean, maternal age, and parity. (From Eden RD, Boehm FH (eds): Assessment and Care of the Fetus: Physiological, Clinical and Medico-legal Principles. Norwalk, CT: Appleton and Lange, 1990; p. 664; with permission.)*

adherence [49,50]. Placental shape can also be affected by the site of the original implantation. As the endometrial blood supply is best away from the lower uterine segment, the placenta preferentially extends its growth upward toward the fundus (trophotropism), leaving the site of cord insertion behind as a marker for the center of the original placental disc [32]. The increased rate of eccentric cord insertion, velamentous insertion of the cord, and vasa previa observed with placenta previa adds support to this theory.

The best-documented and greatest risk to the fetus with placenta previa is prematurity. Fortunately, with modern obstetric management, serious maternal complications of previa are uncommon, although all cases involve morbidity and some risk of serious complications.

### Placenta Accreta/Increta/Percreta

Placental accreta/increta/percreta are related and complex abnormalities of placental implantation. These conditions are strongly associated with both placenta previa and prior cesarean delivery. Clark and coworkers identified the risk of accreta coexisting with placenta previa as 5% in women without a prior cesarean delivery. This rate increased to 67% among women with a placenta previa and four prior cesarean operations (Figure 7.2) [48]. Other studies have reported similar findings [49,50]. In contrast, the risk for placenta accreta in the absence

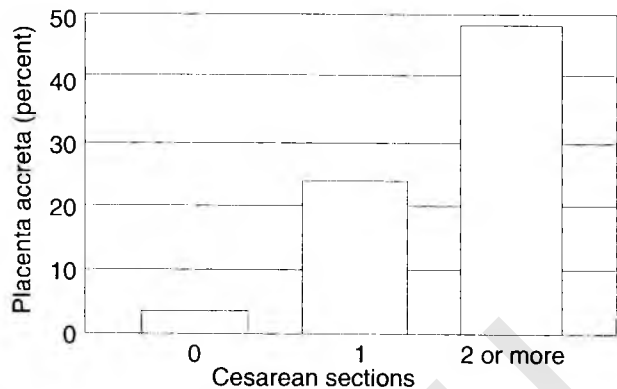


FIGURE 7.2.

*The risk of placenta accreta in patients with a placenta previa and one or more previous cesareans. (From Creasy RK, Resnik R (eds): Maternal-Fetal Medicine: Principles and Practice, 2nd ed. Philadelphia: WB Saunders, 1989; p. 608; with permission.)*

of placenta previa and prior cesarean delivery has been estimated at 1 in 68,000. The prevalence of placenta accreta is rising in the United States and has been attributed to the rising cesarean delivery rate. Among women with placenta previa, advanced maternal age is likely an independent risk factor for placenta accreta [46]. An elevated maternal serum alpha-fetoprotein (AFP) level in the absence of demonstrated fetal anomalies is a potential early biochemical marker for the diagnosis of placenta accreta [51].

Maternal mortality and morbidity are considerably higher with placenta accreta than with previa alone owing to the propensity for sudden severe hemorrhage due to myometrial invasion and subsequent involvement of adjacent tissues, and the additional surgical procedures required for control (Figure 7.3). Fetal outcomes in placenta accreta are similar to the fetus with isolated placenta previa.

### Abruption Placentae

Premature separation of the normally implanted placenta (i.e., abruption placentae, accidental hemorrhage) remains one of the leading causes of fetal and neonatal mortality and significant maternal morbidity. The incidence of abruption is approximately 1%. Severe abruption leading to fetal death occurs in 0.12% of pregnancies (1:830) [34]. Although no single cause has been identified, underlying pathologic alterations of the decidual and uterine

TABLE 7.2 Risk Estimates: Abruption Placentae\*

Risk Factors	No. of Cases of Placenta Previa per 1,000 Cases with Risk Factor <sup>†</sup>	Relative Risks (95% CI)	Attributable Risks (95% CI)
All cases	21 (1140)		
Maternal factors			
Smoked during pregnancy	27 (295), <i>P</i> ≤ 0.001	1.5 (1.2–1.9)	0.07 (0.05–0.09)
Preeclampsia, eclampsia	35 (319), <i>P</i> ≤ 0.001	1.7 (1.4–2.0)	0.09 (0.06–0.12)
Age ≥35 yr	28 (91), <i>P</i> ≤ 0.005	1.8 (1.2–2.6)	0.03 (0.02–0.04)
Fetal factor			
Major fetal malformations	32 (160), <i>P</i> ≤ 0.001	1.6 (1.1–2.2)	0.03 (0.02–0.04)
Placental factor			
Acute chorioamnionitis	39 (269), <i>P</i> ≤ 0.001	1.9 (1.7–2.2)	0.08 (0.05–0.11)
Markers for vigorous fetal motor activity			
Unusually long umbilical cord	24 (517), <i>P</i> ≤ 0.002	1.6 (1.2–2.0)	0.11 (0.09–0.13)
Other indicators			0.01 (0.00–0.02)
Population attributable risk			0.40 (0.36–0.45)
Pregnancy outcomes			
Preterm birth	48 (558), <i>P</i> ≤ 0.001	1.5 (1.4–1.8)	
Fetal growth retardation	23 (110), <i>P</i> ≤ 0.1	1.1 (0.9–1.3)	
Stillbirth	190 (184), <i>P</i> ≤ 0.001	4.1 (3.4–5.2)	
Neonatal death	135 (137), <i>P</i> ≤ 0.001	1.9 (1.0–3.6)	
Neurologic abnormalities at 7 yr	25 (42), <i>P</i> ≤ 0.1	1.2 (0.4–2.1)	

\*Significant values are in boldface.

<sup>†</sup>Numbers of cases are in parentheses.

From Naeye RL: Disorders of the Placenta, Fetus, and Neonate: Diagnosis and Clinical Significance. St. Louis: Mosby Year Book, 1992: 215–9; with permission.

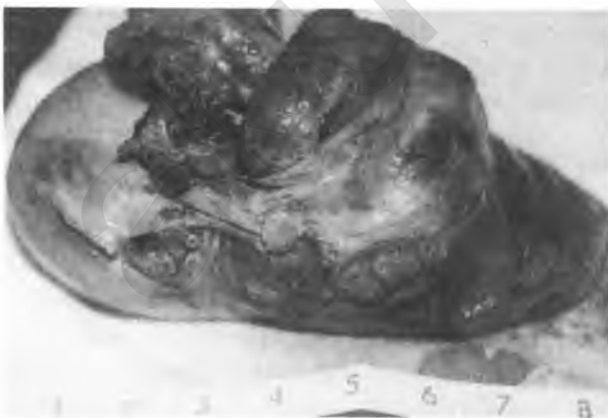


FIGURE 7.3.  
*Hysterectomy specimen showing an anterior placenta increta/percreta bulging through the uterine wall.*

vasculature are doubtless involved [1,11,33,34]. Some have speculated that the underlying mechanisms can differ between preterm and term gravidas affected with placental abruption [52]. Historical factors associated with placental separation include increased parity, prior cesarean delivery, hypertensive disorders, premature prolonged rupture of membranes, multiple gestations, cigarette smoking, cocaine use, and prior abruption. Rarely, direct uterine trauma is a cause of abruption placentae (Table 7.2). Recent studies have not confirmed folic acid deficiency as a causal factor in abruption placentae, and fortification efforts have not changed the prevalence of placental abruption [53].

Drug and cigarette exposure is an area for which there is clearly potential for prevention and intervention. Despite educational efforts, fully 30% of pregnant women still smoke cigarettes, guaranteeing the continued generation of new cases of

abruptio placentae. Drug use is also a continuing and severe problem. Exposure to illicit drugs in pregnancy is estimated to be between 5% and 11%, regardless of socioeconomic class, race, or type of community setting [54]. Trauma is an additional potentially preventable cause of abruptio placentae [36–40]. Appropriate seat belt use can reduce the risk of traumatic abruption and should be part of antepartum education. Partner physical abuse might occur for the first time in pregnancy and since it is often directed at the abdomen, breasts, and genitalia, abruptio placentae could be an unexpected consequence. A high index of suspicion in unusual cases of abruption as well as specific inquiry about physical violence should be incorporated into routine obstetric practice. However, overall trauma as an etiology for abruptio placentae remains uncommon.

Intrapartum risk factors associated with abruptio placentae include rapid decompression of the uterus after rupture of the membranes in cases complicated by hydramnios or, similarly, sudden change in uterine size after the delivery of the first fetus in a multiple gestation pregnancy. External cephalic version and congenital hypofibrinogenemia are additional and unusual risk factors for placental abruption. There is no relationship between abruption at or near term and diagnostic amniocentesis performed earlier during pregnancy [55].

The incidence of abruption appears to be increasing, with a near twofold increase among black women over the past two decades. White women experienced a 15% increase over the same period [56]. The recurrence risk for abruption is 5% to 15% but could be considerably higher if underlying risk factors are present. For this reason, a careful review of the past obstetric history and medical documentation is crucial in preconceptual and early pregnancy risk assessment.

## DIAGNOSIS OF PLACENTAL ABNORMALITIES

### History

Elements of the patient's history are useful in the initial evaluation of bleeding thought to be of placental origin; however, there is overlap in signs and symptoms among patients with placenta previa, abruptio placentae, and bleeding of unidentified etiology

TABLE 7.3 History and Physical Findings with Third-trimester Bleeding\*

History or Physical Finding	Placenta Previa	Abruptio Placentae	Marginal Sinus Bleeding
Recent intercourse	+	-	-
Trauma	-	+	-
Drug ingestion	-	+	±
Severe pain	-	+	-
Uterine hypertonus	-	+	-
Abnormal presentation	+	-	-
Fetal distress	-	+	-
Hemorrhage	+	+	±

\*See text for additional discussion.

(Table 7.3). Most important, an episode of vaginal bleeding suggests, but is not limited to, the diagnosis of a placental abnormality. Bleeding can occur at any point in pregnancy, although the peak incidence is at 34 to 35 weeks of gestation. Bleeding can occur spontaneously or be incited by intercourse, a pelvic examination, trauma, or the onset of term or preterm labor.

Bleeding can be painless or associated with complaints of severe distress. Late third-trimester painless bleeding is classically ascribed to placenta previa. Abruption, can, however, accompany a placenta previa, resulting in acute signs and symptoms that are mixed. Uterine contractions are present in as many as 25% of cases. Bleeding from an abruption is occasionally painless, particularly if the separation is small. As a general rule, the greater the degree of the separation, the more likely are both pain and uterine irritability. Maternal restlessness and discomfort out of proportion to the amount of blood observed, or initial maternal hypotension with or without anemia, strongly suggest the possibility of concealed and intramural bleeding characteristic of abruption.

### Physical Examination

Classically, on examination, the woman with placenta previa has painless bleeding, a soft uterus, and vital signs reflecting the degree of blood lost. During Leopold maneuvers, the presenting part is not engaged. The patient with abruptio placentae usually has recurrent contractions or a tense and often tender uterus and can have signs and symptoms of

hypovolemia. As with the patient's history, there is an overlap of findings in the physical examination of patients with vaginal bleeding. Classic histories are notable for their rarity and atypical presentations are common. If a concealed separation is the problem, the amount of blood seen per vagina can be minimal compared with the patient's complaints or physical findings. A high index of suspicion is often required to promptly establish the correct diagnosis. Initial laboratory test results such as a complete blood count (CBC), clotting studies, or a Kleihauer-Betke stain can fall within the normal range for the laboratory. The use of Kleihauer-Betke testing does not provide diagnostic assistance concerning the mechanism of the observed vaginal bleeding [57].

Confusion caused by the potential overlap in history and physical findings with the various placental abnormalities has led physicians in recent years to rely heavily on ultrasound scanning to help in identifying the cause of ante- and intrapartum hemorrhage.

### Ultrasound Scanning

Despite advances in ultrasound imaging, the likelihood that scanning will identify placental separation or other sites of hemorrhage by ultrasound examination remains low. The principal use for ultrasound in cases of bleeding is to establish gestational age, verify fetal number, evaluate gross fetal normality, determine the volume of amniotic fluid, test for fetal well-being, and *exclude placenta previa*. Improving diagnostic accuracy is extremely important. Inappropriately diagnosing a placental abnormality can lead to unnecessary cessation of work, bed rest, hospitalization, or even an operative delivery. Alternatively, a missed diagnosis can make even a simple pelvic examination potentially dangerous.

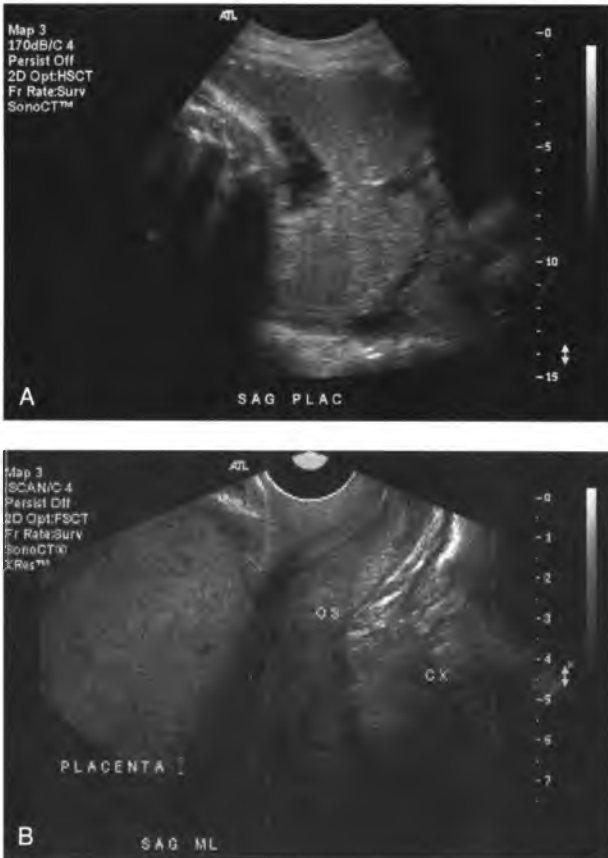
### PLACENTA PREVIA

The placenta is first identified ultrasonographically as a thickening along one surface of the intrauterine gestational sac by about 10 to 14 weeks of gestation. Gross anomalies such as moles, molar degeneration, or amniotic bands can also be identified at this time. In early gestational age the localization of the placenta is frequently centered around the cervix with low-lying placentas or even previas common. However, the edge of the placenta is often difficult to

define with *transabdominal sonography* (TAS) techniques, and *transvaginal sonography* (TVS) imaging must be used to confirm placental location. This early localization does not reflect the future position of the placenta, so that first-trimester studies are poorly predictive for those placenta previa at term.

Placental localization becomes progressively easier using abdominal scanning as gestation proceeds. The overall accuracy of TAS for placental localization ranges from 93% to 97% [58]. False localizations in the second trimester are possible and can result from several causes, including an overly distended maternal bladder, myometrial contractions, and masses such as leiomyomas and extramembranous blood clots, which can have an echo pattern similar to placental tissue. Problems with third-trimester studies are also possible and can result from difficulty in visualizing the cervix secondary to shadowing from the maternal symphysis pubis or overlying fetal parts. Failing to examine the lateral uterine wall can also result in a missed diagnosis for placenta previa. The common factors of obesity, degree of bladder fullness, or overlying presenting part are largely avoided by transvaginal study (TVS). The use of high-frequency TVS probes also provides information regarding the distance from the leading placental edge to the internal cervical os. Properly conducted TVS does not incite bleeding during the investigation of suspected placenta previa. As opposed to speculum placement, the transvaginal probe is performed under direct vision due to the images it provides. Additionally, the angle between the longitudinal axis of the cervix and the vagina (greater than 45°), prevents inadvertent insertion into the cervix [59], and the focal zone of the probe (ranging 2 cm–8 cm) provides images of the lower uterine segment without entering the cervical canal [60]. Aggressive manipulation of the transducer once it is inserted does have the potential for inducing bleeding and is to be avoided, however.

Compared with abdominal ultrasound, the vaginal approach allows more precise delineation of the internal cervical os and the placenta at any stage of gestation (Figure 7.4). The important point is that the diagnosis of previa can be reliably excluded by a normal transvaginal scan. In the mid-trimester, the finding of a leading placental edge more than 2.0 cm from the internal cervical os virtually excludes the occurrence of clinically



**FIGURE 7.4.** *A, Ultrasound image of placenta previa: transabdominal ultrasound technique; B, transvaginal ultrasound technique.*

significant previa at term [61]. At or near term, demonstration of the placental edge within 2.0 cm from the internal cervical os indicates a high-risk situation. Cesarean delivery is indicated for women in whom the leading edge of the placenta is within 1.0 cm from the internal os. The optimal delivery management for those women in whom the placental edge lies within 1.1 cm to 2.0 cm of the internal cervical os, however, is less clear [62]. If the placental edge does not cross the cervix, and there is an interval of time from diagnosis until the anticipated delivery, the greater the possibility for migration, as is discussed below.

*Transperineal sonography* (TPS), also known as *translabial sonography*, offers some of the advantages of the vaginal approach without the potential risk of bleeding and discomfort or the need for a special transducer. Quality of the scan is limited by echoes produced by the symphysis pubis and by soft perineal tissue, and experience is needed to interpret

the images; however, TPS has a high acceptance rate by patients and can be readily repeated as necessary.

Following an abdominal scan that suggests placenta previa or an observed episode of bleeding, the patient is placed in the lithotomy position or with her legs flexed and the knees widely spread. A transducer covered with a plastic sheath or simply an examining glove is placed over the vulva. The maternal bladder should be empty for this examination. With this technique, the internal os of the cervix can be visualized in 97% to 100% of cases [63,64]. When the fetal presenting part or amniotic fluid lies directly over the internal os, placenta previa can be reliably excluded. Early studies on this approach report 90% accuracy with the diagnosis of placenta previa [63]. Although not as precise for diagnosis of previa, this technique has additional value as it can be used during labor as well as in cases of ruptured membranes. Poor positioning, maternal soft tissue and symphysis pubis echoes, uterine contractions, an echogenic hematomas, operator inexperience, and failure to visualize the entire cervix are potential sources of error.

The interval between the diagnosis of placenta previa and the confirmation of that diagnosis at delivery relates to the phenomenon known as placenta migration. Placentas that appear low in the uterus in the second trimester appear to move toward the fundus with advancing gestational age. This is due to the combined effects of preferential placental growth and normal elongation in the lower uterine segment. Longitudinal studies of placental position suggest a migration “speed” of approximately 0.5 cm per week [61]. In fact, 90% of asymptomatic placentas diagnosed in midgestation “resolve” by this normal mechanism by the time of delivery.

### PLACENTA ACCRETA

With the known association of placenta previa and prior cesarean delivery with placenta accreta, and an ever-increasing rate of cesarean delivery, there has been a recent focus on identifying ultrasound findings predictive of abnormal placenta adherence (Figure 7.5) [65–70]. Close attention to placental locale and echo pattern is prudent, especially in women with multiple prior cesarean deliveries and a low and anterior implantation site. Ultrasound features suggestive of placenta accreta include 1) the

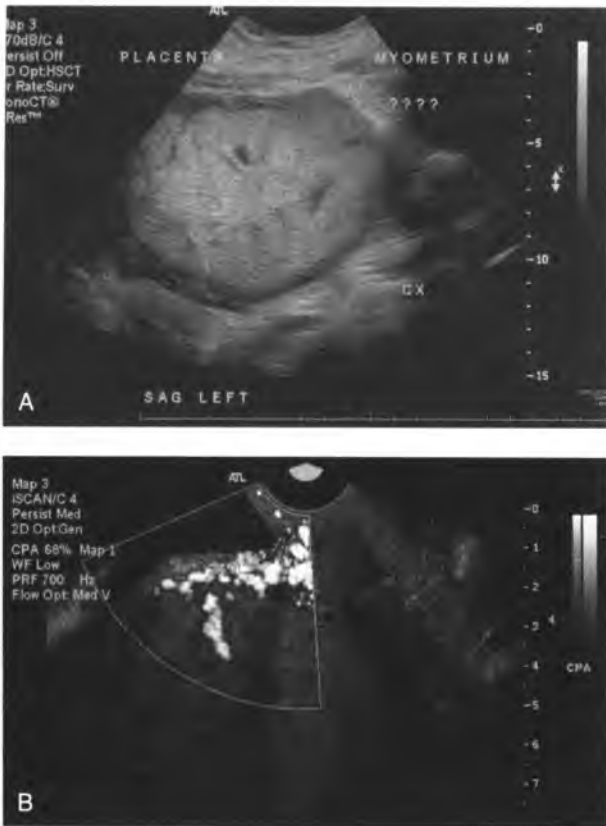


FIGURE 7.5.

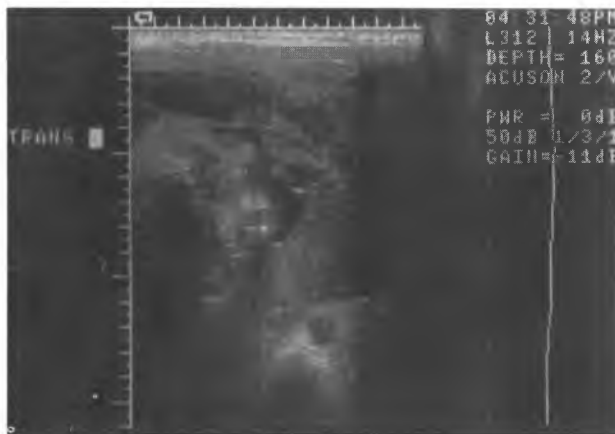
*A, Ultrasound images of placenta accreta: transabdominal ultrasound technique. Note vascular lakes and poorly defined myometrium. B, Transvaginal ultrasound with color Doppler study demonstrating abnormal vessels. For color reproduction see Color Plate 1.*

interruption of the usual maternal bladder-uterine interface (seen as a disruption of the echolucent border between these two structures), 2) obliteration of the clear space between the uterus or myometrium and the placenta, and 3) the finding of numerous lacunar vascular spaces (“lakes”) within placental tissue. In an early prospective study, Finberg and Williams [66] identified eighteen patients with such suggestive sonographic findings, sixteen (89%) of whom ultimately required hysterectomy for accreta. Conversely, of sixteen women with negative findings, only one proved to have an abnormally adherent placenta. More recently, Comstock and colleagues [67] reported on 12 years of study data from a population of 2,002 women at risk for placenta accreta. Fifteen patients were ultimately demonstrated to have clinically proven placenta accreta. Eighty-six percent of patients were noted to

have one or more ultrasound findings suspicious for placenta accreta in the second trimester, and all had ultrasound findings suspicious for placenta accreta in the third trimester, closer to the time of delivery. An additional eighteen patients with ultrasound findings suspicious for placenta accreta were not found to have placenta accreta at the time of delivery. The presence of placenta lacunae (“lakes”) was the most sensitive for predicting placenta accreta (93%, or 14/15 patients). False-positives resulted most frequently from an isolated finding of obliteration of the clear space between myometrium and placenta (16/18 cases). In this study women with placenta percreta could not be distinguished from those with placenta accreta [67]. In the uncommon event of an anterior placenta percreta that invades into the bladder, hematuria (gross or microscopic) is a common finding. At times in such cases a perforating mass can be identified in the bladder. The role of magnetic resonance imaging (MRI) in detecting placenta accreta is still under investigation [70]. With additional study the appropriate role for MRI as a diagnostic tool in evaluating placental adherence will doubtless become better established. (See the discussion that follows for Magnetic Resonance Imaging.)

### ABRUPTIO PLACENTAE

When placenta previa is excluded as the cause for antepartum bleeding, the diagnosis of marginal sinus hemorrhage or abruptio placentae should be entertained. Ultrasound scanning for abruptio can be misleading if the limitations of such studies are not recognized. Blood that has escaped through the vagina or passed into the amniotic cavity or myometrium will not be revealed by ultrasound scanning. This is the situation in three quarters of cases of suspected abruptio. If blood remains within the uterus, a variety of findings are possible. A retained clot has a varied appearance, depending on location, size, and age. Initially, a hematoma appears hyperechoic or isoechoic with the placenta (Figure 7.6). As such, it can be confused with a leiomyoma, a uterine contraction, a chorioangioma, or low-lying placenta or even a succenturiate lobe of the placenta. After several days to a week, the clot usually becomes hypoechoic or sonolucent. Such changes in appearance over time are helpful in differentiating hemorrhage from other diagnoses. Other associated findings suggest abruptio such as



**FIGURE 7.6.**  
*Longitudinal transabdominal ultrasound scan demonstrating clot adherent to lateral edge of the placenta.*

elevation of the adjacent chorion or a spherical shape to the placenta. The use of ultrasound for the diagnosis of abruption must obey the rule of reason. In many instances of placental separation, the clinical presentation of pain, uterine irritability, anemia, or hemorrhage with or without fetal distress strongly suggests the diagnosis of abruption. In extreme cases of a potentially viable fetus in apparent extremis, treatment should never be delayed simply to obtain an ultrasound scan. When clinical circumstances are less pressing, scanning can help establish the correct diagnosis primarily by excluding other possibilities – especially placenta previa. As previously discussed, scanning also permits verification of gestational age and an assessment of fetal anatomic normality, as well as documenting fetal position, assessing amniotic fluid volume, and studying other details of intrauterine anatomy or fetal growth that could prove important in management.

Experience with TVS for the diagnosis of abruptio placentae presents the same difficulties described with abdominal scans. A fresh intrauterine extramembranous hematoma can mimic the placenta or even a leiomyoma. Color Doppler flow study has been suggested as a way to further characterize difficult-to-interpret ultrasonic findings. Prominent venous flow in the hypoechoic areas near the cervix is most consistent with abnormal placentation, although scar windows or increased serosal vascular supply can confuse the interpretation [66].

## Magnetic Resonance Imaging

MRI is potentially well suited to the evaluation of third-trimester bleeding. The technique is noninvasive and is excellent for tissue differentiation and the identification of blood. MRI uses neither ionizing radiation nor a contrast medium, and neither maternal bone nor intestinal gases interfere with the image. Although the published data with MRI in pregnancy continue to grow, many of the problems encountered remain the same since the initial review of this topic for this text almost 10 years ago. Clinical experience with MRI studies in pregnancy remains limited to larger institutions, affecting the availability of knowledgeable physicians for study interpretation. Examination costs remain high. Despite increased availability of MRI mobile suites, the scanner is frequently located at a substantial distance from the labor and delivery suite, requiring patient transport at a time when clinical deterioration could potentially occur. Further, some women poorly tolerate their isolation in the traditional scanning chambers, remaining supine for 30 minutes or more or simply have difficulty fitting comfortably within the magnet.

MRI is usually employed in pregnancy when ultrasonic studies have failed to establish a diagnosis, and the index of suspicion for occult abnormalities remains high. MRI studies are also helpful in the diagnosis of adnexal masses and ectopic gestation in pregnancy, in the evaluation of complex fetal anomalies, and in placental localization. In the largest series specifically addressing bleeding, the location of the placenta with respect to the cervix was correctly identified in all cases [68]. No placental abruptions were identified, but in four cases a suspicion of intrauterine bleeding in the absence of a placenta previa was confirmed by the presence of hematoma at delivery. Cases in which no bleeding source was identified subsequently delivered without further hemorrhage or evidence of placental pathology.

MRI could prove helpful in establishing the diagnosis in an asymptomatic woman believed to be at risk for placenta accreta or percreta based on prior ultrasound scanning (Figure 7.7). If the diagnosis is secure, this allows for the preparation for additional support services such as embolization and subspecialty surgical consultations. A preoperative diagnosis also assists the surgeon by indicating that the



**FIGURE 7.7.**  
*MRI image of placenta accreta indicating marked attenuation of the myometrial wall.*

attempted removal of the adherent placenta is to be avoided [69,70]. The identification of abnormal placental adherence in women with a posterior placenta previa and prior uterine surgery is enhanced with MRI as opposed to ultrasound because the problem of penetration is nonexistent with the latter. Nonetheless, MRI is not without its own technical and physical limitations. Furthermore, in many cases, clinical experience with MRI studies during pregnancy is limited. The use of MRI to evaluate placental anatomy has occurred at the same time as vaginal ultrasound scanning and has become increasingly reliable in determining the existence of abnormal placental adherence. Thus, while there is a role for MRI as a complement to ultrasound scanning in the diagnosis of placenta accreta/increta/percreta, it remains to be established.

### Fetoscopy

For several years, fetoscopy has had a role in the diagnosis of fetal anomalies or genetic disorders. In cases with high perinatal risk, the additional risk of fetoscopy is considered justified to confirm the diagnosis. Its use to evaluate placental abnormalities, however, has been almost exclusively limited to the unique problem of twin-twin transfusion syndrome, where perinatal mortality is extremely

high. In this condition, abnormal vascular relationships between the placentas of monochorionic twins predispose to massive hydramnios, hydrops fetalis, intrauterine growth retardation, and maternal respiratory compromise or preeclampsia. Preterm delivery, often before fetal viability, is a common complication. Therapies include periodic amniocentesis for removal of amniotic fluid, and fetoscopy to identify anastomoses and photocoagulate them with a neodymium: yttrium-aluminum-garnet (Nd:YAG) laser [71]. Because of the risk of fetoscopy, photocoagulation for the twin-twin transfusion syndrome is usually performed in the second trimester, before the period of potential fetal viability. The risks of the procedure have been reduced by improvements in technique, such that fetoscopy and laser obliteration of connecting vessels are becoming an important treatment for this distressing disorder in selected cases [72,73]. (See Chapter 10, Fetal Surgery, for additional discussion.)

### Double Set-up Examination

The double set-up examination was classically considered the final step in the evaluation of placental localization. Owing to the rapid advances in the technologies mentioned previously, however, its use today is quite limited. Patients with complete placenta previa established by other diagnostic tests or extensive hemorrhage do not need the double set-up examination and could be subjected to unnecessary risk because of it. Owing to the potential for increased bleeding following digital manipulation, the double set-up examination is performed only when the usual methods of evaluation are equivocal and additional information is needed to decide the appropriate route for delivery. A double set-up may allow a trial of labor in a patient who desires a normal vaginal delivery in a borderline case of previa that cannot be decided by ultrasonic scanning.

Preparations for operative delivery should be completed before the double set-up examination is begun. This includes having blood available and moving the patient to the operating room with a full surgical team, including the anesthesiologist, present.

In the conduct of the examination an abdominal and/or vaginal ultrasound examination is first performed. With this examination, the clinical decision to proceed or not with the pelvic examination can



be facilitated. Next, the most experienced physician conducts a vaginal examination, palpating the vaginal fornix furthest from where the placenta is anticipated to be located. If the fetal presenting part is clearly felt in that quadrant, the examining finger is gradually moved progressively around the cervix toward the suspect quadrant. If no placenta or boggy sensation is felt, vaginal delivery is probably safe. The membranes are then ruptured, a scalp electrode is placed, and oxytocin is administered, as required. With membrane rupture, the presenting part should descend and often will tamponade a low-lying placental edge; however, if during the cervical examination no presenting part is palpable or if there is a sensation of fullness, suggesting either placenta or clot, or if hemorrhage follows the examination, the patient's legs are drawn down and a cesarean delivery is promptly performed. These examinations should be restricted to women at or about term who are believed to have a favorable cervix and are strongly motivated to have a vaginal trial. Such examinations are now quite uncommon, but circumstances can still occur that favor this procedure.

### Placenta Pathology

In some cases, the nature of the placental abnormality cannot be accurately diagnosed until after delivery. When the pregnancy outcome is preterm delivery or stillbirth, or if an intrauterine growth restriction is present, a microscopic as well as a gross examination of the placenta is recommended. The responsibility for placental examination begins with the delivering physician. A careful description of placental condition at delivery, including the difficulty or ease of removal, integrity of the maternal surface, and the presence of adherent clots, can be vital information for the diagnosis of previa, accreta, or abruptio placentae. Clinical history and patient data must be shared with the pathologist in the laboratory and should be stated on the pathology requisition. If specific gross findings are present at delivery, a photograph for documentation is invaluable. If a formal examination can not be conducted immediately, the placenta can be labeled and stored in a regular refrigerator until the pathologist is available.

Evaluation of the placenta is invaluable for the confirmation of clinical diagnoses. There are numerous articles and books detailing such examina-

tions [1,2,6,74,75]. Even for the pathologist with little experience in examining placentae, evaluation should include a description of placental size, color, and completeness; the presence of infarcts, hematomas, or tumors; and the appearance of the membranes and cord. Cord length, number of vessels, and placental weight should also be recorded.

Aberrations in placental weight are commonly used in speculation regarding perinatal outcome. To standardize placental evaluation, blood should be allowed to drain from the placenta completely, and the cord and membranes should be trimmed prior to weighing. Ideally, the placenta should be weighed before formalin fixation. Variation in placental weight has its origin in genetics and the maternal nutrition state, as well as in the effects of maternal vascular disease, toxin ingestion (e.g., cigarettes and cocaine), and certain environmental influences (e.g., altitude). By definition, a small placenta weighs less than the 10th percentile for that gestational age against a table of normal values. If the placenta is small, minimal pregnancy weight gain and resultant lower-than-average expansion of intravascular blood volume are common associations [75]. Fetal and placental growth are *not* always linked, however, and even in combination, the identified risk factors explain about only one half of small placentas. Interestingly, maternal cigarette smoking, which is a well-known cause of growth retardation, does not cause a reduction in placental growth. In this situation, the ratio of fetal-to-placental weight actually increases.

In small placentas, microscopic findings can provide insight into the etiology of poor growth. For example, stenotic segments, or even occlusion of the spiral arteries, can limit blood flow to the intervillous space, resulting in limited placental growth, placental infarct, or other histologic evidence of chronic hypoxia, as previously discussed. This can occur in association with clinical evidence of abnormal fetal growth.

Placental histopathologic evaluation also includes evaluation for evidence of chorioamnionitis and for disorders of maturation. Villi go through an orderly sequence of events during their life cycle. Premature or "accelerated" maturation sequences have been associated with hypertensive states. Accelerated maturation can be uniform, affecting all villi, or uneven, forming a mosaic. Uneven maturation, presumably on the basis of fluctuating vasoconstriction,

is significantly related to adverse perinatal outcomes [76].

*Maternal floor infarction* is another rare placental condition of clinical importance [77,78]. This disorder is characterized by a heavy deposition of fibrin surrounding the villi, on the maternal side of the placenta. As with accelerated maturation, this process can either be uniform or patchy in distribution. If enough villi are disabled by the process, those remaining cannot support the pregnancy, and the fetus could grow abnormally or even die. Because this process can recur in subsequent pregnancies, the diagnosis is clinically valuable. Maternal floor infarcts also can result in an increase in maternal serum alpha-fetoprotein early in gestation [78].

Placental aneuploidy, mosaicism or chromosomal abnormalities such as paternal isodisomy can account for 3% to 5% of IUGR fetuses. In these cases the placental karyotype is dissimilar to the fetal karyotype [79]. For more information one should refer to specific texts relating to placental pathology.

## MANAGEMENT

The management of placental abnormalities depends on numerous factors, including type of abnormality, gestational age at diagnosis, related prognosis, and associated maternal and fetal status. These must be considered in total when deciding on a plan and when presenting that plan to the patient and her family. The plan should include careful discussion of the presumed diagnosis and management options with the patient and her provider of care.

### **Previable Pregnancy**

Early in gestation, management plans focus primarily on maternal well-being. There are varying opinions on when in gestation that emphasis should change, but currently, prior to 24 weeks of gestation, treatment strategies revolve around establishing the correct diagnosis and assessing its impact on the mother first, and considering the long-term outlook for the pregnancy second. Thus, the options considered prior to the period of potential fetal viability are primarily expectant and supportive. Pregnancy termination should not be overlooked as a management strategy, depending on the severity of the complication, the gestational age, and the maternal wishes.

With serious anomalies, risks to the mother include hemorrhage, surgical complications, blood transfusion, future infertility, emotional and physical stress, and economic losses. Each of these potential issues can weigh heavily in the decision process. The risks and complications of the potential options should be discussed, even if the patient or the physician is uncomfortable with the available choices.

Cases involving missed abortion, blighted ovum, and molar pregnancy offer little controversy. Such pregnancies have failed, and maternal considerations, such as hemorrhage, transfusion, emergency surgery, or persistent trophoblastic disease, are the main issue. If the pregnancy does not end spontaneously soon after the diagnosis is made, suction curettage, medical termination, or expectant management may be considered. Conversely, some structural abnormalities diagnosed by ultrasonography have little if any effect on the mother or developing embryo, and no immediate action is necessary. An example of this would be amniotic sheets or bands where the fetus appears normal and growth is within normal limits.

In some situations, although there is no adverse effect at the time of diagnosis, the potential for subsequent complications must be recognized and discussed with the patient. Chorioangioma of the placenta and placenta previa are two such examples. Both can be associated with fetal intrauterine growth retardation or other complications [1,2]. If a large chorioangioma is present, the fetus could develop hydrops fetalis as a result of the increased cardiac output required by the presence of the arteriovenous shunt. Hydramnios occurs in approximately 30% of cases of grossly identified chorioangiomas, increasing maternal discomfort as well as heightening the risks of preeclampsia, premature delivery, and postpartum hemorrhage.

Bleeding in the first half of pregnancy is frequently alarming to the patient but is rarely life threatening. Initial contact is often by telephone. The physician must attempt to assess the mother's condition quickly. If there is any question concerning the amount of bleeding or if the patient is acutely symptomatic, she is instructed to go immediately to the hospital. Whether to advise transport by ambulance will depend on the patient's particular history as well as local practices. In less pressing circumstances, examination in the office or clinic can be

arranged either the same day or at another mutually convenient time.

Once at the hospital, the patient's condition should be rapidly evaluated. A detailed history and vital signs should be obtained, including postural blood pressure and pulse. Even if vital signs are initially normal, serial measurements are prudent. If bleeding is thought to be serious, one or more large-bore intravenous lines are inserted. Blood should be drawn for diagnostic studies, including coagulation testing and blood type and cross-match. An isotonic salt solution such as lactated Ringer's solution or normal saline is infused with an initial bolus of 500 ml to 1,000 ml. The amount of blood seen in the hospital is correlated with the history and physical findings. The amount of visible blood with abruptio placentae is sometimes less than expected from the apparent severity of the maternal condition, because the bleeding can be concealed. Subsequent steps depend on the history, the laboratory data, the outcome of physical examinations, a bedside real-time ultrasound study, the patient's response to initial therapy, fetal heart monitoring if the pregnancy is viable, and the presence or absence of continued bleeding.

Maternal well-being is paramount for fetal survival, and strenuous efforts are appropriate to improve or sustain her condition if she is found to be hemodynamically unstable. Measures include aggressive fluid therapy as well as blood or blood product transfusion, as required to sustain vascular volume and oxygen-carrying capacity, and to maintain urinary output.

In addition to the steps reviewed previously, interventions at or about the period of potential viability (23–24 weeks gestational age) are critically important. It is imperative to know the gestational age accurately, whether the fetus is alive, and the site of the placenta. With a prompt real-time scan, fetal heart motion is easily detected, a rough gestational age can be determined, and complete placenta previa can often be identified without potentially compromising patient care by transportation to another area. Future management is determined by the cause of bleeding, the clinical course for mother and fetus, and the gestational age.

The initial bleed of a placenta previa is usually self-limited, hence its historical reference as the "sentinel" bleed. If the initial episode of bleeding abates spontaneously, further evaluation is possible.

If the patient is close to fetal viability and the bleeding is not life threatening, the gestation can continue under close observation. If the pregnancy is remote from viability, patient may opt to terminate the pregnancy in the face of continued non-life-threatening bleeding. The source and degree of the bleeding, need for transfusion, potential effect on fetal growth and well-being, and the possibility of long-term hospitalization must be considered in this decision. If at any point the hemorrhage is considered life threatening, the physician must act promptly to end the pregnancy, regardless of the fetal outcome.

### Potentially Viable Gestation

Once there is a possibility of survival of the fetus, management must consider fetal as well as maternal well-being. As noted and previously discussed, the bitter edge of fetal viability in most institutions is now 23 to 24 weeks of gestation with an estimated fetal weight of 350 g to 450 g. Although there is variation between institutions in their survival statistics and difficulty in correctly assigning gestational age, below these limits of gestational age and weight there are usually very few if any intact survivors. In patient counseling, clinicians must know the statistics from their own services as well as an accurate estimate of gestational age and fetal weight. The woman, the family, and the physician must consider the risks and benefits of any management plan, especially when the plan places either the mother or fetus at risk to benefit the other. (See Chapter 26, Ethical Issues.)

### Placenta Previa

The initial bleed of placenta previa is seldom life threatening to mother or fetus. The patient might describe a gush of blood at home and often has minimal bleeding on arrival to the hospital. If she is easily stabilized, the gestational age and the etiology of the bleeding become the critical factors in subsequent management. History, clinical examination, and ultrasonic scanning are used to determine the correct diagnosis (Table 7.3). Most patients with placenta previa who are remote from term can be managed expectantly, with significant prolongation of their pregnancy. If the gestation is at or near term, however, prematurity is not the risk; the problem is

the severity of bleeding. If the patient is 37 weeks' gestation or later by reliable dating, then delivery is the treatment of choice. In cases involving a late preterm gestation (33–36 weeks) in which bleeding stabilizes clinically, amniocentesis and determination of fetal pulmonary maturity can provide additional data to help the clinician reach a management decision. In general, pulmonary maturity in a problematic case should prompt the clinician to favor delivery. If the woman is stable on bed rest and neither she nor the fetus is distressed, however, preterm delivery should not be performed based simply on a finding of pulmonary maturity. If there is a decision to proceed to delivery and the diagnosis of placenta previa is in question, this is almost always resolved by one of the techniques of ultrasonic scanning previously described. In rare instances, a double setup examination can be performed in the operating room. Otherwise, the safest course is to proceed with a cesarean.

Planning for surgery is extremely important. Such cases are often more difficult than performing a cesarean delivery for other indications. Because of the risk of rapid and extensive blood loss, at least four units of cross-matched blood should be available. Although most of the observed blood loss is maternal in origin, approximately 18% of newborns born to mothers with placenta previa are initially anemic or hypotensive. The pediatrician should be aware of the maternal diagnosis and might desire O-negative blood to be available for potential transfusion to the neonate. When obtaining surgical consent, there must be a discussion of the risk(s) of extensive hemorrhage with the possible need for transfusion, the use of additional uterus-sparing procedures to control hemorrhage (i.e., uterine or hypogastric artery ligation, B-Lynch suture placement, embolization), or possibly, hysterectomy. In planning for the case, it is important for the operating physician to know the patient's wishes regarding future childbearing. In difficult cases, it is best to include a note in the medical record preoperatively outlining the clinical circumstances and the management decisions that have been reached. This notation should include statements that make explicit that the possibility of additional intraoperative procedures, blood transfusions, and hysterectomy were discussed.

Prior to surgery, the operator should be aware of the placental location as well as the fetal presen-

tation. When possible, it is desirable to perform a low transverse incision in the myometrium, but this plan could be altered by the discovery of a poorly developed or highly vascularized lower segment or an abnormal fetal lie. The uterine incision should also avoid the placenta if possible. If the placenta is located anteriorly, the surgeon should proceed rapidly around the edge of the chorionic plate rather than through it. If the umbilical cord appears in the incision prior to the infant, it is best clamped before delivery of the child to minimize fetal blood loss. Otherwise, rapid delivery of the fetus and subsequent cord clamping is performed in the usual manner.

Intraoperative and postpartum hemorrhage is a common problem. With low-lying placentation, the lower uterine segment is usually heavily vascularized and characteristically contracts minimally after the fetus is delivered. Oxytocin, methergine/ergotrate, 15-methylprostaglandin F<sub>2</sub>α, (Hemobate), and rectal misoprostol (PGE<sub>1</sub>) may be administered to firm the uterus; however, these drugs are sometimes unsuccessful. Uterine artery or utero-ovarian artery ligation is often performed next and is frequently successful. If uterotonics and ligation are unsuccessful, however, direct ligation of large myometrial venous lakes is occasionally helpful to decrease the overall blood loss and increase visibility in the operative field. Uterine gauze packing or the use of intrauterine balloons can also control bleeding, particularly in cases of a slow, continuous venous ooze; otherwise either technique can assist as a temporizing measure while preparations for more definitive treatment are arranged (i.e., blood obtained, personnel organized, and so forth). Classic gauze packing and other forms of uterine compression treatment are discussed in greater detail in Chapter 11, The Third Stage, and Chapter 18, Cesarean Delivery and Surgical Sterilization.

A hypogastric artery ligation is best reserved for cases in which there is a hemorrhage from a cervical or high vaginal tear that is unresponsive to other treatments. For most obstetric hemorrhage, direct uterine artery ligation (O'Leary suture, see Chapter 11) or combined uterine artery and utero-ovarian artery ligation is best. Vessel ligations rarely engender complications due to ischemia because arterial vessels lack valves and thus retrograde flow is possible through the rich collateral pelvic circulation. The decrease in both pulse pressure and blood

flow to the lower uterine segment following vessel ligation allows local clotting and uterine contractions to control the bleeding. In selected cases, vessel embolization is another technique that is proven to successfully control bleeding. During an episode of hemorrhage, continuous and careful assessment of blood loss and maternal cardiovascular function and urinary output is critical. Delays in initiating transfusion or in performing a hysterectomy while other methods of temporizing treatment are attempted could place the patient at significant risk.

If various measures for the control of bleeding are unsuccessful, or if the patient becomes progressively hemodynamically unstable despite aggressive therapy, the surgeon must proceed to hysterectomy. In a specific case, knowing the patient's future child-bearing plans could allow the surgeon to move more quickly to hysterectomy, often with less blood loss and improved outcome. Owing to the substantially increased morbidity of cesarean hysterectomy, however, this operation should not be planned solely for reasons of permanent sterilization. Total hysterectomy is technically more difficult than the usual supracervical procedure, increasing the risk for blood loss and complications such as ureteric injury. Unfortunately, removal of both the lower uterine segment and the cervix is often required in cases of placenta previa or when unusual placental adherence is present in order to control the hemorrhage. (See Chapter 18, Cesarean Delivery and Surgical Sterilization, for additional discussion.)

The necessity for cesarean hysterectomy is increased significantly if placenta accreta is suspected, either because of ultrasound findings or due to the historical risk factors of a previous cesarean delivery or a prior placenta previa. This should prompt the clinician to make the necessary arrangements if this more extensive surgery is required. If an accreta is encountered intraoperatively and the patient desires sterility, it is best to proceed directly to hysterectomy. If the area of accreta is small and the patient desires more children, however, conservative approaches such as oversewing the implantation site or local excision have been attempted with occasional success, but at the risk of significant complication [47,48].

Management of preterm placenta previa is complex. Prematurity is the major contributor to perinatal mortality. When the diagnosis of other than a central previa is made by ultrasound in an *asymptomatic patient in the second trimester*, no restrictions in activity are recommended because of the high likelihood of the placenta's "migrating" before term, as discussed previously. Minor bleeding after intercourse or digital examination can be managed by cessation of those activities. Spontaneous bleeding is managed initially by hospitalization and extensive evaluation.

Complete placenta previa tends to bleed earlier in gestation and more extensively than partial previa and predisposes to preterm delivery. It is usually the *severity* of bleeding and not the *frequency* that correlates best with the need for early delivery and ultimate perinatal mortality.

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Expectant management in hopes of achieving a mature fetus at the time of delivery has been the standard approach to symptomatic preterm placenta previa for at least 50 years. Fortunately, approximately 50% of patients with placenta previa are delivered after 36 weeks' gestation, even if the bleeding initially began considerably prior to term [33]. When the symptoms occur, the patient is placed on restricted activity and if she is Rh negative, she is evaluated for transplacental hemorrhage and immune globulin is administered, as required. Some clinicians hospitalize women automatically after the first serious bleed. Management in the hospital can be initiated at the time of a second or third bleed if the initial episodes are not serious and the patient is compliant. There are lower maternal costs for out-of-hospital care, but, more important, higher average birthweights and lower rates of neonatal complications among hospitalized patients [80]. A recent randomized controlled study confirmed a significant savings in maternal hospital charges without a demonstrated adverse effect on maternal or neonatal outcomes (Table 7.4) [81]. In all cases, clinicians must individualize care. Based on current data and clinical experience, a role exists for both in- and out-of-hospital care in carefully selected cases. Management should be tailored to the individual patient and not the diagnosis.

If the bleeding is not life threatening and the fetus is stable, ultrasound scan is used to confirm gestational age and document fetal growth. Fetal testing is appropriate if the physician might intervene at that gestational age for a fetal indication. Fetal position and placental position should be studied, while the extent and duration of maternal bleeding are determined by pad counts, clinical observation,

TABLE 7.4 Inpatient Versus Outpatient Management of Placenta Previa

Outcome Measure	Inpatient (n = 27)	Outpatient (n = 26)	p Value
Gestational age at delivery (weeks)	34.5 ± 2.4	34.6 ± 2.3	0.88
Hospital stay (days)			
Mother	28.6 ± 20.3	10.1 ± 8.5	≤0.0001
Neonate	20.9 ± 20.8	17.9 ± 18.4	0.71
Birthweight (g)	2413.7 ± 642.7	2607.8 ± 587.1	0.26

Modified from Wing DA, Paul RH, Millar LK: Management of the symptomatic placenta previa: A randomized control trial of inpatient vs. outpatient expectant management. *Am J Obstet Gynecol* 1996; 175(4):806–11; with permission.

serial assessment of vital signs, and hematocrit values.

If the mother is hemodynamically unstable or if fetal jeopardy is diagnosed at a potentially viable gestational age ( $\geq 23$ –24 weeks,  $\geq 400$ –450 g) and if rapid, aggressive treatment does not promptly improve clinical status, cesarean delivery is indicated. Preparations for surgery and the potential complications in preterm deliveries are similar to those at term. In such cases, there is a higher chance of the need for a nontraditional or classic uterine incision because of fetal malpresentation. Also, there is the likelihood of a poorly developed lower uterine segment and hemorrhage from the placental attachment site.

After the initial stabilization period, the major dilemma facing the clinician is how to balance the risks of prematurity against those of continued bleeding. Several strategies have been attempted to try to prolong gestation without further jeopardizing maternal health, including prophylactic blood transfusion, use of tocolytic agents, and cervical cerclage.

Blood transfusion is clearly indicated for the patient with continued hemorrhage and unstable vital signs. Some patients, however, experience a substantial loss of blood over a short period but then stabilize and experience no further bleeding. In this setting, judicious transfusion might permit a delay in delivery until a more advanced gestational age is reached. There is considerable disagreement over the criteria for blood transfusion. Historically, many advocated maintaining the hematocrit above arbitrary values such as 25%, because of the potential for emergency surgery as well as the theoretical need for oxygen-carrying capacity for the fetus.

Recent reports have emphasized abandoning the use of an absolute value for the hematocrit in favor of careful evaluation of the patient's symptoms and the projected clinical course, using these physiologic criterion as the basis for the decision to transfuse [82]. Ongoing blood loss, postural changes in vital signs, evidence of acute hypoxia, or the imminent need for surgery are examples of additional important criteria to consider. With education of medical personnel and quality assurance audit, the number of transfusions can safely be restricted without increased risk to the patient [83]. Apart from the savings in transfusion costs, the small but real risks of hepatitis, other viral infection, transfusion reactions, and human immunodeficiency virus (HIV) transmission are also reduced or avoided. This is yet another area where the rule of reason must apply. Transfusion must not be unnecessarily withheld by clinicians or rigidly insisted upon by them without a reasonable basis for the decision and a long-term plan for management.

Often when a woman is admitted with bleeding from placenta previa, uterine activity is evident on uterine palpation or external fetal monitoring. It is unclear whether such activity is the cause for the onset of bleeding or is the result of it. Not surprisingly, there have been efforts to attempt to disrupt this cycle with tocolysis. Typically, parenteral  $\beta$ -mimetic drugs have been avoided because of their effects on maternal blood pressure and pulse, which can make it difficult to distinguish drug side effects from signs of hypovolemia due to hemorrhage. For similar reasons, many have avoided calcium channel blockers as tocolytic agents in placenta previa despite their overall favorable profile as a tocolytic agent compared with magnesium

sulfate [84]. One recent trial randomized 60 patients with placenta previa and uterine activity to tocolysis with the  $\beta$ -mimetic ritodrine or expectant management. Rates of maternal complications did not differ significantly between study groups, and the number of blood transfusions was similar [85]. Magnesium sulfate has also been administered in several studies, reportedly with successful prolongation of gestation and without apparent adverse fetal effects [86,87]. However, the risk/benefit profile for magnesium is questionable and this drug has been used less frequently in recent years because of limited efficacy and possible adverse fetal effects. Thus, although tocolysis appears safe in selected cases, there is no ideal agent and tocolysis has not replaced simple expectant management. Tocolysis is reserved for patients remote from term in stable cardiovascular status with normal coagulation studies. In such women, if uterine activity or changes in cervical length as documented by ultrasound scanning suggests preterm labor or if there is increased bleeding associated with contractions, tocolysis may be considered. This issue is discussed in greater detail later.

Cervical cerclages have been used as treatment for various causes of preterm delivery, including placenta previa. Since prophylactic cervical cerclages are placed early in the second trimester there is occasionally a low-lying or placenta previa present. One study randomized patients with placenta previa admitted with bleeding after 24 weeks' gestation to cerclage or conservative treatment [88]. In this small series of twenty-five patients, there was an improvement in gestational age at delivery, mean birthweight, and reduced neonatal complications for those in the cerclage group over those managed in the conventional manner. Maternal bleeding and hospital costs were lower for this group as well. Unfortunately, this study could not differentiate from the placenta that migrated away from the os versus those that were not previa to start with. A more recent randomized study showed a reduction in antenatal hospital stay with cerclage, although no improvement in overall outcome [89]. In sum, the benefits of cerclage in cases complicated by a placenta previa are at best uncertain. Based on available data we do not advocate routine cerclage for placenta previa management.

Whichever strategies are chosen to prolong gestational age with placenta previa, approximately one

half of the cases will deliver prematurely, although delivery with the initial bleeding episode is uncommon. There is usually sufficient time to offer antenatal corticosteroids to reduce the risk of respiratory and other complications of prematurity. The role of antenatal corticosteroids (betamethasone, 12 mg IM, two doses 24 hours apart) is no longer controversial [90]. Traditionally, steroids were administered between 26 and 32 weeks of gestation. Although there are few confirmatory data, these limits have empirically been extended by most clinical services to approximately 24 weeks on the lower end and 34 weeks on the upper. The once common clinical practice of repeating antenatal corticosteroid weekly until 34 weeks after an initial course is now discouraged given the unclear benefit of this therapy and the concern for adverse neonatal complications.

### Placenta Accreta

Although delivery considerations for a suspected placenta accreta are not dissimilar from patients with a placenta previa, the likelihood for cesarean hysterectomy, and the increased potential for significant maternal hemorrhage or extrauterine involvement of placental tissue often requires careful preoperative planning and coordination of a multidisciplinary team.

The timing of delivery in patients with suspected placenta accreta, as with placenta previa, balances the risks for morbidity associated with fetal prematurity against the potential for late or untimely intervention (i.e., after the occurrence of significant maternal blood loss). There is a distinct advantage in this group of patients for a scheduled delivery so that an appropriate obstetric operating team with selected consultants available for assistance can be ready. This team might include a gynecologic oncologist, urogynecologist, or interventional radiologist to assist with surgery and control of hemorrhage. As with placenta previa, delivery at 37 to 38 weeks' gestation is an arbitrary although nonetheless reasonable time frame for asymptomatic patients. One could argue that lung maturity testing is beneficial in these cases, but not essential, since the longer a patient remains undelivered the greater the risk of labor and bleeding. Choice of incision on the uterus will depend on the position of placenta (anterior previa vs. posterior), of the fetus (transverse lie, back down), and the prior number of cesareans.

Decision making for early intervention is problematic if the diagnosis is uncertain. When the placenta is a previa and unusual adherence is strongly suspected, surgical exploration must be considered, and the question becomes the presurgical arrangements in terms of assistants, blood preparation, and so on. If the placenta is not a previa, best management must be individualized as the antepartum diagnosis of unusual adherence is not invariably accurate. Intraoperatively, if the diagnosis appears doubtful after careful inspection, it is appropriate to attempt to express or extract the placenta after delivery of the infant. A major degree of placental adherence or massive maternal hemorrhage in women of advanced parity usually prompts hysterectomy. How best to proceed in other cases depends on the surgical findings, the skill of the surgeons, and the hemodynamic status of the mother. When unusual adherence is strongly suspected, especially if there is a presumptive diagnosis of an anterior accreta, the use of preoperatively placed ureteral stents and a three-way Foley balloon catheter have been recommended to facilitate surgical dissection and limit urologic injury. Preoperative placement of intravascular balloon catheters in the internal iliac arteries and the use of cell-saver technologies have also been described as a means to limit intraoperative blood loss [91]. Despite their apparent reasonableness, the clinical benefit of these interventions in avoiding complications and reducing overall blood loss is yet to be proved.

In some cases the management of placenta accreta without hysterectomy can be contemplated [92–94]. Surgical approaches to provide uterine conservation vary but can include repair of the hysterotomy without disturbance of the attached placenta, focal resection of adherent placenta, or segmental resection with subsequent uterine repair [93,94]. Methotrexate has been administered as an adjunct in this setting [92]. More cases of successful conservative management follow when a vaginal delivery occurs with a placenta accreta, which is then diagnosed and subsequently left in place without efforts at removal. Cases of successful conservative management are few and the rate of complication remains high, however. Because this is an area of continuing study, approaches to placenta accreta that do not involve surgical removal of the placenta or hysterectomy are not considered standard

but might prove appropriate in unique situations with close follow-up.

### **Abruptio Placentae**

The management of abruptio placentae is similar to that for placenta previa and depends largely on the extent of bleeding and the gestational age [95,97]. Once the initial assessment is completed and the patient is stabilized, as described previously, ultrasound scanning is performed to rule out placenta previa. In the absence of previa as an explanation for the bleeding, vaginal and cervical examinations are performed to identify local causes, including labor. If these evaluations are negative, abruptio placentae or the poorly defined entity of marginal sinus separation becomes the presumptive diagnosis. Occasionally, ultrasound diagnosis of abruptio placentae is possible when an extramembranous or retroplacental hematoma is seen. These cases, however, are the exception rather than the rule.

The severity of placental abruptions ranges widely from the clinically insignificant to extremely severe, leading to fetal death and threatening the life of the mother. In cases involving women with prior severe abruptions but who lack established risk factors such as hypertension or a thrombophilia, best management in a subsequent pregnancy is unclear. While it is known that the overall recurrence risk is approximately 10%, the accurate prediction of the risk of recurrence in a specific pregnancy at a specific time is not possible. Neither patient nor clinician can relax until the fetus is successfully delivered. Developing symptoms such as bleeding, cramping, or decreased fetal movements might occasionally be noted and assist the clinician in timing intervention. Yet, in most cases little is observed and often no overt symptoms herald a recurrence. Close observation with frequent clinical visits, serial attention to Doppler flow and fetal growth, and electronic fetal monitoring tests are often performed but are not predictive of recurrence risk. As such cases are followed, consideration of intervention begins once the period of viability is reached. Steroids are often administered after the 28th week and the intensity of observation is increased after the pregnancy advances into the third trimester. When to intervene and how to achieve delivery in such difficult cases cannot be determined by protocol and care must be individualized.



Mild cases of abruption are often diagnosed only in retrospect when the placenta is examined. They are often mistakenly diagnosed as preterm labor or have been described as marginal sinus bleeding. Initial treatment of mild cases without coagulopathy or previa consists of bed rest and observation with appropriate use of fetal surveillance. The clinical course of abruption is unpredictable. There is a concern that the degree of separation could increase, leading to increased maternal symptoms, anemia, coagulation abnormalities, or, in the rare case of chronic abruption, progress to intrauterine growth retardation. To date, there is minimal evidence beyond the anecdotal to support these concerns. Nonetheless, most clinicians favor keeping patients with a diagnosed abruptio placentae at limited activity for the rest of their pregnancy [33].

With a preterm gestation with an abruption, intermittent bleeding is often associated with increased uterine activity. As with placenta previa, tocolysis has been used in selected cases of abruption with the effort to disrupt the cycle of bleeding, contractions, and more bleeding. In selected cases, gestation has been prolonged with tocolysis without apparent adverse effects on the mother or fetus [95]. There remain substantial uncertainties concerning this management strategy, despite its popularity. A concern is the agents available for tocolysis. The sympathomimetic properties of the  $\beta$ -agonists result in maternal restlessness, tachycardia, and blood pressure changes that mimic the symptoms of occult blood loss. Because of such concerns, in the past magnesium sulfate was chosen because of its limited effect on maternal cardiovascular function and maternal symptomatology. In recent years, magnesium has been looked upon much more critically, with calcium channel blockers being more popular as tocolytics.

Any treatment has the limitation of knowing if the applied therapy was in fact successful. The essential difficulty is in evaluating an apparently successful intervention from the natural history of mild-to-moderate abruption as most of the less severe forms will spontaneously improve. Unfortunately, there is no prospective randomized study of mild abruption to demonstrate the benefit of tocolysis over expectant management. When the clinical diagnosis is abruptio placentae, the active inhibition of uterine contractions is best restricted to hemodynamically stable women with minimal symptoms

and no evidence of coagulopathy. Treated pregnancies should be remote from term and the fetus believed to be pulmonically immature.

Atypical presentations for abruption are also possible. Abruption occasionally presents as a chronic process with recurring episodes of minor bleeding and uterine irritability extending over days or even weeks (e.g., Breus' mole). In these cases, close observation in the hospital with serial physical examinations, fetal heart rate monitoring, ultrasound studies, and laboratory tests (e.g., hematocrit, platelet count, and fibrinogen levels) can be necessary to establish the correct diagnosis. As noted, in some instances the only presenting complaint is uterine activity and neither pain nor observed vaginal bleeding is present.

In some instances, the timing or severity of the clinical presentation makes best management easy. If the diagnosis of the abruption is made at or about term, delivery is indicated, usually by induction if labor is not present at the time of admission. Cesarean delivery is appropriate if the fetus does not tolerate the stresses of labor or if there are other obstetric indications.

Immediate clinical management of an active abruption must be swift, with careful attention to both maternal and fetal status. In the acute evaluation of an episode of abruptio placentae, the initial steps are to quantify the degree of bleeding, determine the woman's coagulation status, note fetal condition, and evaluate the maternal cardiovascular status. Several classifications have been developed; these essentially divide patients into a mild, minimally symptomatic category without hematologic abnormalities, and a moderate-to-severe group. The latter cases are marked by vaginal bleeding, uterine tenderness, laboratory abnormalities, or fetal distress [33]. All save the occasional patients with minimal symptoms, remote from term, with stable coagulation studies, require delivery.

Significant hemorrhage, unstable vital signs, pain out of proportion to visible blood loss, fetal distress, and evidence of clotting abnormalities are signs consistent with moderate-to-severe abruption. In such women, two large intravenous lines should be inserted and at least four units of cross-matched blood prepared. Two units of fresh-frozen plasma are also often requested. An indwelling urethral Foley catheter is helpful to evaluate fluid balance and renal perfusion. If there is evidence of acute

fetal jeopardy or if the fetus is dead, the clinician can confidently conclude that the patient has lost sufficient blood to warrant immediate transfusion. On the initial review of laboratory results the extent of the blood loss might not be appreciated, and a drop in fibrinogen concentration, the presence of elevated fibrin split products, or thrombocytopenia could be the first indication of the seriousness of the abruption. Treatment is based largely on clinical observations and must be aggressive. Regarding the laboratory data, the fibrinogen level is usually the best single indication of severity. Levels less than 250 mg/dl are clearly abnormal and demand prompt action. Despite evidence of coagulopathy, there is no evidence that heparin treatment improves outcome. Within 12 hours of delivery, the coagulation abnormality usually resolves or substantially improves. After initial stabilization, a plan for delivery is formulated. Although the clinician's initial response often is to proceed to cesarean delivery, the maternal and fetal status, gestational age, and cervical ripeness or dilatation occasionally permit a vaginal delivery. Especially in the woman with a previable gestation or fetal demise, it is desirable to avoid the additional surgical risk of cesarean delivery unless intervention is required for pressing maternal signs and symptoms. Fortunately, patients in active labor with a normal fetal heart tracing are likely to deliver expeditiously because of the intense uterine activity normally associated with separation.

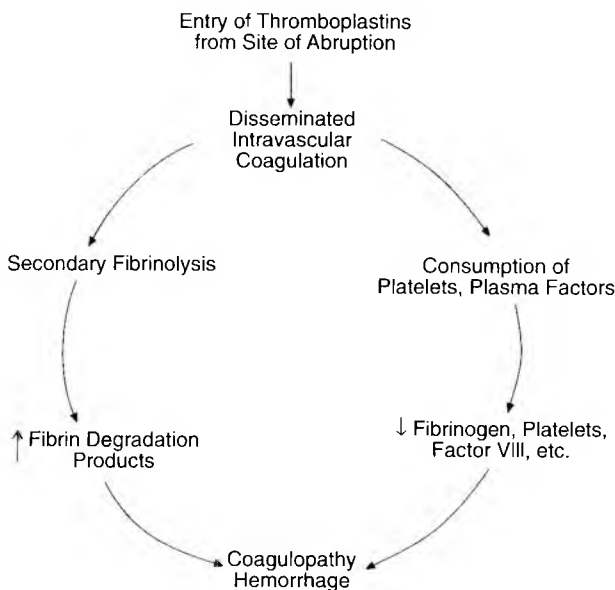
If a trial of labor is to be undertaken, and the membranes remain intact, an amniotomy should be performed. This is thought to decrease blood loss into the myometrium as well as to accelerate or stimulate labor. After amniorrhexis, careful intrapartum monitoring is paramount with both a fetal scalp electrode (for a potentially viable fetus) and an intrauterine pressure catheter. This provides for an accurate assessment of fetal heart rate as well as for measurement of uterine contractility and resting tone. A high resting tone (i.e., >20 mm Hg with an internal pressure transducer) can decrease blood flow to the fetus and predispose to extravasation of blood into the myometrium (Couvelaire uterus). When this occurs, labor is often ineffective and fetal distress is more likely. When fetal monitoring is reassuring and the resulting tone normal, the mother is continually evaluated with serial vital signs, assessment of urine output, hematocrit, platelet counts, and fibrino-

gen concentration determinations while labor progresses. Oxytocin can be added judiciously if contractions are ineffective, since there is an inherent tendency for uterine hyperstimulation with abruption; a low-dose protocol is recommended.

In the absence of immediate fetal or maternal instability necessitating delivery, monitoring for both fetal heart rate abnormalities and uterine activity is helpful in evaluating fetal status and establishing the correct diagnosis. In the presence of bleeding, the uterine tocodynamometer might show frequent low-amplitude contractions, increased baseline resting tone, or prolonged tetanic contractions – all suggesting abruptio placentae as the correct diagnosis. When placental separation is advanced, fetal distress in the form of decreased beat-to-beat variability, recurrent late decelerations or prolonged bradycardia may occur. Particularly in the absence of a definitive diagnostic test for abruptio placentae, such clinical information is invaluable and could lead the physician to intervene for fetal indications even if the mother appears clinically stable. (See Chapter 22, Fetal Assessment, for additional discussion of monitoring and fetal conditions.)

Prior to the routine use of fetal heart rate monitoring, there was an improved perinatal survival with a liberal use of cesarean delivery as opposed to vaginal delivery [33]. Fetal loss was especially high in the first few hours after admission. Today, because electronic fetal monitoring (EFM) permits continuous fetal assessment, circumstances have changed; nonetheless, fetal risks remain high. In moderate abruption, approximately 60% of fetuses will develop EFM evidence of acute fetal jeopardy [96]. In these cases, cesarean delivery is usually best. With reassuring monitor strips in patients without serious coagulopathy, however, induction is possible in selected cases, and labor can result in a successful vaginal delivery. By adopting such protocols, Hurd and coworkers demonstrated a 50% reduction in their cesarean delivery rate for abruptio placentae without an increase in perinatal mortality [97].

From the maternal point of view, surgery is best avoided. Particularly in the presence of a clotting disorder, surgery is often complicated by excessive bleeding, which risks increased maternal morbidity. In the operating room, careful attention must be paid to bleeding sites with liberal use of cautery



**FIGURE 7.8.**  
*Pathogenesis of the coagulation disorder in abruptio placentae.* (From Green JR: *Placenta previa and abruptio placentae*. In: Creasy RK, Resnik R (eds): *Maternal-Fetal Medicine: Principles and Practice*. Philadelphia: WB Saunders, 1989; pp. 592–612; with permission.)

or ligation to provide hemostasis. Additionally, vigorous volume replacement with packed red blood cells and fresh-frozen plasma is required to replace red blood cells and clotting factors. If fibrinogen levels are low (<100 mg/ml), then cryoprecipitate is the best therapy. With prompt diagnosis and active treatment, infusion of other specialized blood products such as platelets is uncommonly required. With rapid restoration of blood volume and prompt delivery, coagulation studies progressively autocorrect. Postoperative and postpartum monitoring remains critical, however, because bleeding and coagulation disorders often require hours for correction. Consultation with the anesthesiologist as soon as the patient is identified ensures a coordinated approach to fluid and blood replacement, particularly if surgery is necessary. In unusual or complex cases, consultation with a hematologist is recommended (Figure 7.8).

In approximately 8% of cesarean deliveries performed for abruptio placentae, massive extravasation of blood into the myometrium is encountered—the Couvelaire uterus [96]. In this condition, blood infiltrates into myometrium and subserosa, discol-

oring the uterus. This condition is associated with postpartum hemorrhage secondary to uterine atony. Classically, hysterectomy was recommended when a Couvelaire uterus was discovered. Fortunately, aggressive use of uterotonics combined with judicious use of vessel ligation has greatly reduced the necessity for removal of even a Couvelaire uterus. Presently, hysterectomy should not be performed solely for a diagnosis of Couvelaire uterus since most do respond to uterotonics and conservative surgery.

## COMPLICATIONS

As expected, the most common complications for both mother and fetus that stem from placental abnormalities are direct consequences of hemorrhage. Acutely, hemorrhagic shock and acquired coagulopathies pose the greatest risk. A nonreassuring fetal heart rate pattern, preterm delivery, and hematologic abnormalities in the neonate are also possible outcomes. Even after the initial threat has passed, Rh isoimmunization, organ damage from hypoperfusion, and intrauterine growth retardation are potential long-term consequences. These risks must be considered when formulating a management plan.

## CONCLUSION

Placental abnormalities can lead to maternal adverse outcomes such as hemorrhage requiring transfusion, more extensive surgery, sterility, emotional consequences, and even death. The physician must be aware of these problems and be sensitive to the fears of the patient and her family. When there is time, detailed discussions with the family are certainly appropriate and can help to put their concerns into perspective.

Newborn outcomes are extremely varied and dependent on prenatal as well as intrapartum and neonatal events. Prematurity, antepartum hemorrhage, intrauterine growth retardation, congenital malformations, antepartum and intrapartum asphyxia, and neonatal complications have all been implicated in placental abnormalities described in this chapter. The placenta must be understood as the key to a successful pregnancy. Unfortunately, we are only just beginning to develop methods of diagno-

sis and treatment for placental disorders based upon scientific knowledge of function and pathophysiology. There are presently limited methods to test placental function. Further, there are few manipulations that are demonstrated to improve pregnancy outcome in cases in which placental dysfunction is believed to be a major component. Improved access to prenatal care, smoking cessation and drug abstinence, better diet, hygiene, and, in highly selected cases, administration of aspirin and anti-clotting agents such as heparin have resulted in better outcomes in some cases. It is hoped that new technologic advances will improve our abilities in coming decades. Until more is known about placental function and its modification, however, we are limited to modifications in prenatal care, close fetal surveillance, and ultimately, timely delivery as the principal means of therapy.

## REFERENCES

1. Benirschke K, Kaufmann P: Pathology of the Human Placenta, 2nd ed. New York: Springer-Verlag, 1990; pp. 1–12.
2. Rushton DI: Pathology of placenta. In: Wigglesworth JS, Singer DB (eds): Textbook of Fetal and Perinatal Pathology. Boston: Blackwell Scientific Publications, 1991; pp. 161–219.
3. McFadyen IR: The pathophysiology of intrauterine growth retardation. In: Kurjak A, Beazley JM (eds): Fetal Growth Retardation: Diagnosis and Treatment. Boca Raton, FL: CRC Press, 1989; pp. 29–51.
4. Kaufmann P, Scheffen I: Placental development. In: Polin RA, Fox WW (eds): Fetal and Neonatal Physiology. Philadelphia: WB Saunders, 1992; pp. 47–56.
5. Fox H: Normal placentation and development. In: Reece AE, Hobbins JC, Mahoney MJ, Petrie RH (eds): Medicine of the Fetus and Mother. Philadelphia: JB Lippincott, 1992; pp. 59–66.
6. Fox H: Placental pathology, abnormal placentation, and clinical significance. In: Reece AE, Hobbins JC, Mahoney MJ, Petrie RH (eds): Medicine of the Fetus and Mother. Philadelphia: JB Lippincott, 1992; pp. 67–77.
7. Baschat AA: Pathophysiology of fetal growth restriction: Implications for diagnosis and surveillance. *Obstet Gynecol Surv* 2004; 59: 617–27.
8. Rushton DI: Placental pathology in spontaneous miscarriage. In: Beard RW, Sharp F (eds): Early Pregnancy Loss: Mechanisms and Treatment. London: Royal College of Obstetricians and Gynecologists, 1988; pp. 149–57.
9. Brosens IA: The utero-placental vessels at term – the distribution and extent of physiological changes. In: Kaufmann P, Miller K (eds): Placental Vascularization and Blood Flow: Trophoblast Research 3. New York: Plenum Press, 1988; pp. 66–8.
10. Sheppard BL: The maternal blood supply to the placenta in pregnancy complicated by intrauterine growth retardation. In: Kaufmann P, Miller K (eds): Placental Vascularization and Blood Flow. Trophoblast Research 3. New York: Plenum Press, 1988; pp. 69–82.
11. Wigglesworth JS, Singer DB (eds): Textbook of Fetal and Perinatal Pathology. Boston: Blackwell Scientific Publications, 1991.
12. Lau TK, Yu SC, Leung TY, To KF, Fung TY, Leung TN: Prenatal embolisation of a large chorioangioma using enbucrilate. *Br J Obstet Gynaecol* 2005; 112(7):1002–4.
13. Quarello E, Bernard JP, Leroy B, Ville Y: Prenatal laser treatment of a placental chorioangioma. *Ultrasound Obstet Gynecol* 2005; 25(3):299–301.
14. Fox H, Sen DK: Placenta extrachorialis: A clinicopathological study. *J Obstet Gynaecol Br Commonw* 1972; 79(1):32–5.
15. Lademacher DS, Vermeulen RC, Hartenn JJ, Arts NF: Circumvallate placenta and congenital malformation. *Lancet* 1981; 1(8222):732.
16. Torpin R: Fetal Malformations Caused by Amnion Rupture. Springfield, Illinois: Charles C Thomas, 1968.
17. Patten RM, Van Allen M, Mack LA, Wilson D, Nyberg D, Hirsch K, Vlamont T: Limb-body wall complex: In utero sonographic diagnosis of a complicated fetal malformation. *AJR Am J Roentgenol* 1986; 146(5):1019–24.
18. Randel SB, Filly RA, Callen PW, Anderson RL, Golbus MS: Amniotic sheets. *Radiology* 1988; 166(3): 633–6.
19. Seki M, Strauss L: Absence of one umbilical artery: Analysis of 60 cases with emphasis on associated developmental aberrations. *Arch Pathol* 1964; 78: 446–53.
20. Chow JS, Benson CB, Doubilet PM: Frequency and nature of structural anomalies in fetuses with

- single umbilical arteries. *J Ultrasound Med* 1998; 17(12):765–8.
21. Woodward PJ, Kennedy A, Sohaey R, Byrne JLB, Oh KY, Puchalski M: Diagnostic Imaging: Obstetrics. Salt Lake City, UT: Amirsys 2005.
  22. Prucka S, Clemens M, Craven C, McPherson E: Single umbilical artery: What does it mean for the fetus? A case-control analysis of pathologically ascertained cases. *Genet Med* 2004 Jan–Feb: 6(1):54–7.
  23. Gornall AS, Kurinczuk JJ, Konje JC: Antenatal detection of a single umbilical artery: Does it matter? *Prenat Diagn* 2003 Feb: 23(2):117–23.
  24. Yovich JL: Embryo quality and pregnancy rates in in-vitro fertilization. *Lancet* 1985; 1(8423): 3–4.
  25. Glass RH, Golbus MS: Pregnancy wastage: Habitual abortion. In: Creasy RK, Resnik R (eds): *Maternal Fetal Medicine: Principles and Practice*. Philadelphia: WB Saunders, 1984; pp. 385–94.
  26. Robertson WB, Brosens I, Dixon HG: Uteroplacental vascular pathology. *Eur J Obstet Gynecol Rep Biol* 1975; 5(1–2):47–65.
  27. Robertson WB, Brosens IA, Dixon HG: Maternal blood supply in fetal growth retardation. In: Van Assche FA, Robertson WB (eds): *Fetal Growth Retardation*. Edinburgh: Churchill-Livingstone, 1981; pp. 126–38.
  28. Anna-Karin Wikström, Anders Larsson, Ulf J. Eriksson, Peppi Nash, Solveig Nordén-Lindeberg, Matts Olovsson: Placental growth factor and soluble FMS-like tyrosine kinase-1 in early-onset and late-onset preeclampsia. *Obstet Gynecol* Jun 2007; 109(6):1368–74.
  29. Levine RJ, Maynard SE, Qian C, Lim KH, England LJ, Yu KF, Schisterman EF, Thadhani R, Sachs BP, Epstein FH, Sibai BM, Sukhatme VP, Karumanchi SA: Circulating angiogenic factors and the risk of preeclampsia. *N Engl J Med* 2004 Feb 12: 350(7):672–83.
  30. Moore-Simas T, Crawford SL, Soltiro MJ, Frost SC, Meyer BA, Maynard SE: Angiogenic factors for the prediction of preeclampsia in high-risk women. *Am J Obstet Gynecol* 2007 Sept: 197(3):244.e1–8.
  31. Giovino GA: The tobacco epidemic in the United States. *Am J Prev Med* 2007 Dec: 33(6 Suppl):S318–26.
  32. Benirschke K, Kaufmann P: *Pathology of the Human Placenta*, 2nd ed. New York: Springer-Verlag, 1990; pp. 373–382.
  33. Green JR: Placenta previa and abruptio placentae. In: Creasy RK, Resnik R (eds): *Maternal Fetal Medicine: Principles and Practice*. Philadelphia: WB Saunders, 1989; pp. 592–612.
  34. Deering SH, Satin A: Abruptio Placentae, eMedicine 2007 (imedicine.com) topic = 6.
  35. Rizos N, Doran TA, Miskin M, Benzie RJ, Ford JA: Natural history of placenta previa ascertained by diagnostic ultrasound. *Am J Obstet Gynecol* 1979; 133(3):287–91.
  36. El Kady D: Perinatal outcomes of traumatic injuries during pregnancy. *Clin Obstet Gynecol* 2007 Sep: 50(3):582–91.
  37. El-Kady D, Gilbert WM, Anderson J, Danielsen B, Towner D, Smith LH: Trauma during pregnancy: An analysis of maternal and fetal outcomes in a large population. *Am J Obstet Gynecol* 2004 Jun: 190(6): 1661–8.
  38. Hyde LK, Cook LJ, Olson LM, Weiss HB, Dean JM: Effect of motor vehicle crashes on adverse fetal outcomes. *Obstet Gynecol* 2003 Aug: 102(2):279–86.
  39. Ali J, Yeo A, Gana TJ, McLellan BA: Predictors of fetal mortality in pregnant trauma patients. *J Trauma* 1997 May: 42(5):782–5.
  40. Rogers FB, Rozycki GS, Osler TM, Shackford SR, Jalbert J, Kirton O, Scalea T, Morris J, Ross S, Cipolle M, Fildes J, Cogbill T, Bergstein J, Clark D, Frankel H, Bell R, Gens D, Cullinane D, Kauder D, Bynoe RP: A multi-institutional study of factors associated with fetal death in injured pregnant patients. *Arch Surg* 1999 Nov: 134(11):1274–7.
  41. Lauria MR, Smith RS, Treadwell MC, Comstock CH, Kirk JS, Lee W, Bottoms SF: The use of second-trimester transvaginal sonography to predict placenta previa. *Ultrasound Obstet Gynecol* 1996; 8(5):337–40.
  42. Dashe JS, McIntire DD, Ramus RM, Santos-Ramos R, Twickler DM: Persistence of placenta previa according to gestational age at ultrasound detection. *Obstet Gynecol* 2002; 99(5 Pt 1):692–7.
  43. Becker RH, Vonk R, Mende BC, Ragosch V, Entezami M: The relevance of placental location at 20–23 gestational weeks for prediction of placenta previa at delivery: Evaluation of 8,650 cases. *Ultrasound Obstet Gynecol* 2001; 17(6):496–501.
  44. Laughon SK, Wolfe HM, Visco AG: Prior cesarean and the risk for placenta previa on second-trimester ultrasonography. *Obstet Gynecol* 2005; 105(5 Pt 1): 962–5.
  45. Rosemond RL, Kepple DM: Transvaginal color Doppler sonography in the prenatal diagnosis of

- placenta accreta. *Obstet Gynecol* 1992 Sep; 80(3 Pt 2):508–10.
46. Miller DA, Chollet JA, Goodwin TM: Clinical risk factors for placenta previa-placenta accreta. *Am J Obstet Gynecol* 1997 Jul; 177(1):210–4.
  47. Faiz AS, Ananth CV: Etiology and risk factors for placenta previa: A review and meta-analysis of observational studies. *J Matern Fetal Neonatal Med* 2003 Mar; 13(3):175–90.
  48. Clark SL, Koonings PP, Phelan JP: Placenta previa/accreta and prior cesarean section. *Obstet Gynecol* 1985; 66:89–92.
  49. Silver RM, Landon MB, Rouse DJ, Leveno KJ, Spong CY, Thom EA, Moawad AH, Caritis SN, Harper M, Wapner RJ, Sorokin Y, Miodovnik M, Carpenter M, Peaceman AM, O'Sullivan MJ, Sibai B, Langer O, Thorp JM, Ramin SM, Mercer BM, National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network: Maternal morbidity associated with multiple repeat cesarean deliveries. *Obstet Gynecol* 2006 Jun; 107(6):1226–32.
  50. Usta IM, Hobeika EM, Abu Musa AA, Gabriel GE, Nassar AH: Placenta previa-accreta: Risk factors and complications. *Am J Obstet Gynecol* 2005 Sep; 193(3 Pt 2):1045–9.
  51. Kupfermink MJ, Tamura RK, Wigton TR, Glassenberg R, Socol ML: Placenta accreta is associated with elevated maternal serum alpha-fetoprotein. *Obstet Gynecol* 1993 Aug; 82:266–9.
  52. Ananth CV, Getahun D, Peltier MR et al: Placental abruption in term and preterm gestations: Evidence for heterogeneity in clinical pathways. *Obstet Gynecol* 2006; 107:785–92.
  53. McDonald SD, Vermeulen MJ, Ray JG: Folic acid and placental pathology. *Epidemiology* 2005 May; 16(3):419–20.
  54. Chasnoff IJ, Landress HJ, Barrett ME: The prevalence of illicit-drug or alcohol use during pregnancy and discrepancies in mandatory reporting in Pinellas County, Florida. *N Engl J Med* 1990 Apr; 26:322:1202–6.
  55. Tabor A, Philip J, Madsen M, Bang J, Obel EB, Norgaard-Pedersen B: Randomised controlled trial of genetic amniocentesis in 4,606 low-risk women. *Lancet* 1986 Jun 7; 1(8493):1287–92.
  56. Ananth CV, Oyelese Y, Yeo L, Pradhan A, Vintzileos AM: Placental abruption in the United States, 1979 through 2001: Temporal trends and potential determinants. *Am J Obstet Gynecol* 2005 Jan; 192(1):191–8.
  57. Balderston KD, Towers CV, Rumney PJ, Montgomery D: Is the incidence of fetal to maternal hemorrhage increased in patients with third-trimester bleeding? *Am J Obstet Gynecol* 2003 Jun; 188(6):1615–8; discussion 1618–21.
  58. Nimrod CA, Petrie RH: Third-trimester bleeding. In: Reece EA, Hobbins JC, Mahoney MJ, Petrie RH (eds): *Medicine of the Fetus and Mother*. Philadelphia: JB Lippincott, 1992; pp. 1363–9.
  59. Timor-Tritsch IE, Yunis RA: Confirming the safety of transvaginal sonography in patients suspected of placenta previa. *Obstet Gynecol* 1993 May; 81(5 Pt 1):742–4.
  60. Farine D, Fox HE, Timor-Tritsch IE: Vaginal approach to the ultrasound diagnosis of placenta previa. In: Chervenak FA, Isaacson GC, Campbell S (eds): *Ultrasound in Obstetrics and Gynecology*. Boston: Little, Brown, 1993; pp. 1503–7.
  61. Oppenheimer L, Holmes P, Simpson N, Dabrowski A: Diagnosis of low-lying placenta: Can migration in the third trimester predict outcome? *Ultrasound Obstet Gynecol* 2001 Aug; 18(2):100–2.
  62. Oyelese Y: Placenta previa and vasa previa: Time to leave the Dark Ages. *Ultrasound Obstet Gynecol* 2001 Aug; 18(2):96–9.
  63. Hertzberg BS, Bowie JD, Carroll BA, Kliever MA, Weber TM: Diagnosis of placenta previa during the third trimester: Role of transperineal sonography. *AJR Am J Roentgenol* 1992 Jul; 159(1):83–7.
  64. Ziliani M, Azuaga A, Calderon F, Redondo C: Transperineal sonography in second trimester to term pregnancy and early labor. *J Ultrasound Med* 1991 Sept; 10(9):481–5.
  65. Hoffman-Tretin JC, Koenigsberg M, Rabin A, Anyaegbunam A: Placenta accreta: Additional sonographic observations. *J Ultrasound Med* 1992 Jan; 11(1):29–34.
  66. Finberg HJ, Williams JW: Placenta accreta: Prospective sonographic diagnosis in patients with placenta previa and prior cesarean section. *J Ultrasound Med* 1992 Jul; 11(7):333–43.
  67. Comstock CH, Love JJ Jr, Bronsteen RA, Lee W, Vettraino IM, Huang RR, Lorenz RP: Sonographic detection of placenta accreta in the second and third trimesters of pregnancy. *Am J Obstet Gynecol* 2004 Apr; 190(4):1135–40.
  68. Kay HH, Spritzer CE: Preliminary experience with magnetic resonance imaging in patients with third-trimester bleeding. *Obstet Gynecol* 1991 Sep; 78(3 Pt 1):424–9.

69. Maldjian C, Adam R, Pelosi M, Pelosi M 3rd, Rudelli RD, Maldjian J: MRI appearance of placenta percreta and placenta accreta. *Magn Reson Imaging* 1999 Sep: 17(7):965-71.
70. Ha TP, Li KC: Placenta accreta: MRI antenatal diagnosis and surgical correlation. *J Magn Reson Imaging* 1998 May-Jun: 8(3):748-50.
71. De Lia JE, Cruikshank DP, Keye WR Jr: Fetoscopic neodymium: YAG laser occlusion of placental vessels in severe twin-twin transfusion syndrome. *Obstet Gynecol* 1990 Jun: 75(6):1046-53.
72. Hecher K, Plath H, Bregenzler T, Hansmann M, Hackeloer BJ: Endoscopic laser surgery versus serial amniocentesis in the treatment of severe twin-twin transfusion syndrome. *Am J Obstet Gynecol* 1999 Mar: 180(3 Pt 1):717-24.
73. Senat MV, Deprest J, Boulvain M, Paupe A, Winer N, Ville Y: Endoscopic laser surgery versus serial amnioreduction for severe twin-to-twin transfusion syndrome. *N Engl J Med* 2004 Jul: 351(2):136-44.
74. Yetter JF: Examination of the placenta. *Am Fam Physician*. 1998: 57.
75. Naeye RL, Tafari N: Risk Factors in Pregnancy and Diseases of the Fetus and Newborn. Baltimore: Williams and Wilkins, 1983.
76. Naeye RL: Pregnancy hypertension, placental evidences of low uteroplacental blood flow, and spontaneous preterm delivery. *Hum Pathol* 1989 May: 20(5):441-4.
77. Clewell WH, Manchester DK: Recurrent maternal floor infarction: A preventable cause of fetal death. *Am J Obstet Gynecol* 1983 Oct 1: 147(3):346-7.
78. Katz VL, Bowes WA, Sierkh AE: Maternal floor infarction of the placenta associated with elevated second tri-mester serum alpha-fetoprotein. *Am J Perinatol* 1987 Jul: 4(3):225-8.
79. Berghella V. Prevention of recurrent fetal growth restriction. *Obstet Gynecol*. 2007 Oct: 110(4):904-12.
80. D'Angelo LJ, Irwin LF: Conservative management of placenta previa: A cost-benefit analysis. *Am J Obstet Gynecol* 1984 Jun 1: 149(3):320-3.
81. Wing DA, Paul RH, Millar LK: Management of the symptomatic placenta previa: A randomized, controlled trial of inpatient versus outpatient expectant management. *Am J Obstet Gynecol* 1996 Oct: 175(4 Pt 1):806-11.
82. U.S. Dept. of Health and Human Services: Indications for the use of red blood cells, platelets, and fresh-frozen plasma. Washington, DC: NIH Publication no. 89-2974A, May 1989.
83. Morrison JC, Sumrall DD, Chevalier SP, Robinson SV, Morrison FS, Wiser WL: The effect of provider education on blood utilization practices. *Am J Obstet Gynecol* 1993 Nov: 169(5):1240-5.
84. King JF, Flenady V, Papatsonis D, Dekker G, Carbonne B: Calcium channel blockers for inhibiting preterm labour: A systematic review of the evidence and a protocol for administration of nifedipine. *Aust N Z J Obstet Gynaecol* 2003 Jun: 43(3): 192-8.
85. Sharma A, Suri V, Gupta I: Tocolytic therapy in conservative management of symptomatic placenta previa. *Int J Gynaecol Obstet* 2004 Feb: 84(2):109-13.
86. Watson WJ, Cefalo RC: Magnesium sulfate tocolysis in selected patients with symptomatic placenta previa. *Am J Perinatol* 1990 Jul: 7(3):251-3.
87. Saller DN Jr, Nagey DA, Pupkin MJ, Crenshaw MC Jr: Tocolysis in the management of third-trimester bleeding. *J Perinatol* 1990 Jun: 10(2):125-8.
88. Arias F: Cervical cerclage for the temporary treatment of patients with placenta previa. *Obstet Gynecol* 1988 Apr: 71(4):545-8.
89. Cobo E, Conde-Agudelo A, Delgado J, Canaval H, Congote A: Cervical cerclage: An alternative for the management of placenta previa? *Am J Obstet Gynecol* 1998 Jul: 179(1):122-5.
90. Crowley PA: Antenatal corticosteroid therapy: A meta-analysis of the randomized trials, 1972 to 1994. *Am J Obstet Gynecol* 1995 Jul: 173(1):322-35.
91. Dubois J, Garel L, Grignon A, Lemay M, Leduc L: Placenta percreta: Balloon occlusion and embolization of the internal iliac arteries to reduce intraoperative blood losses. *Am J Obstet Gynecol* 1997 Mar: 176(3):723-6.
92. Henrich W, Fuchs I, Ehrenstein T, Kjos S, Schmider A, Dudenhausen JW: Antenatal diagnosis of placenta percreta with planned in situ retention and methotrexate therapy in a woman infected with HIV. *Ultrasound Obstet Gynecol* 2002 Jul: 20(1): 90-3.
93. Schnorr JA, Singer JS, Udoff EJ, Taylor PT: Late uterine wedge resection of placenta increta. *Obstet Gynecol* 1999 Nov: 94(5 Pt 2):823-5.
94. Palacios Jaraquemada JM, Pesaresi M, Nassif JC, Hermosid S: Anterior placenta percreta: Surgical approach, hemostasis and uterine repair. *Acta Obstet Gynecol Scand* 2004 Aug: 83(8):738-44.

95. Combs CA, Nyberg DA, Mack LA, Smith JR, Benedetti TJ: Expectant management after sonographic diagnosis of placental abruption. *Am J Perinatal*. 1992 May; 9(3):170-4.
96. Benedetti TJ: Obstetric hemorrhage. In: Gabbe SG, Niebyl JR, Simpson JI (eds): *Obstetrics: Normal and Problem Pregnancies*. New York: Churchill Livingstone, 1991; pp. 573-606.
97. Hurd WW, Miodovnik M, Hertzberg V, Lavin JP: Selective management of abruptio placentae: A prospective study. *Obstet Gynecol* 1983 Apr; 61(4): 467-73.

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## Kevin Giordano

*Hombre apercebido medio combatido.  
(A man well prepared has already half fought  
the battle.)*

Miguel de Cervantes (1547–1616)  
*Don Quixote de la Mancha* (1605–1615)

Scientific advancements have had a profound impact on preconception risk assessment, prenatal diagnostic capabilities, and early intervention. Increased use of sophisticated ultrasound scans and laboratory technology, including the application of recent developments in gene mapping and prenatal blood, tissue, amniotic fluid testing, has resulted in dramatic advancements in antepartum testing. Obstetricians can now identify earlier and with greater accuracy fetal chromosomal and structural anomalies as well as better monitor fetal condition. Continued development of this technology, coupled with improvement in early intervention techniques, will progressively improve the ability to diagnose many conditions early in pregnancy and lead progressively to in utero treatment in selected circumstances. These developments and the important legal points that derive from them in reference to antepartum obstetric management are the subject matter for this chapter.

In recent years, the scientific, ethical, and philosophical implications of these new developments in obstetrics, particularly in the areas of genetic counseling, invasive antepartum testing, new screening protocols, and the ability for fetal treatment, have generated considerable legal commentary and created new legal and ethical challenges. How have these developments affected medical malpractice law and created obligations to the obstetrician? Interestingly, this depends on the level of advancement and the state of the law at any particular point in time. For instance, technologic advancement has given obstetricians greater insight into the uterus and the fetus's development. These advances can also provide pregnant patients with considerable knowledge about the health of their babies. Without the ability to intervene and alter the outcome, however, the right to legal redress is limited, if not nonexistent. The benefit to a patient or practitioner that knowledge without the ability to intervene provides is debatable and depends on the clinical circumstances. There is an independent and largely persuasive argument for the benefits of counseling and family preparation, however. Under any circumstance, legal recovery is limited to the value that

earlier diagnosis and knowledge would have provided. In the absence of technologic capability to treat a specific medical condition, or if pregnancy termination is not an option, damages are generally difficult to identify.

The landscape of obstetric malpractice claims has changed dramatically in circumstances where advances in technologic capabilities have improved the ability for earlier prenatal diagnosis *and* either there is potential for a significantly improved outcome with treatment, or, the diagnosis is made early in pregnancy, when pregnancy termination is still an option. The most significant impact of new technologies in prenatal testing has been in providing parents with greater information about fetal condition or the existence of fetal abnormalities. An important additional factor is the increasing potential for predicting the risk of certain serious fetal abnormalities based on specialized laboratory and other testing, especially those involving abnormal chromosome number and certain serious hereditary diseases. Legal events indicate that the failure to timely utilize technology appropriately to establish the diagnosis of a fetal problem earlier when treatment or termination may have avoided the outcome, is a sufficient and legal recognizable injury for the parents. As the ability to treat in utero improves, there will be an increase in malpractice cases involving failure to diagnose a fetal anomaly when there was a significant chance of intervention or an improved outcome. The U.S. Supreme Court's determination in *Roe v. Wade* [1], that a pregnant woman has a constitutional right to choose to terminate her pregnancy before viability, has created liability issues for failure to detect genetic afflictions or other abnormalities before the fetus is viable. The information that is derived from advancing technology permits parents to make more educated decisions in exercising their right to continue the pregnancy or not. In light of the United States Supreme Court's decision in *Roe v. Wade* and in subsequent rulings, virtually all jurisdictions in medical negligence cases have determined that a physician whose negligence deprived a mother of her right to decide whether her fetus should be aborted, "should be required to make amends for the damage he has proximately caused" [2]. In reaffirming this right in the matter of *Planned Parenthood of Southeastern Pennsylvania v. Casey*, the Supreme Court declared, "The woman's right to terminate

pregnancy before viability is the most central principle of *Roe v. Wade*. It is a *rule of law and a component of liberty* we cannot renounce" [3]; [emphasis added]. (For an expanded review of these issues, see the Appendix of Legal Principles.) Consequently, concomitant with the ability to obtain such information is an expanded legal duty to educate parents of the technologic capabilities and, where indicated, perform the appropriate testing to inform parents about their particular risk(s) in both this and subsequent pregnancies.

### **Genetic Testing**

Soon after the introduction of this new genetic technology, novel theories of physician liability emerged in medical malpractice litigation. The ability to detect genetic anomalies prenatally has given rise to new potential causes of action against physicians. Increasing numbers of plaintiffs now claim that physicians have failed to make appropriate use of established genetic technology and thereby deprived them as parents of essential information necessary for reproductive decision making. This developing theory of liability has provided two unique conceptual bases for litigation: *wrongful birth* and *wrongful life*.

Different from the birth of a normal child, courts recognize that the birth of a severely deformed baby is necessarily an unpleasant and aversive event, causing inordinate financial burden that would not attend the birth of a normal child [4]. An afflicted child requires the expenditure of extraordinary medical, therapeutic, and custodial care expenses by the family, not to mention the additional reserves of physical, mental, and emotional strength that are required of all concerned. If the diagnosis of abnormality is made sufficiently early in pregnancy, those who do not wish to undertake the many burdens associated with the birth and continued care of such a child have the legal right, under *Roe v. Wade* and subsequent decisions, to terminate their pregnancies. Thus, it is the U.S. Supreme Court decision in *Roe v. Wade* that has created the legal underpinnings for wrongful life and wrongful birth cases, by establishing that decisions concerning conception, including the right to terminate a pregnancy, are the private and exclusive rights of the parents. Prior to this decision, an obstetrician practicing in a state where legislation had banned abortion could not be held liable for failing to diagnose any

postconception anomaly or condition in which the outcome could not have been altered with treatment. In other words, in the absence of the ability to treat the anomaly, an obstetrician's negligence for failure to diagnose the fetus's condition in utero could not be causally connected to harm, because the only therapy to avoid the harm, therapeutic abortion, was at that time illegal. This would have been true even if obstetricians had then had access to the advanced diagnostic technology of today.

Given the information that preconception and antepartum testing can provide a prospective parent with information about severely disabling or potentially fatal conditions, which could be either hereditary or acquired, genetic counseling is crucial. Proper management plays an important role in minimizing potential medical malpractice claims. The treating prenatal physician must therefore be aware of general developments within this complex field. In medical malpractice litigation, the locality rule has been essentially abandoned, although consideration is still given to the availability of medical resources. Thus, the present standard to which a physician is held is a national one that takes into account advancements within the specialty as a whole.

Because advanced medical technology has provided the means for detection of many severely disabling genetic conditions, inaccurate or incomplete genetic counseling can have serious legal repercussions. Although these types of cases are commonly labeled *wrongful life*, *wrongful pregnancy*, or *wrongful birth*, they remain at their core medical negligence actions and are determined by application of common-law tort principles. To illustrate the point, consider *Schirmer v. Mt. Auburn Obstetrics and Gynecologic Association* [6]. After previous unsuccessful pregnancies, genetic testing revealed that mother carried a balanced translocation of chromosomes 11 and 22. Although it caused her no harm, there was a one-third chance that she would pass an unbalanced form of the translocation to her child and cause serious birth defects. During the pregnancy, the plaintiff instructed the defendants to perform all necessary testing to determine if her fetus carried this genetic defect. The results of the testing would have permitted the parents to decide whether to terminate the pregnancy rather than to bring a severely mentally and physically disabled child into the world.

The defendants recommended and conducted a chorionic villus sampling (CVS) test, which was the recognized and accepted test to determine the genetic makeup of a fetus by sampling fetal cells. The test result indicated that the fetus was probably a female, with the same balanced chromosome translocation as the mother. At term, the plaintiff gave birth to a son who had inherited a partial trisomy of chromosome 22 from his mother and was profoundly mentally and physically disabled. The child requires round-the-clock care. The evidence showed that the chromosome analysis sample was derived from maternal as opposed to fetal cells. In bringing the lawsuit, the plaintiffs' claimed that the defendants failed to take the further steps necessary to validate the CVS data results, resulting in the wrongful birth of their child. The trial court initially dismissed the case on the basis that it was determined that the plaintiff's claim for damages was not permitted under state law. The trial court reasoned that the determination of damages would necessitate a fact-finder weighing the value of existence with a disability against nonexistence, there could be no claim for consequential economic or noneconomic damages. In reversing this lower court's decision, the appellate court opined that the defendants had been consulted to obtain information and medical guidance with a direct bearing on whether the prospective mother would carry her pregnancy to term or exercise her constitutionally protected right not to procreate and to terminate the pregnancy. Consequently, the economic harm that the parents sustained as a consequence of having to raise their disabled child over and above the ordinary child-rearing expenses was compensable.

The court recognized that the defendants neither caused the defective condition, nor could they have prevented the child from being born without disability. In coming to its decision, the *Schirmer* court nonetheless undertook an analysis not dissimilar from that of a traditional medical negligence case. The duty owed by the physicians in such circumstances, that being, to inform a patient of the diagnosis and the known or inherent risks so that the patient can make an intelligent decision regarding the course of treatment, arises as a matter of law from the physician-patient relationship. The facts of this case were that because of the negligent medical advice and testing of the defendants, the

plaintiffs were prevented from making an informed decision about whether to proceed with the pregnancy and a delivery that would result in a severely disabled child. Thus, as the court noted, the very reason she undertook extensive genetic testing under the guidance of the defendants was to determine if the fetus she carried was affected by the trisomy 22 defect. If the plaintiffs thus established a departure from the standard of care, that departure deprived them of precisely the information needed to make an informed decision whether to terminate the pregnancy or carry the fetus to term. In the court's analysis, therefore, the failure of one or more of the defendants to conduct follow-up testing to verify the accuracy of the CVS testing gave rise to a set of facts that would have permitted the plaintiffs' recovery, if the plaintiffs were able to prove at trial that their physicians and the laboratory technicians had breached their duty. The court upheld the rationale of precluding recovery when the measure of damages requires a valuation of being versus non-being. Consequently, the parents *could not* recover damages for the emotional and physical tolls resulting from raising and supporting a disabled child as it would require a valuation of the child's being versus nonbeing.

Many wrongful birth cases arise because of the failure of the practitioner to stay current with development in identifying genetic risk. The case of *Howard v. Lecher* [7] is an example of a situation in which early in the development of genetic science, the obstetrician was not fully cognizant of the risks associated with the family history. In *Howard*, the claimants were both Eastern European Jews, and their infant daughter was born with Tay-Sachs disease. The parents claimed that had a proper genealogical history been performed, the physician would have recognized the potential risk to the fetus and thus would have ordered the appropriate tests for Tay-Sachs for the parents. It was further alleged that such testing would have revealed that the fetus was afflicted with this neurologic impairment and would have been aborted. Unlike *Schirmer*, in which the plaintiffs claimed further antepartum testing should have been performed to diagnose a potentially serious genetic abnormality in an unborn child, the plaintiffs in *Howard* claimed that the physician failed to appreciate the risk inherent in the relationship between their heritage and Tay-Sachs disease during preconceptional counseling.

As in any malpractice case, expert testimony on both the applicable standard of care and causation is highly determinant of litigation outcome [8]. In establishing proximate cause for wrongful birth, plaintiffs must show that the resulting birth defect was reasonably foreseeable, that is, not too remote in relation to the defendants' negligence, and that had the defendants not been negligent, the pregnancy would have been terminated.

The primary legal considerations for the obstetrician relating to genetic testing and counseling are as follows:

- With enhanced screening techniques, genetic technology has provided physicians with widely available diagnostic testing that can detect many common genetic disorders [9,10,11]. These conditions include Tay-Sachs disease, sickle cell anemia, cystic fibrosis, Down syndrome, and other major trisomies (e.g., trisomies 13, 15, and 18) and sex chromosome deletion syndromes (e.g., Turner syndrome, Klinefelter syndrome). Some prenatal tests are invasive (e.g., amniocentesis, chorionic villus biopsy) and involve substantial risks. For example, the risks associated with amniocentesis include fetal-maternal hemorrhage, membrane rupture, various types of direct fetal injury, and spontaneous abortion. Ordinarily, to be within the standard of care, such invasive testing is performed only when analysis indicates that the benefits outweigh the risks. This analysis must also take into account the fact that the specific treatment of most genetic diseases, either medically or surgically, is either not possible or is experimental.
- The practicing obstetrician must be aware of the advances in genetic testing and implement procedures that ensure appropriate genetic counseling and testing. Performing testing procedures that are experimental or considered as yet diagnostically unreliable is ordinarily not considered to be within the scope of accepted or standard medical care. Ultimately, the decision to perform any particular test is based on a consideration of the sum of benefits weighed against the potential risks.

The American College of Obstetricians and Gynecologists (ACOG) recommends advising all pregnant women about the availability of maternal serum alpha-fetoprotein testing [10]. In many locales, routine testing for all pregnancies in the first trimester is progressively becoming

a practice norm [11]. Currently, antepartum tests for human chorionic gonadotropin, alpha-fetoprotein, inhibin, and estriol (commonly known as the *triple* or *quad test*, depending on the number of tests in the battery), are now routinely recommended by clinicians in the mid-trimester. The newest testing protocols combine ultrasonic findings in early pregnancy (i.e., nuchal lucency at approximately 11–13 weeks of gestation) with biochemical screens, sometimes retesting with additional studies after the 15th week. These protocols result in the identification of up to 90% of pregnancies with a Down syndrome fetus. The physician should be aware of and understand the significance of these test results and to consider further diagnostic testing when a test result is interpreted as revealing an increased risk, especially if these data correlate with other genetically relevant risk factor (s) identified by clinical evaluation, medical history, or ultrasonic scan.

- At present, routine testing of pregnant women for aneuploidy risk is becoming standard medical practice. The decision to recommend more specialized or focused testing depends on the clinical features of each case evaluated in terms of the potential risks and benefits to the patient and her unborn child.
- An important implication is that patients with complex or unusual histories (e.g., familial mental retardation, recurrent early childhood deaths, or family members with structural abnormalities suspected to be hereditary) are often best referred to genetics specialists for in-depth counseling and case review.
- The significant risks associated with invasive testing have encouraged the increasing popularity of other forms of genetic testing such as diagnostic ultrasonography, which is safe, and, for some conditions, sufficiently accurate to establish a diagnosis reliably [16,18,24]. As these and other laboratory tests become more reliable, the use of traditional invasive procedures will decline, and wider use of various genetic tests performed on maternal serum for screening purposes will occur.
- Documentation is critical; the physician must document in the medical record the relevant aspects of patient counseling, and in particular the infor-

mation communicated regarding potential risks of genetic disease, the patient's options, and whether a referral to a specialist was offered and either accepted or refused. The physician must keep abreast of the literature, particularly as more information becomes known regarding the correlation between genetic diseases and certain ethnic, racial, and religious groups, as well as other clinical and genetic factors such as maternal age or other siblings born with known or suspected genetic diseases. Obviously, informed consent should be obtained and documented for all invasive tests that are performed.

- Because the primary means for treatment for serious genetic disorders or structural defects remains therapeutic abortion, some practitioners are uncomfortable in discussing testing and its implications with patients. The standard of care does not necessarily require physicians to conduct testing, but it does require that pregnant women be informed of the possibility of testing and how such testing might be obtained.

### *Ectopic Pregnancy*

Ectopic pregnancy is a serious risk to the general and reproductive health of a woman. The incidence of ectopic pregnancy appears to have steadily and persistently risen since 1970. Between 1970 and 1992, the rate of ectopic pregnancy increased from 4.5 to 19.7 per 1,000 reported pregnancies (including live births, legal abortions, and ectopic pregnancies) [15]. This trend, likely due to the emergence of several key elements, including enhanced diagnostic capability to detect ectopic pregnancies early in gestation, the rising incidence of gonorrhea and chlamydial infections in reproductive-aged women, and the growing use of treatments to circumvent infertility, including in vitro fertilization, declined significantly over time. For additional discussion of ectopic pregnancy see Chapter 4.

A ruptured ectopic pregnancy is a true medical emergency and a leading cause of maternal mortality during the first trimester. Overall, ectopic gestations account for 10% to 15% of all maternal deaths [12,13,15]. Short of mortality, morbidity is also a very significant concern, as evidenced by the case of *Roberts v. Mecosta County Gen. Hosp* [14].

In *Roberts*, the plaintiff, while pregnant with her first child, presented herself to Mecosta County General Hospital complaining of severe pain. At that time, a diagnosis of a spontaneous abortion was made and a dilatation and curettage (D and C) was performed. She was sent home at that time. Over the course of the next few days the claimant continued to experience pain and cramping and, 3 days later, was again seen at Mecosta County General Hospital. The plaintiff was told that the pain she was experiencing was due to uterine cramping from the D and C. Again, she was sent home. She returned to the hospital the next day, wherein it was discovered that the plaintiff had not had a spontaneous abortion but had an ectopic pregnancy in her left tube, which had ruptured. Emergency surgery was performed and due to damage to her left fallopian tube, it was removed. She had previously had surgery to remove her right tube and thus, as a result of the alleged negligence, she claimed that she was unable to have children.

Because of the potential for catastrophic injury as evidenced in the *Roberts* case, there are myriad important medicolegal issues connected with the clinical problem of ectopic pregnancy. These include the indications for performance of appropriate diagnostic procedures, issues of informed consent, and the obligation to provide warnings regarding the necessity for follow-up care in uncertain cases. Modern advances in ultrasound technology and the use of serum beta-subunit human chorionic gonadotropin ( $\beta$ -hCG) and, to a lesser degree, progesterone levels have made ectopic pregnancies easier to identify than in prior decades [16,18,19,21]. The incidence of rupture and the fatality rates have also declined significantly over the past three decades. Nonetheless, establishing the correct diagnosis remains a challenge. Often, the clinical history as received or recorded does not lead to appropriate testing, or the tests that are performed are interpreted as equivocal. It has been reported that as many as 40% to 50% of ectopic pregnancies are misdiagnosed at the initial visit to an emergency department [15]. Treatment depends not just on the actual medical condition of the woman but also on her particular circumstances. For example, a woman with an ectopic pregnancy who is unconcerned about sterility might be treated in one fashion, such as tubal excision, whereas another patient who desperately wishes to

preserve her fertility might be managed quite differently, with efforts at tubal conservation.

- Given the relative frequency of misdiagnosis and relative high mortality rate, failure to diagnose ectopic pregnancy is frequently a cause of malpractice litigation. Often the physician's failure to identify risk factors is cited as the reason for misdiagnosis [16,18,20]. Ultimately, the treating physician must make certain that the clinical history includes both the positive and negative historic data that might affect the risk of ectopic pregnancy. Among the most important risk factors for ectopic pregnancy include a history of a prior ectopic pregnancy or surgical sterilization, prior tubal surgery, history of infertility, advancing age, smoking, a history of pelvic inflammatory disease and, now increasingly rarely, prior DES exposure. The presence or absence of these and other risk factors must be elicited from the patient to determine the likelihood of alternative diagnoses and prevent a delay in diagnosis.

Most often, malpractice cases involving ectopic pregnancy stem from the physician's failure to recognize the risk and exclude the diagnosis. Typically, the diagnosis is accurately made when the classic triad of symptoms, pelvic pain, amenorrhea, and abnormal bleeding are present. The difficulty is that up to 50% of patients will not present with this classic constellation of symptoms. This makes clinical suspicion of paramount importance in the detection of an ectopic pregnancy. Although some women present acutely with a ruptured ectopic pregnancy and a hemoperitoneum, up to 80% of diagnoses are made among outpatients, where the signs and symptoms of the ectopic are much less dramatic. Because the rupture of an ectopic pregnancy is such a medical emergency, any delay in processing the diagnostic algorithm for ectopic pregnancy might be catastrophic for both the patient and for the defense of a subsequent medical negligence case. Thus, before excluding the diagnosis of ectopic pregnancy, the patient's clinical presentation should dovetail with the history provided. If in order to complete the assessment, prior records or laboratory data exist that could shed light on a patient's history are available, the physician may well be under a duty to consult such records.

Cases have also been brought in which the physician has made the diagnosis erroneously. The case of *Coffman v. Roberson* [17] is an example.

In *Coffman*, a woman went to her obstetrician/gynecologist, after a home urine pregnancy test showed she was pregnant. The obstetrician performed a serum hCG test, which revealed that the human chorionic gonadotropin hormone level in her blood was elevated, suggestive of pregnancy, although his physical exam “did not show a pregnancy in the uterus.” The physician referred her for an ultrasound examination. Following the ultrasound scan, the radiologist suspected that she had an ectopic pregnancy. The obstetrician was called in his car on the way to vacation and was read the ultrasound report over the phone. He never reviewed the ultrasound personally. The report stated that the radiologist “strongly suspect[ed]” an ectopic pregnancy. While still driving to vacation, the obstetrician called the patient to discuss the ultrasound. Because of the danger from an ectopic pregnancy, to the covering obstetrician, who, based on the information he was provided, prescribed the administration of a shot of methotrate to terminate the pregnancy. Later the next month, an ultrasound revealed an intrauterine pregnancy without a heartbeat. A dilation and evacuation procedure was performed.

Communication is an additional factor that all too often plays a role in an unfavorable outcome. The potential for communication errors exists at several levels: physician to patient, physician to nurse, and physician to physician. Because the signs and symptoms of ectopic pregnancy can be subtle and serial study with close attention to laboratory data can be critical to early diagnosis, a breakdown in any of these interactions can have disastrous consequences. No matter how well trained, a nurse or nurse practitioner is not in as good a position as a doctor to appreciate the subtle symptoms or evaluate the potential signs of ectopic pregnancy. In addition, too much reliance on untrained office personnel responding to telephone calls might result in a patient with important symptoms experiencing an inappropriate delay by being scheduled for a routine examination rather than being seen immediately.

The primary legal considerations for the obstetrician relating to ectopic pregnancy and counseling for possible ectopic pregnancy are as follows:

- Ectopic pregnancy is still a leading cause of maternal mortality; therefore, a high index of suspicion is required when a physician is faced with a woman of childbearing age who presents with otherwise unexplained low abdominal pain, a history of menstrual irregularities, or suspected pregnancy with or without vaginal bleeding. However, it must be remembered that up to 50% of patients will not present with the classic constellation of symptoms. This makes clinical suspicion of paramount importance in the early detection of an ectopic pregnancy, because up to 80% of diagnoses are made in the outpatient setting, usually based on blood testing and ultrasound scanning.
- It is crucial that the obstetrician obtain a complete and accurate medical history. This is just as critical as properly executing diagnostic tests. A physician considering a diagnosis of ectopic pregnancy must be careful to elicit both positive and negative historic data that might support an increased risk of ectopic pregnancy. Detailed questioning and medical record documentation concerning the clinically significant risk factors are essential not only for potential patient care but also to provide the foundation for good malpractice claim defense. Details concerning all prior pregnancies, prior infertility treatments, and methods of contraception should be elicited and recorded in the medical record. The medical history review should also reflect questioning concerning prior episodes of pelvic inflammatory disease, and fallopian tube surgery, as well as other potential risk factors.
- The physician must explain in detail to the patient any treatment recommendations and appropriate precautions. The woman must be advised of not only the options available but also the potential risks, benefits, and probability of success of any proposed surgical procedure or course of medical therapy. Care must be taken to convey accurately to the patient the pros and cons of all available alternative treatments and to ensure that choices are made with a full understanding of the risks and benefits. For example, even when fertility can be preserved in the treatment of a properly diagnosed ectopic gestation, the patient must clearly understand that she could be at risk for recurrent ectopic pregnancies owing to tubal damage or dysfunction.

- Unfortunately, there are no symptoms or signs of sufficient reliability to permit clearly distinguishing between a normal pregnancy with symptoms, an abnormal intrauterine pregnancy, and an ectopic. Moreover, there are multiple gynecologic and nongynecologic diagnoses that can be confused with an ectopic pregnancy. Thus, diagnostic tests have gained increasing importance as complements to history and physical examination in the timely diagnosis of early abnormal pregnancies. The most important of these diagnostic tests are serial serum beta-human chorionic gonadotropin (B-hCG) and high-resolution transvaginal ultrasound scanning. Even these diagnostic tests have limitations, however, which may compel the use of invasive procedures such as laparoscopy to finally establish a correct diagnosis.
- Good communication is essential. Whenever ectopic pregnancy is suspected or cannot be reliably excluded, and it is decided to follow a woman as an outpatient, she should be instructed to be attentive to and report specific symptoms to the physician. These standard precautions may be recorded on a form that is reviewed with the patient and that she takes home for subsequent study. Furthermore, women defined as at risk should be followed until either the diagnosis is confirmed or confidently excluded. A surgeon, emergency physician, or other specialist treating a patient for ectopic pregnancy who might not be the follow-up physician must make sure that the patient understands the need for continued observation and the signs and symptoms suggestive of acute trouble. This communication should be documented in the medical record, and preferably these women should be provided with written discharge instructions or other written information concerning what to be aware of and when, and contact personnel for problems or questions. There are, of course, reasonable limitations to any system of notification or communication. In the case of failure to appear for follow-up evaluation, some physicians send one or multiple certified letters to former patients advising of the signs and symptoms to be wary of and the need for prompt follow-up evaluation or treatment. Such practice should be sufficient to avoid any claim of inadequate communication. The primary goal of any practitioner

handling a possible ectopic pregnancy is to avoid a bad medical outcome. In so doing, a bad legal outcome is also avoided.

- Patients who have undergone surgery, especially those receiving medical treatment for ectopic pregnancy, need to know that seemingly successful treatment must be followed closely by serial hCG testing to exclude a persistent ectopic pregnancy [22].
- Nonphysician medical and office personnel should continually be trained simply to receive and record facts from patient telephone calls and immediately transmit them to the physician for appropriate follow-up recommendations. Any greater use of discretion by nonphysicians can be severely criticized in subsequent legal proceedings. Ultimately, the physician could be held responsible for any unwarranted delay that has a bad result.

### *Ultrasonography*

Advances in ultrasound technology have both improved the capability to detect fetal anomalies during antepartum testing as well as expanded its potential to include applications in offices, the labor and delivery suite, and the operating suite and triage areas [23,24]. Concomitant with the increased capacity of ultrasound imaging comes increased litigation for the failure to utilize this technology appropriately in the prenatal and antepartum evaluation and management of patients.

Presently, lawsuits related to obstetric ultrasound are more common than claims arising from gynecologic or other abdominal studies [23]. Although previously obstetric malpractice cases in which ultrasonography was a principal component of the allegations frequently related to the failure to diagnose ectopic pregnancy, today the most common allegation is the failure to detect fetal anomalies. A significant number of these cases involve allegations that a study was misinterpreted. An example of this type of case would be an obstetric ultrasound first performed in the third trimester and interpreted as indicating a fetus of 38 to 39 weeks' gestation, without noting caveats of potential inaccuracy in terms of both gestational age and the ability to diagnose certain types of fetal abnormalities. The problem arises if the same or another clinician then acts upon these data and either induces labor or performs a cesarean.



If at delivery the infant is determined to have respiratory distress syndrome (RDS) or intracranial hemorrhage and the gestational age by examination proves to be 34 to 35 weeks, there is a potential for a legal claim.

Many of the claims involving ultrasound concern system-related problems such as improper supervision of the ultrasound technologist, poor physician-patient communication, and failure to consult with a radiologist or maternal-fetal medicine physician concerning scan interpretation. The following cases illuminate these problems [23].

A woman developed gestational diabetes during her pregnancy and insulin was administered. She presented to the hospital in her 39th week because she could no longer feel any fetal movement. At the hospital, clinical assessment included ultrasound, electronic fetal monitoring, and a biophysical profile. Those tests revealed that there was fetal movement but the fetal heart rate was abnormal and the amniotic fluid volume was noted to be low. Following cesarean delivery, the child needed immediate resuscitation and subsequently died from a series of complications four days later. Suit was brought against the obstetrician alleging that he was negligent in failing to use appropriate methods to monitor the fetus' condition, resulting in the death of the baby. Apparently, throughout the pregnancy, the obstetrician did not use equipment that was in his office to perform ultrasound examinations, fetal monitoring, or biophysical profiles to monitor the health and development of the fetus. Instead, it was determined that he relied upon clinical examinations and fundal height measurements to determine fetal health and growth. Of interest, this case involved neither a question of misinterpretation of data, nor that of physician judgment. The issue was whether the failure of the physician to implement standard antepartum testing for diabetes was in and of itself, medical negligence [25].

Another case presented a different systemic issue. In this proceeding, the plaintiff reported that her first pregnancy resulted in a child born with hydrocephalus and severe mental and motor retardation. The child required extensive medical care until her death at four months of age. When the parents became pregnant, they were fearful of bearing another child with congenital defects. Testing showed the pregnancy was normal. The antenatal

course and the birth proceeded without complication and in fact this second child proved normal. Ultimately, the plaintiff's conceived a third child. In this pregnancy an amniocentesis performed at 19½ weeks gestation and was interpreted as normal. However, an ultrasound scan performed the same day revealed a larger-than-normal measurement for the ventricles of the brain as well as an unusual head shape. The maternal-fetal medicine physician who performed the testing requested her staff to schedule the patient for follow-up testing. Due to an office error, however, the patient was not scheduled nor was the ultrasound report forwarded to the patient's treating physician. At 33 weeks' gestation, the treating physician performed his own ultrasound scan and discovered that the unborn child had advanced ventriculomegaly (hydrocephalus). At that point it was too late to terminate the pregnancy. The mother subsequently gave birth to a child with multiple birth defects who died as a result of these abnormalities four months later. Thus, the failure to arrange for appropriate follow-up and the failure to inform the primary care obstetrician allowed the patient to "fall through the cracks" and serious management errors to occur [26].

Issues of interpretation are potentially a problem as the following case indicates. During the twentieth week of a woman's pregnancy, the patient's obstetrician ordered a complete pregnancy ultrasound examination. A hospital-employed technician performed the scan. Permanent recorded images were made of various anatomical structures. These images were reviewed, interpreted, and reported by a radiologist who had contracted with the hospital to provide imaging services. The technician who performed the examination recorded nothing abnormal about the cerebral ventricles. The plaintiffs were aware a hospital technician had actually performed the sonogram but the ultrasound report indicated that it was interpreted by the radiologist. The radiologist did not perform his own independent assessment of the ventricles. Subsequently he testified at deposition that it was the policy of both the hospital and the radiological medical group that the hospital's technician held the responsibility to measure the cerebral ventricles and interpret those measurements. The plaintiffs alleged the cerebral ventricles were abnormal at the time of the ultrasound examination and that had competent care been provided,

this abnormality would have been properly reported and subsequently acted upon. The court determined that regardless of whether the radiologist performed the examination or made the measurements him or herself, the radiologist could still be held liable for the quality of the examination and the proper interpretation of the measurements [27].

It is important to understand that although obstetricians and obstetric practices routinely use ultrasound technology in patient management, referral to a maternal-fetal specialist, a fetal evaluation unit, or an experienced radiologist is indicated when clinical circumstances or atypical studies warrant. This is particularly true given the level of sophistication possible with focused ultrasound studies as performed by experts. Although scans do depend on the quality of the equipment used in the procedure, their reliability and accuracy are much more dependent on the training and experience of the interpreting physician. Many general obstetric practices seek to perform even non-routine ultrasound studies in house, partly for convenience but also because of the reimbursement potential for these studies. Health insurance plans and hospital systems might encourage, if not require, that referrals be made to physicians who participate in the plan or are within the health system. Such internal referrals can create a conflict if the “in-plan” studies lack the competent personnel or technical sophistication possible in an “out-of-plan” referral. Juries are loathe to accept failure to refer a patient in need of a higher-level physician, or at least offer such a referral to the patient, when the motivation for the failure to do so appears to be either a reimbursement potential or a cost savings.

Conducting advanced scanning procedures is potentially hazardous from a legal point of view when the personnel involved have limited expertise or limited exposure to high-risk conditions. In the ordinary course of interpreting and developing a management plan based on routine ultrasound testing, an obstetrician is held to the standard to provide care consistent with that provided by the reasonably prudent obstetrician, and not that of an appropriately trained and experienced specialist in maternal-fetal medicine or radiology. Should the obstetrician neglect to obtain an appropriate consultation with a specialist when the standard of care requires it, however, the obstetrician will be held

accountable for any harm that is caused by that failure. When a physician undertakes to perform a procedure or evaluate a patient's condition that is ordinarily reserved for a particular specialty or requires the training commensurate with that specialty, the physician can then be held to a higher standard. Thus, in the event that the obstetrician or obstetric practice chooses to perform more complex ultrasound studies beyond the routine when the standard of care normally requires referral and bases their management plan on their own interpretation, they probably will be held to the standard of care of the consultant (usually a maternal-fetal medicine specialist or radiologist with advanced training).

The primary legal considerations for the obstetrician relating to ultrasonic scanning and management predicated on the results of scans are as follows:

- Both the capabilities and limitations of ultrasound and its role in antenatal diagnosis must be understood by clinicians, as well as its capability to provide important information during labor and delivery and postpartum care. For example, sonographic assessment of cervical length is important in the evaluation of women for whom the differential diagnosis includes the risk of either cervical insufficiency versus preterm labor. How to use these data and when to recommend or perform cerclage as opposed to other possible treatments remains controversial, however. Furthermore, the support in the literature for cerclage is problematic, and all of the cerclage procedures carry potentially important risks. Which patients to follow clinically without intervention and which to offer surgery has not been clearly established. Thus, there are substantial risks of both over- and undertreatment. (See Chapter 5, Cervical Insufficiency.)
- Remaining current with the literature is necessary. This is particularly true where the technology is such that once accepted, it can be used as a tool allowing more informed decision making, especially about whether to continue with the pregnancy. In today's climate, failure to provide informed information or to perform indicated antepartum or preconceptional tests (and thus arguably deprive patients of the ability to make informed decisions) is a significant cause

of malpractice claims. Jurors can easily relate to a woman who is deprived of potentially crucial information by her obstetrician at such a critical time.

- Similarly, the physician must understand the capabilities and limitations of various types of ante- and intrapartum fetal surveillance tests. Biophysical profile testing (BPP) for example, is generally recognized as the gold standard for assessing the risk of acute fetal jeopardy in antenatal cases. Newer tests involving study of blood flow within the umbilical or central cerebral vessels or other specific parts of the body also are used to aid decision making in cases involving known or suspected abnormalities in fetal growth. Ultrasound or electronic monitoring to assess fetal well-being during labor remains controversial, however. An obstetrician does not want to be behind the learning curve on best practice in fetal evaluation. Juries can understand that not all adverse events in pregnancy are the result of negligence, but failing to use reliable available tests or to conduct procedures recognized as effective methods of surveillance, and arguably part of the standard of care, creates a significant problem in the defense of a case if a tragic outcome has occurred. This is true even though it takes a certain time for new information to filter through the specialty and become recognized as standard.
- Communication between the physician interpreting ultrasound studies and the obstetrician is critical to providing good patient management. If the obstetrician does not understand the significance of certain findings, or the interpreter's report is unclear, the expert and the ordering physician should directly discuss the findings and their potential significance. Although certain findings might not affect immediate management, they could affect the need for either subsequent or serial assessments or specific postpartum evaluations.
- The obstetrician must discuss test results with the patient. In so doing, the clinician must fully understand both the anticipated level of accuracy as well as the limitations of the study relative to any findings or absence of findings, so that the patient can be apprised of any potential risk. It is for the patient to determine the significance of

any risk that is judged more than remote. (See the Informed Consent Discussion in the Appendix of Legal Principles for additional information and discussion.)

### ***Therapeutic Abortion***

The most controversial area of obstetric care is abortion. Litigation has primarily surrounded the legality of abortion and the state's ability to impose legislation that might impede or restrict a person's right to choose termination as established by *Roe v. Wade* [1] (For an expanded discussion of this complex and contentious issue, see the Appendix of Legal Principles). Abortion also plays a central role in wrongful birth and wrongful life cases. As discussed previously, in these cases the suit is predicated upon the potential harm caused by depriving the patient of her right to an abortion. Thus, a pregnancy termination would have avoided the economic harm and emotional turmoil that the parents sustained as a consequence of having to raise a disabled child over and above the ordinary child-rearing expenses.

Although rare, there are cases in which the technique used to perform the abortion is at issue. These types of cases are termed *wrongful pregnancy*. Usually arising out of the negligent performance of a sterilization procedure in a wrongful pregnancy or in the case of failed sterilization, *wrongful conception* claims, that the parents, on their own behalf, seek damages they suffered as a result of giving birth to a normal, healthy, yet unplanned and unwanted child. As with wrongful life and wrongful birth, the recoverable damages are often a central issue on appeal. Most often, the issue is whether damages in such cases are limited to the cost of delivery or, despite giving birth to a normal healthy baby, the parents should be entitled to an award for the financial burden of child-rearing given the expense of raising a child, as well as the possible disruptive effect of the unplanned child on the finances of the family. Those courts that follow a "limited-damages" approach provide for recovery of the medical costs of the pregnancy and delivery, damages for emotional distress due to pregnancy, lost wages due to pregnancy, damages for the husband's loss of consortium during pregnancy, and damages for the mother's pain and suffering during pregnancy and delivery. These courts foreclose recovery of the costs of raising the

child most often because of the public policy belief that the birth of a normal, healthy child cannot be an injury to their parents. Meanwhile, courts that allow for the award of child-rearing expenses usually subscribe to the theory that the parents' injury is not the birth of her child but rather is the invasion of their interest in the financial security of her family and the attendant desire to limit her family size, and the deprivation of her right to limit procreation [19c]. For a more detailed discussion of this topic, please refer to the appendix).

Consider the case of *Sheppard-Mobley v. King*, however [28]. In July 1999, the mother sought treatment for abdominal discomfort from her obstetrician. The obstetrician examined the mother, performed a sonogram, and determined that she was pregnant. The obstetrician advised the mother that because of fibroid tumors in her uterus that could not be surgically removed while she was pregnant, the mother would probably be unable to carry a child to term. The physician recommended that the mother undergo an abortion. The patient was referred to a fertility specialist, who also advised her that the fibroids might abort the pregnancy. He recommended that she not undergo a surgical abortion because the fibroids would likely complicate the procedure. Rather, he opined that the pregnancy should be terminated via injections of methotrexate, a cytotoxic drug commonly administered for ectopic pregnancy and, infrequently, for the induction of abortion. The mother returned to her obstetrician who, after consulting with the specialist, administered the methotrexate. As would later be deduced, allegedly the dose of methotrexate was insufficient, and as a result, the pregnancy was not terminated. When the mother's doctors finally discovered that the mother was still pregnant but was probably now carrying a compromised fetus, the mother grappled with emotionally painful choices, including whether to undergo an out-of-state, late-term abortion. Instead, the mother gave birth to the infant plaintiff, who is afflicted with serious defects.

The court determined that no cause of action could be maintained on behalf of the infant plaintiff for wrongful life, in other words, that that he would never have been born but for the negligence of the defendants. The court did allow both the child and parents to pursue an action based on the more tra-

ditional obstetric negligence concept that permits a cause of action on behalf of a born child for injuries sustained in utero resulting from a tort committed during pregnancy. This claim was premised on allegations that the defendants were negligent in recommending an abortion and that, had the defendants not been negligent in their advice regarding the mother's ability to carry the fetus to term and in advising her to undergo a chemical abortion, the mother would have given birth to a healthy child. Thus, the infant plaintiff claimed that the defendants breached the duty of care they owed to him when they gave the mother this improper advice. In addition, the parents also claimed that in any event, the defendants were negligent in the manner in which they failed adequately to diagnose, treat, observe, manage, and care for the mother during her pregnancy. Consequently, they pursued an action for the extraordinary medical costs and emotional distress caused by raising this severely impaired child, for the negligent performance of the abortion, and for the subsequent management of her condition.

Other actions involving therapeutic abortion stem from issues surrounding the decision to terminate the pregnancy. Subsequent remorse or depression can develop, leading the woman to question her initial decision. Informed consent is a very important concept in these situations. The outcome of an informed consent case in this setting depends on establishing the information that was provided to the patient, including the risk of sadness and possible serious depression. Consider *Martinez v. Long Island Jewish Hillside Medical Center* [29]. In *Martinez*, the plaintiff alleged that she had had an abortion solely because she was falsely informed that the fetus was disabled. She claimed that otherwise would not have considered having an abortion. Her claim was that she made the abortion decision on misinformation, and that she was persuaded that an abortion would be beneficial. A distinction can be drawn between this case and *Perez v. Park Madison Professional Laboratories, Inc.* [30]. In *Perez*, the plaintiff acknowledged that she was accurately informed as to the status of her pregnancy and was fully aware that the abortion would terminate it. Having received accurate information concerning her medical condition at the time of her abortion, as well as all the physical and emotional risks attendant upon the procedure,

the plaintiff was the only person in a position to know whether an abortion under those circumstances was in violation of her personal convictions, according to the court. The court went on to find that the plaintiff “. . . cannot seek to hold the defendant liable because those convictions have apparently changed since she consented to and underwent the procedure.”

Cases involving therapeutic abortion are not common where the issue is the actual performance of the procedure. Given the intense emotional and religious issues surrounding abortion and pregnancy loss, physicians should approach these cases with prudence and err on the side of full documentation especially in terms of patient counseling.

The primary legal considerations for the obstetrician relating to pregnancy termination and counseling are as follows:

- A physician must be aware of the regulatory enactments in the state where he/she practices. Regardless of the individual beliefs that are held by the physician, he/she must recognize both the rights of the patient in pregnancy termination as well as the state's interest in preserving potential life as reflected in statutory law.
- Given the intensity of the medical, moral, ethical, and legal controversies that are involved, therapeutic abortion is a highly volatile issue and one in which both an uneducated patient and an unwitting physician can both become entangled. There is a potential that a woman who undergoes a therapeutic abortion could develop postabortion psychological difficulties related to her experiences. Even without this psychological trauma, the patient might become remorseful over the decision to terminate. In either scenario, she might subsequently question whether her decision was in fact informed. It should not go unrecognized that the person who sought the right to have an abortion as “Jane Roe” in *Roe v. Wade* has since changed her position and is now an advocate for making abortion illegal. Often, malpractice cases stem from a patient's claim that she did not receive appropriate preabortion counseling, thus it is argued, her consent was uninformed. Although often the woman remembers signing a consent form acknowledging that the required information was made known to her,

she will claim that either she did not read it or if she did read it, she could not understand the form.

- Because of a perceived risk of psychological trauma, states are now mandating changes in abortion decision making. In particular, women's right-to-know laws have been enacted that precisely determine the content of information to be given to potential abortion patients and when this information should be made available before an abortion may be performed. Obstetricians must be aware of such state mandates and ensure that the person(s) responsible for counseling, who might not be the obstetrician actually performing the abortion, has been educated on specific requirements and undertaken them conscientiously.
- If informed consent is not obtained prior to an abortion, there are potential grounds for medical malpractice litigation. The following elements have been deemed important in preabortion counseling and often compose the requirements of any state mandated right-to-know law: the risks of the proposed treatment, the probable gestational age of the unborn child, the alternative risks associated with carrying the baby to term, the availability of medical assistance benefits if childbirth is carried out, and the father's financial responsibility. If information regarding abortion alternatives is conspicuously absent in the counseling process, arguably it is not possible for a woman to weigh the benefits and risks of electing an abortion. Preabortion counseling should occur in a private office and not an examination room or another area where privacy might in any way be impeded. Sufficient time should be reserved for the patient so that information is delivered unhurriedly and the patient clearly understands that there is plenty of opportunity to ask questions privately.
- Abortion cases involving minors are high risk and must be treated delicately. The obstetrician must know and understand any state requirements for parental notification. Several states now have such parental consultation statutes, requiring minors seeking abortions to involve their parents in their decision making. These regulations usually have exceptions for mature minors or in the circumstance when advising the parent(s) might be harmful to the patient.

### **Placental Abnormalities**

Malpractice claims are filed every year against obstetricians who permit mothers with an undiagnosed placenta previa to proceed to a vaginal delivery that then results in bleeding complications leading to maternal or fetal injury. In this context, evidence of ultrasonographic confirmation of the diagnosis of placenta previa often becomes a major trial battleground. The plaintiff claims that the scans demonstrate previa, whereas the physician claims the opposite.

It might also be asserted that a full examination to exclude previa (usually by transvaginal scanning) was either not performed or improperly performed when it was indicated owing to clinical signs and symptoms or a prior history. A common difficulty is the timing of the scans during pregnancy with a failure to follow up for suspected or partial previa or a failure to visualize the cervix in suspected cases, when only abdominal scanning was performed in potentially high-risk patients.

Although less common, abruptio placentae is another abnormality that occasionally finds its way into the courtroom. This complication, if unrecognized and not treated promptly, has the potential for horrendous clinical consequences and resultant litigation. The typical case involves a physician's alleged failure to consider or exclude the diagnosis of abruptio in a patient who presents with minimal expressed bleeding but vital signs or other physical findings that suggest occult hemorrhage. After inevitable delays, delivery occurs, accompanied by maternal or fetal injuries, leading to a claim of failure to diagnose and failure to act appropriately in the face of a potentially life-threatening obstetric emergency.

Similarly, a physician's management of other placental abnormalities can result in legal exposure if the patient suffers permanent injury. Placenta accreta, percreta, and increta are each conditions that can lead to both serious patient injury and liability if improperly managed.

Exposure for management of these abnormalities usually arises if the physician either fails to recognize the condition or fails to treat it properly. Although a physician can find assistance in the medical literature that speaks eloquently of the difficulty in management, physician cannot shield him- or herself from malpractice if there was

the opportunity to perform an adequate evaluation.

### **Cervical Insufficiency**

Despite major advances in the scientific body of knowledge about preterm birth and efforts to diagnose and treat this condition, the rate of preterm birth has not declined. The incidence of *preterm birth*, defined as before 37 weeks' gestation, remains between 7% and 11% [31]. Preterm birth is the major cause of perinatal mortality. The etiology of preterm delivery is recognized as multifactorial. Cervical insufficiency is thought to be among the causes of preterm births, especially those occurring in the second and third trimesters [32].

These cases involve early pregnancy losses or preterm deliveries. If the patient experiences a preterm birth prior to the period of viability, the only claim that could be advanced is that the obstetrician could have undertaken steps to prevent delivery. Otherwise, although the physician might be negligent in diagnosing a condition known to be associated with premature delivery, if it were probable that the child would not survive, the negligence cannot be said to have been a cause of the harm. In cases in which prolonging gestation might have created a significant chance for survival, the obstetrician potentially can be held responsible for depriving the patient of a loss of chance, although there remained uncertainty surrounding viability. (See Appendix 1 for a complete discussion of the Loss of Chance Doctrine.)

In any case involving preterm delivery when delaying delivery was an option, one must determine whether a legitimate clinical basis for the decision existed, and whether timely and appropriate tests were performed to confirm fetal well-being during the delay. Furthermore, the outcome will be significantly affected by whether the parents were advised of the potential options and their attendant risks. Principal risks to markedly premature infants are preexisting chromosomal or structural defects, mechanical birth injuries, and hypoxia/asphyxia.

The philosophy used during the prosecution of any malpractice case involving a pregnancy within the period of presumed viability in which asphyxia is the presumed cause of a neurologic injury is that

the fetus should have been removed from a “hostile environment” while there was time to avoid insult or reduce the extent of injury. Thus, the plaintiff will suggest that had the physician complied with the standard of care and effected delivery, the degree of injury to the infant from hypoxia/asphyxia could have been lessened or eliminated.

When failure to diagnose cervical insufficiency is the plaintiff’s claim, the case is based either on the presence of risk factors or the results of various clinical evaluations or ultrasonic scans. Unfortunately, symptoms of cervical insufficiency are nonspecific and can be consistent with normal pregnancy or numerous other conditions. Further, both physical examination and sonographic evaluation might not identify the condition accurately. The law responds to the problem of diagnostic challenges by advising jurors that not all mistakes or errors in judgment equate to malpractice. Thus, if in evaluating the patient’s condition, the obstetrician undertook the appropriate evaluation and exercised judgment consistent with that of the average practicing obstetrician, the physician was not negligent even though the correct diagnosis was not established.

The primary legal considerations for the obstetrician relating to cervical insufficiency and counseling are as follows:

- Obtain an adequate history at the time of the initial prenatal history. The obstetrician must recognize the possibility of risk for a woman with a prior history of cervical insufficiency or a history including recurrent mid-trimester or early third-trimester losses. In the event of recurrent mid-trimester or early third-trimester losses, cervical insufficiency, undiagnosed Müllerian anomalies, idiopathic premature labor, and other problems must be considered. In a patient with complex history of preterm losses or deliveries, it is best that the obstetrician obtain the patient’s medical records documenting her prior losses rather than relying on the patient’s understanding or recollection.
- Obstetricians must recognize the limitations inherent in both clinical and laboratory data. These limitations must be considered if the obstetrician undertakes to confirm or exclude the diagnosis of cervical insufficiency or recommends treatment if the diagnosis is suspected.
- The absence of an available test to diagnose cervical insufficiency does not relieve the obstetrician of the duty to investigate when clinical suspicion should exist. In patients who are considered at risk, serial physical examinations, laboratory testing (i.e., fetal fibronectin [FNF]) or serial ultrasound assessment of the cervix are the tests recommended to evaluate cervical change or predict the likelihood of preterm delivery.
- In women with a known history of recurrent mid-trimester or early third-trimester losses, serial transvaginal ultrasound scans might contribute to the clinical evaluation. When these studies are best conducted has not been established, however. In patients with a history of first-trimester loss, even if losses are recurrent, transvaginal ultrasonography is not necessarily indicated because the diagnosis in such cases is unlikely to be cervical insufficiency. Thus, when there is a history of early losses, transvaginal ultrasound studies are usually not helpful in establishing an etiology, unless a rare uterine structural defect such as a Müllerian anomaly is identified.
- When the diagnosis of cervical insufficiency has been made, a conservative approach to treatment is recommended unless the risk is considered high because of the prior history or other data such as the results of an ultrasound scan. Although cervical cerclage is the standard treatment for known or suspected insufficiency, its advantage over nonsurgical therapies has not yet been clearly established, given the difficulty in conducting properly controlled prospective and randomized studies and in properly identifying women who might benefit from this form of treatment. Furthermore, cerclage is not without risk. The more common risks are infection, rupture of membranes, bleeding (most notably in transabdominal cerclage), the need for resuturing, and possibly chorioamnionitis. Emergency cerclages especially are associated with an increased incidence of complications and of failure.
- Given the attendant risk, the obstetrician should know the recommended management of patients with suspected cervical insufficiency and the different clinical considerations that must be considered at a specified stage of pregnancy. Reference to the ACOG practice bulletin on clinical

management of cervical insufficiency is an appropriate starting place [32].

- Patient education is important, not only for purposes of management but also to inform the patient of the risks associated with the diagnosis and the possible complications of any recommended treatment plan. Alternatives, including the alternative of no treatment, and the risks of such alternatives must be discussed. An obstetrician must have a frank discussion with the woman about possible fetal/neonatal morbidity and mortality when it is believed that there is a high risk of preterm delivery. Documentation of these discussions is important given the potential for a dire outcome.

## REFERENCES

1. *Roe v. Wade*, 410 U.S. 113 (1973).
2. *Berman v. Allan*, 404 A.2d 8 (1979).
3. *Planned Parenthood of Southern Pennsylvania v. Casey*, 505 U.S. 833 (1992).
4. *Howard v. Lecher*, 366 N.E. 2d 64 (1977).
5. *Reed v. Campagnolo* 332 Md. 226, 241 (1993).
6. *Schirmer v. Mt. Auburn Obstetrics and Gynecologic Assoc* 155 Oho App. 3d 640 (2003).
7. *Howard v. Lecher* 366 N.E.2d 64 (1977).
8. Meadow W, Sunstein CR: Statistics, not experts. *Duke Law Journal* 2001; 51(2):629–646.
9. American College of Obstetricians and Gynecologists Practice Bulletin Number 27, May 2001: Prenatal diagnosis of fetal chromosomal abnormalities. Washington, DC: American College of Obstetricians and Gynecologists, 1998.
10. American College of Obstetricians and Gynecologists Practice Bulletin No. 44, July 2003: Neural Tube Defects. Washington, DC: American College of Obstetricians and Gynecologists.
11. American College of Obstetricians and Gynecologists Committee on Genetics. Committee Opinion No. 296 July, 2004: First-trimester screening for fetal aneuploidy. Washington, DC: American College of Obstetricians and Gynecologists, 2004.
12. Koonin LM, MacKay AP, Berg CJ: Pregnancy-related mortality surveillance – United States, 1987–1990.
13. MMWR CDC Surveillance Summaries 1997; 46 No. SS-4: 17–36.
14. *Roberts v. Mecosta County Gen. Hosp.*, 610 N.W.2d 285 (2000).
15. Anderson FW, Hogan JG, Ansbacher R: Sudden death: Ectopic pregnancy mortality. *Obstet Gynecol.* 2004 Jun;103(6):1218–23.
16. Kaplan BC, Dart RG, Moskos M, Kuligowska E, Chun B, Adel Hamid M, Northern K, Schmidt J, Kharwadkar A: Ectopic pregnancy: Prospective study with improved diagnostic accuracy. *Ann Emerg Med* 1996 Jul;28(1):10–7.
17. *Coffman v. Roberson*, 571 S.E. 2d 255 (North Carolina Court of Appeals, 2002).
18. Cartwright PS: Diagnosis of ectopic pregnancy. *Obstet Gynecol Clin North Am* 1991 Mar;18(1): 19–37.
19. Silva C, Sammel MD, Zhou L, Gracia C, Hummel AC, Barnhart K: Human chorionic gonadotropin profile for women with ectopic pregnancy. *Obstet Gynecol* 2006 Mar;107(3):605–10.
20. Barnhart KT, Sammel MD, Gracia CR, Chittams J, Hummel AC, Shaunik A: Risk factors for ectopic pregnancy in women with symptomatic first-trimester pregnancies. *Fertil Steril* 2006 Jul;86(1):36–43. Epub 2006 May 30.
21. Barnhart KT, Sammel MD, Rinaudo PF, Zhou L, Hummel AC, Guo W: Symptomatic patients with an early viable intrauterine pregnancy: HCG curves redefined. *Obstet Gynecol* 2004 Jul;104(1):50–5.
22. American College of Obstetricians and Gynecologists Practice Bulletin No. 3, December, 1998: Medical management of tubal pregnancy. Washington, DC: American College of Obstetricians and Gynecologists, 1998.
23. Sanders RC: Changing patterns of ultrasound-related litigation: A historical survey. *J Ultrasound Med* 2003 Oct;22(10):1009–15. No abstract available.
24. American College of Obstetricians and Gynecologists Practice Bulletin No. 58, December, 2004: Ultrasonography in pregnancy. Washington, DC: American College of Obstetricians and Gynecologists, 1998.
25. *Das v. Thani* 795 A.2d 876 (2002).
26. *Krishnan v. Sepulveda*, 916 S.W.2d 478, 483 (Tex. 1995).
27. *McArthur v. St. Mary's Hosp.*, 717 N.E.2d 501 (1999).
28. *Sheppard-Mobley v. King* 778 N.Y. S. 2d (2004).
29. *Martinez v. Long Island Jewish Hillside Medical Center* 519 N.Y.S. 53 (1987).



30. *Perez v. Park Madison Professional Laboratories, Inc*, 212 A.D. 2d 271, 630 N.Y.S. 2d 37 (1st Dep't 1995).
31. Iams JD, Goldenberg RL, Mercer BM, Moawad A, Thom E, Meis PJ, McNellis D, Caritis SN, Miodovnik M, Menard MK, Thurnau GR, Bottoms SE, Roberts JM: The Preterm Prediction Study: Recurrence risk of spontaneous preterm birth. National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. *Am J Obstet Gynecol* 1998 May;178(5): 1035-40.
32. American College of Obstetricians and Gynecologists Practice Bulletin No. 48, November, 2003: Clinical Management Guidelines on Cervical Insufficiency. Washington, DC: American College of Obstetricians and Gynecologists.

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# INTRAPARTUM AND POSTPARTUM

## Chapter 9 OBSTETRIC ANESTHESIA

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*... this work... treats of a noble subject – the remedy of pain. After ages of suffering, and of frequently and long intermitted pursuit of such a remedy, one has been discovered. It remains with the profession to say whether it shall take its place among the permanent and most important agents in the treatment of disease, and in abolishing pain; or whether it shall pass away with the unimportant and undeserving, until another and a truer age shall revive and give it a wider sphere of usefulness and a surer perpetuity.*

Walter Channing (1786–1876)  
*A Treatise on Etherization in Childbirth*  
Boston: W.D. Ticknor, 1848, p25.

Historically understood to encompass all of childbirth in its application, *obstetric anesthesia* is popularly more closely identified with labor and (non-operative) vaginal delivery than with surgery. In this way it differs decisively from surgical anesthesia – its value is widely disputed, and its rationale is ill defined. This condition has continued unresolved for nearly a century and a half. Charles Meigs, impressed with childbirth as an essentially natural function, explained in 1849:

I have always regarded a labor-pain as a most desirable, salutary, and conservative manifestation of life-force. I have found that women, provided they were sustained by cheering counsel and promises, and carefully freed from the distressing element of terror, could in general be made to endure, without great complaint, those labor-pains which the friends of anaesthesia desire so earnestly to abolish and nullify for all the fair daughters of Eve [1].

Less impressed with labor as a natural function than with the carnage he associated with severe and protracted labor pain, James Simpson protested in defense of anesthesia that

almost as often as the human intellect has been thus permitted to obtain a new light, or strike out a new discovery, human prejudices and passions have instantly sprung up to deny its truth, or doubt its utility, and thus its first advances are never welcomed as the approach of a friend to humanity and science, but contested and battled as if it were the attack of an enemy [1].

## ANESTHESIA VERSUS ANALGESIA

From the outset, the debate concerning relief of pain during childbirth failed to differentiate *analgesia* and *anesthesia*. Confusion about the distinctions between obstetric pain management and operative obstetric anesthesia has had far-reaching consequences for patient and practitioner alike. For example, popular perception of obstetric anesthesia as having to do with childbirth rather than “surgery” has led to widespread second-cousin status for the patient having operative obstetric intervention compared with her counterpart in the main operating suite. This devalued status is expressed in terms of cast-off and substandard anesthesia equipment cluttering outmoded delivery rooms, few and inexperienced staff, and the disregard for many intra- and postoperative procedures routinely afforded any other surgical patient. Although regulatory authorities have begun to address this problem, signs of popular discontent have been sporadic (often following the occasional catastrophe) and misdirected (apt to elicit a plush, modern décor for the same substandard facilities). This is the atmosphere that invites sarcasm from disgruntled anesthesiologists regarding the advisability of “natural” cesareans, and that withered exhortation (usually from academics) for “greater enthusiasm and sense of duty” toward obstetric anesthesia practice. Absent a turn in popular thinking, in some institutions regulatory centralization of surgical and anesthetic services to a single operating suite might be the only way of elevating the patient requiring operative obstetric intervention to full surgical status.

As much as operative obstetric anesthesia has suffered from failure to distinguish labor analgesia from surgical anesthesia, obstetric pain management has fared far worse. Simpson surmised a difference: “. . . the application of anesthesia to midwifery involves many more difficult and delicate problems than its mere application to surgery. New rules must be established for its use, its effects upon the action of the uterus, upon the state of the child and on the puerperal state of the mother” [2]. Perhaps the rudimentary development of both the analgesic and anesthetic management of his day retarded any further distinction. In any event, the popular perception of (surgical) anesthesia being applied to obstetric (nonsurgical) pain management was firmly planted. From it has grown a widespread (and sometimes justified) discomfort with the use of so drastic

a measure for the relief of what is understood to be a natural function [3]. Indeed, the American College of Obstetricians and Gynecologists (ACOG) found it necessary to issue a Committee Opinion rebutting the view that a “medical indication” should be required to justify the expense of anesthesia during labor [4]. Simpson was right, however: anesthesia modifies or suspends a variety of normal functions, can affect labor, involves risks and complications, and necessitates further efforts to circumvent these effects. Recent advances in the study of pain as distinct from the study of anesthesia, however, have promoted both analgesia and the preservation or restoration of normal functions through the control of pain. Such management finds natural application and popular acceptance in childbirth.

Consideration of pain management exclusive of surgical anesthesia has been the principal advance in obstetric analgesia of the last quarter-century, leading to improvements in maternal satisfaction and safety, labor progress and outcome, and fetal/neonatal condition. Popular expectations correspond with this distinction and anticipate a new practice paradigm, that obstetric analgesia does not imply operative anesthesia, even if both use an epidural catheter. *Epidural anesthesia* for delivery (e.g., rather than perineal infiltration) is predicated on the need for operative intervention and not necessarily precedent epidural analgesic management. Distinguishing obstetric analgesia from operative obstetric anesthesia encourages the anesthesiologist’s rational exercise of consultative pain management skills, limits anesthetic management to true operative obstetric cases, and stimulates obstetric reconsideration and tailoring of labor management for the parturient who (unlike her predecessors in previous generations) enjoys potent analgesia but is not anesthetized. Free of a misconceived connection to operative obstetric anesthesia, obstetric pain management has developed a reasonable utilization rate, distinct practice and staffing patterns, and become self-sustaining by satisfying popular expectations.

This chapter begins with a discussion of the pharmacology of both new and accepted drugs in obstetric anesthesia management. Obstetric pain management (i.e., neuraxial analgesia) is naturally the focus of the authors’ work; the practice of obstetric anesthesia based on its division into obstetric analgesia and operative obstetric anesthesia is critically reviewed, and principles by which this is best

technically accomplished are presented. The implications of this distinction as they extend to the organization and management of an obstetric service are also presented.

## PHARMACOLOGY OF OBSTETRIC ANALGESIA/ANESTHESIA

Drugs administered to the parturient to provide analgesia or anesthesia for childbirth can affect not only maternal physiology but also fetal condition and neonatal well-being. Therapeutic strategies must be formulated with consideration for these effects, as well as the compounding influences of obstetric agents and illicitly consumed substances.

### Analgesic/Anesthetic Agents and Adjuvants

#### *Premedicants*

Glycopyrrolate (Robinul), an anticholinergic agent used to increase heart rate, is a quaternary ammonium compound that does not cross the placenta. In contrast, both scopolamine and atropine cross readily and can suppress normal fetal heart rate variability [5]. Because of the potential for hallucinations and delirium caused by scopolamine, there is little to recommend its use as a sedative. Anticholinergic agents reduce lower esophageal sphincter tone and can increase the possibility of gastric reflux. Cimetidine (Tagamet) and metoclopramide (Reglan) are used to decrease gastric acidity and promote gastric emptying, respectively, prior to induction of general anesthesia. Although their efficacy is controversial, they appear to be without adverse side effects when used alone in parturients [6].

#### *Tranquilizers*

Diazepam (Valium) is seldom administered to parturients in the United States because of its long duration of action and association with neonatal depression, hypotonia, and hypothermia. Small intravenous doses (2.5 mg–10 mg) do not cause neonatal sedation or alter acid–base status but do decrease fetal heart rate variability and neuromuscular tone, the latter persisting for several hours [7,8]. The anterograde amnestic effect of midazolam (Versed) is generally undesirable because of its potentially negative effect on maternal bonding. Additionally, the commonly used combination

of intravenous midazolam (Versed), 0.05 mg/kg, together with a small amount of fentanyl (Sublimaze), results in a significant potential for hypoxemia and apnea [9].

#### *Analgesics*

Meperidine (Demerol) remains the most widely used opioid for labor analgesia. In contrast to other opioids, in doses of 2 mg to 2.5 mg/kg it decreases myocardial contractility and can lower arterial blood pressure, peripheral resistance, and cardiac output, while increasing heart rate. Meperidine produces greater cardiovascular depression when used with nitrous oxide than does morphine. Meperidine rapidly crosses the placenta after intravenous dosing and can lead to depressed Apgar scores and a delay in establishing spontaneous ventilation [10]. Adverse effects in the fetus and newborn are related to timing and dose. When the drug-to-delivery interval (DDI) is between 1 and 3 hours, a 50-mg intramuscular dose causes a significantly higher incidence of neonatal depression than is seen after a longer or shorter DDI [11]. After a 100-mg dose, heightened risk of neonatal depression persists even with a DDI exceeding 3 hours.

Morphine is rarely used for labor pain relief because it results in greater neonatal ventilatory depression than does an equianalgesic dose of meperidine. Similarly, morphine is seldom used alone for epidural or spinal analgesia because of an unacceptable incidence of side effects at doses required for satisfactory pain relief.

Fentanyl (Sublimaze) is a potent synthetic opioid with rapid onset and short duration of action. Fentanyl causes little, if any, depression of myocardial contractility or change in pulmonary or systemic vascular resistance. It seldom produces significant decreases in blood pressure when given alone, even in patients with poor left-ventricular function. It appears to cause more maternal vomiting and sedation than morphine at equianalgesic doses but has a lower incidence of neonatal respiratory depression [12]. Its ability to depress ventilation is synergistic with that of benzodiazepines, however, and care must be exercised with use of this combination. Combined with 0.125% bupivacaine (Marcaine, Sensorcaine) epidurally, fentanyl shortens onset time and prolongs duration of action compared with local anesthetic alone, without untoward maternal/fetal or labor effects (see Epidural Blockade).

Sufentanil (Sufenta), like fentanyl, has also been added to epidural bupivacaine. Specific attention must be directed to the avoidance of unintentional intravenous injection, because very little sufentanil is required to cause profound maternal ventilatory depression. Alfentanil (Alfenta) is a synthetic opioid with a rapid onset and short duration of action. It is highly protein bound in the blood. Initial experience suggests that an intravenous dose of 10  $\mu\text{g}/\text{kg}$  during induction of general anesthesia has no immediate adverse effect on the neonate [13]. Because of lower fetal levels of binding protein ( $\alpha_1$ -acid glycoprotein), however, free fractions of drug are typically as high (or higher) in fetal as in maternal blood. This warrants caution in the use of the drug. Compared with fentanyl for epidural use, sufentanil and alfentanil are more likely to produce maternal and fetal/neonatal side effects, respectively [14].

Butorphanol (Stadol) and nalbuphine (Nubain) are synthetic agonist-antagonist narcotics that are popular for use during labor because, unlike morphine, there is a ceiling effect (of dubious significance, see Systemic Analgesia) on the ventilatory depression that they produce [15]. Nalbuphine has not been associated with neonatal side effects [16]. Butorphanol, 1 mg or 2 mg intramuscularly, is equally safe for the neonate when compared with equianalgesic doses of meperidine [17]. Unlike fentanyl, sufentanil, alfentanil, or nalbuphine, butorphanol can increase systemic and pulmonary artery pressures, pulmonary capillary wedge pressure, pulmonary vascular resistance, and cardiac work [18]. Butorphanol therefore should be used cautiously, if at all, in parturients with cardiomyopathy of pregnancy or other conditions in which pulmonary artery pressures are elevated.

Opiates that cause less hemodynamic instability, are more potent, and are faster in onset and shorter in duration than existing drugs would be of interest for obstetric use. Remifentanyl, the newest synthetic opioid, can produce rapid onset of intense analgesia. A unique ester linkage enables it to undergo rapid metabolism [19]. It has a small volume of distribution and an extremely short half-life (a calculated 50% reduction in concentration at the effector site in 3.7 minutes). The time to recovery of patients receiving an infusion does not appear to be influenced by the duration of administration or total dose, or by patient age, weight, or sex [20,21]. Its potential clinical advantages include 1) rapid titra-

tion of the drug to a desired clinical effect, 2) lack of accumulation, 3) reduced incidence of postinfusion narcotic side effects, 4) safe use in obstetric analgesia, where its unique metabolism and pharmacokinetics could significantly reduce fetal exposure and all but preclude undesirable side effects in the neonate, and 5) a route of metabolism unaffected by renal or hepatic disease, with implications for use in medically complicated parturients. A recent clinical trial compared a patient-controlled IV infusion of remifentanyl with an IV infusion of meperidine; remifentanyl produced greater pain relief and patient satisfaction, less sedation and hemoglobin desaturation, fewer crossovers to epidural analgesia, and no differences in mode of delivery and neonatal outcome [22].

Ketorolac (Toradol), a nonsteroidal anti-inflammatory drug, is an attractive alternative to narcotics because of its lack of opiate side effects. A recent report, suggesting that 60 mg intravenously following cesarean delivery provides pain relief similar to spinal morphine, differs from others that show that intramuscular ketorolac alone fails to provide adequate postcesarean analgesia [23]. Such drugs must be administered with caution to pregnant women owing to potential adverse effects of prostaglandin inhibition on amniotic fluid volume production and closure of the ductus arteriosus.

Two novel analgesics, applicable only to epidural or intrathecal dosing and investigational use in the United States, are indicative of the analgesic (rather than anesthetic) approach to obstetric pain management. Neostigmine, a cholinesterase inhibitor, promotes analgesia by stimulating an acetylcholine-mediated spinal cord mechanism. Not sufficiently potent for use alone, its role as an adjuvant for opioid analgesia remains in doubt owing to nausea and vomiting caused by even very small doses. Clonidine, a centrally acting alpha-adrenoceptor agonist, also produces analgesia by stimulating a spinal cord mechanism; it is not associated with motor blockade or respiratory depression. Mild degrees of maternal sedation and hypotension remain to be fully elucidated, but an adjuvant role in local anesthetic/opiate mixtures appears promising.

### **General Anesthesia: Induction Agents**

A standard dose of thiopental (Pentothal) for induction of general anesthesia produces a decrease in

maternal systolic and diastolic blood pressure, a reduction in uterine and intervillous blood flow, and transient fetal hypoxia; nevertheless, Apgar scores at 1 minute are typically seven or greater [24–26]. Although intravenous ketamine (Ketalar), 0.9 mg/kg to 5 mg/kg, produces a dose-related increase in maternal blood pressure, an increase in uterine tone with a concomitant decrease in uterine blood flow, and fetal acidosis in pregnant ewes, intravenous induction with 1 mg/kg in humans seems to have an insignificant effect on blood flow [27]. Induction and maintenance until cesarean delivery with ketamine produces only mild elevations of maternal heart rate and blood pressure and does not result in maternal recall of events if the incision-to-delivery time is less than 10 minutes. A low Apgar score and umbilical pO<sub>2</sub> correlates with the interval from uterine incision to delivery of the infant, rather than with anesthetic management [28].

Studies evaluating propofol (Diprivan) have shown its effects on maternal hemodynamics to be similar to those of thiopental, but with equal or lower neonatal neurobehavioral scores [29,30]. This agent is often used as a component of a total intravenous anesthetic technique. An initial report showed that maintenance infusion of propofol from 150 µg/kg/min to 450 µg/kg/min does not alter uterine blood flow [31]. Subsequent studies have shown, however, that maintenance infusion during cesarean delivery is associated with a lower neurologic and adaptive capacity score at 15 minutes [32] and at 2 hours of age, secondary to rapid transfer across the placenta and accumulation in the fetus [33]. Thus, propofol appears suitable as a substitute induction agent for thiopental when the latter is contraindicated but probably should not be used for maintenance of anesthesia.

Etomidate (Amidate) is commonly used for induction in patients with compromised cardiovascular status. Its use in obstetrics has not become widespread, perhaps because of the occurrence of pain on injection in peripheral veins and because it causes involuntary muscle movement in unpremedicated patients. It is noteworthy that these effects can be prevented by judicious use of intravenous lidocaine and premedication, respectively. The suppression of cortisol production that can occur after a single bolus for anesthetic induction in adults also has been demonstrated in the neonate following maternal dosing for cesarean delivery, however [34].

### *Volatile General Anesthetic Agents*

Inhalational anesthetics do not significantly affect uteroplacental blood flow or fetal acid-base status when used to produce light planes of anesthesia. Deeper anesthesia produces a reduction in cardiac output, which in turn can cause a decrease in uterine blood flow, fetal acidosis, and bradycardia [35]. Halothane (Fluothane) is a potent uterine muscle relaxant. Even analgesic doses diminish uterine activity, and anesthetic amounts virtually abolish it [36]. Enflurane (Ethrane), isoflurane (Forane), desflurane (Suprane), and sevoflurane (Ultane) will also relax the uterus. Uterine relaxation with inhalation agents, which is usually performed at cesarean delivery or for a breech extraction or other obstetric maneuvers, can increase blood loss following delivery. It is therefore important to decrease the concentration and effect of these agents as rapidly as possible once the obstetric procedure is completed. Inhalation agents that permit the most rapid changes in alveolar concentration, like sevoflurane, might therefore offer a clinical advantage. In any event, the uterus will respond normally to oxytocin as anesthetic concentrations are reduced [37,38].

Nitrous oxide, when administered alone, causes an increase in sympathetic activity, which might decrease placental perfusion secondary to an increase in vascular resistance. This effect appears to be blunted when nitrous oxide is used in combination with a major inhalational agent. Nitrous oxide does not directly alter uterine tone. Inspired concentrations should not exceed 50%, and duration of exposure should be kept to a minimum (preferably under 20 minutes) to avoid anesthetization of the neonate.

### *Agents to Support Circulation*

Conditions that lower perfusion pressure, such as profound sympathetic blockade during regional anesthesia, cardiovascular depression with general anesthesia, hypovolemia, or supine hypotensive syndrome, can result in reduced uterine blood flow. Hypertension, which can arise from endogenously released (as during endotracheal intubation for general anesthesia) or exogenously administered vasoconstrictors, also decreases uterine blood flow secondary to an increase in uterine vascular resistance.

Ephedrine, a mixed  $\alpha$ - and  $\beta$ -adrenergic agonist, is considered the pressor agent of choice for anesthetic-induced hypotension because it lacks negative effects on uterine blood flow or fetal acid-base status [39]. Phenylephrine (Neo-Synephrine), an  $\alpha_1$ -agonist, has also been used to treat hypotension in otherwise normal parturients during regional anesthesia without affecting fetal acid-base balance [40,41]. Further study is indicated before its use in compromised parturients can be recommended. It could be of value in patients with cardiac disease in whom tachycardia caused by ephedrine is not tolerated, however. Effects of epinephrine are dose and route related. Although epinephrine used in small doses as a component of epidural local analgesics does not impair placental perfusion, use of intravenous epinephrine to support the maternal circulation can profoundly decrease uterine blood flow and must be considered only in critical situations.

There is continued debate over choice and volume of fluid to use for prevention or treatment of hypotension during regional anesthesia. There is no apparent difference between the effects of colloid or crystalloid solutions when used for circulatory preload [42]. Maternal administration of large amounts of dextrose-containing solutions is potentially detrimental to the neonate, however. Large amounts of colloid solution can theoretically cause allergic reactions and coagulation defects, whereas massive amounts of crystalloid solution can occasionally lead to pulmonary edema. Understanding these limitations, the authors recommend the infusion of 500 ml to 1500 ml of a dextrose-free, isotonic crystalloid solution (e.g., lactated Ringer's solution) prior to administering the block.

### ***Neuromuscular Blocking Agents***

Although plasma pseudocholinesterase is reduced in parturients, implying a decreased rate of metabolism for succinylcholine (Anectine), doses administered to facilitate maternal intubation have no depressant effect on neonatal ventilation. Prolonged maternal and neonatal neuromuscular blockade is a potential complication, however, in mothers with an atypical cholinesterase. The nondepolarizing agents – curare, pancuronium (Pavulon), vecuronium (Norcuron), atracurium (Tracrium), and rocuronium (Zemuron) – commonly used to prevent fasciculations from succinylcholine and to maintain surgical relaxation,

have minimal placental transfer and consequently have no adverse neonatal ventilatory, neuromuscular, or neurobehavioral effects.

### ***Local Anesthetics***

Although epidural anesthesia has been shown not to affect placental perfusion unless associated with systemic hypotension, local anesthetic use is not free of potential hazards to mother and fetus. The hormonal changes of pregnancy enhance the potency and toxicity of local anesthetics [43,44]. Local anesthetics become potentially toxic when accidentally injected intravascularly, or if there is systemic absorption of an excessive dose from the injection site. Absorption can be decreased by use of a vasoconstrictor (epinephrine) with the local anesthetic. Central nervous system (CNS) toxicity ranges from dizziness, tinnitus, and light-headedness, to seizures followed by CNS and ventilatory depression. Obstruction of the airway, the potential for aspiration of gastric contents, and increased oxygen demand during seizure activity, coupled with the decreased vital capacity and oxygen reserve of pregnancy, places the mother and fetus at risk for hypoxemia. Although most of the commonly used local anesthetics are equally toxic to the CNS, there is a profound difference in their duration of action. The effect of chloroprocaine (Nesacaine) is usually short lived because of its rapid ester hydrolysis in the blood, whereas seizures resulting from the amide drugs such as lidocaine (Xylocaine) and bupivacaine (Marcaine, Sensorcaine) can last for several minutes.

There is a general correlation between the anesthetic potency and toxicity of local anesthetics; however, bupivacaine produces cardiotoxicity of greater degree and duration at relatively lower dosages than lidocaine, ropivacaine, and levobupivacaine (the L-isomer of bupivacaine). Ventricular dysrhythmias including fibrillation are more likely to occur following rapid intravenous injection of bupivacaine than lidocaine [45]. Cardiac resuscitation is more difficult following bupivacaine-induced cardiovascular collapse and can require massive amounts of fluid, epinephrine, and antiarrhythmics. Consideration of toxicity is vitally important when any of these agents is used in doses sufficient to produce surgical anesthesia; however, doses used to produce neuraxial analgesia are well below the toxicity threshold for all of these local anesthetics.

## Drug Interactions

### *Tocolytic Agents*

A variety of agents is used for tocolysis, including  $\beta$ -sympathomimetics (ritodrine [Yutopar], terbutaline [Brethine]), direct smooth muscle relaxants (magnesium sulfate), calcium antagonists (nifedipine [Procardia]), and prostaglandin synthetase inhibitors (indomethacin [Indocin]). Drug classification helps to clarify their potential for causing serious side effects alone or in combination with other drug therapy. The most commonly used drugs with uterine relaxant properties are the  $\beta_2$ -agonists, ritodrine and terbutaline. Important side effects include arrhythmias (7%), palpitation (53%), hyperglycemia (30%), hypokalemia (39%), pulmonary edema (1%), tremor (39%), and headache (23%) [46]. Pulmonary edema is associated with multiple gestations, glucocorticoid and magnesium sulfate (a negative inotrope), or betamimetic administration, maternal infection, and, especially, injudicious fluid administration [47,48]. Arrhythmias are especially likely when  $\beta_2$ -agonists provide a pharmacologic background for the administration of anesthetic agents, such as halothane [49], anticholinergics [50], and possibly ketamine.

Slow calcium channel ion antagonists inhibit transmembrane influx of calcium ions into cardiac and vascular smooth muscle. This causes varying degrees of systemic vasodilation, negative chronotropy (reduced heart rate), and negative inotropy (decreased myocardial contractility). Nifedipine is the most potent vasodilator of the calcium channel antagonists and can have additive vasodilating effects with inhalation agents, narcotics, and epidural anesthesia. Verapamil (Calan) can interact with  $\beta$ -adrenergic blockers and inhalational anesthetics to further depress myocardial contractility and heart rate but is rarely used in obstetric practice as a tocolytic agent.

Magnesium sulfate is a divalent ion that competes with the calcium ion presynaptically at the myoneural junction, with a resultant decrease in release of the neurotransmitter acetylcholine. Postsynaptically, magnesium decreases the sensitivity of the receptor to acetylcholine. These actions result in the well-documented abnormal neuromuscular function seen in preeclamptic women receiving magnesium sulfate [51]. Magnesium can act in synergy with reduced plasma cholinesterase

(<50% of normal) seen in preeclampsia to prolong the neuromuscular blockade of an intubating dose of succinylcholine [52]. Magnesium also has negative chronotropic and inotropic effects, inhibits catecholamine release, and blunts the vascular response to vasoconstrictors [53]. Magnesium therefore can interact with both general and epidural anesthesia to cause hypotension and decreased uteroplacental perfusion. Although both phenylephrine and ephedrine have been shown to correct epidural-induced hypotension in hypermagnesemic ewes, the former causes an increase in uterine vascular resistance, whereas ephedrine does not [54].

### *Uterotonic Agents*

Oxytocin (Pitocin) is most often associated with a dose-related hypotension but occasionally causes hypertension, even in the absence of contamination by vasopressin [55]. The risk for untoward effects is greatest when bolus intravenous doses are given. When oxytocin is combined with regional or inhalation anesthesia, the potential for severe hypotension exists. Prolonged administration of high-dose oxytocin, particularly if administered in hypotonic solutions (e.g., D5W), can cause antidiuresis, fluid retention, and rarely, water intoxication. Administration of vasoconstrictor drugs in this setting could precipitate serious hypertension. Anesthesiologists should always administer oxytocin in an isotonic salt solution such as lactated Ringer's solution.

Prostaglandin 15-methyl-F $2\alpha$  (15M-PGF $_2$ , Hemabate), used to control postpartum hemorrhage, or prostaglandin E $_2$ , used to induce labor, can produce hemodynamic instability and bronchospasm in normal as well as asthmatic parturients [56,57]. PGF $_2$  can cause pulmonary artery vasoconstriction with resultant hypoxemia, systemic vasoconstriction, and cardiac arrhythmias [58]. Pulmonary effects are prominent, because most PGF $_2$  is removed in a single pass through the lungs. Any hemodynamic instability resulting from either regional or general anesthesia will compound management difficulties. Capnography and pulse oximetry, bronchodilating and vasodilating drugs, and provision for invasive cardiovascular monitoring should be readily available to manage cardiovascular collapse and refractory bronchospasm if PGF $_2$  is administered parenterally.



### ***Therapy for Pregnancy-induced Hypertension***

A variety of drugs is used in therapy for pregnancy-induced hypertension (PIH), many of which have significant interaction with agents commonly administered by anesthesiologists. Cimetidine can interfere with metabolism of drugs by cytochrome P-450, reduce hepatic blood flow, and thereby prolong the clearance and clinical action of drugs dependent on hepatic elimination. This can augment the hepatic dysfunction already present in PIH. Among drugs used for PIH that interact with cimetidine are phenytoin (Dilantin), propranolol (Inderal), lidocaine, and labetalol (Normodyne). Ranitidine (Zantac), in contrast, has less potential for significant interaction.

Labetalol, a combined  $\alpha_1$ - and nonselective  $\beta$ -adrenergic blocker, is commonly used in both chronic and acute management of PIH and to decrease the hypertensive response to endotracheal intubation during general anesthesia. Its  $\beta$ -blocking potency is approximately seven times greater than its  $\alpha_1$  blockade. Dose requirements vary, however, and titration is required. Injudicious use has the potential for precipitous drops in blood pressure when combined with potent vasodilators such as nitroglycerin (Tridil) or nitroprusside (Nipride), or whenever sympathetic tone is diminished by regional or inhalational anesthesia.

Verapamil has more potent negative chronotropic and inotropic properties than does nifedipine. It can cause severe bradycardia and decreased cardiac output when used in patients receiving  $\beta$ -adrenergic blockers. Caution must be exercised when the likelihood of using inhalational anesthetics (negative inotropic action) or regional anesthesia (vasodilation) is present. The depressant effects on uterine contractility of inhalational anesthetics and verapamil are additive, with enflurane being the most potent and halothane the least, in this regard. General anesthesia therefore can represent a higher risk of uterine atony and postpartum hemorrhage in patients treated with this and possibly other calcium antagonists [59].

Both nitroglycerin, which primarily dilates capacitance vessels, and nitroprusside, a mixed arteriolar and venous dilator, carry the risk of severe hypotension when combined with any anesthetic. These agents can increase intracranial pressure, of importance to parturients with severe preeclampsia or

other seizure disorders. Prolonged administration of nitroprusside can cause maternal and perhaps fetal cyanide toxicity. Short courses of nitroprusside therapy appear to be well tolerated. Tachyphylaxis to the vasodilating effect of nitroglycerin is well known. Hydralazine (Apresoline), a direct arteriolar dilator, has a much slower onset of action and can precipitate unexpected hypotension when used with other drugs that depress the cardiovascular system.

Low-dose aspirin, used to inhibit lipid peroxide and thromboxane production, and thus vasoconstriction and pathologic clotting, can affect bleeding time and has been a source of concern with respect to intraspinal hemorrhage, complicating regional anesthesia. Recent studies demonstrate that a bleeding time is not the "gold standard" it was once thought to be, and that the thromboelastogram (TEG) could more closely indicate clinical reality. The absence of clinical coagulopathy, typically associated with a normal TEG [60], removes the traditional (and rather arbitrary) 100,000-platelet count restriction on use of regional anesthesia; on these grounds, low-dose aspirin exposure is no longer judged an impediment to regional anesthetic procedures. PIH-associated coagulopathy remains a relevant consideration, however (see Contraindications to Neuraxial Procedures).

### ***Illicit Drugs***

Use of illicit drugs can have life-threatening consequences for both parturient and fetus and can require critical intervention. Although alcohol and marijuana continue to be the substances most commonly abused by women, the abuse of cocaine is significant in some obstetric populations. Cocaine users often abuse other drugs; the authors frequently see young expectant women who combine cocaine with alcohol, marijuana, amphetamines, or narcotics. Cocaine's half-life is unchanged in parturients (20 min–60 min), but the cardiovascular system is particularly susceptible to its effects during pregnancy [61]. The relation of cocaine to preterm labor, placental abruption, premature rupture of membranes, compromise of uterine blood flow and fetal distress, cardiac arrhythmias, and cerebrovascular accidents exposes the parturient to and compounds the attendant risks of emergency anesthesia. Acutely, cocaine inhibits neuronal uptake and deactivation of neurotransmitters,

resulting in vasoconstriction, tachycardia, hypertension, and CNS stimulation. These effects increase anesthetic requirements. Agents such as halothane and ketamine, which are by themselves arrhythmogenic, can potentiate cocaine's cardiovascular toxicity.  $\beta$ -adrenergic blockers have been used to treat cardiovascular complications, but the resulting unopposed  $\alpha$ -adrenergic stimulation can worsen hypertension. Labetalol could be the wiser therapy [62]; however, large doses could be required, which carry the risk of sudden cardiovascular collapse if epidural or general anesthesia is added. This combination can suddenly unmask an otherwise unrecognized hypovolemic state in a previously vasoconstricted cocaine abuser. Conversely, epinephrine used in epidural anesthesia can act synergistically to cause severe hypertension. Chronic cocaine abuse can deplete catecholamine stores, thus decreasing anesthetic requirements while increasing the risk of hypotension. Catecholamine depletion also decreases the response to indirect sympathomimetic drugs (ephedrine) used to treat hypotension. A direct-acting vasopressor (phenylephrine) might be required.

Like cocaine, amphetamines enhance release of catecholamines. Acute and chronic abuse can increase and decrease anesthetic requirements, respectively. The anesthesiologist should take precautions similar to those used in managing the cocaine addict. Unfortunately, whereas the effects of "crack" cocaine can last 20 to 30 minutes, those of methamphetamine ("ice") can last 8 to 24 hours.

Marijuana is the most commonly used illicit drug among women of childbearing age. It can produce euphoria and sleeplessness; as a  $\beta$ -adrenergic agonist it can cause tachycardia and increased cardiac work. The cardiovascular effects can be obtunded by  $\beta$ -adrenergic blockers. Data regarding prolongation or arrest of labor are inconclusive. Regional analgesia/anesthesia is a good choice for labor and delivery, especially since marijuana use is associated with acute and chronic bronchitis [63].

A wide variety of complications arises from narcotic abuse, including intravenous drug-related infection, endocarditis, pneumonia, pulmonary emboli, pulmonary edema, renal insufficiency, cardiac arrhythmias, and hypotension [64]. In narcotic-habituated pregnant women, methadone therapy should be continued or initiated when appropriate. Naloxone can be required to treat coma and ven-

tilatory depression. Regional analgesia/anesthesia is the obvious choice for these patients, but the dehydration, hypovolemia, and lack of cooperation that accompany narcotic withdrawal can complicate its use. Agonist-antagonist drugs should be avoided lest they precipitate acute withdrawal. Conversely, the parturient with increased tolerance can require large doses of narcotics for pain control. The addition of the parenteral nonsteroidal agent ketorolac has been helpful in the authors' management of postoperative pain in these patients

Alcohol continues to be the most abused drug in the United States. Complications include gastritis, hepatitis, cardiomyopathy, hepatic failure, malnutrition, cardiac arrhythmias, sudden death, peripheral neuropathy, and pancreatitis. Regional anesthesia can be advantageous, but the chronic alcoholic can be hypovolemic, and attention must be paid to volume status before beginning a regional procedure. The parturient's coagulation status should be checked because alcoholic liver disease can seriously elevate the prothrombin time and increase the risk for epidural hematoma. Peripheral neuropathies should be documented but do not constitute a contraindication to regional anesthesia. The risks complicating administration of general anesthesia, such as electrolyte imbalance, myocardial depression, unknown volume status, and aspiration outweigh the hypothetical risk of exacerbating a peripheral neuropathy by spinal or epidural anesthesia. A continuous epidural infusion, allowing for more gradual and controlled anesthetic spread, can be preferable to an intrathecal technique. In the acutely intoxicated or uncooperative patient undergoing alcoholic withdrawal, however, all anesthetic techniques are difficult.

### ***Obstetric Pain Management***

Management of obstetric pain has long been the subject of impassioned controversy. Disagreement arises over the severity and significance of labor pain, and hence the choice between various steps taken to relieve it. In practice, both "naturalists" and "interventionists" (for lack of better terms) have difficulty resisting overstatement of their respective positions. Some childbirth educators, minimizing the severity of labor pain and exaggerating both the effectiveness of "natural" techniques and the complications of intervention, exhort their pupils to avoid "drugs"

in favor of natural simplicity. Many practitioners consider it only natural to desire relief from pain, and all the more commendable the more technologically sophisticated, intellectually appealing, and costly the means (a 100% epidural rate!). Never mind that accommodation to pain can sometimes suffice, or that an improvement in outcome from the latest technique is no more than theoretical.

Recognition of individual differences, rather than adherence to preconceived regimens, is the basis of successful pain management. Seen in this light, "prepared childbirth training and skillfully administered epidural analgesia are compatible, complementary procedures that allow recognition of the individuality of each woman" [65].

### Labor Pain: Characteristics and Consequences

Accurate, unbiased instruction is the first step in satisfying the patient's expectations for obstetric pain management, regardless of the technique ultimately used [66,67]. Although a heavy responsibility is placed on the educator, the consequences of inappropriate expectations befall the medical practitioner who fails to understand and support this effort. Early natural childbirth enthusiasts asserted that childbirth, being a normal physiologic function, is not inherently painful. Any pain perceived was the consequence of a fear-tension-pain sequence ultimately attributable to faulty childbirth expectations stemming from modern society. Correction of these expectations and progressive relaxation would all but eliminate labor pain. Intrigued by its social implications, anthropologists and psychologists took up the question. A cooperative study of reproduction in 64 primitive peoples, however, found that "the popular impression of childbirth in primitive society as painless and easy is definitely contraindicated by our cases. As a matter of fact, it is often prolonged and painful" [68]. Nevertheless, the need to purge societal expectations to achieve painless, natural childbirth encounters formidable obstacles.

Pain is the result of a multitude of extrinsic and intrinsic factors (including expectations) and ultimately is as severe as the patient says it is. Applied to childbirth, the McGill Pain Questionnaire permits a more rational appreciation for the severity and characteristics of obstetric pain. On average, labor pain is severe, comparable to that of causalgia

and traumatic amputation of a digit, and exceeding that of cancer and postherpetic neuralgia [69]. Variability is significant, however; "about 25% of primiparas and 10% of multiparas have extremely severe pain," whereas "about 10% of primiparas and 25% of multiparas have very little pain" [70]. This recalls the importance of recognizing individual differences to rational pain management. At least a percentage of parturients is therefore likely to achieve acceptable pain management by natural techniques. The effectiveness of these techniques is variable and limited, however. For example, Melzack found that, where skillfully administered anesthesia was available, women with Lamaze training were no less likely than those without such training to request epidural anesthesia [69]. Wuitchik and coworkers observed that "although PCT (prenatal childbirth training) is known to reduce subjective pain during labor, the relief obtained is generally modest... the psychological management of active labor may be much more difficult than generally assumed... childbirth training information is most often presented in a group context without due regard for individual differences" [71]. As with any approach to pain management, dogged persistence with an ineffective therapy invites complications [72]. Severe labor pain and stress are associated with endogenous catecholamine release and predispose to dysfunctional or prolonged labor, decreased uterine blood flow, fetal acidosis and heart rate abnormalities, and a higher incidence of instrumental and cesarean delivery [65,73-75].

### Epidural Blockade

The term *epidural block* has, with the advent of neuraxial pain management, become the source of much confusion and misconception in obstetric practice and is now probably best avoided. The term no longer defines a specific therapy any more than does *parenteral*, for example. In an attempt to restore some meaning to the discussion, the authors draw a distinction between epidural *analgesia* and epidural *anesthesia*. Although recent developments in pain therapy have emphasized this distinction, the boundaries of these two therapies abut and sometimes blur. Nevertheless, fundamental differences between the two must be understood if physicians are to exploit the benefits and avoid potential disadvantages of each. In particular, practitioners who

misjudge the depth of this dichotomy, picking and choosing elements of *analgesic* therapy for use in their familiar *anesthetic* paradigm, should not be surprised to reap the benefits of neither. An analgesic regimen is presented in detail to illustrate the principles of this alternative management.

### *Analgesia Versus Anesthesia*

Epidural analgesia is powerful, providing unparalleled relief of even the most severe labor pain [76]. Many parturients experience satisfactory analgesia from less potent management techniques, however. Historically, the principal drawbacks to the use of an epidural have been an increase in malpresentation, dystocia, and instrumental delivery, as well as confinement to bed and need for bladder catheterization. The studies and experience in support of this view have been based on the use of epidural anesthesia for labor, relying solely on local anesthetics to provide conduction blockade of not only sensory but also motor and autonomic innervation. By contrast, epidural analgesia makes comparatively little and discretionary use of local anesthetics. These two epidurals are by no means interchangeable nor can the effects and management requirements of one be carelessly ascribed to the other, which profoundly affects interpretation of past and current scientific literature. Flexible obstetric management incorporating a simple epidural analgesic regimen can retain the pain-relieving potency of its anesthetic predecessor. Because epidural analgesia shuns any infringement on obstetrically important reflexes and functions, it can offer a solid gain to the parturient with severe labor pain and stress [77–79].

There are positive effects from relief of severe pain with epidural analgesia. Pain relief can improve maternal and fetal/neonatal well-being, uterine function, and, under certain circumstances, blood flow. For example, maternal hyperventilation in response to prolonged, painful labor can jeopardize fetal acid–base status; both maternal and fetal conditions return to normal with epidural analgesia [80,81]. Relief also forestalls maternal exhaustion and promotes cooperation. Additional advantages stem from avoidance of many of the well-known negative effects of epidural anesthesia, which must be understood if analgesic regimens are to be optimally applied.

### *Negative Effects of Epidural Local Anesthetics: Problems*

Local anesthetics alone, in sufficient dosage to block intense sensory input, have confounding effects on motor and autonomic transmission, accounting for the important disadvantages of epidural anesthesia in labor. Sympathetic and motor blockade together require the parturient's confinement to bed. Sympathetic vasodilation produces relative hypovolemia and orthostatic hypotension, which can jeopardize uterine blood flow and function. Careful prehydration and recumbency do not by themselves ensure adequate compensation for these sympathetic effects, however. Supine aortocaval compression, amplified by the reduction in vasomotor tone, can lead to pelvic hypotension not reflected by routine blood pressure measurements. Thus, sympathectomy from local anesthetic conduction blockade demands unremitting attention to hydration and lateral uterine displacement (during recumbency) if adverse consequences are to be avoided.

Myriad advantages have been ascribed to upright posture and ambulation during labor. Although the literature to date is rather ambiguous concerning the significance of these effects, no detrimental consequences have been described as long as appropriate fetal monitoring (by telemetry if necessary) is maintained. Marked motor blockade from local anesthetics prohibits ambulation and excludes any of its potential benefits. Voluntary bearing down, normally an important adjunct to uterine activity during expulsion, is often diminished by local anesthetic-induced muscle weakness. Less obvious, although potentially more important, is the effect of motor blockade on the tone of the intrinsic pelvic musculature. Midpelvic cranial rotation results from the interaction among downward-expulsive force, the size and attitude of the fetal head, and the bony and muscular architecture of the pelvis. Loss of muscle tone and conformation can delay or preclude rotation and descent of the head. Motor blockade from local anesthetics therefore raises several potential impediments to normal progress in labor.

The lower extent of local anesthetic conduction blockade from a lumbar epidural anesthetic descends, both with time and with repeated doses, to include the sacral segments. Pelvic autonomic (parasympathetic) transmission is thus interrupted. Sensory inputs conveyed along pelvic autonomic

nerves, however, are vital to normal progress late in labor [82]. Clinically, the strength of uterine contractions increases in the second stage of labor during the transitional phase, in association with cranial descent and distension of the birth canal. This effect culminates in the urge to bear down. Both animal and human data indicate that this bearing-down reflex depends on the release of endogenous oxytocin. The afferent limb of this neurohumoral reflex arc consists of sensory transmission along pelvic autonomic nerves stimulated by distension of the birth canal. This distension is the consequence of progressive descent of the presenting part; it is not a result of the attainment of full cervical dilatation, although these events can roughly coincide in time. The efferent limb of the reflex consists of the pulsatile release of oxytocin by the pituitary, leading to more powerful uterine contractions and further descent of the presenting part.

Local anesthetic blockade of pelvic autonomic interferes with the automatic reinforcement of expulsive powers by disrupting this bearing-down reflex arc. The blockade also predisposes to maternal exhaustion when the parturient, deprived of both her neurohumoral reflex and motor strength, is encouraged to bear down before cranial descent is well established. Allowing local anesthetic blockade to wear off before delivery should restore this reflex, but the accompanying resurgence of pain, with all of its attendant disadvantages, can well thwart the desired outcome. There is another alternative, however [82]. Epidural analgesic techniques, such as those described here, routinely and reliably deliver potent analgesia throughout labor while avoiding or minimizing the various disadvantages and untoward effects of local anesthetic blockade.

## An Epidural Analgesic Regimen for Labor

### *Neuropharmacology of Pain*

Advances in analgesic management have focused attention on the neuropharmacology of pain processing at the spinal level. Current therapy emphasizes manipulation of these processes through interaction with spinal opiate and  $\alpha$ -adrenergic receptors and minimizes reliance on the indiscriminate axonal effects of local anesthetics. Addition of fentanyl, a short-acting synthetic opiate, to bupivacaine produces epidural analgesia of greater quality and dura-

tion than is achieved with either agent alone [83]. Combination therapy therefore permits substantial bupivacaine dose reduction through replacement by fentanyl in the epidural solution, while preserving analgesic potency. Does medication of spinal opiate receptors require epidural administration of the fentanyl component, or would intravenous dosing of the narcotic prove equally effective and simpler? Vella and coworkers tested this hypothesis in parturients and observed that "... despite slightly higher plasma fentanyl concentrations in the intravenous fentanyl group, epidural fentanyl produced analgesia which was more complete, more rapid in onset and slightly longer lasting." Although fentanyl, like bupivacaine, is subject to absorption from the epidural space, "the presence of fentanyl in the systemic circulation makes a negligible contribution to analgesia..." [84]. D'Angelo and coworkers demonstrated that epidural but not intravenous fentanyl produced a large dose reduction in patient-controlled bupivacaine [85].

The role of epidural epinephrine has traditionally been considered to include reduction in vascular absorption and extension of duration of action of local anesthetics; however, research has defined a new role for  $\alpha$ -adrenergic agonists in spinally mediated analgesia. Descending inhibitory mechanisms normally impinge on the processing of a painful stimulus in the dorsal horn of the cord, acting to impede its further transmission. Epinephrine activates these inhibitory mechanisms to block spinal pain transmission. A small dose of epinephrine, ineffective by itself for pain suppression, acts with fentanyl to produce profound suppression [86]. Low-dose epinephrine in epidural analgesic solutions enhances spinal opiate effects, intensifies analgesia, and further reduces the dose of bupivacaine.

### *Technique*

The authors present an epidural analgesic mixture and management technique they have used, for more than 20 years, in over 40,000 parturients. From this the reader can presume that it has met with a reasonable measure of success and satisfaction but should conclude neither that it is demonstrably the best such approach nor that it might be employed indiscriminately. The literature is replete with epidural "cocktails" composed of virtually every permutation and combination of local

anesthetic (bupivacaine, ropivacaine, levobupivacaine), opiate (fentanyl, sufentanil, alfentanil), and adjuvant (epinephrine, clonidine, neostigmine) in varying concentrations and dosing rates. Each has its advocates and, with careful attention to the peculiarities of the components and their interactions, can be used with success. Cataloging the alternatives would not be useful; instead, the authors point out the fundamentals of epidural analgesia exemplified in the technique they know best and encourage other practitioners to adopt and become well versed in the details of a regimen consistent with these goals and suited to their own environments. Intrathecal analgesia, which has become a relevant alternative in selected circumstances, is discussed later.

### Initiation

Prior to epidural placement, the rate of intravenous infusion is accelerated, and at least 500 ml of lactated Ringer's or another glucose-free isotonic salt solution is given – more if the parturient's hydration is deficient or in doubt. A catheter is then introduced into the epidural space using standard technique and secured at a lumbar level. There is enormous personal variation in the technical details of catheter placement; however, what matters is that there should be a low incidence of complications (e.g., dural puncture, multiple punctures with backache) in the quality control tracking data [87]. An important exception to some placement techniques is that no local anesthetic or *test dose* should be injected through the needle into the epidural space. As can be seen, relative to low-dose local anesthetic-opiate- $\alpha$ -adrenergic agonist combinations used for epidural analgesia, standard epidural anesthetic test doses contain more local anesthetic than the therapeutic analgesic dose; their use, although appropriate for surgical anesthesia, defeats the purpose of local anesthetic dose reduction in epidural analgesia. Furthermore, a local anesthetic test dose through the needle can obscure the effect of a subsequent analgesic test dose through the catheter, needlessly complicating assessment of catheter position and further analgesic management.

With the catheter in place, an initial 10-ml bolus dose is administered in 5-ml increments. The dose consists of 12.5 mg bupivacaine (0.125%), 50  $\mu$ g fentanyl, and 16.5  $\mu$ g epinephrine (1:600,000) in saline solution [77,88]. This constitutes both the

*test dose* (confirming catheter position) and *loading dose* preparatory to infusion. If the catheter is properly sited, analgesia begins within 5 minutes of dosing, has achieved near-maximal effect at 15 minutes [89], and after 20 minutes, dullness to pinprick over the lower third of thoracic dermatomes with intact ankle plantar flexion strength can be demonstrated bilaterally on careful inspection. Absence of these effects after 20 minutes indicates catheter malposition – in a vein, outside the epidural space, or infelicitously threaded (laterally or caudally) within the epidural space – and the need for prompt catheter replacement.

Injection of this initial small analgesic dose through a catheter unintentionally sited in an epidural vein results in a striking absence of potent pain relief, or any other characteristic effect, and constitutes a marker for intravascular injection. Much has been written concerning the sensitivity and specificity of various markers (i.e., catecholamines, air, local anesthetics) added to epidural doses to detect intravascular injection, none of which is contributory under these circumstances, and all of which are best avoided. The initial bupivacaine dose is too small to produce toxic symptoms, whereas the fentanyl can at most result in a short-lived and inadequate analgesia unaccompanied by evidence of sensory blockade (dullness to pinprick). The epinephrine dose was the subject of theoretical concern based on research showing a transient decline in uterine blood flow (without fetal effect) after intravenous injection in healthy pregnant ewes [90]. Youngstrom and coworkers injected this analgesic dose (bupivacaine-fentanyl-epinephrine) intravenously into ewes in the presence of severe fetal asphyxia (from umbilical cord compression and placental infarction), and detected no changes in fetal or maternal condition [91]. Absence of the desired analgesic effects described previously after this initial dose is a sensitive and specific marker of the need to avoid further dosing until the catheter is replaced.

A subdural position is the only other destination for a wayward catheter. This initial analgesic dose also constitutes a safe and effective test for detection of injection through a subdural catheter. Van Zundert has reported on the safety of subarachnoid injection of 12.5 mg bupivacaine (10 ml of 0.125%) for cesarean delivery [92]. Ng et al. observed a T5 sensory level and lower-limb motor blockade,

without cardiovascular or respiratory depression, within 5 minutes after unintentional spinal injection of a similar bupivacaine-fentanyl combination [93]. In the authors' experience, subdural injection of the suggested initial dose results, within 5 to 10 minutes, in an upper thoracic sensory level and, most distinctively, marked motor blockade in the lower extremities. Loss of ankle plantar flexion strength from spinal bupivacaine at this dosage is reliably seen after 10 minutes [94]. Observation of the parturient for 20 minutes after injection shows that these effects are unmistakable; once recognized, untoward effects of subsequent subdural injection and high or total spinal anesthesia are avoided.

Twenty minutes after injection, the initial analgesic dose affords the parturient significant pain relief while maintaining cardiovascular stability and muscle strength. To maintain this state of affairs throughout labor, this initial dose can be viewed as a loading dose, preparatory to an epidural infusion designed to produce steady-state, patient-specific analgesia. Continuous infusion (by a volumetric pump) is preferred to intermittent bolus dosing; the latter 1) is predicated on "roller-coaster" analgesia (intermittent dosing equals intermittent analgesia), repeat doses being given upon the return of pain, 2) predisposes to hemodynamic instability from waxing and waning sympathetic effects, 3) entails heightened risk from a misdirected bolus should the catheter stray to an unacceptable location, 4) complicates management with the approach or onset of second stage labor (i.e., should another bolus be given?), and 5) makes unnecessarily heavy and ongoing demands on the time of scarce personnel [95,96].

### **Maintenance**

Many epidural infusion solutions are described in the literature. The earliest schedules used surgical concentrations (e.g., bupivacaine 0.5%) of local anesthetics. Although it was found that concentrations could be decreased and conduction blockade maintained, even those regimens relying on bupivacaine 0.125% remain holdovers from anesthetic (rather than analgesic) labor management. Epidural analgesic management rarely includes concentrations greater than bupivacaine 0.0625% for labor infusion. In the authors' experience, an infusion of bupivacaine 0.044%, fentanyl 1.25  $\mu\text{g}/\text{ml}$ ,

and epinephrine 1:800,000 effectively continues the analgesia produced by the loading dose. The dermatomal levels affected by the standardized loading dose differ among patients owing to biologic variability and differences in procedural technique. This individuality of effect can be used to gauge a patient-specific rate for infusion: the more (or less) extensive the spread of the loading dose, the lower (or higher) the rate of infusion. Extension to the eighth thoracic dermatome and an infusion rate of 14 ml/hr are typical [77,88]. This results in an hourly epidural infusion dose of 6.16 mg bupivacaine, 17.5  $\mu\text{g}$  fentanyl, and 17.5  $\mu\text{g}$  epinephrine.

Procedures for recognizing catheter dislodgment and malposition during epidural infusion are similar to those followed after the loading dose. Intravenous infusion from erosion of the catheter into a vein leads to dissipation of analgesia in a previously comfortable patient, without untoward effect. Resurgence of pain calls for reevaluation. Investigation of the site and quality of the pain can suggest its origin, such as breakthrough pain from a uterine rupture in a patient with a previous low-transverse incision, from perineal distension and imminent delivery, from a one-sided disparity in the level of analgesia (lateral catheter displacement), or from a symmetric loss of analgesic effect (intravenous erosion). Suspicion of an intravascular position calls either for catheter replacement or for testing its position in the same manner used initially after its insertion.

Subdural erosion of a catheter is rare but of potentially great consequence. With the hourly dosage described above, over 2 hours of subdural infusion would be required to produce high spinal anesthesia. This gradual onset of spinal blockade results in progressive motor blockade of the lower extremities. It is therefore useful to monitor lower-limb mobility hourly during infusion and immediately reevaluate or discontinue infusion should obvious impairment of muscle strength develop.

Tangible pain relief restores maternal confidence and encourages a resumption of thought and behavior that is no longer pain dominated – "freed from the distressing element of terror." Motor strength and postural stability (dorsal column function) remain essentially intact [97,98]; Breen and coworkers have demonstrated the ability of about 70% of parturients to ambulate safely during low-dose epidural infusion analgesia. (Orthostatic

hypotension and inability to perform a standing partial knee bend, indicative of a more pronounced anesthetic effect, are contraindications [99].) Nearly one half of ambulatory parturients were able to void in the bathroom (in preference to use of a bed pan), thus avoiding or delaying the need for urinary catheterization. Whereas evidence for shorter labor with ambulation remains equivocal [100], Melzack and coworkers have demonstrated that upright posture (sitting or standing) is associated with less intense labor pain [101]. Thus a low-dose epidural infusion, by encouraging movement out of bed and its related reduction in pain intensity, can promote additional local anesthetic dose reduction (achievable when the infusion is patient controlled), enhancing both success of analgesic therapy and patient satisfaction and reducing the episodic need for analgesic supplementation and perhaps assisted delivery [102,103].

For many parturients, pain relief affords the opportunity for much-needed rest and recuperation. As anxiety levels decline, support staff must remain vigilant concerning two potential anesthesia-related hazards. First, the supine position entails the risk of aortocaval compression by the gravid uterus, which can be more severe with sympathetic blockade. During recumbency, lateral uterine displacement must be ensured. Second, the laboring parturient (even the low-risk variety) is substantially more likely than her nongravid counterpart to undergo intraabdominal surgery in the very near term. For this reason, oral intake during labor must be curtailed [104]. Indeed, restriction of oral intake on a surgical ward in a patient with a similar likelihood of going to surgery is unquestioned. Prudence dictates that oral intake be further reduced in patients with predictable airway difficulty. Preference for regional anesthesia, although laudable, does not ensure the ability to actually provide satisfactory regional anesthesia when necessary. Concern for pulmonary aspiration is an important element underlying this policy and primarily reflects the difficulty in protecting the airway when the patient is unable to do so herself. This being the case, the notion that the problem of aspiration is avoided completely by routine administration of preoperative antacid is profoundly misconceived [105]. *There is no evidence that antacid therapy has influenced either morbidity or mortality from pulmonary aspiration, much less the ease of airway management.* Those who correctly

observe and lament the fact that airway disasters are more frequently associated with sparse or inexperienced anesthetic staffing on the obstetric ward than in the surgical operating suite should consider other ways in which the parturient having operative intervention might benefit from surgical management [106]. Uniform quality of care should be afforded all patients having surgery – the notion that operative childbirth is something less than surgery should not be allowed to continue.

Epidural infusion should be continued through delivery. Lowering the infusion rate to reduce numbness and restore an urge to push is rarely necessary, although easily accomplished. An episodic request for heightened analgesia is made in about 20% of cases; after ruling out pathology, imminent delivery, and catheter malposition (described previously), several options can be considered to restore acceptable relief. Most often the test dose used to reaffirm correct catheter position is sufficient to regain satisfactory analgesia. Alternatively, a small additional intravenous dose of opiate can be considered in the rapidly progressing, transitional-stage parturient.

### ***Delivery Management***

The distinction between analgesia and anesthesia must be borne in mind at delivery. Episiotomy, perineal repair, forceps delivery, and, less frequently, vacuum extraction all require anesthesia. Epidural analgesia might well suffice for spontaneous delivery, but perineal infiltration with local anesthetic (or less frequently, pudendal block) is often added, especially if an episiotomy is planned or repair required; epidural anesthesia, however, is rarely necessary under these circumstances. Anesthesia for most outlet forceps and vacuum operations is managed in the same manner. The uncommon difficult or rotational forceps procedures warrant induction of epidural anesthesia (e.g., 100 mg–200 mg lidocaine); in a patient with an epidural analgesic infusion of at least 2 to 3 hours' duration (and a primed epidural space), the onset of anesthesia after a perineal dose (or a larger dose for an urgent cesarean) is frequently rapid (<5 min).

No adverse neonatal effects have been ascribed to low-dose epidural analgesia [107]. Cumulative bupivacaine dosage is low, comparable to that known to be without neonatal neurobehavioral



effects [108]. Similarly, fentanyl dosage is low; less than one half that in other regimens found to be free of neonatal depression [99,109]. Epidural analgesia does not interfere with breastfeeding success [110].

A low-dose epidural infusion regimen of the kind described is recommended for labor analgesia in most medically complicated parturients (e.g., multiple gestation [111], preterm [112], breech presentation [113], diabetes [114], vaginal birth after cesarean delivery [VBAC; 115], obesity [116], and many forms of cardiac disease [117]), and especially those with PIH [118]. Multiple sclerosis [119], recurrent herpes genitalis [120], asymptomatic HIV infection [121], and treated chorioamnionitis [122], although once controversial, are no longer considered contraindications to epidural analgesia. Spinal deformity and previous surgery are technical obstacles to epidural placement but do not preclude a skillful attempt [123]. Traditional contraindications remain however. (See Contraindications to Neuraxial Procedures.)

## Implications for Obstetric Management of Labor

### *Timing of Analgesia*

Individual differences in labor pain and progress confound attempts to determine a point in labor at which epidural analgesia ought to be introduced. This is especially troublesome when severe pain and distress are experienced in the latent phase. Wuitchik and coworkers investigated the influence of early pain on the subsequent course of labor, controlling for obstetric factors and epidural anesthesia [74]. Latent-phase pain was rated from *discomforting* to *horrible/excruciating*, and cognitive activity from *coping* to *distress-related*. These two measures were found to be prognostic of obstetric outcome. In the horrible/excruciating pain group, 68% required instrumental delivery, compared with 30% in the discomforting pain group. Subjects in the distress-related cognitive group had two to six times the incidence of instrumental delivery, five times the incidence of abnormal fetal heart rate patterns, and four times the requirement for pediatric assistance for the neonate than subjects in the coping group [74].

Assessment of pain and cognitive activity during latent labor identified obstetrically normal women at risk for prolonged and complicated labor. There

was no such correlation, however, between labor efficiency and either pain or thought assessed during midactive or transition phases.

**Early experience of severe pain and distress-related thought may predispose the parturient to an inefficient labor pattern, which could continue through the second stage. High pain during latent labor may precipitate pathogenic increases in catecholamines and cortisol, which have been found to be higher in anxious women and which can attenuate uterine activity [74].**

Latent-phase psychobiologic influences set the tone for the subsequent course of labor and recall the importance of prenatal childbirth training in dispelling faulty expectations and promoting psychoprophylaxis. Some parturients, however, despite optimal preparation, will continue to suffer severe pain and distress in latent labor.

This subgroup of women might benefit psychologically from commencement of epidural analgesia earlier in labor [124]. Timely control of severe pain and distress and restoration of confidence with an effective analgesic (rather than an anesthetic, or temporizing intravenous analgesics) are opportunities to modify important risk factors in the pathogenesis of dystocia and should not await achievement of an arbitrary degree of cervical dilation [125]. Delay predisposes to later labor inefficiency and complications, regardless of the kind of analgesic management subsequently required, and leads to a distraught and depleted parturient whose management can become increasingly difficult and demanding. In contrast, epidural analgesia afforded the same parturient can simplify, and certainly does not negate the potential advantages of, active labor management [126].

Early epidural analgesia is also recommended for those parturients likely to have operative intervention or in whom urgent general anesthesia is likely to be unusually hazardous. The morbidly obese parturient meets both criteria: she is much more likely to undergo cesarean delivery, to require emergent intervention, and to be difficult to intubate [116]. Although epidural catheter placement is challenging (more time, more frequent second attempts, and an earlier start are required), success rates are high, and the need for general anesthesia (even for emergent

intervention) is all but eliminated. Similarly, early epidural analgesia is recommended for the parturient with severe preeclampsia [118] but without coagulopathy (see later). When each technique is optimally applied, epidural anesthesia is safer than general anesthesia should cesarean delivery be required and is less likely to compromise fetal/neonatal well-being [127], outworn opinions to the contrary notwithstanding [128,129].

### **Second-stage Management and Labor Outcome**

Epidural analgesia is continued through the second stage because significant sacral anesthetic blockade (remedied, if necessary, by a reduction in the infusion rate) is unusual. As with any analgesic technique, provision for perineal anesthesia is a separate issue. Maintaining the infusion – and relief from contraction pain balanced against perception of perineal pressure and the urge to bear down – leads to no more instrumental deliveries than does allowing analgesia to wear off [130,131]. In this way, both the bearing-down reflex and maternal fortitude are preserved.

This highlights the difference between epidural analgesia and traditional epidural anesthetic management techniques. Low-dose analgesia preserves a higher degree of pelvic floor sensation [132] and motor strength and is associated with a lower rate of instrumental and cesarean delivery compared with its high-dose, anesthetic predecessor [133–137].

A tendency toward prolongation of the second stage with epidural analgesia (and an attendant increase in instrumental delivery) led to adoption of a lengthening of the allowed duration of the second stage, consistent with ACOG's 2000 recommendations concerning instrumental delivery [138,139]. In addition, advocacy of *delayed pushing* encourages the parturient not to bear down unless she feels the urge to do so, or, at the earliest, until the presenting part is powerfully distending the perineum [104]. Evidence for such management is equivocal; neither time spent pushing nor total second-stage duration appears to be significantly reduced. There is somewhat less maternal fatigue, probably a reduction in midpelvic instrumentation, and no difference in fetal/neonatal condition, however [140–142]. If epidural analgesia has unintentionally become excessively dense (numb, heavy legs), allowing time for sacral regression (and hopefully restoration of the

bearing-down reflex) by turning down the infusion appears reasonable.

### **Parturient-controlled Epidural Analgesia**

Parturient-controlled epidural analgesia (PCEA) represents a technologic enhancement to the continuous infusion of epidural analgesic mixtures. The patient is permitted to titrate, within prescribed parameters, epidural dosage to desired pain relief. After initiating epidural analgesia in the usual way, the infusion solution is delivered through a programmable pump capable of administering a basal rate, bolus dose, lockout interval, and hourly dose limit. The principal drawback is the considerable additional cost of programmable pumps and their required infusion cartridges. Compared with continuous infusion, PCEA produces similar overall analgesia and no apparent difference in labor and fetal/neonatal outcome. Cumulative doses and motor blockade are consistently less, patient satisfaction is somewhat higher, and workload of anesthesia personnel is reduced (a matter of consequence to both the busy obstetric center and the thinly staffed community hospital) [143–146].

### **Spinal Analgesia**

Exploitation of spinally mediated pain mechanisms and the emerging availability of very-small-gauge needles sparked a reconsideration of intrathecal analgesia for labor. Early investigations with fentanyl and sufentanil found analgesia to be potent but of short duration; attempts to prolong it by addition of epinephrine or morphine only increased an already substantial incidence of side effects (e.g., transient neurologic effects, pruritus, nausea and vomiting) [147–150]. Local anesthetic/opiate combination (usually bupivacaine 1.25 mg–2.5 mg plus fentanyl 10 µg–25 µg) demonstrates the expected potentiation/dose-sparing effect, but duration remains in the 60- to 90-minute range [151]. Spinal analgesia alone appears best suited to the rapidly progressing parturient in advanced labor, for whom rapid onset is paramount and short duration is probably acceptable. Complications remain a consideration; transient (1–3 days) neurologic symptoms were recently reported in 4.2% of parturients, and an 8.5% incidence of postdural-puncture headache using 27-gauge

needles [152]. Headache was the third most common complication among the obstetric claims identified in the American Society of Anesthesiologists (ASA) Closed Claims Project and resulted in payments in over one half of the cases [153].

### Combined Spinal/Epidural Analgesia

In the case of the rapidly progressing parturient posed above, which options would be available if labor outlasted the spinal analgesic, or vaginal delivery failed and a cesarean were required? Either eventuality could be managed with an epidural catheter introduced in the same procedure as the spinal. Combined spinal/epidural analgesia (CSE) is just such a procedure, designed to exploit the rapid onset of spinal analgesia while preserving the option of long duration from epidural analgesia. The technique involves siting an epidural needle in the usual fashion, introducing a longer, narrower needle through the epidural needle to administer the spinal, and removing that needle to subsequently thread the epidural catheter.

Enthusiasts note the advantage of rapid-onset CSE analgesia late in labor, a time when epidural onset is likely to be most prolonged. Accompanying transient nonreassuring fetal heart rate changes or severe hypotension (at twice the rate seen with epidural analgesia [154]) do not typically lead to an increased incidence of cesarean delivery, and neonatal outcome has been good [155]. In another large series, additional side effects requiring treatment in 1.6% of patients prompted a recommendation that continuous pulse oximetry for 1 to 2 hours and "prompt treatment with intravenous naloxone for severe drowsiness, low oxygen saturation ( $\text{PaO}_2 \leq 90\%$  unresponsive to mask oxygen), or dysphagia should be used to minimize the risk of apnea" [156]. Portable pulse oximeters are available, should the parturient wish to ambulate. Because a minority of parturients deliver before the intrathecal component of CSE has worn off and been replaced by epidural analgesic management, it would seem unlikely that the intrathecal component would have much impact on labor progress and outcome. Indeed, CSE has not been found to influence the duration of labor or mode of delivery, compared with epidural analgesia [157,158]. Finally, a reduction in manpower requirements has been proposed with CSE: intrathecal analgesia is so profound

that "anesthesia personnel need only return to evaluate patients that subsequently develop inadequate analgesia from the epidural portion of the technique (usually less than 20% of patients)" [159].

Detractors point out that, compared with epidural analgesia, onset time for CSE is shorter by 10 minutes or less; although this might or might not achieve statistical significance, its clinical relevance is dubious [160]. After onset, analgesia from the two techniques is not significantly different [161], and neither leads to fewer cesareans [162]. Intrathecal analgesia carries a higher incidence of such side effects as pruritus, nausea, somnolence, and hypotension [163] and is distinctively associated with reports of severe respiratory depression or arrest [164,165]. The magnitude of the increase in incidence of postdural-puncture headache from CSE is operator dependent: low for inexperienced trainees more apt to puncture the dura during epidural needle placement (performed in both techniques), but several-fold higher for experienced practitioners with very little "wet-tap" risk. In both cases, however, the headache risk *must* increase with CSE.

Probably the most troubling deficiency in CSE is reflected in the necessity (referenced earlier as an advantage) to manage inadequate analgesia (i.e., an unsuitable epidural catheter) discovered only after the intrathecal effect has waned. With epidural analgesia, successful catheter position is tested and confirmed by the pain relief produced by the initial dose at catheter placement; in CSE, the analgesic effect of the intrathecal dose precludes such catheter confirmation. One consequence of the CSE technique, then, is the need to replace an unsuitable epidural catheter at a later (probably more painful) stage of labor in a previously comfortable patient, and to do so quickly. Another, more serious problem arises if the patient requires urgent operative intervention before recession of intrathecal analgesia has unmasked an ineffective catheter. For this reason, CSE should be avoided in patients at risk for urgent operative delivery or difficult airway management, including "those with morbid obesity, severe preeclampsia, history of placenta previa or an abruption, multiple gestations, abnormal presentation, or those attempting VBAC" [159].

Finally, CSE materials cost more. As with any technique, use of CSE should be confined to those circumstances wherein the benefits outweigh the

costs and disadvantages; at present, this certainly does not include generalized use for labor analgesia.

### Contraindications to Neuraxial Procedures

There are circumstances, listed later, in which neuraxial (spinal, epidural) procedures should not be performed or offered. Among them, coagulopathy poses the principal clinical challenge: the risk of causing unchecked bleeding in noncompressible epidural veins can be difficult to quantify, yet the consequence of an expanding epidural hematoma – permanent neurologic injury – is devastating. When the risk-benefit balance favors a neuraxial procedure but the threat of epidural bleeding is not trivial, steps should be taken to ensure early detection of an epidural hematoma, because surgical decompression can restore neurologic function if undertaken within 8 hours. Severe back pain, loss of bowel and bladder control, and progressive sensory and motor deficit are symptoms necessitating urgent neurosurgical consultation. Neuraxial analgesic techniques with little motor blockade are recommended; for anesthesia, short-acting agents are preferred, and the patient must be closely observed for normal block resolution. Techniques involving prolonged epidural catheterization (as for postoperative analgesia) are best avoided; catheter removal poses the same bleeding risk as insertion and can require reexamination of coagulation status. After block recession and catheter removal, neurologic checks should be monitored (especially for a spontaneous “return of the anesthesia”) at least every 2 hours until coagulation status has normalized.

Preeclampsia-induced thrombocytopenia is the most common of the pathologic coagulopathies. If several hours have elapsed since a platelet count  $\leq 150,000/\text{mm}^3$  was initially observed, it should be repeated. A stable count of  $70,000/\text{mm}^3$  (as in ITP) is less worrisome than a rapidly falling count of  $80,000/\text{mm}^3$  in a preeclamptic patient. When the platelet count is  $\leq 100,000/\text{mm}^3$ , many practitioners will check PT and PTT values, and add D-dimer assay in instances of severe preeclampsia, abruptio placentae, or intrauterine fetal demise; abnormality in any of these is not consistent with neuraxial intervention. Thromboelastograph studies and platelet function analysis suggest that platelet function can remain acceptable with counts as low as

$60,000/\text{mm}^3$  in preeclampsia, although few practitioners would accept a value below  $70,000/\text{mm}^3$  [166]. Finally, and perhaps most important, the patient should be evaluated for any clinical signs of coagulopathy: bruises, petechiae (under the blood pressure cuff), bleeding gingiva (or reported occurrence with tooth brushing) or IV sites, and hematoma at an IM injection site, before proceeding. The same considerations apply before removal of a neuraxial catheter.

Guidelines have been published regarding anesthetic management in patients receiving drug therapy that might impact coagulation status [167]. Low-dose aspirin therapy and NSAID use are not considered contraindications. Subcutaneous (mini-dose) thromboprophylaxis with unfractionated heparin is also considered low risk, and this can be confirmed by PTT testing; however, patients receiving heparin for more than 4 days should have a platelet count assessed to rule out heparin-induced thrombocytopenia. Prophylaxis with low-molecular-weight heparin (LMWH) is problematic because the anticoagulant effect is difficult to quantify (monitoring anti-Xa is no longer recommended). Early communication between obstetrician and anesthesiologist regarding patients receiving thromboprophylaxis is essential to optimal management; general anesthesia has been implicated as an associated factor in a maternal death, the anticoagulation regimen having precluded use of the preferred neuraxial technique [168]. Patients receiving low-dose LMWH therapy should not undergo neuraxial procedures for at least 12 hours after the last dose; higher-dose therapy (e.g., enoxaparin  $1.5 \text{ mg/kg}$  daily) requires an interval of at least 24 hours. Postoperatively, a twice-daily dosing regimen should not be started after less than 24 hours; single-daily dosing can be started 8 hours postoperatively, with the second dose no sooner than 24 hours after the first. Therapeutic dosing with any of the heparins, oral anticoagulants, or thrombolytic agents is, of course, an absolute contraindication.

### Systemic Analgesia

Systemic opiates and agonist/antagonists provide less potent pain relief than does epidural analgesia. Their efficacy is limited by maternal side effects and neonatal depression (all cross the placenta) at increasing doses. They are useful in uncomplicated

obstetrics for pain of moderate degree or short duration, however. Their essential pharmacology is briefly reviewed here.

Meperidine (Demerol) remains the most commonly used agent, typically in doses of 25 mg to 50 mg IV at least one hour apart. Accumulation of its active metabolite, normeperidine, can have significant and prolonged effects on the newborn. Analgesia from intravenous fentanyl (50  $\mu$ g–100  $\mu$ g) compares well with that from meperidine and is associated with fewer side effects [12]. In acceptable doses, however, neither drug alone affords adequate analgesia for transitional phase labor. Nalbuphine and butorphanol have each been found to be comparable to meperidine in terms of analgesia and neonatal effects [16,17]. Both also produce sedation and have been associated with sinusoidal fetal heart rate patterns [169,170]. The touted ceiling effect for agonist/antagonist respiratory depression is matched by their ceiling effect for analgesia and is thus of little clinical advantage. The limited analgesia and respiratory depression from the agonist/antagonists is clinically comparable to that from acceptable, equianalgesic doses of opiates. Meperidine, fentanyl, and nalbuphine have also been used in intravenous patient-controlled analgesia (PCA) for labor [171–173]. The PCA approach can exert a modest dose-sparing effect and reduce the incidence of side effects, while often improving patient satisfaction.

The limited role and effectiveness of systemic analgesia for labor has not been substantively altered by any new drug or dosing regimen until recently. Remifentanyl (whose unique opiate pharmacology was previously described) would appear to offer the potential for an improvement in the scope and efficacy of systemic labor analgesia. With onset and offset in a matter of minutes, intense analgesia, and limited fetal/neonatal exposure, the goal of useful IV-PCA labor analgesia begins to sound plausible [22].

### Paracervical Block

Submucosal local anesthetic injection into the lateral fornices anesthetizes the visceral sensory nerves (Frankenhauser's plexus) from the uterus, cervix, and upper vagina. The perineum is not anesthetized and dosing after the cervix has reached 8 cm dilation is not recommended because of the risk of direct

fetal injection. Repeated blocks are often required during labor, compounding the potential for maternal local anesthetic toxicity, from each injection and from the cumulative dose. In a recent review, low-grade anesthetic toxicity (tinnitus and oral paresthesias) was the most common maternal side effect [174].

The overriding complication of paracervical block (PCB) is fetal bradycardia. The etiology is uncertain and could be variable (toxic local anesthetic effect on the fetal myocardium, decreased uteroplacental blood flow from local anesthetic-induced vasoconstriction). Depending on its duration, it is associated with decreased fetal oxygenation, fetal acidosis, and an increased likelihood of neonatal depression [175]. Occurring in 2% to 40% of cases, bradycardia usually resolves after 10 to 20 minutes, with intrauterine resuscitative maneuvers (i.e., lateral uterine displacement, supplemental oxygen, and hydration). Persistent bradycardia can necessitate operative delivery and full neonatal support. Some recommend the ester local anesthetic 2-chloroprocaine for use in PCB because of its rapid metabolism [176]; however, it also has a short duration of action, about 40 minutes. Use of bupivacaine, the most cardiotoxic of the local anesthetics, in PCB is contraindicated by its manufacturers in the United States.

PCB might have its greatest appeal where support services (e.g., anesthesia) are limited, yet other support (surgical and neonatal) could be required should bradycardia develop and persist. When epidural analgesia is contraindicated or unavailable, a decision between PCB and systemic analgesia could ensue. Under these circumstances, the greater analgesic potency of PCB recommends it in cases of severe maternal pain and stress. Evidence suggests that neonatal status is unlikely to be adversely affected thereby, although only when PCB is administered by experienced practitioners in healthy mothers with a normal, full-term fetus [177].

### Pudendal Block/Perineal Infiltration

The analgesic techniques considered thus far focus primarily on the pain of labor, although (apart from PCB) they exert some effect on pain at delivery as well. Bilateral pudendal nerve block, or perineal infiltration, is the most common intervention for

management of pain at uncomplicated vaginal delivery. It is rational that this should be so regardless of analgesic management during labor, whether non-pharmacologic, systemic, paracervical, or epidural. Skillfully done, pudendal block can suffice for spontaneous and most low and outlet forceps or vacuum deliveries, episiotomy, and repair [178]. It can be safely performed by the obstetrician during continuous low-dose epidural analgesia, and propitiously so owing to a degree of reduced perineal sensation often conferred by the latter. Lidocaine and 2-chloroprocaine are the agents commonly used.

As blockade affects only the lower vagina, vulva, and perineum, provision must be made for induction of major regional or general anesthesia should more extensive intervention be required. Before surgical anesthesia, with its attendant risks and requirements, is begun, indication for a commensurate degree of obstetric intervention should be identified. If mid-forceps delivery or profound perineal relaxation is required, for example, epidural anesthesia can be induced when epidural analgesia has already been established. Circumstances that can involve intrauterine manipulation have traditionally been understood to warrant general anesthesia; however, when analgesia is otherwise sufficient (as with low-dose epidural), general anesthesia is no longer the only or safest alternative (see Anesthesia and Urgent Uterine Relaxation).

## **OBSTETRIC ANESTHESIA**

Obstetric anesthesia, comprising major regional conduction blockade and general anesthesia, is designed to facilitate operative intervention. It is quantitatively and qualitatively different from obstetric analgesia as described previously, must conform to the published guidelines and standards for anesthesia care, and is almost always conducted in a fully equipped operating suite [179]. Operative anesthesia must be appropriately adapted to the special requirements of surgery during pregnancy but is rarely a legitimate alternative for analgesic management.

### **Epidural Anesthesia**

Epidural anesthesia for cesarean delivery avoids the direct depressant effects of fetal exposure to general anesthetics, a distinction of increasing impor-

tance the greater the duration of exposure. Consequently, neonatal depression after elective cesarean delivery is less with epidural than general anesthesia [180]. Hypotension (more common in the non-laboring parturient) is avoided or treated with lateral uterine displacement, appropriate volume loading, and vasopressors [181]. Epidural anesthesia is less likely to be associated with severe maternal injuries [153] than is general anesthesia and is increasingly preferred for the obstetrically complicated parturient (e.g., preterm, multiple gestation, and severe preeclampsia) [127,182].

Closed-claims analysis revealed that pain during anesthesia was the third most common source of maternal injury (after death and headache), and almost all of these claims involved cesarean operations under regional anesthesia [153]. These claims could stem from poor communication (among parturient, anesthetist, and obstetrician), deficient prenatal preparation, unrealistic expectations, and reluctance by anesthesia personnel to convert to general anesthesia despite an inadequate epidural block. Several steps can be recommended to improve the quality of epidural anesthesia [183]. Addition of epinephrine, in concentrations up to 1:200,000, to lidocaine 2% intensifies the anesthetic blockade and reduces peak blood levels of local anesthetic. Absorbed epinephrine from the epidural space does not affect uterine blood flow [184,185]. Alkalinization of lidocaine 2% by addition of bicarbonate (usually 1 mEq bicarbonate to 10 ml lidocaine) both intensifies blockade and speeds its onset [186]. Finally, addition of fentanyl (up to 100  $\mu$ g) to the local anesthetic solution potentiates the anesthetic and decreases the incidence of nausea and vomiting during uterine manipulation, without adverse maternal or neonatal effects [187–189].

It is often the case, however, that an inadequate anesthetic encountered intraoperatively is a consequence of a malpositioned epidural catheter, a circumstance that cannot be relieved by any dosing schedule or supplementation, and one that requires general anesthesia. This occurrence is usually prevented by appropriate testing of catheter position preoperatively; however, traditional test-dosing regimens, designed to confirm that the catheter is not sited intravascularly or subdurally, fail to confirm that the catheter is sited so as to permit dense, bilateral blockade. A symmetric effect from the analgesic test dose described previously signifies a

well-positioned catheter and portends an effective anesthetic. Clinically, this necessitates catheter placement and testing in a labor or (anesthetic) induction room to accommodate the requisite observation period. This is logistically advantageous for elective surgery in a busy obstetric suite because subsequent anesthetic blockade can be quickly and reliably achieved while operating room time is minimized. The same advantages pertain, of course, when epidural labor analgesia has been previously established.

Recognizing the increased risks associated with general anesthesia, ACOG has advocated greater use of regional anesthesia for emergency cesarean delivery [190]. Actually, this recalls Simpson's remark after the first maternal death attributed to general anesthesia: "If we could induce local anaesthesia without that absence of consciousness which occurs in general anaesthesia, many would see it as a still greater improvement" [191]. Although general anesthesia is still preferred under certain circumstances (e.g., prolapsed cord and massive hemorrhage), a diagnosis of *fetal distress*, a term that is imprecise, nonspecific, and that lacks positive predictive value, is not incompatible with reliance on epidural anesthesia for cesarean delivery [192]. When a low-dose epidural analgesic infusion is already in use, conversion to anesthetic blockade is quick, reliable, and safe. Epidural anesthesia can be initiated in other circumstances in which immediate delivery is not essential (e.g., failure of induction, failure to progress, repeat cesarean delivery in early labor). The ACOG Committee Opinion also addresses the importance of antepartum risk assessment to minimizing the complications of emergency anesthesia. For patients in whom general anesthesia would be especially hazardous (e.g., because of obesity, airway abnormality, severe asthma, or preeclampsia), appropriate risk management often entails prophylactic epidural placement. Use of a low-dose analgesic regimen is well suited to this end.

### Combined Spinal/Epidural Anesthesia

Spinal anesthesia has long been an established alternative to general anesthesia in obstetrics and is experiencing a resurgence in popularity [193]. Risks associated with maternal airway management are diminished, and depressant effects of general anesthetics on the newborn are avoided [194]. Hypoten-

sion from rapid and extensive sympathectomy is a potential complication that must be quickly recognized and corrected to avoid neonatal acidemia, particularly in the diabetic patient [195,196]. Maternal effects of sympathectomy contraindicate the technique in several forms of maternal heart disease. Dural puncture headache is less common with the use of narrow-gauge pencil-point needles but remains a source of patient dissatisfaction [153]. Nevertheless, consistent with the ACOG Committee Opinion, the advantages of spinal anesthesia outweigh the drawbacks for most parturients when compared to general anesthesia and its attendant risk from airway management [190].

Compared with epidural anesthesia, perceived advantages of spinal anesthesia are reliability and speed. The former has less to do with successful block performance (failure rates for the two techniques are about the same [197]) than with less need for supplementation (greater patient comfort) once the block is established [198] and is important if pain during anesthesia and the need to convert to general anesthesia are to be minimized. As with epidural anesthesia, addition of epinephrine and fentanyl is recommended to enhance the success of spinal anesthesia [199–201], the latter being particularly effective in reducing perioperative nausea [202]. Rapid onset makes spinal anesthesia advantageous in urgent circumstances as a means of avoiding use of general anesthesia [203]. A potential drawback to this rapid onset is the accompanying sympathectomy, requiring careful attention to intravascular volume, position (uterine displacement), and vasopressor support to avoid hypotension. Nevertheless, spinal anesthesia can be advocated for urgent operative delivery even in instances of severe preeclampsia [204], although under more elective circumstances, an epidural catheter ideally has been placed and tested prophylactically in such high-risk parturients.

Traditional spinal anesthesia, being a "single-shot" technique, has a limited duration of action. This poses a problem in those circumstances (especially training settings) when surgery might be expected to be prolonged. CSE finds application here; if the spinal block begins to wane, the epidural catheter can be incrementally dosed to maintain anesthesia for the duration of the procedure. As a further refinement, sequential CSE can be used to advantage in complex parturients (those with cardiac

disease, pheochromocytoma, severe toxemia, or diabetes), in whom a more gradual onset of sympathectomy is preferred. In this case, a smaller spinal dose is given, with the intent to achieve only a lower thoracic level (leaving most of the sympathetics unblocked); incremental epidural doses are then used to raise the level of anesthesia gradually, giving more time to stabilize the changing hemodynamics [205,206]. Epidural anesthesia alone, however, can achieve the same result as sequential CSE, with perhaps fewer side effects (no dural puncture) and greater reliability (a confirmed catheter position).

### General Anesthesia

Circumstances such as massive maternal hemorrhage, coagulopathy, and severe sudden fetal distress continue to require general anesthesia to accommodate operative intervention. Deep inhalational anesthesia has also traditionally been used for acute uterine relaxation to facilitate removal of a retained placenta, extraction of the second of twins or a trapped after-coming head, and management of acute uterine inversion. Demand for participatory childbirth, improved understanding of the anesthetic implications for maternal morbidity and maternal/fetal physiology, and emergence of alternative means of acute tocolysis have all contributed to a marked decline in the use of general anesthesia in obstetrics [191].

General anesthesia is considered to be less safe for the parturient and more depressant for the fetus/neonate than regional anesthesia [207]. Where skilled anesthesia and neonatology support are available, maternal safety becomes the overriding issue – most anesthesia-related maternal death is a consequence of airway disasters: failed intubation, bronchospasm, or pulmonary aspiration [208–211]. Significant reduction in anesthesia-related death corresponds to increased use of regional anesthesia, with essentially no improvement in mortality risk from general anesthesia. General anesthesia is therefore chosen only for an ever-smaller group of special indications. Its reputation as unsafe and second best is particularly enhanced, however, where the anesthesiologist is called at the last possible moment to “give a whiff” to a parturient who has not been anesthesiologically evaluated, in an inadequately equipped or prepared setting, and with no realistic opportunity to pursue alternative management. Such a sce-

nario is virtually unthinkable in the surgical operating suite; rather, it smacks of casualty management in the field, where awake intubation is more liberally applied to secure the airway. Such circumstances are rarely justified in obstetrics and imply failure of organization, management, or communication.

Safe anesthesia for operative obstetrics allows no relaxation in the practice standards prescribed for surgical anesthesia. Many of these are organizational issues – preanesthetic patient evaluation, equipment and staffing – and are considered further later in this chapter. Cooperation between anesthesiologists and obstetricians is necessary if safe facilities, support, and protocols are to be designed for emergency general anesthesia. Communication and interaction are essential components of safe practice. The need for general anesthesia and the incidence of “emergencies” are reduced, and careful induction of necessary general anesthesia under better-controlled circumstances is promoted [212].

Among the hazards of obstetric anesthesia is the notion that antacid prophylaxis provides a simpler solution to the risks of general anesthesia than the approach outlined previously. For those who feel compelled to raise gastric pH on the basis of pregnancy alone, a variety of regimes (including sodium citrate, H<sub>2</sub> antagonists, and metoclopramide) are available; however, any reduction in clinical morbidity or mortality remains to be demonstrated [213]. Improvement is based instead on careful and timely patient evaluation, adequate equipment, experienced staffing, and competent assistance with airway management (e.g., cricoid pressure).

### Anesthesia and Urgent Uterine Relaxation

Deep inhalational anesthesia, although an effective means of providing uterine relaxation, carries risks of considerable morbidity in obstetrics. Deep inhalational anesthesia predisposes to neonatal depression and acidosis, increases maternal blood loss and cardiovascular depression, and entails the well-known hazards of maternal airway management. A potent, fast-acting, short-duration intravenous alternative to deep general anesthesia is preferable.

Intravenous nitroglycerin produces uterine relaxation [214], and it has been used successfully for this purpose in the management of retained placenta [215,216], inverted uterus [217], and twin [218], breech [219], and premature delivery [220].



Intravenous nitroglycerin is used in conjunction with intravenous sedation, regional anesthesia, or general anesthesia. The effect is apparent within about 1 minute after dosing, with recovery of uterine tone after another minute. Boluses of at least 50  $\mu\text{g}$  to 100  $\mu\text{g}$ , repeated as necessary, appear to be effective and well tolerated. Perhaps because of its evanescent effect, the fetus and neonate appear to be unaffected by its antenatal administration [221,222].

Intravenous nitroglycerin is faster than inhalational anesthesia in both onset and offset and avoids the risks of general anesthetic induction. Because it is also a vasodilator, however, it shares with deep inhalational anesthesia the risk of hypotension, especially in the presence of uncorrected hypovolemia. Caution must be exercised under these circumstances with either technique; rapid intravenous volume infusion, careful monitoring, and immediate availability of vasopressor support are essential. Nevertheless, the brevity of nitroglycerin's effect is an important comparative advantage.

### Massive Hemorrhage

Most obstetric hemorrhage occurs postpartum; uterine atony is the most common cause, others being inversion, retained placenta, lacerations, and coagulopathy (e.g., disseminated intravascular coagulation [DIC], either alone or superimposed). Pharmacologic management of atony escalates from oxytocin to methyergonovine to prostaglandins; retained placenta and inversion can also require intravenous nitroglycerine for transient uterine relaxation. Conservative techniques including uterine packing, balloon tamponade, vessel ligation, placement of compression sutures, or embolization should be considered before more radical options for continued bleeding [223,224]. Other causes of peripartum hemorrhage include placenta previa, abnormal attachment (placenta accreta/increta/percreta), abruption, and uterine rupture. Surgical management is the rule and, to the extent hemorrhagic risk can be anticipated, early anesthetic consultation is vital. Autologous blood predonation is appropriate and typically sufficient for volume replacement during elective surgical intervention in these high-risk cases. Epidural anesthesia can be selected to mitigate red cell loss – intravenous volume loading to maintain normovolemia produces

hemodilution as it compensates for vascular dilation during block induction – and is associated with less operative blood loss and greater survivability from hemorrhage [225,226]. Other preemptive steps exist to reduce blood loss but might not be available in many obstetric units. Interventional radiologists can preposition intraarterial catheters to provide embolization or balloon occlusion of selected vessels; this is less effective when undertaken emergently after the fact, however. Another, although controversial step, is autologous blood salvage; even when aspiration of amniotic fluid is avoided, removal of all potentially hazardous supernatant and cellular material cannot be proved. Its use could nevertheless be justified in life-threatening hemorrhage; however, the difficulties of providing this infrequently used service distant from the operating rooms, at odd hours and error free, may be significant.

Anesthetic management of ongoing obstetric hemorrhage focuses on patient resuscitation and accommodation of invasive maneuvers to control bleeding. If the patient has an epidural catheter in place, extension of the block to accommodate surgery in the face of continued bleeding and hypovolemia is ill-advised, because hemodynamic stability could be disrupted by the reduction in sympathetic tone. General endotracheal anesthesia is indicated if there is impairment of consciousness, respiratory distress, or cardiovascular instability, and to provide for surgical intervention in the unanesthetized patient. This is accomplished in a manner consistent with the principles of trauma anesthesia, protecting the airway while avoiding agents associated with cardiac depression and vasodilation (and bearing in mind that oxytocin is such an agent in the presence of cardiovascular compromise). At least two large intravenous catheters and an effective blood/fluid warmer are required. An intraarterial catheter aids in both pressure monitoring and obtaining samples for cross-match, repeated blood gas analysis, hemoglobin and fibrin degradation product estimation, and coagulation screen. One should prepare for massive transfusion; a rule of thumb is that by the time transfusion is contemplated, the patient is several units behind. Patient response to induction of general anesthesia can be indicative of the extent of cardiovascular compromise. Aggressive colloid and crystalloid infusion and vasopressor support are indicated to maintain pressure but are no substitute for

replacing the oxygen-carrying capacity of red blood cells. The blood bank should be alerted to stay several units ahead and of the potential need for fresh-frozen plasma and platelets as these coagulation factors are diluted out or consumed. A period of surgical compression or clamping might be necessary to allow time for restoration of a viable blood volume. A minimum of a second trained pair of hands is essential for anesthetic management under these circumstances, and recovery in an intensive care unit is required [227].

Many avoidable hemorrhagic deaths have been associated with the “too little, too late” phenomenon – severity of hemorrhage recognized too late, too little expert (obstetric, anesthetic, intensivist) assistance, too little interspecialty communication, too little warmed blood transfused, and too few support staff (to carry samples and blood products to and from laboratories and blood bank) [228]. This recalls the previously mentioned misperception of operative obstetrics as childbirth rather than surgery – cast-off equipment and sparse staffing in outmoded delivery rooms, far (in so many ways) from the operating suite and intensive care unit. Regular “fire drills” at all hours – to ensure rapid availability of requisite equipment and personnel, and effective communication and organization – are essential to meet this challenge.

### Anesthesia for Surgery During Pregnancy

Surgical intervention during pregnancy can affect fetal outcome; early obstetric and anesthesiologic consultation is beneficial. Evidence suggests an increased risk of fetal wastage or preterm delivery and early death but does not support a heightened frequency of congenital anomalies [229,230]. Against this risk is weighed the threat posed by the surgical pathology; on balance, elective surgery is deferred until after delivery, and more urgent surgery postponed until the second trimester if possible. For emergent surgery in the first trimester, or any surgery during pregnancy, maintenance of maternal and fetal homeostasis is of paramount importance.

Surgery requiring intraoperative uterine manipulation or retraction carries the greatest risk of adverse fetal outcome and calls for exceptional precision and finesse on the part of the surgical team. Regional anesthesia is recommended; it probably

carries the least risk of teratogenicity in the first trimester and avoids the risk of airway management later in pregnancy. No particular anesthetic technique enjoys demonstrated superiority with respect to fetal outcome, however. General anesthesia, using agents with an established record of safe use, is often an acceptable alternative. Regardless of technique, anesthetic management must account for the altered physiology of pregnancy and placental perfusion.

Obstetric anesthesia precepts regarding airway management and protection are applicable on entry into the second trimester. By 18 to 20 weeks' gestation, positioning should include lateral (usually left) uterine displacement to avoid aortocaval compression and the threat it poses to maternal hemodynamic stability and fetoplacental perfusion. Maternal blood pressure is ideally maintained within the patient's preanesthetic range; this entails appropriate volume loading and use of a mixed agonist such as ephedrine, as necessary. Regarding general anesthesia, hyperventilation is undesirable as it can impair fetoplacental gas exchange [231]. Ketamine in large doses increases uterine tone and is best avoided. Similarly, uterine vasoconstriction, whether exogenous from  $\alpha$ -adrenergic agonists or endogenous from light, balanced anesthesia, can have an adverse impact on placental perfusion.

Monitoring specific to the pregnant surgical patient includes fetal heart rate (FHR) and uterine activity. Ongoing obstetric consultation is preferred in the interpretation of these data. By the period that presumed fetal viability is reached, arbitrarily defined as 23 to 24 weeks of gestation, an electronic fetal heart rate (FHR) tracing should be obtained before, after, and, if possible, continuously during the procedure. Untoward changes might suggest adjustment of uterine displacement, maternal hyperoxygenation, alteration of maternal ventilation, or reassessment of blood pressure and volume. The concomitant use of upper- and lower-extremity oxygen saturation monitors (i.e., finger/toe) might be helpful in judging the adequacy of these measures. When the incision site precludes FHR monitoring during surgery, careful assessment of fetal condition by EFM or intrasonic techniques after anesthetic induction and positioning but prior to the incision can be particularly worthwhile. When the uterus is of sufficient size, monitoring its activity with an external tocodynamometer is similarly

recommended and could suggest addition or augmentation of a volatile agent for its uterine relaxant effect. Postoperatively, this could provide early warning of preterm labor and guide tocolytic therapy. (See Appendix III, Fetal Heart Rate Monitoring: Surgical Procedures for additional discussion.)

In the event of maternal cardiopulmonary arrest or massive trauma, manual uterine displacement (to the left and slightly cephalad) after 18 to 20 weeks' gestation must not be overlooked in the attempt to restore circulation. Prior to fetal viability (at least 23 weeks – specific criteria, recognizing institutional neonatology capabilities, should be determined prospectively) maternal survival drives resuscitation efforts and represents the best chance for fetal survival. If the point defined as potential fetal viability has been reached, expedited delivery during CPR must be considered. In some circumstances (particularly in late pregnancy) emergency delivery might be lifesaving not only for the infant but also improves maternal circulation and prospects for recovery [232]. Perimortem delivery carries a better infant prognosis the earlier it is performed but should be attempted up to at least 25 minutes after maternal arrest [233]. (See Chapter 18.)

### Postoperative Pain Management

Intravenous patient-controlled analgesia (IV-PCA) and neuraxial analgesia have been extensively investigated and developed over the past 20 years in response to deficiencies with intramuscular meperidine “as needed” for postcesarean pain management. In this regard, both have registered a marked and lasting improvement. Patients having general anesthesia receive better pain relief with IV-PCA than with conventional IM opiates, and satisfaction and side effects with IV-PCA compare favorably with those from neuraxial analgesics (although pain scores are not as low) [234,235]. Neuraxial analgesia with spinal or epidural morphine is more effective and longer lasting than with parenteral opiates [236], safe in recommended regimens, and accompanied by few, easily managed side effects (notably pruritus). Preservative-free morphine 0.1 mg, added to a spinal anesthetic (bupivacaine plus fentanyl), provides postcesarean analgesia of roughly 24 hours' duration; larger doses only add to side effects [237]. Ketorolac (30 mg IV, not to exceed 120 mg/24 hr)

is particularly effective should supplemental pain relief be requested during this time [238] and is considered safe for use in breastfeeding women by the American Academy of Pediatrics [239]. Nalbuphine 2 mg to 3 mg IV is preferred for treatment of pruritus when necessary [240]; distinct from naloxone or diphenhydramine, relief is achieved without reversal of analgesia, sedation, or recurrence. Similar analgesia in a comparable regimen is afforded patients having epidural anesthesia; morphine 4 mg is administered before the catheter is removed [241]. Larger doses are not more effective. Alternatively, the catheter is secured and a continuous epidural infusion of fentanyl-epinephrine provided for 24 to 48 hours; this has been shown to be an equally effective analgesic technique with fewer side effects and heightened patient satisfaction [242].

Ever more sophisticated, complex, and costly regimens are proposed. Postcesarean epidural PCA infusion produces better pain relief with less nausea than spinal morphine, but with similar satisfaction; it is, however, more expensive [243]. With growing emphasis on the cost-effectiveness of therapy, where do these techniques fit in? Demonstrations of improvements in outcome or length of stay have become conspicuous by their absence. Statistically significant reductions in pain scores might be more gratifying to the physician than the patient, especially if achieved at the price of an unpleasant side effect or annoying inconvenience, and are unpersuasive. Further research should address the marginal cost of improvements in patient satisfaction from new technologies [244].

### AN OBSTETRIC ANESTHESIA SERVICE

An obstetric anesthesia service requires a director with interest and skill in management; clinical, educational, or research success is not a substitute. Challenges include provision for appropriate staffing and equipment and fostering effective communication among professional staff from multiple disciplines whose timely, coordinated input is essential to safe, high-quality outcomes.

#### Staffing and Equipment

There continues to be a shortage of qualified anesthesiologists interested in providing care for

obstetric patients, and the demand exceeds availability [245,246]. The size of the patient population is a major determinant in the availability of obstetric anesthesia coverage. Gibbs and coworkers reported that there was an anesthesiologist "in house" for 24-hour coverage in only 3% and 38% of small and large hospitals, respectively [246]. This emphasizes the impracticality of small maternity units: they lose economies of scale, overtax the resources of the anesthesiology department and the hospital, and exert negative pressure on quality of care. Health service contracts that require a hospital to be a full-service institution are in conflict with this reality. Small obstetric units, if they cannot be consolidated, must be subsidized.

The American Society of Anesthesiologists' (ASA) Guidelines for Regional Anesthesia in Obstetrics state that an anesthesiologist should initiate a regional anesthetic, whereas a CRNA is permitted to monitor its effect [247]. Such idealism does not correspond to the realities or logistics of clinical practice in either rural America or many metropolitan areas. An unpublished 1991 survey of members of the Society for Obstetric Anesthesia and Perinatology reported that a large percentage of conduction anesthetics for obstetrics were done by CRNAs with or without anesthesiologist supervision [248]. In the authors' view, it is rational that a CRNA who has met the technical and intellectual requirements established by a hospital administer an epidural analgesic to appropriately selected patients. The anesthesiologist can be called to address those who are more seriously ill or difficult. There are major logistical and cost advantages: 1) duplication of personnel is avoided, 2) personnel are better used during the on-call period, and 3) anesthesiologist time is applied more effectively.

It is a regulatory requirement in the United States that anesthesia and monitoring equipment in the obstetric suite must be of the same quality as that in the general operating room. Provision must be made (preferably in the obstetric suite or by expeditious transfer to the ICU) for intensive care facilities, which should include monitoring equipment for direct measurement of arterial, central venous, and pulmonary artery pressures; evaluation of cardiac output; and for measurement of arterial and mixed venous blood gases. Intensive care ventilator(s) and respiratory therapists should be designated for obstetric service. Ideally, a satellite labora-

tory should be located in a busy high-risk obstetric suite.

### Communication and Coordination

A high-reliability obstetric unit is characterized by robust communication among obstetricians, anesthesiologists, neonatologists, midwives, and nurses that solidifies the perception of team membership. "What if" scenarios should be emphasized and discussed. Emergencies, including hemorrhage and shoulder dystocia, are rehearsed, and drills include interaction with laboratories, blood bank, respiratory therapy, ICU, and the main operating rooms as necessary. In an academic setting, this can be promoted by resident and faculty attendance at daily teaching rounds, as well as obstetric and neonatology conferences. In nonacademic settings, the director must attend obstetric and nursing department meetings and ideally combined quality assurance meetings. Attention and input at shift changes by a member of the anesthesia service benefits practice and morale in both settings.

### Outpatient Anesthesiology Clinic

Communication among obstetricians, midwives, and anesthesiologists is enhanced by providing access to an outpatient anesthesiology clinic for prenatal patients. The logistical advantages are several and have been shown to include benefits to all concerned [249].

The preanesthetic evaluation focuses on a review of personal and familial medical and surgical histories, allergies and adverse drug reactions, current illness (often overlooked), medication use and abuse, potential for airway difficulties, and other complicating anesthetic factors such as obesity or spinal deformity. Medically complicated parturients and those likely to require urgent or operative intervention should be identified and discussed in advance. This is particularly important in conditions with the potential for blood loss (placenta previa or accreta, and prior cesarean delivery), which occur more frequently as the cesarean delivery rate has increased [250]. In patients receiving thromboembolism prophylaxis, this opportunity for early planning and regimen coordination can be crucial to avoiding compromise of optimal anesthetic management. (See Contraindications to Neuraxial Procedures.)

Relevant laboratory assessment should be confirmed and should include arrangements for autologous blood donation.

This prenatal visit also affords a valuable opportunity to discuss anesthetic options, risks, benefits, expectations, and fears [66]; it can be supplemented by information pamphlets and video tapes, which visually describe the typical experience of a parturient having an epidural analgesic or anesthetic. These efforts pay dividends during intrapartum management and can help ensure informed consent.

### ***Informed Consent***

The pertinent risks, benefits, and expectations of the anesthetic plan must be fully discussed [251]. Although of concern to many anesthesiologists, informed consent may be legally obtained during labor and is appropriate and necessary [252]. Explanation of an anesthetic plan is intended to inform, not coerce or frighten. When a detailed discussion of risks is called for, the obstetric nurse is often well suited to assuage unwarranted anxiety and curb unreasonable expectations. Documentation should acknowledge the following: a description of the planned management, patient acknowledgment of the salient risks and expected benefits, a statement that patient inquiries were invited and answered, and finally that she wishes to proceed.

### **Quality Assurance**

#### ***Traditional Approaches***

Traditional intradepartmental guidelines regarding credentialing, policies, procedures, risk management, and quality care can be found in standard textbooks, statements by the Joint Commission on Accreditation of Healthcare Organizations (JCAHO), and in the Standards, Guidelines, and Statements of the ASA. ASA guidelines and JCAHO standards are frequently employed, at least in part, by virtually all anesthesiology departments and appear in their policies and procedures manuals. In the United States, compliance with JCAHO standards is functionally mandatory, because most states require JCAHO accreditation to qualify for licensure. JCAHO quality assurance (QA) focuses on 1) documentation of preoperative and postoperative anesthetic evaluation, and 2) presence of an

assessment process that records "indicator" (adverse event) occurrence, investigates the circumstances, determines deviation from acceptable standards of care, and communicates directly back to the responsible person(s) with a plan for corrective action. The ASA has an occurrence-driven QA process in which a QA committee determines relevancy to anesthetic management, classifies the type of management error, determines the nature of the underlying error (i.e., none, mechanical, human), grades the clinical severity according to a negative outcome score, and reports corrective action back to the practitioner [253]. This process facilitates tracking of individual performance, as well as comparison with department norms and with other practitioners, at significant administrative cost [254].

These QA approaches, along with legal closed-claims analysis, have helped to improve care, decrease risk, and decrease legal and financial liability. Both are based on retrospective analysis of identified adverse outcomes, however. There are no guidelines for the measurement of variations in clinical practice that underlie these events. Both count adverse events but provide no measure of cause and effect and thereby little stimulus for continuous quality improvement.

#### ***Continuous Quality Improvement***

Continuous quality improvement (CQI) or TQM (total quality management) is founded on statistical analysis of an activity (process)-based accounting system and provides a framework for continuous process and product improvement. Three important tenets of this statistical process control are 1) outcome (quality) must be measured for improvement to occur, 2) analysis of variation found in outcome reveals information that can be used to improve quality, and 3) control charts and flow diagrams help to identify process variation and facilitate statistically valid improvements in outcome.

The concept of statistical process control can form the basis of CQI through integration with cause-and-effect diagrams. This is a management tool that tracks all of the events in a manufacturing process and enables them to be related to various outcome measurements [255]. Similar flowcharts, in the form of algorithms or critical paths, have been used in medical textbooks for years as teaching aids. The CQI technique involves creation of a flowchart

TABLE 9.1 Labor and Delivery Anesthesia Survey

Please circle YES or NO for each question.

1. Did you have epidural analgesia for labor pain relief?	YES	NO
2. Were risks and benefits of epidural analgesia explained clearly?	YES	NO
3. Did your epidural give you satisfactory pain relief?	YES	NO
4. You did not have an epidural because:		
a. You did not want one.	YES	NO
b. It was not offered.	YES	NO
c. The anesthesia team was too busy.	YES	NO
d. The nurses were too busy.	YES	NO
e. Did not have time – too close to delivery.	YES	NO
f. Wanted one, but changed mind after hearing the risks.	YES	NO
5. Would you have an epidural for your next delivery?	YES	NO
6. Did you have a spinal anesthetic?	YES	NO
7. Were you satisfied with your spinal anesthetic?	YES	NO
8. Did you have a vaginal delivery (from below)?	YES	NO
9. Did you have a cesarean delivery?	YES	NO
10. Did you have a general anesthetic (put to sleep)?	YES	NO
11. Were you satisfied with your general anesthetic?	YES	NO
12. How would you rate the anesthesia team member who took care of you?		
a. Professional	YES	NO
b. Pleasant	YES	NO
c. Considerate	YES	NO
d. Indifferent	YES	NO
e. Inconsiderate/rude	YES	NO

Comments:

Name (optional):

Date of delivery (optional):

diagram that is used to describe how the process of clinical care should be organized. Such a system, used by one of the authors [256], served to define areas of quality improvement, such as when a control chart run of low rates of narcotic-related side effects began when fentanyl replaced morphine for postcesarean epidural pain management. Outcome based on this change in clinical practice (use of fentanyl) was then used to reset (raise) the performance level and control limits of the service [87].

A simple but useful way to introduce this approach to QA is the patient satisfaction survey (Table 9.1). Filled out at the time of discharge, this survey can provide valuable outcome results that might suggest modifications in the service. This highlights the goal of CQI: to improve the process of care, not to identify persons or cast blame for the responsibility of substandard care. More powerful

application of CQI requires investment in information technology but offers the prospect of continuous advances in patient safety and care.

## REFERENCES

1. Farr AD: Early opposition to obstetric anaesthesia. *Anaesthesia* 1980;35:896–907.
2. Reid DE: Foreword. In: Bonica JJ (ed): *Principles and Practice of Obstetric Analgesia and Anesthesia*. Philadelphia: FA Davis, 1967; pp. vii.
3. *Birth of Babies in the '90s*. FDA Consumer, December 1992.
4. The American College of Obstetricians and Gynecologists: *Pain relief during labor*. ACOG Committee Opinion #118. Washington, DC: American College of Obstetricians and Gynecologists, 1993.

5. Abboud TK, Raya J, Sadri S, et al: Fetal and maternal cardiovascular effects of atropine and glycopyrrolate. *Anesth Analg* 1983;62:426-430.
6. Hodgkinson R, Glassberg R, Joyce TH, et al: Comparison of cimetidine (Tagamet) with antacid for safety and effectiveness in reducing gastric acidity before elective cesarean section. *Anesthesiology* 1983;59:86-90.
7. Scher J, Hailey DM, Beard RW: The effects of diazepam on the fetus. *J Obstet Gynaecol Br Commonw* 1972;79:635-638.
8. Yeh SY, Paul RH, Cordero L, et al: A study of diazepam during labor. *Obstet Gynecol* 1974;43:363-373.
9. Bailey PL, Pace NL, Ashburn MD, et al: Frequent hypoxemia and apnea after sedation with midazolam and fentanyl. *Anesthesiology* 1990;73:826-830.
10. Shnider SM, Way EL, Lord MJ: Rate of appearance and disappearance of meperidine in fetal blood after administration of narcotic to the mother. *Anesthesiology* 1966;27:227-228.
11. Shnider SM, Moya F: Effects of meperidine on the newborn infant. *Am J Obstet Gynecol* 1964;89:1009-1015.
12. Rayburn WF, Smith CV, Parriott JE, et al: Randomized comparison of meperidine and fentanyl during labor. *Obstet Gynecol* 1989;74:604-606.
13. Dann WL, Hutchinson A, Cartwright DP: Maternal and neonatal responses to alfentanil administered before induction of general anesthesia for caesarean section. *Br J Anaesth* 1987;59:1392-1396.
14. de Sousa H, Stiller R: Cisternal CSF and arterial plasma levels of fentanyl, alfentanil and sufentanil after lumbar epidural injection. Annual Meeting of the Society for Obstetric Anesthesia and Perinatology Abstracts of Scientific Papers, Seattle, Washington, 1990.
15. Romagnoli A, Keats AS: Ceiling effect for respiratory depression by nalbuphine. *Clin Pharmacol Ther* 1980;27:478-485.
16. Frank EJ, McAteer R, Cattermole B, Loughnan BA, Stafford LB, Hitchcock AM: Nalbuphine for obstetric analgesia: A comparison of nalbuphine with pethidine for pain relief in labour when administered by patient-controlled analgesia (PCA). *Anaesthesia* 1987;42:697-703.
17. Maduska AL, Hajghassemali M: A double-blind comparison of butorphanol and meperidine in labor: Maternal pain relief and newborn outcome. *Can Anaesth Soc J* 1978;25:398-404.
18. Popio KA, Jackson DH, Ross AM, et al: Hemodynamic and respiratory effects of morphine and butorphanol. *Clin Pharmacol Ther* 1978;23:281-287.
19. Glass SA, Hardman D, Kamiyama Y, et al: Preliminary pharmacokinetics and pharmacodynamics of an ultra-short-acting opioid: Remifentanil (GI87084B). *Anesth Analg* 1993;77:1031-1040.
20. Talmage D, Egan MD, Lemmens HJ, et al: The pharmacokinetics of the new short-acting opioid remifentanil (GI87084B) in healthy adult male volunteers. *Anesthesiology* 1993;79:881-892.
21. Westmoreland CL, Hoke JF, Sebel PS, et al: Pharmacokinetics of remifentanil (GI87084B) and its major metabolite (GI90291) in patients undergoing elective surgery. *Anesthesiology* 1993;79:893-903.
22. Evron S, Glezerman M, Sadan O, et al: Remifentanil: A novel systemic analgesic for labor pain. *Anesth Analg* 2005;100:233-238.
23. Cohen SE, Desai JB, Ratner EF, et al: Ketorolac and spinal morphine for postcesarean analgesia. *Anesthesiology* 1993;79:A1025.
24. Cossmi EV, Condorelli S, Scarpelli EM: Fetal asphyxia induced by sodium thiopental, thiamylal, and methohexital. Fourth European Congress of Perinatal Medicine, Prague, Czechoslovakia, 1974 (abstr).
25. Shnider SM, Wright RG, Levinson G, et al: Plasma norepinephrine and uterine blood flow changes during endotracheal intubation and general anesthesia in the pregnant ewe. Annual Meeting of the American Society of Anesthesiologists Abstracts of Scientific Papers, Chicago, IL, 1978.
26. Jouppila P, Kuikka J, Hollmen A: Effect of induction of general anesthesia for cesarean section on intervillous blood flow. *Acta Obstet Gynaecol Scand* 1979;58:249-253.
27. Marx GF: Postpartum uterine pressures with different doses of ketamine. *Anesthesiology* 1979;50:163-166.
28. Baraka A, Louis F, Dalleh R: Maternal awareness and neonatal outcome after ketamine induction of anaesthesia for caesarean section. *Can J Anaesth* 1990;37:641-644.
29. Gin T, Gregor A, Oh TE: The haemodynamic effects of propofol and thiopentone for induction

- of cesarean section. *Anaesth Intens Care* 1990;18:175-179.
30. Dailland P, Cockshott ID, Lirzin JD, et al: Intravenous propofol during cesarean section: Placental transfer, concentrations in breast milk and neonatal effects. *Anesthesiology* 1989;71:827-834.
  31. Alon E, Rosen MA, Shnider SM, et al: Maternal and fetal effects of propofol anesthesia in the ewe. *Anesthesiology* 1991;75:A1077.
  32. Gin T, Yau G, Chan K, et al: Disposition of propofol infusions for cesarean section. *Can J Anaesth* 1991;38:31-36.
  33. Yau G, Gin T, Ewart MC, et al: Propofol for induction and maintenance of anaesthesia at cesarean section: A comparison with thiopental/enflurane. *Anaesthesia* 1991;46:20-23.
  34. Reddy BK, Pizer B, Bull PT: Neonatal serum cortisol suppression by etomidate compared with thiopentone for elective cesarean section. *Eur J Anaesth* 1988;5:171-176.
  35. Palahnuik RJ, Shnider SM: Maternal and fetal cardiovascular and acid-base changes during halothane and isoflurane anesthesia in the pregnant ewe. *Anesthesiology* 1974;41:462-472.
  36. Vasicka A, Kretchmer H: Effect of conduction and inhalation anesthesia on uterine contractions: Experimental study of the influence of anesthesia on intraamniotic pressures. *Am J Obstet Gynecol* 1961;82:600-622.
  37. Abadir AR, Humayen SG, Calvillo D, et al: Effects of isoflurane and oxytocin on gravid human uterus in vitro. *Anesth Analg* 1987;66:S1.
  38. Marx GF, Kim YO, Lin CC, et al: Postpartum uterine pressures under halothane or enflurane anesthesia. *Obstet Gynecol* 1978;51:695-698.
  39. James FM, Greiss FC, Kemp RA: An evaluation of vasopressor therapy for maternal hypotension during spinal anesthesia. *Anesthesiology* 1970;33:25-34.
  40. Ramanathan S, Grant GJ: Vasopressor therapy for hypotension due to anesthesia for cesarean section. *Acta Anaesth Scand* 1988;32:559-565.
  41. Moran DH, Perillo M, LaPorta RF, Bader AM, Datta S: Phenylephrine in the prevention of hypotension following spinal anesthesia for cesarean delivery. *J Clin Anesth* 1991;3:301-305.
  42. Murray AM, Morgan M, Whitwam JG: Crystalloid versus colloid for circulatory preload for epidural cesarean section. *Anaesthesia* 1989;44:463-466.
  43. Datta S, Hurley RJ, Naulty JS, et al: Plasma and cerebrospinal fluid progesterone concentrations in pregnant and nonpregnant women. *Anesth Analg* 1986;65:950-954.
  44. Morishima HO, Pedersen H, Finster M, et al: Bupivacaine toxicity in pregnant and nonpregnant ewes. *Anesthesiology* 1985;63:134-139.
  45. Strichartz GR, Covino BG: Local anesthetics. In: Miller RD (ed): *Anesthesia*. New York: Churchill Livingstone, 1990; p. 463.
  46. The Canadian Preterm Labor Investigators Group: Treatment of preterm labor with the beta-adrenergic agonist ritodrine. *N Engl J Med* 1992;327:308-312.
  47. Armson BA, Samuels P, Miller F, et al: Evaluation of maternal fluid dynamics during tocolytic therapy with ritodrine hydrochloride and magnesium sulfate. *Am J Obstet Gynecol* 1992;167:758-765.
  48. Katz M, Robertson P, Creasy R: Complications associated with terbutaline for preterm labor. *Am J Obstet Gynecol* 1981;139:605-608.
  49. Shin YK, Kim YD: Ventricular tachyarrhythmias during cesarean section after ritodrine therapy: Interaction with anesthetics. *South Med J* 1988;81:528-530.
  50. Simpson JJ, Giffin JP: A glycopyrrolate-ritodrine drug-drug interaction. *Can J Anaesth* 1988;35:187-189.
  51. Ramanathan J, Sibai B, Pillai R, et al: Neuromuscular transmission studies in preeclamptic women receiving magnesium sulfate. *Am J Obstet Gynecol* 1988;158:40-46.
  52. Kambam J, Mouton S, Entman S: Effect of preeclampsia on plasma cholinesterase activity. *Can J Anaesth* 1987;34:579-581.
  53. Zaloga G, Eisenach JC: Magnesium, anesthesia, and hemodynamic control. *Anesthesiology* 1991;74:1-2.
  54. Sipes SL, Chestnut D, Vincent RD Jr, et al: Which vasopressor should be used to treat hypotension during magnesium sulfate infusion and epidural anesthesia? *Anesthesiology* 1992;77:101-108.
  55. Weis F, Markello R, Mo B, et al: Cardiovascular effects of oxytocin. *Obstet Gynecol* 1975;46:211-214.
  56. Partridge BL, Key T, Reisner LS: Life-threatening effects of intravascular absorption of PGF2-alpha during therapeutic termination of pregnancy. *Anesth Analg* 1988;67:1111-1113.



57. Brenner WE, Hendricks CH, Braaksma JT, et al: Intra-amniotic administration of prostaglandin F<sub>2α</sub> to induce therapeutic abortion: Efficacy and tolerance of two dosing schedules. *Am J Obstet Gynecol* 1972;141:781-787.
58. Secher NJ, Thyssen PA, Olsen J: Effect of prostaglandin E<sub>2</sub> and F<sub>2α</sub> on the systemic and pulmonary circulation in pregnant anaesthetized women. *Acta Obstet Gynaecol Scand* 1982;61: 213-218.
59. Laszo A, Buljubasic N, Zsolnai B, et al: Interactive effects of volatile anesthetics, verapamil, and ryanodine on contractility and calcium homeostasis of isolated pregnant rat myometrium. *Am J Obstet Gynecol* 1992;167:804-810.
60. Orlikowski CEP, Payne AJ, Moodley J, et al: Thromboelastography after aspirin ingestion in pregnant and nonpregnant subjects. *Br J Anaesth* 1992;69:159-161.
61. Woods JR, Plessinger MA: Pregnancy increases cardiovascular toxicity to cocaine. *J Obstet Gynecol* 1990;162:529-533.
62. Ramoska E, Sacchetti AD: Propranolol-induced hypertension in treatment of cocaine intoxication. *Ann Emerg Med* 1985;14:1112-1113.
63. Wu TC, Tashkin DP, Djahed B, et al: Pulmonary hazards of smoking marijuana as compared with tobacco. *N Engl J Med* 1988;318:347-351.
64. Giuffrida JG, Bizzarri DV, Saure AC, et al: Anesthetic management of drug abusers. *Anesth Analg* 1970;49:272-278.
65. Melzack R: The myth of painless childbirth. *Pain* 1984;19:321-337.
66. Paech M, Gurrin L: A survey of parturients using epidural analgesia during labour: Considerations relevant to antenatal educators. *Aust N Z J Obstet Gynecol* 1999;39:21-25.
67. McCrea B, Wright M: Satisfaction in childbirth and perceptions of personal control in pain relief during labour. *J Adv Nurs* 1999;29:877-884.
68. Ford CS: *A Comparative Study of Human Reproduction*. New Haven: Yale Medical Press, 1945.
69. Melzack R, Taenzer P, Feldman P, Kinch R: Labour is still painful after prepared childbirth training. *Can Med Assoc J* 1981;125:357-363.
70. Melzack R, Kinch R: Labour is still painful. *Can Med Assoc J* 1982;126:357.
71. Wuitchik M, Hesson K, Bakal D: Perinatal predictors of pain and distress during labor. *Birth* 1990; 17:186-191.
72. Stanton ME: The myth of "natural" childbirth. *Journal of Nurse-Midwives* 1979;24:25-29.
73. Lederman RP, Lederman E, Work BA, McCann D: Anxiety and epinephrine in multiparous women in labor: Relationship to duration of labor and fetal heart rate pattern. *Am J Obstet Gynecol* 1985;153: 820-827.
74. Wuitchik M, Bakal D, Lipshitz J: The clinical significance of pain and cognitive activity in latent labor. *Obstet Gynecol* 1989;73:35-42.
75. Stewart D: Psychiatric symptoms following attempted natural childbirth. *Can Med Assoc J* 1982; 127:713.
76. American Academy of Pediatrics, American College of Obstetricians and Gynecologists: *Guidelines for Perinatal Care*, 3rd ed. Elk Grove Village, IL: American Academy of Pediatrics; Washington, DC: ACOG, 1992; p. 73.
77. O'Grady JP, Youngstrom P: Must epidurals always imply instrumental delivery? *Contemporary Obstet and Gynecol* 1990;35:19-27.
78. Thallon A, Shennan A: Epidural and spinal analgesia and labour. *Curr Opin Obstet Gynecol* 2001; 13:583-587.
79. Marucci M, Fiore T: Epidural analgesia for labour and delivery. *Minerva Anestesiologica* 2004;70: 643-647.
80. Bergmans M, Geijn H, Hasaart T, et al: Fetal and maternal transcutaneous PCO<sub>2</sub> levels during labour and the influence of epidural analgesia. *Eur J Obstet Gynecol Reprod Biol* 1996;67:127-132.
81. Reynolds F, Sharma S, Seed P: Analgesia in labour and fetal acid-base balance: A meta-analysis comparing epidural with systemic opioid analgesia. *Br J Obstet Gynaecol* 2002;109:1344-1353.
82. Goodfellow CF, Hull MGR, Swaab DF: Oxytocin deficiency at delivery with epidural analgesia. *Br J Obstet Gynaecol* 1983;90:214-219.
83. Youngstrom P, Eastwood D, Patel H, et al: Epidural fentanyl and bupivacaine in labor: Double-blind study. *Anesthesiology* 1984;61:A414.
84. Veila LM, Willatts DG, Knott C, et al: Epidural fentanyl in labour: An evaluation of the systemic contribution to analgesia. *Anaesthesia* 1985;40:741-747.
85. D'Angelo R, Gerancher J, Eisenach J, et al: Epidural fentanyl produces labor analgesia by a spinal mechanism. *Anesthesiology* 1998;88:1519-1523.
86. Collins JG, Kitahata LM, Matsumoto M, et al: Spinally administered epinephrine suppresses

- noxiously evoked activity of WDR neurons in the dorsal horn of the spinal cord. *Anesthesiology* 1984;60:269–275.
87. Youngstrom P, Boyd D, Rhoton F: Statistical process control (SPC) in OB anesthesia service: Six years experience. *Anesthesiology* 1992;77:A1020.
  88. Youngstrom P, Sedensky M, Frankmann D, et al: Continuous epidural infusion of low-dose bupivacaine-fentanyl for labor analgesia. *Anesthesiology* 1988;69:A686.
  89. Harms C, Siegmund M, Marsch S, et al: Initiating extradural analgesia during labour: Comparison of three different bupivacaine concentrations used as the loading dose. *Fetal Diagn Ther* 1999;14:368–374.
  90. Hood DD, Dewan DM, James FM: Maternal and fetal effects of epinephrine in gravid ewes. *Anesthesiology* 1986;64:610–613.
  91. Youngstrom P, Hot M, Veille JC, et al: Effects of intravenous test dose epinephrine on fetal sheep during acute fetal stress and acidosis. *Reg Anesth* 1990;15:237–241.
  92. Van Zundert AA, DeWolf AM, Vaes L, Soetens M: High-volume spinal anesthesia with bupivacaine 0.125% for cesarean section. *Anesthesiology* 1988;69:998–1003.
  93. Ng A, Shah J, Smith G: Is continuous spinal analgesia via an epidural catheter appropriate after accidental subarachnoid administration of 15 ml of bupivacaine 0.1% containing fentanyl 2 µg/mL? *Int J Obstet Anesth* 2004;13:107–109.
  94. Daoud Z, Collis R, Ateleanu B, et al: Evaluation of S1 motor block to determine a safe, reliable test dose for epidural analgesia. *Br J Anaesth* 2002;89:442–445.
  95. Hanson B, Matouskova-Hanson A: Continuous epidural analgesia for vaginal delivery in Sweden. *Acta Anaesthesiol Scand* 1985;29:712–715.
  96. Lamont RF, Pinney D, Rodgers P, et al: Continuous versus intermittent epidural analgesia. *Anaesthesia* 1989;44:893–896.
  97. Abrahams M, Higgins P, Whyte P, et al: Intact proprioception and control of labour pain during epidural analgesia. *Acta Anaesthesiol Scand* 1999;43:46–50.
  98. Parry M, Fernando R, Bawa G, et al: Dorsal column function after epidural and spinal blockade: Implications for the safety of walking following low-dose regional analgesia for labour. *Anaesthesia* 1998;53:382–403.
  99. Breen TW, Shapiro T, Glass B, et al: Epidural anesthesia for labor in an ambulatory patient. *Anesth Analg* 1993;77:919–924.
  100. Vallejo M, Firestone L, Mandell G, et al: Effect of epidural analgesia with ambulation on labor duration. *Anesthesiology* 2001;95:857–861.
  101. Melzack R, Belanger E, Lacroix R: Labor pain: Effect of maternal position on front and back pain. *J Pain Symptom Manage* 1991;6:476–480.
  102. Russell R: Assessment of motor blockade during epidural analgesia in labour. *Int J Obstet Anesth* 1992;1:230–234.
  103. Murphy JD, Henderson K, Bowden MI, Lewis M, Cooper GM: Bupivacaine versus bupivacaine plus fentanyl for epidural analgesia: Effect on maternal satisfaction. *Br Med J* 1991;302:564–567.
  104. Crawford JS: The stages and phases of labour: An outworn nomenclature that invites hazard. *Lancet* 1983;2:271–272.
  105. Cunningham F, Leveno K, Bloom S, Hauth J, Gilstrap L, Wenstrom K: *Williams Obstetrics*, 22nd ed. New York, McGraw-Hill, 2005.
  106. Elkington KW: At the water's edge: Where obstetrics and anesthesia meet. *Obstet Gynecol* 1991;77:304–308.
  107. Capogna G, Camorcia M: Epidural analgesia for childbirth: Effects of newer techniques on neonatal outcome. *Pediatr Drugs* 2004;6:375–386.
  108. Kangas-Saarela T, Jouppila R, Alahuhta S, et al: The effect of lumbar epidural analgesia on the neurobehavioural responses of newborn infants. *Acta Anaesthesiol Scand* 1989;33:320–325.
  109. Jones G, Paul DL, Elton RA, et al: Comparison of bupivacaine and bupivacaine with fentanyl in continuous extradural analgesia during labour. *Br J Anaesth* 1989;63:254–259.
  110. Halpern S, Levine T, Wilson D, et al: Effect of labor analgesia on breastfeeding success. *Birth* 1999;26:83–88.
  111. Crawford JS: A prospective study of 200 consecutive twin deliveries. *Anaesth* 1987;42:33–43.
  112. Rolbin S, Tey S, Ananthanarayan C: Anaesthesia for preterm labour and delivery. *Curr Opin Anaesthesiol* 1992;5:360–365.
  113. Van Zundert A, Vaes L, Soetens M, et al: Are breech deliveries an indication for lumbar epidural analgesia? *Anesth Analg* 1991;72:399–403.
  114. Writer WDR: The pregnant diabetic. In: Hood DD (ed): *Anesthesia in Obstetrics and Gynecology*.

- Philadelphia: JB Lippincott, 1989; pp. 69–89.
115. Johnson C, Oriol N: The role of epidural anesthesia in trial of labor. *Reg Anesth* 1990;15:304–308.
  116. Hood DD, Dewan DM: Anesthetic and obstetric outcome in morbidly obese parturients. *Anesthesiology* 1993;79:1210–1218.
  117. Mangano DT: Anesthesia for the pregnant cardiac patient. In: Shnider SM, Levinson G (eds): *Anesthesia for Obstetrics*, 3rd ed. Baltimore: Williams and Wilkins, 1993; pp. 485–523.
  118. Jouppila P, Jouppila R, Hollmen A, et al: Lumbar epidural analgesia to improve intervillous blood flow during labor in severe preeclampsia. *Obstet Gynecol* 1982;59:158–161.
  119. Crawford JS: Epidural analgesia for patients with chronic neurological disease. *Anesth Analg* 1983;62:620–621.
  120. Crosby ET, Halpern SH, Rolbin SH: Epidural anaesthesia for caesarean section in patients with active recurrent genital herpes simplex infections: A retrospective review. *Can J Anaesth* 1989;36:701–704.
  121. Gershon R, Manning-Williams D: Anesthesia and the HIV-infected parturient: A retrospective study. *Int J Obstet Anesth* 1997;6:76–81.
  122. Bader AM, Gilbertson L, Kirz L, et al: Regional anesthesia in women with chorioamnionitis. *Reg Anesth* 1992;17:84–86.
  123. Cheek TG, Banner RN: Orthopedic/neurologic diseases. In: Hood DD (ed): *Anesthesia in Obstetrics and Gynecology*. Philadelphia: JB Lippincott, 1989; pp. 112–129.
  124. Wuitchik M, Bakal D, Lipshitz J: Relationships between pain, cognitive activity and epidural analgesia during labor. *Pain* 1990;41:125–132.
  125. Vahratian A, Zhang J, Hasting J, et al: The effect of early epidural versus early intravenous analgesia use on labor progression: A natural experiment. *Am J Obstet Gynecol* 2004;191:259–265.
  126. Rogers R, Gilson G, Miller A, et al: Active management of labor: Does it make a difference? *Am J Obstet Gynecol* 1997;177:599–605.
  127. Ramanathan J, Coleman P, Sibai B: Anesthetic modification of hemodynamic and neuroendocrine stress responses to cesarean delivery in women with severe preeclampsia. *Anesth Analg* 1991;73:772–779.
  128. Lindheimer MD, Katz AI: Hypertension in pregnancy. *N Engl J Med* 1985;313:675–680.
  129. Pritchard JA, MacDonald PC, Gant NF: *Williams Obstetrics*, 17th ed. Norwalk, CT: Appleton-Century-Crofts, 1985; p. 551.
  130. Phillips KC, Thomas TA: Second stage of labour with or without extradural analgesia. *Anaesthesia* 1983;38:972–976.
  131. Chestnut DH, Laszewski LJ, Pollack KL, et al: Continuous epidural infusion of 0.0625% bupivacaine-0.0002% fentanyl during the second stage of labor. *Anesthesiology* 1990;72:613–618.
  132. Olofsson C, Ekblom A, Ekman-Ordeberg G, et al: Obstetric outcome following epidural analgesia with bupivacaine-adrenaline 0.25% or bupivacaine 0.125% with sufentanil – a prospective randomized controlled study in 1000 parturients. *Acta Anaesthesiol Scand* 1998;42:284–292.
  133. Comparative Obstetric Mobile Epidural Trial (COMET) Study Group UK: Randomized controlled trial comparing traditional with two “mobile” epidural techniques. *Anesthesiology* 2002;97:1567–1575.
  134. Gaiser R: Labor epidural and outcome. *Best Pract Res Clin Anesthesiol* 2005;19:1–16.
  135. Wong C, Scavone B, Peaceman A, et al: The risk of cesarean delivery with neuraxial analgesia given early. *N Engl J Med* 2005;352:655–665.
  136. Zhang J, Klebanoff M, DerSimonian R: Epidural analgesia in association with duration of labor and mode of delivery: A quantitative review. *Am J Obstet Gynecol* 1999;180:970–977.
  137. Sharma S, McIntire D, Wiley J, et al: Labor analgesia and cesarean delivery. *Anesthesiology* 2004;100:142–148.
  138. Maresh M, Choong KH, Beard RW: Delayed pushing with lumbar epidural analgesia in labour. *Br J Obstet Gynaecol* 1983;90:623–627.
  139. American College of Obstetricians and Gynecologists Practice Bulletin Number 17, June 2000. Operation Vaginal Delivery. Washington, DC: The American College of Obstetricians and Gynecologists.
  140. Roberts C, Torvaldsen S, Cameron C, et al: Delayed versus early pushing in women with epidural analgesia: A systematic review and meta-analysis. *Int J Obstet Gynecol* 2004;111:1333–340.
  141. Plunkett B, Lin A, Wong C, et al: Management of the second stage of labor in nulliparas with continuous epidural analgesia. *Obstet Gynecol* 2003;102:109–114.
  142. Vause S, Congdon H, Thornton J: Immediate and delayed pushing in the second stage of labour for

- nulliparous women with epidural analgesia: A randomized controlled trial. *Br J Obstet Gynaecol* 1998;105:186–188.
143. Sia A, Chong J: Epidural 0.2% ropivacaine for labour analgesia: Parturient-controlled or continuous infusion? *Anaesth Intensive Care* 1999;27:154–158.
  144. Collis R, Plaat F, Morgan B: Comparison of midwife top-ups, continuous infusion and patient-controlled epidural analgesia for maintaining mobility after a low-dose combined spinal-epidural. *Br J Anaesth* 1999;82:233–236.
  145. Van der Vyver M, Halpern S, Joseph G: Patient-controlled epidural analgesia versus continuous infusion for labour analgesia: A meta-analysis. *Br J Anaesth* 2002;89:459–465.
  146. Eriksson S, Gentile C, Olofsson C: PCEA compared to continuous epidural infusion in an ultra-low-dose regimen for labor pain relief: A randomized study. *Acta Anaesthesiol Scand* 2003;47:1085–1090.
  147. Cohen SE, Cherry CM, Holbrook RH, et al: Intrathecal sufentanil for labor analgesia – sensory changes, side effects, and fetal heart rate changes. *Anesthesiology* 1993;77:1155–1160.
  148. Camann WR, Mintzer BH, Denney RA, et al: Intrathecal sufentanil for labor analgesia: Effects of added epinephrine. *Anesthesiology* 1993;78:870–874.
  149. Grieco WM, Norris MC, Leighton BL, et al: Intrathecal sufentanil labor analgesia: The effects of adding morphine or epinephrine. *Anesth Analg* 1993;77:1149–1154.
  150. Caldwell LE, Rosen MA, Shnider SM: Subarachnoid morphine and fentanyl for labor analgesia. *Reg Anesth* 1994;19:2–8.
  151. Stocks G, Hallworth S, Fernando R, et al: Minimum local analgesic dose of intrathecal bupivacaine in labor and the effect of intrathecal fentanyl. *Anesthesiology* 2001;94:593–598.
  152. Vitanen H, Porthan L, Vitanen M, et al: Postpartum neurologic symptoms following single-shot spinal block for labour analgesia. *Acta Anaesthesiol Scand* 2005;49:1015–1022.
  153. Chadwick HS, Posner K, Caplan RA, Ward RJ, Cheney FW: A comparison of obstetric and nonobstetric anesthesia malpractice claims. *Anesthesiology* 1991;74:242–249.
  154. Van de Velde M, Teunkens A, Hanssens M, et al: Intrathecal sufentanil and fetal heart rate abnormalities: A double-blind, double placebo-controlled trial comparing two forms of combined spinal epidural analgesia with epidural analgesia in labor. *Anesth Analg* 2004;98:1153–1159.
  155. Van de Velde: Combined spinal epidural analgesia for labor and delivery: A review. *Acta Anaesth Belg* 2004;55:17–27.
  156. Albright G, Forster R: The safety and efficacy of combined spinal and epidural analgesia/anesthesia (6,002 blocks) in a community hospital. *Reg Anesth Pain Med* 1999;24:117–125.
  157. DeBalli P, Breen T: Intrathecal opioids for combined spinal-epidural analgesia during labour. *CNS Drugs* 2003;17:889–904.
  158. Russell R: Combined spinal epidural analgesia is the preferred technique for labour analgesia. *Acta Anaesth Belg* 2002;53:331–334.
  159. D'Angelo R: New techniques for labor analgesia: PCEA and CSE. *Clin Obstet Gynecol* 2003;46:623–632.
  160. Nickells J, Vaughan D, Lillywhite N, et al: Speed of onset of regional analgesia in labour: A comparison of the epidural and spinal routes. *Anaesthesia* 2000;55:17–20.
  161. Hepner D, Gaiser R, Cheek T, et al: Comparison of combined spinal-epidural and low dose epidural for labour analgesia. *Can J Anesth* 2000;47:232–236.
  162. Nageotte M, Larson D, Rumney P, et al: Epidural analgesia compared with combined spinal-epidural analgesia during labor in nulliparous Women. *N Engl J Med* 1997;337:1715–1719.
  163. Vernis L, Duale C, Storme B, et al: Perispinal analgesia for labour followed by patient-controlled infusion with bupivacaine and sufentanil: Combined spinal-epidural vs. epidural analgesia alone. *Eur J Anaesthesiol* 2004;21:186–192.
  164. Greenhalgh C: Respiratory arrest in a parturient following intrathecal injection of sufentanil and bupivacaine. *Anaesthesia* 1996;51:173–175.
  165. Pan P, Moore C, Ross V: Severe maternal bradycardia and asystole after combined spinal-epidural labor analgesia in a morbidly obese parturient. *J Clin Anesth* 2004;16:461–464.
  166. Mandal N, Surapaneni S: Regional anaesthesia in pre-eclampsia. *Drugs* 2004;64:223–236.
  167. Horlocker T, Wedel D, Benzon H, et al: Regional anesthesia in the anticoagulated patient: Defining the risks (The Second ASRA Consensus Conference on Neuraxial Anesthesia and Anticoagulation). *Reg Anesth Pain Med* 2003;28:172–197.

168. Crowhurst J, Plaat F: Why mothers die – Report on confidential enquiries into maternal deaths in the United Kingdom 1994–96. *Anaesthesia* 1999;54:207–209.
169. Feinstein SJ, Lodeiro JG, Vintzileos AM, Campbell WA, Montgomery JT, Nochimson DJ: Sinusoidal fetal heart rate pattern after administration of nalbuphine hydrochloride: A case report. *Am J Obstet Gynecol* 1986;154:159–160.
170. Hatjis CG, Meis PJ: Sinusoidal fetal heart rate pattern associated with butorphanol administration. *Obstet Gynecol* 1986;67:377–380.
171. Rayburn W, Leuschen P, Earl R, Woods M, Lordovic M, Gaston-Johansson F: Intravenous meperidine during labor: A randomized comparison between nursing and patient-controlled administration. *Obstet Gynecol* 1989;74:702–705.
172. Kleiman SJ, Wiesel S, Tessler MJ: Patient-controlled analgesia (PCA) using fentanyl in a parturient with a platelet function abnormality. *Can J Anaesth* 1991;38:489–491.
173. Podlas J, Breland BD: Patient-controlled analgesia with nalbuphine during labor. *Obstet Gynecol* 1987;70:202–204.
174. Goins JR: Experience with mepivacaine paracervical block in an obstetric private practice. *Am J Obstet Gynecol* 1992;167:342–345.
175. Baxi LV, Petrie RH, James LS: Human fetal oxygenation following paracervical block. *Am J Obstet Gynecol* 1979;135:1109–1112.
176. Weiss RR, Halevy S, Almonte RO, Gunderson K, Hinsvark ON, O'Brien JE: Comparison of lidocaine and 2-chloroprocaine in paracervical block: clinical effects and drug concentrations in mother and child. *Anesth Analg* 1983;62:168–173.
177. Kangas-Saarela T, Jouppila R, Puolakka J, Jouppila P, Hollmen A, Puukka M: The effect of bupivacaine paracervical block on the neurobehavioural responses of newborn infants. *Acta Anaesthesiol Scand* 1988;32:566–570.
178. Bonica JJ: Principles and Practice of Obstetric Analgesia and Anesthesia. Philadelphia: F. A. Davis Company, 1967; pp. 487–506.
179. American Society of Anesthesiologists: Basic Standards for Preanesthesia Care (October, 1987); Standards for Basic Anesthetic Monitoring (October, 1986, amended 1993); Standards for Postanesthesia Care (October, 1988, amended 1992); and Guidelines for Patient Care in Anesthesiology (October, 1967, amended 1985).
180. Evans CM, Murphy JF, Gray OP, Rosen M: Epidural versus general anaesthesia for elective caesarean section: Effect on Apgar score and acid–base status of the newborn. *Anaesthesia* 1989;44:778–782.
181. Brizgys RV, Dailey PA, Shnider SM, Kotelko DM, Levinson G: The incidence and neonatal effects of maternal hypotension during epidural anesthesia for cesarean section. *Anesthesiology* 1987;67:782–786.
182. Dewan DM: Anesthesia for preterm delivery, breech presentation, and multiple gestation. *Clin Obstet Gynecol* 1987;30:566–578.
183. Portnoy D, Vadhera R: Mechanisms and management of an incomplete epidural block for cesarean section. *Anesthesiol Clin North Am* 2003;21:39–57.
184. deRosayro AM, Hahrwold ML, Hill AB: Cardiovascular effects of epidural epinephrine in the pregnant sheep. *Reg Anesth* 1981;6:4–7.
185. Albright GA, Jouppila R, Hollmen AI, Jouppila P, Vierola H, Kiovula A: Epinephrine does not alter human intervillous blood flow during epidural anesthesia. *Anesthesiology* 1981;54:131–135.
186. DiFazio CA, Carron H, Grosslight KR, Moscicki JC, Bolding WR, Johns RA: Comparison of pH-adjusted lidocaine solutions for epidural anesthesia. *Anesth Analg* 1986;65:760–764.
187. Ackerman WE, Juneja MM, Colclough GW, Kaczorowski DM: Epidural fentanyl significantly decreases nausea and vomiting during uterine manipulation in awake patients undergoing cesarean section. *Anesthesiology* 1988;69:A679.
188. Preston PG, Rosen MA, Hughes SC, et al: Epidural anesthesia with fentanyl and lidocaine for cesarean section: Maternal effects and neonatal outcome. *Anesthesiology* 1988;68:938–943.
189. Benlabed M, Dreizzen E, Ecoffey C, Escourrou P, Migdal M, Gaultier C: Neonatal patterns of breathing after cesarean section with or without epidural fentanyl. *Anesthesiology* 1990;73:1110–1113.
190. The American College of Obstetricians and Gynecologists: Anesthesia for emergency deliveries. ACOG Committee Opinion #104. Washington, DC: American College of Obstetricians and Gynecologists, 1992.
191. Crowhurst JA: General anaesthesia in obstetrics. *Curr Opin Anaesthesiol* 1991;4:349–355.
192. Ramanathan J, Ricca DM, Sibai BM, Angel JJ:

- Epidural vs general anesthesia in fetal distress with various abnormal fetal heart rate patterns. *Anesth Analg* 1988;67:S180.
193. Jenkins J, Khan M: Anaesthesia for caesarean section: A survey in a UK region from 1992 to 2002. *Anaesthesia* 2003;58:1101–1118.
  194. Abboud TK, Nagappla S, Murakawa K, et al: Comparison of the effects of regional and general anesthesia for cesarean section on neonatal neurologic and adaptive scores. *Anesth Analg* 1985;64:996–1000.
  195. Norris MC: Hypotension during spinal anesthesia for cesarean section: Does it affect neonatal outcome? *Reg Anesth* 1987;12:191–194.
  196. Datta S, Kitzmiller JL, Naulty JS, Ostheimer GW, Weiss JB: Acid–base status of diabetic mothers and the infant following spinal anesthesia for cesarean section. *Anesth Analg* 1982;61:662–665.
  197. Crawford JS: There is only a limited place for spinals in obstetrics. *Acta Anaesthesiol Belgica* 1988;39:181–184.
  198. Vegfors M, Cederholm I, Gupta A, et al: Spinal or epidural anaesthesia for elective caesarean section? *Int J Obstet Anesth* 1992;1:141–144.
  199. Abouleish EI: Epinephrine improves the quality of spinal hyperbaric bupivacaine for cesarean section. *Anesth Analg* 1987;66:395–400.
  200. Hunt CO, Naulty JS, Bader AM, et al: Perioperative analgesia with subarachnoid fentanyl-bupivacaine for cesarean delivery. *Anesthesiology* 1989;71:535–540.
  201. Shende D, Cooper G, Bowden M: The influence of intrathecal fentanyl on the characteristics of subarachnoid block for caesarean section. *Anaesthesia* 1998;53:702–710.
  202. Manullang T, Viscomi C, Pace N: Intrathecal fentanyl is superior to intravenous ondansetron for the prevention of perioperative nausea during cesarean delivery with spinal anesthesia. *Anesth Analg* 2000;90:1162–1166.
  203. Marx GF, Luyckx WM, Cohen S: Fetal-neonatal status following cesarean section for fetal distress. *Br J Anaesth* 1984;56:1009–1013.
  204. Hood D: Spinal anesthesia is the preferred technique for cesarean section in severe preeclampsia: Proponent position. *Acta Anaesthe Belg* 2002;53:305–310.
  205. Rawal N, Holmstrom B, Crowhurst J, et al: The combined spinal-epidural technique. *Anesthesiol Clin North Am* 2000;18:267–295.
  206. Dresner M, Freeman J: Anaesthesia for caesarean section. *Best Pract Res Clin Obstet Gynaecol* 2001;15:127–143.
  207. Marx GF, Schwalbe SS: Obstetric anesthesia and analgesia. *Curr Opin Obstet Gynecol* 1989;1:156–164.
  208. Morgan M: Anaesthetic contribution to maternal mortality. *Br J Anaesth* 1987;59:842–855.
  209. Hawkins J: Anesthesia-related maternal mortality. *Clin Obstet Gynecol* 2003;46:679–687.
  210. McClur J, Cooper G: Fifty years of confidential enquiries into maternal deaths in the United Kingdom: Should anaesthesia celebrate or not? *Int J Obstet Anesth* 2004;87–89.
  211. Munnur U, Suresh M: Airway problems in pregnancy. *Crit Care Clin* 2004;20:617–642.
  212. Morgan BM, Magni V, Goroszeniuk T: Anaesthesia for emergency caesarean section. *Br J Obstet Gynaecol* 1990;97:420–424.
  213. Goudsouzian N: Anesthesia for cesarean section and acid aspiration prophylaxis. *Anesth Analg* 1999;89:533.
  214. Ley SJ, Scheller J, Jones BR, Slotnick N: Intrauterine pressure during administration of nitroglycerin for extraction of retained placenta. *Anesth Rev* 1993;20:95–97.
  215. Peng ATC, Gorman RS, Shulman SM, et al: Intravenous nitroglycerin for uterine relaxation in the postpartum patient with retained placenta. *Anesthesiology* 1989;71:172–173.
  216. DeSimone CA, Norris MC, Leighton BL: Intravenous nitroglycerin aids manual extraction of a retained placenta. *Anesthesiology* 1990;73:787.
  217. Altabef KM, Spencer JT, Zinberg S: Intravenous nitroglycerin for uterine relaxation of an inverted uterus. *Am J Obstet Gynecol* 1992;166:1237–1238.
  218. Mayer DC, Weeks SK: Antepartum uterine relaxation with nitroglycerin at caesarean delivery. *Can J Anaesth* 1992;39:166–169.
  219. Rolbin SH, Hew EM, Bernstein A: Uterine relaxation can be life saving. *Can J Anaesth* 1991;38:939–940.
  220. Rolbin SH: Commentary. *Can J Anaesth* 1992;39:168–169.
  221. Wheeler A, James F, Meis P, et al: Effects of nitroglycerin and nitroprusside on the uterine vasculature of gravid ewes. *Anesthesiology* 1989;52:390–394.

222. Hood D, Dewan D, James F, et al: The use of nitroglycerin in preventing the hypertensive response to tracheal intubation in severe preeclampsia. *Anesthesiology* 1985;63:329–332.
223. Condous G, Arulkumaran S: Medical and conservative surgical management of postpartum hemorrhage. *J Obstet Gynaecol Can* 2003;25:931–936.
224. Tamizian O, Arulkumaran S: The surgical management of postpartum haemorrhage. *Best Pract Res Clin Obstet Gynaecol* 2002;16:81–98.
225. Parekh N, Husanani S, Russell I: Caesarean section for placenta praevia: A retrospective study of anaesthetic management. *Br J Anaesth* 2000;84:725–730.
226. Shibata K, Yamamoto Y, Murakami S: Effects of epidural anesthesia on cardiovascular responses and survival in experimental hemorrhagic shock in dogs. *Anesthesiology* 1989;71:953–959.
227. Clutton-Brock T: Maternal deaths from anaesthesia. An extract from *Why Mothers Die 2000–2002: The Confidential Enquiries into Maternal Deaths in the United Kingdom*. *Br J Anaesth* 2005;94:424–429.
228. Cooper G, McClure J: Maternal deaths from anaesthesia. An extract from *Why Mothers Die 2000–2002: The Confidential Enquiries into Maternal Deaths in the United Kingdom*. *Br J Anaesth* 2005;94:417–423.
229. Duncan PG, Pope WDB, Cohen MM, Greer N: The safety of anesthesia and surgery during pregnancy. *Anesthesiology* 1986;64:790–794.
230. Mazze RI, Kallen B: Reproductive outcome after anesthesia and operation during pregnancy: A registry study of 5405 cases. *Am J Obstet Gynecol* 1989;161:1178–1185.
231. Levinson G, Shnider SM, deLorimier AA, Steffenson JL: Effects of maternal hyperventilation on uterine blood flow and fetal oxygenation and acid–base status. *Anesthesiology* 1974;40:340–347.
232. Marx GF: Cardiopulmonary resuscitation of late-pregnant women. *Anesthesiology* 1982;56:156.
233. Doan-Wiggins L: Cardiopulmonary resuscitation during pregnancy. In: Benrubi GI (ed): *Obstetric and Gynecologic Emergencies*. Philadelphia: JB Lippincott, 1994; pp. 77–85.
234. Cade L, Ashley J: Towards optimal analgesia after caesarean section: Comparison of epidural and intravenous patient-controlled opioid analgesia. *Naesth Intens Care* 1993;21:696–699.
235. Eisenach J, Grice S, Dewan D: Patient-controlled analgesia following caesarean section: A comparison with epidural and intramuscular narcotics. *Anesthesiology* 1988;68:444–448.
236. Youngstrom P, Cowan RI, Sutheimer C, Eastwood DW, Yu JCM: Pain relief and plasma concentrations from epidural and intramuscular morphine in post-caesarean patients. *Anesthesiology* 1982;57:404–409.
237. Yang T, Breen T, Archer D, et al: Comparison of 0.25 mg and 0.1 mg intrathecal morphine for analgesia after caesarean section. *Can J Anaesth* 1999;46:856–860.
238. Pavy T, Paech M, Evans S: The effect of ketorolac on opioid requirement and pain after caesarean delivery. *Anesth Analg* 2001;92:1010–1014.
239. American Academy of Pediatrics Committee on Drugs: The transfer of drugs and other chemicals into human milk. *Pediatrics* 1002;108:776–785.
240. Somrat C, Oranuch K, Ketchada U: Optimal dose of nalbuphine for treatment of intrathecal-morphine induce pruritus after caesarean section. *J Obstet Gynaecol Res* 1999;25:209–213.
241. Palmer C, Nogami W, Van Maren G, et al: Postcaesarean epidural morphine: A dose-response study. *Anesth Analg* 2000;90:887–891.
242. Youngstrom P, Moraes JM, Neves JF, et al: Postcaesarean epidural analgesia: Morphine bolus or fentanyl infusion? *Anesthesiology* 1993;79:A1024.
243. Vercauteren M, Vereecken K, La Malfa M, et al: Cost-effectiveness of analgesia after Caesarean section: A comparison of intrathecal morphine and epidural PCA. *Acta Anaesthesiol Scand* 2002;46:85–89.
244. Youngstrom P: Financial aspects of patient-controlled analgesia – stimulus for development in a changing environment. *Proceedings of the Patient-Controlled Analgesia Symposium, the University of Wales College of Medicine, Naples, FL, 1993*.
245. Levinson G, Shnider SM: Editorial: Obstetric anesthesia coverage – a continuing problem. *Anesthesiology* 1986;65:245–246.
246. Gibbs CP, Krischer J, Peckam BM, et al: Obstetric anesthesia: A national survey. *Anesthesiology* 1986;65:298–306.
247. American Society of Anesthesiologists: *Guidelines for Regional Anesthesia in Obstetrics (October, 1988, amended 1991)*.

248. Downing JW, Brown WU: Standards of care: Managing an obstetric anesthesia service. In: Norris MC (ed): *Obstetric Anesthesia*. Philadelphia: JB Lippincott, 1993; p. 808.
249. Rosaeg OP, Yarnell RW, Lindsay MP: The obstetrical anesthesia assessment clinic: A review of six years' experience. *Can J Anaesth* 1993;40:346–356.
250. Clark SL, Koonings PP, Phelan JP: Placenta previa/accreta and prior cesarean section. *Obstet Gynecol* 1985;66:89–92.
251. Thomas TA: Obstetric anaesthesia and analgesia: Editorial overview. *Cur Opin Anaesthesiol* 1991; 4:325–328.
252. Knapp RM: Legal view of informed consent for anesthesia during labor. *Anesthesiology* 1990; 72:211.
253. Vacanti CJ, Vitez TS: How quality assurance and the peer review process can help your department. In: *Peer Review in Anesthesiology*. Park Ridge, IL: American Society of Anesthesiologists, 1992: 60.
254. Eagle CJ, Pagenkopf D: The cost of an established quality assurance programme: Is it worth it? *Can J Anaesth* 1994;41:813–17.
255. Ishikawa K: Cause and Effect Diagram (CE Diagram). In: Ishikawa K (ed): *Guide to Quality Control*, 2nd (rev. Eng. ed.). Tokyo: Asian Productivity Organization, 1986; pp. 18–29.
256. Youngstrom P, Boyd D, Rhoton F: Computerized quality assurance assessment of obstetric anesthesia care: New outcome indicators. In: Ikeda K, Doi M, Kazama T, et al (eds): *Computing and Monitoring in Anesthesia and Intensive Care*. Tokyo: Springer-Verlag, 1992; pp. 304–305.



## Lucy A. Bayer-Zwirello

*However uncertain we may be of the efficient cause . . . we are taught by long experience, that about the fortieth week of gestation, an effort is made by the uterus to expel its contents; and this effort is called labor.*

William Potts Dewees (1768–1841)  
*Compendious System of Midwifery*  
Philadelphia: Carey and Lea  
Second Edition, 1826, xi, p. 170

### CONDUCT OF NORMAL LABOR

The conduct of labor is a physiologic, social, and medical event in which the practitioner is one of several participants. The clinician's role is to monitor the natural process of parturition while detecting and treating any important deviations from normal. The physician or midwife collates and interprets data from the heart rate/auscultation or electronic monitor record, laboratory data, and the results of various maternal evaluations and examinations. Clinical information from the observations of nurses or other attendants, as well as data directly obtained by evaluation of the progression of labor from examinations and their graphic recording on the partogram, are combined, analyzed, and then discussed. If the course of labor is less than ideal and problems are identified, the clinician evaluates the issue, considers the best response, confers with the family, and initiates an appropriate intervention. The accoucheur must strike a balance between excessive optimism and unremitting pessimism, providing reassurance when possible and support at all times. In sum, the principal birth attendant must be an active participant in the process of labor and not simply an observer.

Obstetric labor management begins when the woman is first admitted. A careful review of the history is performed, and the events leading to admission are discussed. Vital signs are reviewed, and a urine dipstick test is performed. In the initial evaluation, an abdominal examination is conducted, including Leopold's maneuvers (Table 10.1). A pelvic examination follows, and depending on the circumstances, it is either simply a digital or by a sterile speculum.

A digital vaginal examination notes cervical effacement, dilation, and fetal position and station. Cranial positioning – flexion/deflection – is checked, as is the presence or absence of molding or caput succedaneum. An estimate of pelvic capacity is also conducted.

If the history is suspicious for membrane rupture, but uterine activity is neither reported nor apparent, a sterile speculum vaginal examination is in

TABLE 10.1 Leopold's Maneuvers, Modified\*

Procedure	
First maneuver	The operator stands at the patient's side (traditionally the right) and palpates the uterine fundus. Given this manipulation, the fetal size is estimated, the contents of the fundus evaluated, and the lie is determined.
Second maneuver	Using both hands, the surgeon judges the contents of the midportion of the uterus. The fetal back versus small parts can normally be distinguished by kneading the uterus back and forth gently, noting the contour of the fetal body and the increased resistance to digital pressure when the back is palpated.
Third maneuver	The operator grasps the lower uterine segment with the right hand and attempts to move it back and forth. This helps to judge engagement and to identify the presenting part, establishing the presentation.
Fourth maneuver	The operator turns toward the patient's feet and passes his/her hands longitudinally along the presenting part, noting whether the fingers diverge immediately suprapubically (indicating engagement) or dip into the pelvis, displacing the presenting part (suggesting non-engagement). Lateral masses (occiput, in a face presentation, etc.) are also palpable during this examination.

\*If abnormalities are suspected, a bedside real-time ultrasound scan is performed for verification and additional data, at the clinician's discretion.

order. The cervix should be visualized, the condition of the membranes judged, and culture samples obtained, which might or might not be submitted with test samples to confirm or refute amniorrhexis. If membrane rupture is suspect and labor has not begun, digital examination should not be performed, because it increases the risk for infection while not providing information that can be obtained more safely by visual inspection.

Many obstetricians or midwives are now comfortable using ultrasound in the labor and delivery suite to verify the clinical examination. If membrane rupture has occurred without labor, or if fetal malpresentation is suspected, bedside real-time ultrasonic examination is in order. In some parturients in whom Leopold's maneuvers are difficult or inadequate or the presentation is high, an ultrasound scan easily confirms the presentation. In experienced hands, ultrasound scans can also detect more complex malpresentation such as face, brow, or occiput posterior. These women are candidates for close observation; their labors might require oxytocin augmentation or eventually result in cesarean delivery.

Management of premature rupture of membranes (PROM) depends on the gestational age. If the PROM is preterm ( $\leq 37$  weeks), the volume of the remaining fluid is estimated and to evaluate fetal status a biophysical profile (BPP) is performed,

including a non-stress test (NST). The lag time until the onset of labor can be hours or even days; therefore a baseline study of fetal condition is prudent. When membrane status is uncertain, simple reexamination by ultrasound scan or by direct repeat pelvic examination might be necessary. Transvaginal ultrasound examination can also assist in the diagnosis of PROM. Such studies apparently do not increase the risk of infection. In selected cases, an amniocentesis with indigo carmine dye installation to test for fluid leakage and laboratory analysis of an aspirated amniotic fluid sample can be considered. If PROM has occurred at term ( $>37$  weeks), the methods of diagnosis are similar, but management is generally more active. Spontaneous labor commences within 24 hours in 80% cases, and a further 10% of women deliver spontaneously within 48 hours. A minority of term PROM cases will not go into labor until  $>72$  hours. As there is no advantage to expectant management at term, an oxytocin or prostaglandin induction can be performed without delay [1].

Particular attention to the diagnosis of labor is required. In this evaluation, the frequency, persistence, and strength of contractions; the presence or absence of bloody show; and membrane status are important features. With allowance for parity, recurrent uterine contractions with cervical dilatation beyond 2 cm to 3 cm or contractions accompanying

documented membrane rupture are the criteria frequently used to diagnose labor. The most important feature of normal, active labor is, however, progressive cervical dilatation. Thus, serial examinations can be required to establish the correct diagnosis, unless the woman presents initially with advanced dilation and ruptured membranes [2].

With the sum of these clinical data – direct observation, abdominal and pelvic examination, notation of the uterine contraction pattern, and the heart rate pattern – the experienced accoucheur rapidly develops a clinical “snapshot” of the labor. Such clinical impressions, combined with the results of fetal monitoring, serial pelvic examinations, and observation of how the mother is tolerating her contractions, are the basis for clinical decisions concerning any required actions. As long as the labor progresses normally, the likelihood of significant difficulty is minimal. When progress is desultory or becomes arrested in either dilation or descent, the potential morbidity for both mother and fetus dramatically increases, and often the clinician must act.

### **Monitoring and Analgesia**

During labor, the fetal heart rate is monitored either by electronic means or by intermittent auscultation, following the protocol of each institution. The author has usually avoided the administration of epidural anesthesia until the active phase of labor is established; however, this delay might not be necessary. Recent data suggest that modern low-dose mixed epidural anesthetics, combining a narcotic and an anesthetic agent in very low concentration, administered by continuous-pump infusion epidurals do not significantly prolong labor [3,4]. Thus, early use of epidural analgesia by a combined-agent continuous-infusion technique is appropriate in markedly uncomfortable women even in latent phase labor. When and if an epidural agent is administered, its level and intensity should be gauged to provide labor analgesia and not surgical anesthesia. *Anesthesia* (as opposed to *analgesia*) is unnecessary during labor and seriously interferes with normal second-stage progress (see Chapter 9 Obstetric Anesthesia). The goal of epidural analgesia should be explained to the woman clearly, so that surgical anesthesia is neither expected nor requested. The advantage of the low-dose continuous-pump infusion technique is less motor dysfunction and reduced interference with the second stage of labor

[5]. In some institutions, a continuous low-dose pump infusion with self-administered boluses for reinforcement is now being tried with success. In these management plans, the patient is started on a low-dose continuous infusion, and she can initiate boluses if there is pain breakthrough [6]. This self-control is popular both with parturients and the nursing staff.

Occasionally it is appropriate to administer parenteral doses of an analgesic such as nalbuphine hydrochloride (Nubain). A popular method is to administer 2 mg every 20 minutes until pain relief is achieved (maximal dose of 10 mg) or to simply commence with 10 mg. Narcotics are administered with an antiemetic, which can improve analgesia by adding sedation [7]. Owing to their potential adverse effects on the fetus, intermittent injections of parenteral narcotics should be administered sparingly. (See Chapter 9, Obstetric Anesthesia.)

Other parturients do extremely well with prepared childbirth techniques. Such techniques are acceptable for women who are motivated to labor without the administration of chemical analgesics.

### **Stages of Labor**

For purposes of discussion, labor is arbitrarily divided into stages. Although the process of normal labor is dynamic and continuous, the division into stages is a useful method of focusing attention on the physiology of the process and its usual progression. Viewing labor as a continuous process also emphasizes its dynamic features which are best evaluated by graphic representation as a partogram.

The period from the onset of contractions through complete dilation of the cervix is termed the *first stage* of labor. The *second stage* extends from complete dilation of the cervix through delivery of the fetus. The *third stage* of labor is the period from the delivery of the fetus to the delivery of the placenta. The 2-hour period immediately following the delivery of the placenta is less formally termed the *fourth stage*. Normative values for the first stage are recorded in Table 10.2 with a synopsis of common abnormalities in Table 10.3.

### **Station**

A brief discussion of station is important for an understanding of both the older as well as recent

TABLE 10.2 Characteristics of Labor in Both Nulliparous and Multiparous Patients\*

Normative Values	Nulliparas		Multiparas	
	All Patients	Ideal Labor	All Patients	Ideal Labor
Duration of first stage (hr)				
Latent phase	6.4 ( $\pm 5.1$ )	6.1 ( $\pm 4.0$ )	4.8 ( $\pm 4.9$ )	4.5 ( $\pm 4.2$ )
Active phase	4.6 ( $\pm 3.6$ )	3.4 ( $\pm 1.5$ )	2.4 ( $\pm 2.2$ )	2.1 ( $\pm 2.0$ )
Total	11.0 ( $\pm 8.7$ )	9.5 ( $\pm 5.5$ )	7.2 ( $\pm 7.1$ )	6.6 ( $\pm 6.2$ )
Maximal rate of descent (cm/hr)	3.3 ( $\pm 2.3$ )	3.6 ( $\pm 1.9$ )	6.6 ( $\pm 4.0$ )	7.0 ( $\pm 3.2$ )
Duration of second stage (hr)	1.1 ( $\pm 0.8$ )	0.76 ( $\pm 0.5$ )	0.39 ( $\pm 0.3$ )	0.32 ( $\pm 0.3$ )

\*All values given are  $\pm$ SD.

Data derived from Friedman EA: Labor: Clinical Evaluation and Management, 2nd ed. New York: Appleton-Century-Crofts, 1978.

publications in the medical literature concerning labor and descent. Many practitioners originally were taught to report station in thirds, to describe the distance from the plane of the ischial spines to the presenting part. In 1989, the American College of Obstetricians and Gynecologists (ACOG) instituted a technique of reporting station in centimeters [16,17]. (The correlation between the

centimeter versus the classic method is indicated in Table 10.4. See also Figure 10.1.) The intention of this system was to standardize nomenclature and provide a more reproducible terminology for reporting instrumental procedures. The greatest problem is in estimating station in centimeters by palpation alone from the imaginary plane of the ischial spines. In communication among

TABLE 10.3 Abnormal Labor Patterns, Diagnostic Criteria, and Potential Treatment\*

	Diagnostic Criterion		Treatment and Comments
	Nulligravida	Multipara	
Disorders of dilation:			
Prolonged latent phase	>20 hr	>14 hr	Rest (medicated or unmedicated); oxytocin infusion
Protracted active phase	$\leq 1.2$ cm/hr	$\leq 1.5$ cm/hr	Oxytocin, if contractions are inadequate, and disproportion and malpresentation are both excluded by abdominal-pelvic examination <sup>†</sup>
Arrest of dilation	>2 hr	>2 hr	Oxytocin, if contractions are inadequate and disproportion and malpresentation are both excluded by abdominal-pelvic examination; otherwise cesarean delivery
Disorders of descent:			
Protracted descent	$\leq 1.0$ cm/hr	$\leq 2.0$ cm/hr	Forceps or vacuum extraction, if disproportion is excluded and the presenting part is low; otherwise cesarean delivery, failure of descent. In selected patients with epidural anesthesia, oxytocin administration <sup>‡</sup>
Arrest of descent	>1 hr	>1 hr	
Failure of descent			

\*See text and Figs. 10.1 and 10.2 for details.

<sup>†</sup>Oxytocin stimulation in a protracted active phase might not prove successful [74].

<sup>‡</sup>Slow second-stage progress is often related to use of epidural anesthesia and can herald outlet/shoulder dystocia; thus, instrumental delivery must be used with circumspection in this setting, especially if the fetus is believed to be large.

TABLE 10.4 Estimation of Station of the Presenting Part: Comparison of Methods\*

Classic Three-station Scale	ACOG Centimeter Scale	Position of Bony Presenting Part
-3	-5	Pelvic inlet
-2	-4	
-1	-3	
0	0	Ischial spines (engagement)
+1	+1	
+2	+3	On the perineum
+3	+5	

\*Station is estimated by palpation of the bony segment of the presenting part during a vaginal examination and determining the distance from the plane of the ischial spines. See text for details.

Modified from Rosen MG: Management of Labor. New York: Elsevier, 1990.

clinicians, and in the analysis of the medical literature, it must be kept clear which system is used for reporting. Adhering to recent convention, this textbook reports station by two numbers (e.g., +2/5 cm). The first number indicates the positive or negative station in centimeters; the second number reminds the reader that it is the five-centimeter scale that is being used. (See Chapter 17, Instrumental Delivery, for additional discussion.)

With the onset of regular uterine contractions, there is initially minimal change in cervical dilatation, despite frequent and sometimes even strong contractions. During this time, progressive effacement normally occurs. At approximately 3 cm to 4 cm of dilatation, a more rapid rate of cervical change normally develops [8,9]. This initial slow dilatation or preparatory phase is termed the *latent phase of labor*, whereas the interval of more rapid dilatation is termed the *active phase of labor*. The duration of these phases depends on both obstetric management and parity. Latent phase or prodromal labor can last up to 20 to 24 hours in a nullipara but is usually shorter for the multipara. In some respect, the latent phase is a "retrospective" phase, established by review of the partogram once the active phase is identified. Otherwise, if the latent phase does not progress into active phase, this process would be

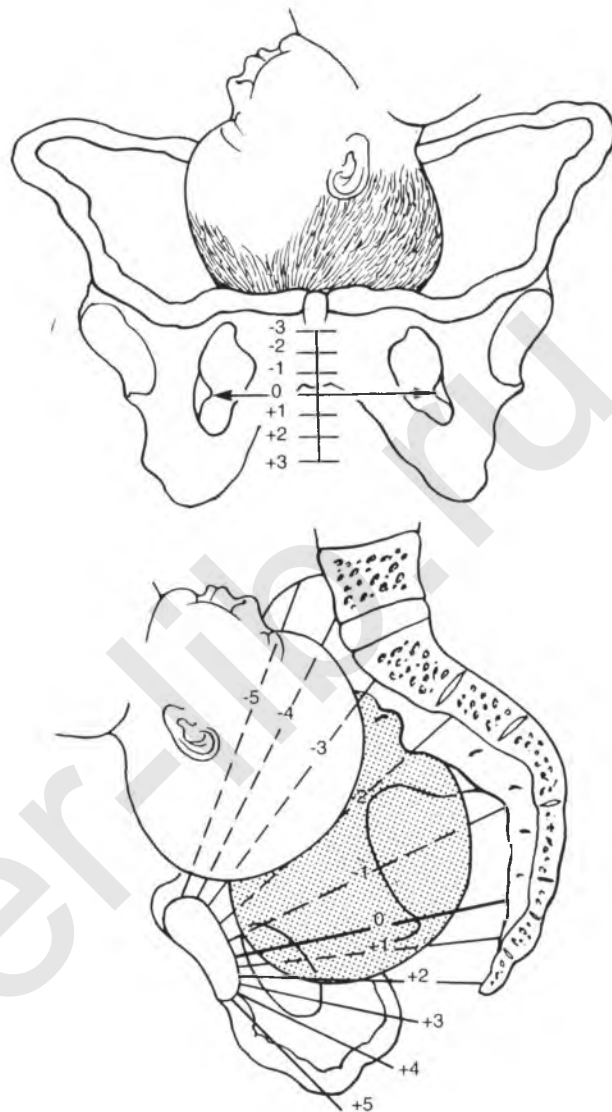


FIGURE 10.1. Estimation of station by different techniques: traditional three station system (top); current ACOG centimeter system (bottom). See text for details.

deemed *false labor*. In the active phase of labor, the rate of progress in terms of cervical dilatation is a function of parity [10]. Based on classic studies of labor, nulliparas normally dilate at a rate of greater than 1.2 cm/hr. In multiparas, the rate of cervical dilatation is faster, with a rate of 1.5 cm/hr or more (Tables 10.2 and 10.3; Figure 10.2).

This classic analysis of basic labor patterns is not universally accepted. O'Driscoll's concept of the active management of labor removes the latent phase from the labor vocabulary. He believes that this allows for a more controlled environment on

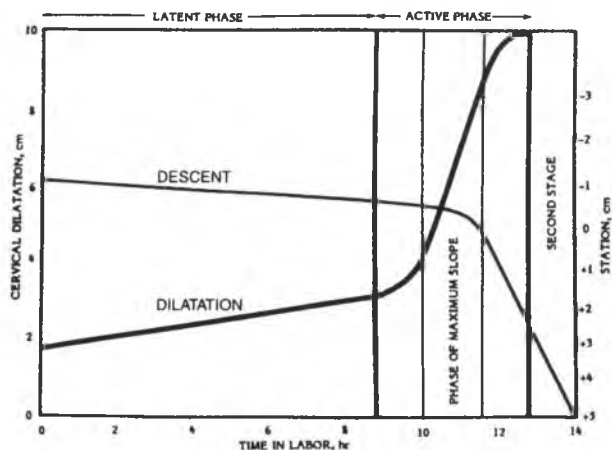


FIGURE 10.2.

*Standard partogram indicating curves of cervical dilatation, presenting part descent, and phases of labor (see text for details).*

labor and delivery [2]. Only patients expected to deliver within 12 hours are admitted to the labor and delivery floor. Patients considered not in labor or prodromal are either sent home or to another floor for observation. Active management of labor is discussed further at the end of this chapter.

### Partogram

Progress in labor is commonly evaluated by plotting cervical dilatation and the descent of the presenting part against time [12–14]. With the resulting labor curve, or *partogram*, arrested or slow progress can be easily detected. Friedman introduced partograms to American obstetric thinking and reported mean and normal ranges for the duration of various divisions or phases of labor based on his statistical analysis of accumulated cases [13]. Partograms remain in common use, although most clinicians do not adhere rigidly to the norms originally established by Friedman. In preparing such graphs, cervical dilatation is plotted on a graph that includes a vertical scale from 0 to 10 cm. Station of the presenting part is plotted from –5 (unengaged and floating), through 0 (engagement), to +5 (crowning). The convention for reporting station is the centimeter scale recommended by ACOG. To create the plot, repeat pelvic examinations at intervals during the active phase of labor, and the cervical dilatation and station are simply recorded against time (see Figure 10.2). Because abnormalities in the progress of labor are common,

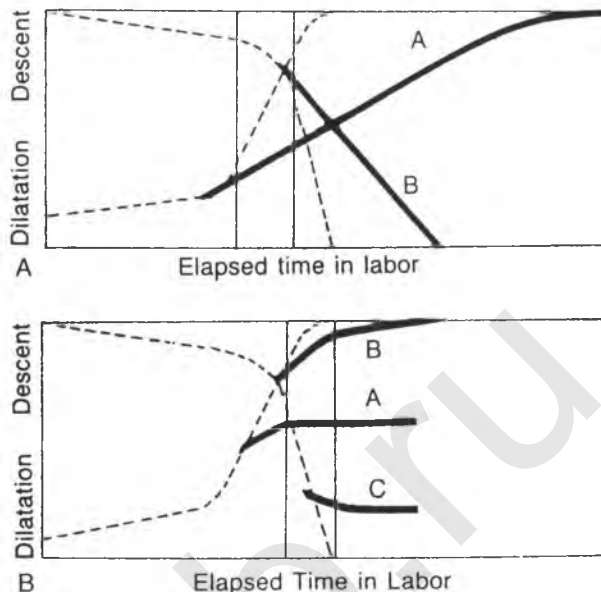


FIGURE 10.3.

*A, Protraction disorders of labor: protracted active-phase dilatation pattern (A); protracted descent pattern (B). Mean normal dilatation and descent curves are shown (broken lines) for comparison. B, Arrest disorders of labor: secondary arrest of dilatation (A); prolonged deceleration phase (B); arrest of descent (C). Normal dilatation and descent curves (broken lines) are illustrated. See text for details. (From Friedman EA: Protraction disorder. In: Friedman EA, Acker DB, Sachs BP (eds): *Obstetrical Decision Making*, Toronto: BC Decker, 1987; pp. 238–240; with permission.)*

it is advisable to routinely follow cases by this simple technique (Figure 10.3, A and B). Several authors and the World Health Organization have recommended changes to what is accepted as a normal partogram, either lengthening the active phase by slowing the rate of dilation or allowing more time to dilate from 4 cm to 7 cm or 8 cm [4]. Regardless of the norms chosen, the use of the partogram to plot labor's progress over time assists in the management of labor.

The other important components of normal progress are *cervical effacement and change in station or descent of the presenting part*. The duration of labor and of the various stages of labor are greatly influenced by the status of the membranes, duration of gestation, strength and frequency of uterine contractions, and medications administered, as well as by other considerations, including fetal size, presentation, and positioning [13,15]. Here, parity again plays a role. Approximately 80% of nulliparas

begin the onset of labor with the presenting part at station 0 to  $-1/5$  cm. In nulliparas, the presenting part normally descends into the pelvis at a relatively constant rate during the active phase of labor, paralleling the progress in cervical dilation. In contrast, in the labor of many multiparas, the presenting part can remain at high station until complete dilation of the cervix occurs. Thereafter, the presenting part descends rapidly, with the second stage of labor often complete in less than 1 hour. In contrast, in nulliparas, the second stage of labor usually begins at station  $+2$  or  $+3/5$  cm, and, under epidural anesthesia, often 2 or more hours are required to complete the delivery.

### The Two-hour Rule

Clinicians have long known the risks of prolonged labor. In the 1920s, such concerns influenced DeLee to recommend limiting the length of the second stage by shortening it by instrumental delivery. Maternal and fetal complications observed in prolonged labors were also part of the original impetus for the Dublin group to develop their protocol for the active management of labor.

It can be difficult to assess progress in descent. In selected cases, ultrasound examination can be useful in this determination. Transperineal scanning, using the symphysis as a landmark, is the most common technique. Experience is necessary to conduct such examinations, but the results improve the diagnostic accuracy of clinical evaluation. Prospective study is needed before such techniques can be verified to improve outcomes, however. A similar technique is useful prior to forceps application if there is a molded head and it is difficult to palpate the standard landmarks, or if a more difficult rotational delivery is attempted (See Chapter 17, Instrumental Delivery [20]).

The appropriate length for the second or expulsive stage of labor is controversial [13,19,21]. Prior obstetric teaching held that the second stage should not exceed an arbitrary time limit, which was established at 2 hours. Many clinicians, including the author, were originally taught to electively terminate the second stage – if necessary by a forceps operation – when 2 hours had transpired unless spontaneous delivery was imminent. How this specific interval came to be enshrined in obstetric practice is unclear. In previous generations, how-

ever, when electronic fetal monitoring (EFM) was nonexistent, epidural anesthesia was uncommon, and many aspects of obstetric and pediatric management were different from current practices, such an arbitrary limitation on the length of the second stage had a measure of clinical validity. In the early 1950s, Hellman and Prystowsky reported a direct relationship among the length of the second stage, infant mortality, and maternal infection/hemorrhage [21]. In this study, the median duration of the second stage was 20 minutes for multiparas and 50 minutes for nulliparas. Only 3% of labors exceeded a 2- to 3-hour second stage, because the second stage was usually terminated by prophylactic forceps operations. These authors also reported an association between abnormalities in the first stage of labor and subsequent difficulties in the second stage. Newer studies suggest that with or without regional anesthesia, the median duration of the second stage has not changed: 19 minutes for multipara and 54 minutes for nulliparous patients [22].

In contrast, modern studies do not correlate serious fetal problems with length of the second stage if adequate monitoring is conducted. Cohen [23] and others [23–25] have studied second-stage length and perinatal mortality and have found no significant relationship (Table 10.5). Maternal febrile morbidity, the likelihood of instrumental or cesarean delivery, and puerperal hemorrhage do however increase

TABLE 10.5 Duration of the Second Stage of Labor and Perinatal Outcome

Duration (min)	No. of Patients	Perinatal Mortality (per 1,000)	Neonatal Mortality (per 1,000)	Low 5-minute Apgar (%)
0–29	623	6.5	0.0	0.6
30–59	1,257	4.8	2.4	0.7
60–89	1,007	3.0	1.0	0.3
90–119	599	0.0	0.0	0.2
120–149	425	2.4	0.0	0.0
150–179	237	0.0	0.0	0.8
180+	255	3.9	0.0	1.6
Total	4,403	3.4	1.8	0.5

Modified from Cohen W: Influence of the duration of second-stage labor on perinatal outcome and puerperal morbidity. *Obstet Gynecol* 1977;49:266–269; with permission.

when the second stage exceeds 3 to 4 hours (see Chapter 17, Instrumental Delivery).

An important influence on second-stage length is epidural anesthesia. If an epidural block has been administered, the acceptable length of the second stage is extended by 1 hour [26,27]. Epidural anesthesia commonly accompanied by major motor blockade, as opposed to modern epidural analgesia, which should not have a significant motor blockade, prolongs the second stage and increase the incidence of instrumental and cesarean delivery. Modifications in both obstetric and anesthetic protocols can greatly influence outcomes (see Chapter 9, Obstetric Anesthesia).

Although a 2-hour second stage is no longer considered a required point for routine intervention, it remains an important marker. Even with epidural anesthesia, with either delayed or immediate pushing, the second stage usually does not exceed 1 hour [28]. Thus, when the second stage exceeds the 2-hour mark, this is when the clinician should carefully judge the progress of the labor and the condition of the mother and fetus. There is a point at which intervention in a prolonged second stage is appropriate; however, this point is not marked by a specific time interval, but it is determined by the dynamics of labor and the evaluation of maternal and fetal condition. In the absence of maternal or fetal distress and as long as reasonable progress continues, close observation, encouragement and, on occasion, oxytocin are the best management techniques for the second stage. If progress stops, the fetal condition becomes uncertain, or maternal exhaustion develops, medical or surgical intervention is indicated. Such interventions could consist of maternal repositioning, rest, provision of improved analgesia, encouragement, oxytocin augmentation, cesarean delivery, or a forceps or vacuum extraction operation. The appropriate type of intervention is the subject of this and subsequent chapters.

### Uterine Activity

Measurements of uterine activity or quantitation of the amount of uterine work during labor requires determination of the onset, duration, frequency, and strength of contractions [27,29,30]. Historically, this task was accomplished by manual palpation by a bedside birth attendant. Clinical estimation of the strength of contractions was based on the

knowledge that the uterus was not easily indented by finger pressure once the intrauterine pressure reached approximately 40 mmHg. To semiquantitate uterine activity, an external tocodynamometer is now frequently used. This device measures the onset of contractions from an established baseline but is capable of recording only the relative intensity of uterine contractions. When tocodynamometry is used in conjunction with a strip chart recorder, a graphic representation of uterine activity over time results. These data, combined with the instantaneous fetal heart rate (FHR) tracing, describe the classic EFM tracing. After membrane rupture, a pressure catheter can also be inserted between the uterine wall and the presenting part to record uterine pressure directly. With these data, the clinician can record the both the onset and duration of contractions as well as monitor their frequency and intensity.

Summation of the area under the pressure catheter deflection curve for uterine contractions over a 10-minute interval constitute the Montevideo units, perhaps the most familiar of the several published measures for uterine work [27,29]. Although it is possible to calculate the amount of uterine work in a given labor by this method, it is not commonly used. In fact, in active, normally progressing labor there is no well-defined normal pattern for contractions, and many variations exist. The range for normal is so wide that labor is best followed routinely by observing the work that the uterus performs – specifically, cervical dilation, effacement, and descent of presenting part.

### Effects of Maternal Posture

Although the tradition in obstetric management has been to position the parturient in supine position with a partial left-lateral position for labor, there is interest in other positions for labor/delivery. There are data to support the idea that upright maternal postures enlarge some pelvic diameters and can shorten the course of labor [31–35]. Upright positioning, including squatting, apparently results in changes in pelvic dimensions because of flexibility of the bony pelvis at its various articulations. An increase in interspinous diameter, the sagittal diameter of the outlet, and posterior rotation of the iliac bones at the sacroiliac joint apparently accompany maternal repositioning. It is possible that these



changes, combined with the added effect of gravity-assisted parturition, could shorten the second stage of labor [32,33]. Newer techniques of epidural analgesia permit patients to retain substantial muscle tone, and repositioning and occasionally even ambulation are possible. As ambulatory fetal monitoring has become technically possible, there is an increasing potential to apply these concepts in labor management. Today many obstetric units have the capability of telemetry, which permits unfettered continuous monitoring of the fetal heart rate. Telemetry encourages walking during the first stage of labor, and because the upright posture can enhance contraction strength, it has the potential to shorten the first stage of labor. Old studies actually showed stronger contractions in the Montevideo units with the parturient standing or sitting as compared with her in the supine position [31,33–35].

## NORMAL LABOR

### **Mechanism of Labor**

Normally, close to the onset of labor, the term fetus is positioned longitudinally in a cephalic presentation, with the head flexed. The arms are flexed and folded across the chest and the knees are brought up against the lower abdomen or chest. Stating that the presentation is a vertex implies knowledge of cranial positioning, that is, the head is flexed. In contrast, stating that it is a cephalic presentation simply indicates that the head is the leading part.

As the fetal head negotiates the passage through the pelvis, it undergoes a series of positional changes termed the *cardinal movements of labor*. These movements include *engagement, flexion, descent, internal rotation, extension, and restitution*. For poorly understood reasons, some fetuses traverse the birth passage with difficulty. Often, subtle changes in fetal position are a factor. For example, in a partially deflexed presentation, the presenting part is larger, additional pelvic space is required, and dystocia is frequently the result. Other common impediments to labor include inefficient uterine activity, soft tissue or cervical dystocia, or combinations of subtle fetal malpositioning combined with other factors.

A spontaneous delivery can occur from any of the anterior or posterior classic presentations, with one major exception. A fetus in *face* presentation, with the chin directed toward the sacrum (*mentum posterior*), is usually undeliverable vaginally because

extension is not possible. Occasionally, in such mentum posterior positions, the fetal head rotates spontaneously or, rarely, in modern practice, it is instrumentally rotated anteriorly, permitting vaginal delivery. A fetus in the brow presentation should also be considered as in an undeliverable position if this cranial position is fixed. Occasionally, a brow presentation is intermittent in a fetal head that is in the process of extending to a face or when a very small fetal head is presenting as in a markedly premature delivery. These brow malpresentations, which are quite uncommon, are too large to allow for normal delivery without flexion to a vertex or extension to a face presentation. In virtually all cases, a brow presentation therefore must undergo spontaneous flexion to permit vaginal delivery, since the diameter presented to the pelvis by the extended head of a term-sized baby is on average 12.5 cm to 13 cm (occipital-frontal). Brow presentation is diagnosed by palpating the nasal bridge and the upper portion of the orbits or the brow during a pelvic evaluation. If the nose is palpable in its entirety, the presentation is most likely face. A brow or face presentation is easily confirmed by combined transabdominal and transperineal ultrasound scan.

The usual plan for labor management includes repeated clinical examinations at regular intervals, noting the rate of dilation and descent of the presenting part. As long as the active phase and descent portions of the labor curve are within normal limits, no intervention is indicated [Figures 10.1–10.3]. If the progress of labor is inadequate, details of fetal presentation, the fetopelvic relationship, and uterine activity are assessed. If uterine work is not optimal, uterine stimulation by oxytocin is usually administered in an attempt to restore uterine activity to normal (see Abnormal Labor). If normal progress does not resume with uterine stimulation, but slow continuous changes are noted (a *protracted* labor) the use of oxytocin must be reevaluated. The important decision is whether the progress is real versus only increasing molding of the fetal caput. As discussed more fully later, in the technique of active management of labor, the membranes are routinely ruptured and uterine activity is augmented using a rapidly advancing oxytocin protocol.

### **Fetopelvic Relationship**

Except in the unusual situation of a gross fetopelvic disproportion, a fixed brow, or a face presentation

(posterior), no available diagnostic technique except labor can establish with high reliability which fetus will or will not successfully negotiate the maternal pelvis. All experienced clinicians have had the unsettling experience of confidently predicting dystocia only to witness a rapid, uncomplicated labor! Nonetheless, clinical pelvimetry and other examinations remain useful for identifying cases at risk for problems and in making management decisions. In 1933, Caldwell and Moloy produced a classification of pelvic types that has since been used throughout the obstetric literature and has been taught to generations of students [36,37]. In this scheme, the shape of the pelvic inlet defines one of several types of pelvis (e.g., gynecoid, android, anthropoid, and platypelloid). As the initial cardinal movement of cranial engagement occurs, the pattern that the head takes in rotation, flexion, and descent is largely determined by reference to pelvic bony anatomy. In the current understanding of labor, the fetus is a passive passenger in this process, simply propelled by uterine contractions. In theory, study of these types and their many clinical variants permits prediction of the mechanism of fetal cranial descent expected during the course of labor. Classically, disproportion between the fetus and the maternal pelvis was evaluated by clinical pelvic examination (i.e., clinical pelvimetry), abdominal estimation at or near term for fetal size and engagement, examination of fetal lie/position at the onset of labor, and the notation of progress during labor (especially descent). Radiographic or x-ray pelvimetry, a technique rarely practiced or indicated today, was used as a method of evaluation pelvic adequacy and occasionally station.

At the time of cranial engagement, the smallest fetal skull diameter usually enters the maternal pelvis in the narrowest available diameter. Thus, in gynecoid pelvis, the fetal head commonly engages in an occiput transverse position. Following engagement, as rotation and descent of the presenting part proceeds, the posterior portion of the fetal head moves anteriorly, leading to the usual occiput anterior position at the time of delivery [38]. Knowledge of pelvic architecture is of some but limited assistance in predicting dystocia at the onset of labor. Clinically significant dystocia is uncommon in women with gynecoid pelvis unless fetal macrosomia, an occiput posterior presentation, or a markedly deflexed fetal head are present. In contrast, android pelvis are associated with labor difficulties in up to 40% of cases. An anthropoid pelvis,

with its restricted transverse diameter, predisposes to occiput posterior positions, predicting a longer labor with greater likelihood for a prolonged second stage and the need for assistance.

Part of this evaluation process includes the estimation of fetal size. The most common methods are palpation (Leopold's maneuvers), the measurement of the height of the uterus from symphysis pubis to fundus, ultrasound examination, a review of the prior obstetric history, and in multiparas, maternal report. Normally, the uterine fundus grows linearly from approximately 24 to 38 weeks of gestation, with the fundal height in centimeters approximately equal to the gestational age in weeks. Thus, consistent fundal growth provides some indirect information regarding fetal size, particularly when the growth exceeds 40 cm or, alternatively, if it severely lags. Unfortunately, as routinely performed, such estimates are rarely accurate and are strikingly operator dependent. Based on palpation alone, clinicians can usually categorize fetuses only as small, medium, or large. Other commonly used techniques are also problematic. Because of the wide deviation, weight estimates by current ultrasound techniques are disappointingly inexact, especially when either very small or very large infants are measured [39]. The American College of Obstetricians and Gynecologists (ACOG) recommends elective cesarean delivery only if the estimated fetal weight is greater than 5,000 g in the nondiabetic or 4,500 g in the glucose-intolerant patient [40]. With these estimates, the clinician is approximately 90% certain that the true fetal weight is greater than or equal to 4,500 g and 4,000 g, respectively. In multiparous women, another method for weight estimation is simply to ask the mother whether the fetus is perceived to be larger, smaller, or the same size as her prior infants. Such reports are often as reliable as other methods of weight evaluation. Given the poor accuracy of these methods, an important question is whether estimation of fetal weight should have any influence on management apart from insulin-requiring diabetics or when the quite uncommon markedly macrosomic infant (>5000 g) is encountered.

### Clinical Pelvimetry

*Clinical pelvimetry* is a traditional technique of physician examinations that is controversial and often poorly taught to most obstetric residents. The

reason for this is the belief by many clinicians that pelvimetry is inconsequential to modern obstetric management. In addition, the measurements of clinical pelvimetry are not easy for many students of obstetrics to perform, because they require “blind” estimates in patients who can be made uncomfortable by the various manipulations, especially in a teaching situation.

In theory, the clinician mentally constructs an image of the pelvis from the results of pelvic palpation. This anatomic review, when combined with the assessment of fetal bulk and presentation, allows the accoucheur to predict the labor mechanism likely for that specific patient’s anatomy and determine how to achieve vaginal delivery. These determinations were of more immediate utility in the era when extensive obstetric interventions, especially midforceps operations, and rotations were commonly performed. With the disappearance of most of these operative procedures – some replaced by cesarean delivery, others avoided by more aggressive use of oxytocin or by simply extending the second stage of labor – many traditional obstetric skills, including clinical pelvimetry, have waned in popularity. Certainly, the ranks of the practitioners experienced in these estimations have been thinned by age and retirement. With the increasing complexity of obstetric practice and the progressive move toward technical knowledge, it is not surprising that training in pelvimetry has suffered. Nonetheless, the author and other traditionally trained obstetricians believe that these data remain potentially useful in labor management.

All birth attendants should minimally be able to evaluate the diagonal conjugate, the prominence of the ischial spines, and the anatomy of the sacrum during a pelvic examination. The diagonal conjugate indirectly measures the size of the pelvic inlet by estimating the distance from the underside of the pubic symphysis to the sacral promontory. Estimated lengths less than 11.5 cm suggest pelvic inadequacy and could suggest why the presenting part has not engaged. If the fetal head is deeply engaged, however, this measurement is neither possible nor necessary. If descent and engagement of the fetal head are verified by pelvic and abdominal examination, this is good evidence that at least the *pelvic inlet* is adequate for that fetus. The conjugate measurement also aids in determining pelvic type, as discussed later.

During palpation for the ischial spines, the shape of the pubic arch is usually easily appreciated as well. If the arch is *roman*, the operator’s fingers easily pass back and forth across the forepelvis during palpation. A wide or roman arch is considered normal, suggesting adequate room in the forepelvis. Such architecture, combined with a deep conjugate and nonprominent spine, documents a gynecoid pelvic configuration. These are the primary markers for a clinically adequate bony pelvis.

The transverse diameter between the ischial spines roughly indicates the size of the midpelvis and serves as a marker of the plane of least pelvic dimensions. When the leading bony edge of the fetal head reaches this point, the largest diameter of the fetal cranium has successfully traversed the pelvic inlet, and the fetal head is engaged. If on pelvic examination the spines are prominent, midpelvic size is suspect, and descent and rotation of the fetal head might be delayed or might not occur at all. Prominent spines might require that the fetal head rotate to an occiput posterior or oblique position rather than occiput transverse, to permit cranial engagement and subsequent descent.

The posterior pelvis is also evaluated with attention to the sacrosciatic notch and the sacrum. In palpating the sacrosciatic notch, the examiner’s finger sweeps from the ischial spines posteromedially toward the sacrum, along the sacrospinous ligament. If the notch is narrow ( $\leq 4$  cm or approximately 2 to 2.5 fingerbreadths), there is limited room in the posterior pelvis. This examination is often limited by patient discomfort unless an anesthetic/analgesic has been administered. After this examination, the sacrum/coccyx is evaluated. A flat or anterior sacrum that juts forward and is elevated toward the plane of the ischial spines restricts the space available at the posterior outlet. This suggests pelvic inadequacy, prior coccygeal fracture/dislocation, or at least a limited amount of posterior pelvic room.

These are important limitations to these techniques. Traditional clinical pelvimetry does not evaluate fetal size nor does it necessarily reflect what happens in the dynamics of labor; thus, the finding of one or more borderline measurements does not necessarily mean that labor will be obstructed. Conversely, normal pelvimetry does not guarantee the

vaginal delivery of a large or malpositioned infant. To be useful, the data obtained from these pelvic examinations must be combined with the results of other physical examinations and observation of the course of spontaneous or stimulated labor to reach decisions concerning disproportion and the appropriate response to poor or limited progress.

Does any role remain for classic clinical pelvimetry? The author believes so. Evaluation of pelvic architecture, when combined with the data from palpation of the fetal cranium, provides the basis for the dynamic evaluation of pelvic adequacy during labor. Furthermore, for the experienced accoucheur contemplating an instrumental delivery, these data are useful for all midpelvic vacuum or forceps procedures.

### Radiographic Pelvimetry

The radiographic techniques for pelvic evaluations once traditionally performed with regularity in obstetric management have disappeared. There is currently little, if any, indication for radiographic fetograms or fluoroscopic examinations during pregnancy. Trauma is, of course, a separate issue. When fetal visualization is required, ultrasonography or magnetic resonance imaging (MRI) has replaced radiographic methods.

Radiographic or x-ray pelvimetry has no role in evaluating pelvic adequacy except in rare instances of prior pelvic fracture, congenital skeletal deformity, or breech presentation. Radiographic pelvimetry does evaluate pelvic measurements but carries the risk of exposure of the fetus to ionizing radiation. Computed tomography (CT) scans of the pelvis have of late found advocates. In the breech fetus, radiographic information from CT studies documents fetal lie and cranial attitude in addition to the usual measurements of the major pelvic diameters. In the now-rare instance when pelvimetry is appropriate, CT scan has replaced the classic x-ray studies owing to its simplicity and low radiation exposure. Current protocols for breech management can include CT radiographic pelvimetry to ensure that all pelvic dimensions are adequate [41]. Vaginal trials under these circumstances are more likely to be successful and atraumatic. (See Chapter 12, Breech Presentation.)

Ultimately, successful transit of the birth canal depends on the ability of the fetal calvarium to

flex, mold, descend, and rotate through an irregular bony and muscular passage. It is clear that none of the traditional means of evaluation – ultrasonography, Leopold's maneuvers, radiographic or clinical pelvimetry – can alone reliably predict vaginal delivery. The ultimate test of pelvic adequacy is a trial of labor. Given the risk, difficulty, and poor predictive value of classic radiographic pelvimetry, this technique is rightly relegated to the category of historic interest for pregnancies with cephalic presentations and has as well been largely superseded by CT pelvimetry in breeches [27].

### Real-time Ultrasonography

The most useful tool for immediate evaluation of fetal anatomy is real-time ultrasonography. Although it cannot evaluate the anatomy of the maternal pelvis, real-time ultrasound scan does have the ability to easily document the lie, presentation, position and station of the fetus, to estimate gestational age, to evaluate fetal anatomy and, with a limited degree of reliability, to estimate fetal weight.

Unfortunately, neither ultrasound estimations of fetal weight nor calculation of ratios between specific fetal measurements have proved useful in eliminating the risk of traumatic delivery. It is precisely those cases for which these data are most critical (i.e., the suspected macrosomic fetus and the very small, perhaps previable fetus) that ultrasonic fetal weight estimates are most difficult to obtain and are the least accurate. Nonetheless, bedside real-time ultrasonic examinations are useful in cases of dystocia in evaluating gross fetal size, presentation, cranial deflection, and, in experienced hands, station. The plane of the fetal orbits is normally identified with ease, especially when the true station is high and the occiput is posterior. In certain other settings, such as second-twin deliveries, ultrasound scanning is valuable both in antepartum assessment and, especially, in intrapartum delivery room management. Estimation of fetal size remains a problem. Even in a diabetic patient, the inaccuracies of ultrasonic weight estimates only marginally improve decision making. A more specific method could be measuring subcutaneous fat at the level of the thigh, abdomen, and cheeks. A fat bulge of greater than 10 mm denotes excessive deposition. If the estimated fetal weight and the fat layer are both more than the

90th percentile, these data might warrant an elective cesarean delivery [42]. Such interesting applications of ultrasonography await supportive clinical trials for validation.

### Importance of Cranial Flexion

Evaluation of fetal cranial flexion is an important issue that is often peculiarly absent from the discussion of disproportion or safe instrumental delivery. Cranial deflection is an important sign for clinicians. If marked cranial deflection is accompanied by abnormal labor progression, disproportion is likely, and cesarean delivery is normally the best management choice. Lesser degrees of deflection are common in many ultimately successful labors, especially in posterior and transverse presentations. In a normally sized term infant, with the chin flexed on the chest, the suboccipitobregmatic diameter is presented to the pelvic inlet (approximately 9.5 cm). This is the smallest presenting diameter for a term-size (approximately 3,500 g) fetus. As the fetal head progressively deflexes from this position, ever-larger diameters are presented to the birth canal. In a brow presentation, which is normally an undeliverable position, the head presents the occipitomenal diameter to the pelvis, measuring approximately 12.5 cm. Many cranial deflections correct spontaneously as labor progresses. For the rare persistent brow and face presentations, clinical associations for deflection should include anencephaly and other fetal anomalies, true disproportion, high maternal parity, prematurity, and premature membrane rupture. Brow, but not face, presentations virtually always result in obstructed labor unless the position is transitional, the baby is very small, or the maternal pelvis is unusually large. Although there are operative techniques to correct brow presentation, these manipulations are rarely performed nor are they appropriate. If a brow presentation is diagnosed, the use of oxytocin is contraindicated. In selected cases, if the pelvis is clinically adequate and the baby is small, a reasonable course might be to observe labor to determine if conversion to a face or vertex presentation occurs. Failure to convert to either a face or a vertex presentation within 2 hours of active phase labor is a reasonable indication for a cesarean. A face presentation with the mentum persistently posterior is an undeliverable posi-

tion vaginally, and cesarean delivery should be performed.

### Epidural Anesthesia

With epidurals, the aim for both the obstetrician and the anesthesiologist is to provide analgesia for labor, not surgical anesthesia. A surgical level of anesthesia in labor is unnecessary, inhibits effective labor progress, and predisposes to unnecessary operative deliveries. (See Chapter 9, Obstetric Anesthesia.)

Epidural blockade has physiologic effects that potentially alter the course of labor. Epidural anesthesia-induced vasodilation can lead to maternal hypotension that might not be necessarily reflected in the usual brachial artery blood pressure determinations. Pulse oximetry is probably a more accurate means of identifying occult utero-pelvic hypoperfusion than routine blood pressure determinations [43,44]. Preparing for and preventing the combination of maternal hypotension with associated fetal bradycardia is essential. Many unnecessary cesareans still occur because of this association. Positioning the mother laterally after epidural placement and sustaining circulating volume with isotonic salt solutions can prevent this complication. Fortunately, epidural-related FHR changes are normally transient, responding to simple remedial measures such as positional changes.

Despite occasional problems, there are also several potential beneficial effects from epidural blockade. Adequate maternal pain relief enhances cooperation, limits exhaustion, and reduces stress-related elevations in catecholamine levels that accompany labor. Adverse neonatal effects of epidurally administered drugs for pain relief are minimal. (See Chapter 9, Obstetric Anesthesia.)

Important potential effects of epidural blockade in labor include adverse effects on labor progression and the related increased risk for operative intervention. Epidural blockade interferes with the mother's voluntary and involuntary expulsive efforts by changing abdominal and pelvic muscle tone and attenuating reflex arcs. Normally, uterine contractions increase in strength concurrent with cranial descent in the second stage, a finding that is specially marked in unmedicated labors in this transitional phase of labor. More intense uterine contractions occur at or near full dilation and are usually followed rapidly by the mother's spontaneous urge to bear down. This effect is believed to result

from stimulation of sensory output by the pelvic autonomic nerves with the endogenous release of pulses of oxytocin from the pituitary [46]. Epidural blockade interferes with this process by interference with nerve transmission. The more profound the motor and sensory blockade, the greater the likelihood of inhibiting this bearing-down reflex. This is another major reason for avoiding dense epidural anesthesia. It is likely that consistently effective analgesia cannot be provided by the epidural technique throughout the second stage of labor without some increase in the incidence of instrumental delivery and perhaps in the requirements for a cesarean [46–48]. Appropriate management protocols for oxytocin and epidural anesthetic use, however, make it possible to provide adequate analgesia for a large percentage of labors and permit nearly normal labor progression with a low level of intervention. Oxytocin should be administered without hesitation in the second stage if progress under epidural blockade is slow, assuming that maternal-fetal status is acceptable and that disproportion has been excluded [49].

Coaching, administration of oxytocin, and extension of the time allowed for second stage when using epidural anesthesia are helpful in promoting either spontaneous delivery or in achieving a lower station of the fetal head prior to an instrumental delivery. In the author's opinion, voluntary bearing down in the second stage is best deferred until spontaneous descent occurs and the mother perceives pelvic pressure and the spontaneous desire to bear down occurs [26]. This more closely simulates normal second-stage progression. Pushing should not be initiated based only on full cervical dilation. Maternal expulsive efforts initiated at the time of full dilation, despite being widely practiced, are often of limited benefit in speeding descent and can tire the mother unnecessarily. The second stage is approximately the same length whether the parturient pushes immediately or waits until the bearing-down reflex is perceived. As discussed, squatting or partial upright positioning can also be beneficial in gaining station.

## ABNORMAL LABOR

### Dystocia

Common terms used to describe patterns of inadequate labor progress, or *dystocia*, include *cephalopelvic disproportion* (CPD) and *failure to*

TABLE 10.6 Dystocia: Common Terms in Use\*

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Failure to progress (FTP)
Lack of progressive cervical dilation
Lack of descent of the fetal head
Fetal macrosomia/excessive fetal size
Contracted pelvis
Dysfunctional or obstructed labor
Cephalopelvic disproportion (CPD)
Absolute disproportion
Relative disproportion
WCO (won't come out!)

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\*See text for details.

Modified from Rosen MG: Management of Labor. New York: Elsevier, 1990.

*progress* (FTP), (Table 10.6). Classically, dystocia is described as resulting either from true or relative disparity between the capacity of the maternal pelvis and the fetal head because of bony architecture, soft tissue or cervical resistance, fetal malpositioning (e.g., face, mentum posterior, brow, or deep transverse arrest), or a combination of these conditions [52]. The greatest cause of dystocia leading to the failure of vaginal delivery in many labors is simply inadequate uterine activity.

In clinical management, the three classic issues (the 3 "Ps") to be considered include 1) The *pelvis*, excluding true fetopelvic disproportion; 2) the *passenger*, determining the fetal presentation, size, and cranial orientation; and 3) the *powers*, establishing if adequate uterine activity is present and that the mother is capable of adequately bearing down when required. The initial examination directs management. For example, if the fetus presents as a fixed mentum posterior, or a transverse lie (shoulder presentation), cesarean delivery is obligatory. Some cases do not present a diagnostic challenge.

The relative fetopelvic relationship is another issue. Disproportion is a statement about the size of the fetal presenting part compared with the amount of space available in the bony pelvis. Classically, disproportion is stated as either *absolute* or *relative*. In absolute disproportion, the fetal head cannot transit the maternal pelvis because it is too large or the pelvis is too small. In this situation, engagement does not occur, and failure of descent or dilatation is inevitable. True disproportion is a *rara avis*, however. In the much more common condition of relative disproportion, a relatively large or possibly

malpositioned fetus exists. Under these circumstances, delivery from below is often possible, but the labor can be prolonged or difficult.

Rather than absolute disproportion, clinicians are much more likely to encounter other problems. A large fetus in an average-sized pelvis or the malpresentation of a normal or average-sized fetus in an otherwise adequate pelvis are common. Because of this, routine clinical pelvimetry is of limited assistance in deciding which parturients should not have an *initial* trial of labor or receive oxytocin stimulation. In the common situation of an arrest of an established labor, a more comprehensive fetopelvic evaluation is required. (See Chapter 17, Instrumental Delivery.)

### Diagnosis of Malpresentation and Disproportion

The clinical evidence for true classic disproportion (CPD) is progressive molding of the presenting part without true descent. That is, the molded caput can be felt to descend, but the actual biparietal stays high. If CPD is present, vaginal delivery is unacceptably dangerous or impossible. But, true CPD is rare. Furthermore, this diagnosis is always suspect without an adequate trial of labor. The clinician's challenge is to identify cases in which disproportion exists and cesarean delivery is indicated versus those in which the dystocia is relative or likely to prove inconsequential and can be safely overcome by oxytocin stimulation or an operative vaginal delivery.

Using a partogram is helpful to establish the correct diagnosis. Protraction or arrest disorders are common with disproportion (see Fig. 10.3, *A* and *B*). Because most cases of dystocia are caused by relative disproportion, they respond promptly to simple amniorrhexis or oxytocin stimulation. After this, the labor should resume with progress, and eventual vaginal delivery should occur. In contrast, in poorly progressing or arrested labors, the need for oxytocin when paired with epidural anesthesia often results in a higher incidence of cesarean or instrumental delivery [46,53].

When dystocia occurs, it can be difficult to establish the etiology. If extensive cranial molding is present, the clinician might not be able to determine whether it is the actual fetal head or the molded and edematous caput that is descending deeper into the maternal pelvis. In this setting, station cannot be accurately judged based solely on palpation of the leading edge of the presenting part, and other clin-

ical findings become important. Increasingly in this setting, transabdominal, vaginal, or labial ultrasound examinations can assist the clinician.

Initially, it is prudent to perform Leopold's maneuvers abdominally (see Table 10.1) and follow with the Müller-Hillis maneuver during a pelvic examination [54]. The Müller-Hillis maneuver is a simple clinical examination that judges descent of the fetal head with fundal or suprapubic pressure. With this maneuver, the cervix should be dilated to 4 cm to 5 cm or more. Fundal pressure is applied with one hand at the height of a contraction during a vaginal examination. The clinician notes the movement of the presenting part elicited during the maneuver. Acceptable descent with this maneuver is one station (or 1 cm). Although the Müller-Hillis maneuver is useful, the clinical importance of the information it provides should not be overstated. If a contraction combined with abdominal pressure results in additional descent of the presenting part, it simply suggests that there is additional space available in the pelvis. At best, the Müller-Hillis maneuver is a simple, rough estimate of pelvic adequacy. The test is meaningless unless interpreted with the additional information derived from the partogram and progress of labor.

Other clinical data are important. The initial abdominal palpation can reveal a high presenting part, a head overriding the pubic symphysis, or a face or brow presentation. Failure of the fetal head to fill the posterior hollow of the sacrum, despite a heavily molded cranial mass beyond the plane of the ischial spines, is a strong suggestion that the head has not negotiated the midpelvis and is unengaged. Similarly, failure to easily palpate the fetal ear also suggests high station [55]. Careful estimation of how much of the fetal head is present abdominally is another possible means of evaluation [56]. In this technique, the extent of cranial descent into the pelvis is estimated in fifths, using a palpation technique akin to the classic Leopold's maneuvers. Engagement of the fetal head has occurred when no more than one fifth of the fetal head remains palpable abdominally. Obviously, anesthesia, patient size, and the skill and experience of the operator contribute to the success of such examinations.

Philpott [57] and Vacca [58] describe an additional and useful technique of gauging the extent of disproportion. In this method, the degree of cranial molding is estimated during pelvic examination by judging the overlap of the fetal cranial bones at

the occipital-parietal and parietal-parietal junctions. The extent of this overlap and the ease of reduction by simple digital pressure are noted. If the bones are overlapping and cannot be separated easily by simple digital pressure, molding is judged as advanced or extreme (+3), and there is probably true disproportion. (See Chapter 17, Instrumental Delivery.)

Real-time ultrasound scanning is a new and important tool in assessing fetal positioning and station. Transperineal or transvaginal ultrasound scans can easily identify specific landmarks, including the maternal symphysis and the fetal calvarium, the fetal orbits, and edema of the scalp (caput). An experienced sonographer can rapidly determine the position of the fetal head and if it is engaged. The degree of cranial molding and caput formation are also evaluated, as is the station of the presenting part. Caput is easily diagnosed by observing an echo-free space between the fetal skull and fetal scalp [59]. As clinicians become more experienced with these methods, the accuracy of the clinical diagnosis of position and station will improve.

### Management

The management of labor dystocia depends on the type of specific abnormality, the maternal-fetal condition, and the results of the evaluation of the fetopelvic relationship. Abnormalities of the latent phase should be treated with either therapeutic rest (with or without sedation) or amniorrhexis and oxytocin infusion. As previously discussed, it is possible but uncommon to discover a fetal presentation at the onset of labor that is undeliverable and for which a trial of labor is inappropriate. Narcotic-induced sleep, an old technique, is still useful in latent labors. After a dose of morphine, the parturient often sleeps for a several hours and then when she awakens is often either in active-phase labor or the contractions have abated and the diagnosis of false labor is made.

For active-phase labor abnormalities when progress is poor, the presentation is cephalic, and absolute disproportion and malpresentation have been excluded by the suggested examinations, the best measure of pelvic adequacy is a trial of oxytocin labor stimulation under close maternal-fetal observation. Oxytocin can safely be administered to nulliparas by various standard protocols with minimal risk. Dystocia in multiparas requires more consideration, because malpresentation is more common and the risks of oxytocin stimulation are greater than

those for nulliparous women [3,38,59]. Whereas Friedman and Cohen [61] reported high failure and complication rates for oxytocin stimulation for dystocia, others, including Cardoza and Pearce [62], did not find this to be true. In the two studies, however, the patient population might not have been the same, and the definitions used to describe the labor problems were different. One group could have had protracted active phase and dysfunctional labor as opposed to an arrest of active phase labor. The latter responds poorly to oxytocin, and the response to stimulation is important. In the 10% to 20% of dysfunctional labors that fail to respond to oxytocin stimulation, there is a high incidence of nonreassuring fetal heart rate patterns and cesarean delivery.

Thus, in second-stage arrests in patients with epidural anesthesia, augmentation with oxytocin should be considered. When second-stage progress is tardy, patient repositioning, use of epidural analgesia as opposed to anesthesia, simply prolonging the second stage, and patient encouragement are often successful in achieving vaginal delivery or, minimally, in advancing the fetal head to a lower station to avoid a complex or rotational instrumental delivery. Because slow second-stage progress can herald shoulder dystocia, heroic efforts at instrumental delivery in women known or suspected to be carrying macrosomic infants are to be avoided.

When oxytocin stimulation/augmentation is performed, labor progress is judged by frequent serial vaginal examinations with careful recording of cervical dilation, station, and position of the fetal head. Although arrests or tardy descent requires close attention, the risks of a trial of oxytocin stimulation for dystocia are minimal [62–64].

Resumption of progress is the critical variable. If the fetal head fails to descend or the cervix fails to dilate following adequate oxytocin stimulation (usually defined as a *minimum* of a 2-hour trial), maternal encouragement, or repositioning, vaginal delivery becomes progressively less likely. The clinician must then decide between modes of operative delivery: cesarean or trial of instrumental delivery. Fetal and maternal condition, cervical dilation, station of the presenting part, skill of the operator, and relative fetopelvic size all figure into the decision. (See Chapter 17, Instrumental Delivery.)

Trials of labor augmentation require especially close attention to possible maternal and fetal stress. The pattern of uterine activity is commonly documented by continuous monitoring using an



intrauterine pressure catheter or transducer (IUPC), while the FHR is recorded electronically. Such invasive monitoring is not required in all cases, at least in nulliparous patients, however. The Dublin group has safely used oxytocin stimulation in thousands of cases with "one-on-one" nursing/midwifery clinical observation and intermittent auscultation without use of electronic detectors to detect or document either uterine activity or fetal heart rate patterns [2]. In U.S. practice, the use of electronic monitoring for women receiving oxytocin is, however, the routine standard. When oxytocin is administered to a patient with a previous uterine scar, or in multiparous women with arrest disorders, the monitoring of uterine contractions and the fetal heart rate response is more critical. In these settings, the frequency of uterine contractions are monitored electronically and if there are concerns an intrauterine pressure catheter may be placed. The risk in oxytocin administration to such patients is uterine rupture. Although a pressure catheter is helpful in determining that an adequate contraction pattern has been established without overstimulation, thus establishing limits for the rate of the oxytocin infusion, it is not a reliable method for the diagnosis of a rupture.

## UTEROTONICS: OXYTOCIN

### **Physiology of Normal Labor**

Normal labor is a complex endocrinologic event that is believed to be initiated by the fetus [67,68].

Oxytocin is a naturally occurring octapeptide that is produced by the posterior pituitary. It is a facilitator of uterine contractions and plays an important but limited role in initiation of normal term labor. The role for fetal oxytocin in the onset of normal labor remains unclear. Oxytocin is produced by the fetus in relatively large amounts. As oxytocin is a relatively small molecule (molecular mass  $\leq 1,000$  Daltons) oxytocin of fetal origins is able to pass from placenta into the maternal circulation. The placenta does degrade oxytocin by a specific oxytocinase enzyme.

The prostaglandins are believed to be essential to the initiation and the normal progress of labor. Clinically, the uterus is able to respond to prostaglandin stimulation at any time during pregnancy. Increases in prostaglandin concentration and

that of their metabolites are observed in both the active phase of labor and in late pregnancy. Prostaglandin F<sub>2</sub> synthesis occurs in the decidua with prostaglandin E<sub>2</sub> produced in both decidua and membranes. The levels of prostaglandin F rise rapidly during active labor. Amniotomy also results in a rapid rise in prostaglandin concentration, presumably by stimulating prostaglandin production in the membranes and decidua. There also is an important prostaglandin effect on the cervix. Primarily mediated by E prostaglandins, progressive collagen degradation and alteration of the connective tissue ground substance prepare the cervix for labor, resulting in softening and effacement [70]. Because of these important effects, prostaglandins have found a role in cervical ripening for labor induction, in treatment of postpartum atony, and in the termination of pregnancy [71–72]. (See Chapter 6, Pregnancy Termination, and Chapter 18, Cesarean Delivery and Surgical Sterilization.)

Oxytocin and the prostaglandins play complementary roles in human parturition. As term is reached, the concentration of myometrial oxytocin-receptors rises sharply. This lowers the level at which contractions are evoked by circulating oxytocin, the levels of which do not change with the onset of contractions. Oxytocin also increases the decidual production of prostaglandins. Prostaglandins in turn stimulate the myometrium. Oxytocin also increases intracellular calcium flux, increasing myometrium contractions [67].

Although complex endocrine changes are involved in the onset of normal term labor, clinicians have long recognized that several other factors, including excessive uterine distension, Müllerian anomalies, placental separation, premature membrane rupture, intrauterine or endocervical infection, and other unknown influences, can also result in the early and inappropriate onset of labor. The precise mechanism leading to most cases of preterm labor, however, remains unknown.

### **Labor Induction and Augmentation**

Induction of labor is now second only to cesarean delivery as the most common obstetric procedure. Induction of labor is indicated when the maternal or fetal benefits of delivery outweigh the risks of continuing the pregnancy. Nationally there has been an increasing trend toward labor induction, the rates

rising from 9.5% in 1990 to 20.6% in 2003 [73,74]. Nulliparous women who were non-Hispanic, white, college educated, and born in the United States were more likely to undergo induction of labor in the years 1989 to 1998. Additionally, women with pregnancies complicated by medical conditions such as hypertension, preeclampsia, and renal disease were more likely to be induced. Despite its popularity, the induction of labor is not risk free. It has been associated with an increased incidence of cesarean delivery and iatrogenic prematurity. Cesarean delivery is increased particularly in nulliparous women undergoing labor stimulation for poor progress.

Physicians should discuss with their patients the indications, methods, and the increased possibility of cesarean delivery prior to proceeding with a trial of induction. The gestational age, an estimate of fetal size, and notation of presentation, a clinical statement concerning pelvic adequacy, and a cervical examination should be included in the hospital admission documents. ACOG has specific guidelines to assist in choosing a date for induction.

Delivery by induction should be limited to specific indications. Potential maternal and fetal reasons for induction include but are not limited to post-dates pregnancy (>41 weeks), fetal demise, known or suspected chorioamnionitis, intrauterine growth restriction, premature rupture of membranes, preeclampsia or eclampsia, isoimmunization, or maternal medical conditions, such as diabetes mellitus, renal disease, or chronic hypertension. With maternal diabetes, the requirement for lung maturity testing is higher than with nondiabetic pregnancies, since fetuses of diabetic mothers often have delayed pulmonary maturation. Thus, respiratory distress is more common, especially if the fetus is delivered by a cesarean without intervening labor.

Contraindications to labor induction include but are not limited to a prior classic uterine incision, active genital herpes infection, known or suspected vasa-previa or placenta previa, and an undeliverable fetal position (e.g., a transverse fetal lie). Suspected fetal macrosomia is a surprisingly common but invalid indication for labor induction [40]. A cautious approach is recommended in induction involving multiple gestations, pregnancies complicated by poor fetal growth, macrosomia, and hydramnios, or maternal heart disease or hypertension. Cases involving prior low transverse cesarean birth(s) and a trial of labor, or a trial of vaginal birth after cesarean

(VBAC), also require close scrutiny. Logistic indications such as history of rapid labor, living a great distance from the hospital, and other social issues are legitimate considerations to include in the decision for induction, depending on circumstances. For all elective inductions, fetal pulmonic maturity is a concern. Criteria suggested by ACOG for determination of term gestation requires fetal heart tones documented for 30 weeks by Doppler (or 20 weeks by nonelectronic fetoscope), a positive urine or serum HCG test documented at a minimum of 36 weeks from the time of induction, ultrasound measurements of crown-rump length at 6 to 12 weeks, or standard ultrasound measurements between 13 and 20 weeks confirming a gestational age of at least 39 weeks. For elective inductions prior to the 39th week, a lung maturity test by amniocentesis is recommended.

A pelvic examination is mandatory prior to beginning an induction. Cervical effacement and dilation are reasonable predictors of successful vaginal delivery. The frequently used Bishop pelvic scoring system assigns a numeric value to dilation, effacement, consistency, and position of the cervix. The likelihood of a vaginal delivery is similar to that after spontaneous labor if the total score is greater than eight. A low score documents an unfavorable cervix that should undergo ripening as part of the induction process. A low score of less than 5 also increases the risk of failure of the induction.

A positive fetal fibronectin (fFN) test is also predictive of a successful induction. Women with an unfavorable cervix examination and a negative fFN have almost a 50% increased risk of a cesarean for failed induction versus those with a similar examination but a positive fFN.

Mechanical methods such as membrane stripping, amniotomy, the placement of intracervical or extraamniotic balloon catheters, the use of cervical dilators such as Laminaria or prostaglandin administration are common methods for cervical ripening. In specific circumstances such as a VBAC, induction with a mechanical device is safer than the administration of prostaglandins. Mechanical methods provide either cervical dilation or simply disrupt the fetal membranes. They have the advantage of low cost and fewer systemic side effects. The goal is to achieve a favorable Bishop's score to improve the likelihood for a successful induction and ultimately a vaginal delivery.

Membrane sweeping or stripping is easily performed after 38 weeks. Some clinicians perform stripping membranes beginning at 38 weeks on a weekly basis. Studies have shown a significant decrease in postterm deliveries with this technique [75].

*Amniotomy*, or intentional rupture of membranes, is a common induction procedure used alone or with other induction agents. It is performed when the membranes are accessible and the fetal head is well applied to the cervix. Although a common procedure in labor induction, modern data are lacking about the value of amniotomy alone for induction of labor. Older studies indicate that up to 60% of women with favorable cervical examinations will go into labor with amniotomy alone [76]. Amniotomy as an adjunct to prostaglandin or oxytocin administration is common.

Induction using a Foley catheter is a modern technique that refines a method long used in obstetric practice. A No. 16 Foley catheter is passed through the partially dilated cervix, and the 30-cc balloon is inflated. The balloon is placed so that it rests against the internal os in the extraamniotic space. Pressure can be applied against the internal os of the cervix by attaching a weight to the end of the catheter, although this is not necessary. The infusion of extraamniotic saline or isotonic infusion through the catheter can decrease induction-to-vaginal delivery time with no increase in the cesarean rate. This technique can also be used in women with a prior cesarean delivery undergoing a VBAC trial, without increased risk of uterine rupture [77,78].

Both hygroscopic cervical dilators (Dilapan) and osmotic dilators (Laminaria) can be placed intracervically to dilate and soften the cervix. These dilators work to improve the Bishop score; however, successful labor and cesarean delivery rates are apparently unchanged, and the risk of postpartum infections is increased [79]. For these reasons, use of such cervical dilators at or about term is not recommended.

Because normal labor begins following a period of preliminary cervical ripening changes caused by prostaglandins, mimicking this process by cervical pretreatment with prostaglandin E2 is a popular approach to labor induction [80,81]. Normally, two to five doses of prostaglandin E2 gel are administered intracervically every 4 to 6 hours. This increases the chances for a successful induction and shortens the duration of labor. The Bishop score (Table 10.7) is commonly used to clinically assess the need for administration of cervical prostaglandins [82]. With a Bishop score of five or greater, treatment with a prostaglandin is usually unnecessary.

Preparations of prostaglandins E1 and E2 are widely available for pharmacologic cervical ripening. The E2 analogue, dinoprostone, is available in a 0.5-g gel form (Prepidil) or 10-mg vaginal insert (Cervidil) [73]. Misoprostol (Cytotec) has also been used for labor induction. Misoprostol is a form of prostaglandin E. This compound is the best prostaglandin preparation to choose if the patient has reactive airway disease. This medication, first used to prevent stomach ulcers and protect the stomach lining, is also a dilator of bronchial muscle and does not induce bronchospasm. Misoprostol

TABLE 10.7 Pelvic Examination: Bishop Score

Clinical Feature	Points*			
	0	1	2	3
Cervical dilatation	0	1–2	3–4	5–6
Cervical effacement (%)	0–30	40–50	60–70	80+
Station <sup>†</sup>	–5	–4	–2 or 0	+1 to +3
Cervical consistency	Firm	Medium	Soft	–
Cervical position	Posterior	Mid	Anterior	–

\*The final score is the sum of the points assigned for the various clinical parameters

<sup>†</sup>Based on ACOG centimeter scale, see text discussion and Table 10.4.

Modified from Bishop EH: Pelvic scoring for elective induction. *Obstet Gynecol* 1964;24:266–269.

is contraindicated in a scarred uterus (VBAC or prior myomectomy scar), and its administration results in more uterine tachysystole than dynoprostone. In two studies, the use of misoprostol had to be discontinued secondary to uterine ruptures in VBAC inductions. A reliable risk for uterine rupture related to misoprostol induction in VBACs is not known; however, misoprostol is now contraindicated in VBAC trials. This drug is safe, however, in second-trimester abortions.

In regular inductions, despite the increased risk of tachysystole with misoprostol, there is no increase in cesarean delivery rates for fetal distress. In addition, misoprostol can be used either orally (50  $\mu\text{g}$ –100  $\mu\text{g}$ ) or per vagina (25  $\mu\text{g}$ –50  $\mu\text{g}$ ). When misoprostol is administered orally, it has the peak effect in a few minutes, with the effect lasting 1 hour. In contrast, when misoprostol is given vaginally, the peak effect is slower to develop, but it lasts for approximately 4 hours. Either form of administration has similar successful vaginal delivery rates [80,81].

Oxytocin (Pitocin) remains the primary drug for labor induction and augmentation and it can be used as an adjunct to a cervical ripener (e.g., Foley catheter) or in conjunction with amniotomy. Amniotomy is effective but should not be performed in special instances, such as inductions in HIV-positive patients or in premature pregnancies. With a favorable cervix, oxytocin can be used alone. For induction, as discussed below oxytocin is administered intravenously using one of several regimens. In terms of an individual case, cervical dilatation, parity, and gestational age are the best predictors of a favorable response.

Oxytocin dosing is variable and many schemes for administration exist [27,85–86]. Because oxytocin requires 40 minutes to reach steady plasma levels, it has been argued that the popular protocols of rapidly increasing the dose (e.g., every 15–20 minutes), as opposed to slowly increasing doses (e.g., every 45–60 minutes), offer no advantages and only increase complications. Despite these theoretic arguments, the use of progressive oxytocin dosing at 15- to 30-minute intervals is near universal. Oxytocin infusion increases amplitude, duration, and frequency of contractions. The dose-response curve flattens, however, at higher doses ( $\geq 24$  mU/min).

Oxytocin is now provided in premixed solutions of 2 ml (10 units) in 500 ml of D5W, for a final concentration of 20 mU/ml. The usual low-

dose oxytocin regimen begins with 0.5 mU/min to 1 mU/min and is increased by 1 mU to 2 mU at 20- to 60-minute intervals. The high-dose regimen commonly starts at 4 mU/min to 6 mU/min and increases the dose by rapid progression (4 mU–6 mU) at 15- to 20-minute intervals.

In a study of 2,788 consecutive single fetuses, cephalic-presenting pregnancies by Satin and coworkers, both the high- and low-dose oxytocin regimens were evaluated for specific benefits or risks for labor augmentation and induction [86]. All solutions used resulted in satisfactory delivery rates; however, there were differences. Induction failed less often with the high-dose regimen (6 mU/min, increased by 6 mU every 20 minutes). Augmentation with the high-dose regimen also minimized the number of cesarean deliveries performed for dystocia and resulted in significantly fewer forceps deliveries. Labors augmented with the high-dose regimen were significantly shortened (by  $>3$  hr), but uterine hyperstimulation was more common with this regimen and cesarean delivery was performed more frequently for fetal distress when the high-dose as opposed to the low-dose protocol was followed. There were no consistent adverse fetal effects. Thus, the positive results of a high-dose oxytocin protocol includes shorter labors (largely by shortening of the latent phase), fewer failed inductions, and a decreased incidence of neonatal sepsis (presumably by shortening labor). The negative result is an increased incidence of cesarean delivery.

Based on these and other data, many clinicians believe that, when faced with poor progress/dysfunctional labor, the higher-dose augmentation protocol, involving pharmacologic doses of oxytocin (e.g., 4 mU/min–6 mU/min, increased by 3 mU–6 mU every 15–20 min, maximum  $\leq 42$  mU/min) is indicated in nulliparas. The evidence suggests that this is the best treatment for dystocia. The data also can be fairly read to favor low-dose protocols or use in multiparas (e.g., 1 mU/min–2 mU/min, increased by 1 mU–2 mU every 30–40 minutes, maximum 20 mU/min) for labor induction.

For labor inductions the author favors a low-dose induction protocol with oxytocin increments at 20- to 30-minute intervals. In contrast, for the augmentation of either dysfunctional or arrested labors, a higher-dose, rapid advancing augmentation protocol is employed. As noted, the higher-dose rapid progression protocol should be used circumspectly in

multiparas. In each case, the administration of oxytocin is an individual titration; the response depends on both previous uterine activity and individual sensitivity.

Timing of induction is important. A small study suggests that inductions started in the morning as opposed to other times in the day have a higher vaginal delivery rate and a greater success rate [88]. If a morning induction succeeds, most women deliver by the early afternoon than do those commencing later. Some studies suggest two peaks in cesareans, the first at about 23:00 and the second about 04:00. The first interval is associated with the nighttime change of shift and, presumably patient reevaluation, the second with an increased likelihood of the diagnosis of a nonreassuring fetal status (presumed fetal jeopardy, fetal distress).

### Active Management of Labor

The labor management technique as practiced in Dublin by the group at the National Maternity Hospital has been uniquely successful in their hands [2,83]. Their system, termed *the active management of labor*, employs early amniotomy and liberal oxytocin administration. A rapidly progressive (every 15 minutes), high-dose oxytocin protocol (6 mU/min–44 mU/min) is preferred. In their technique, the importance of defining the commencement of labor is emphasized. Cephalopelvic disproportion is diagnosed only after a labor trial, and no labors are permitted to extend beyond 12 hours. The system depends on one-on-one nursing, using highly experienced personnel, as well thorough education of their patients and a strong team approach. The dedication and expertise of the Dublin group are as impressive as their success.

This kind of control is hard to achieve in the American labor and delivery services. Beds are often occupied by women who might or might not be in labor, might be being induced, or might be merely under observation. Furthermore, each obstetrician or midwife follows a unique protocol for labor management, and the use of oxytocin stimulation is far from standardized. Furthermore, it is often the least experienced person who examines new patients, and multiple delays preclude prompt action. An important component of all successful active management plan programs is the belief and assistance of the nursing staff and strong physician leadership.

Potential American and Canadian institutions that have attempted active management of labor protocols saw their cesarean rates decline, but as soon as the interested fellow or director of labor and delivery left, the rate would climb again [89]. In recent years, active management of labor programs have fallen from popularity, replaced by the contentious debates over elective cesarean (cesarean on demand) and proper management of VBAC trials.

### COMMENT

Many factors influence the progress of labor. Among these are adequacy of uterine activity, size of the fetus in relation to the birth canal, fetal positioning, bony and soft tissue anatomy of the birth canal, maternal labor position, coaching by experienced personnel, and certain confounding factors such as uterine infection, hydramnios, and the administration of analgesia or anesthesia (especially epidural anesthesia).

Progress in labor is best evaluated by meticulous clinical evaluation accompanied by charting cervical dilation and descent of the presenting part, using a standard partogram. If progress is arrested, knowledge of pelvic architecture, review of the course of labor, fetal size, and appreciation of position and maternal condition is necessary to decide whether oxytocin stimulation, instrumental delivery, or cesarean delivery is best. For example, a deeply engaged, deflexed occiput posterior head in a multiparous woman with a gynecoid pelvis and arrested progress might lead to vacuum extraction failure but a successful delivery following a forceps application. Alternatively, a fetus with a heavily molded head in an occiput transverse, deflexed position at 0 to +1/5 cm station in a nulliparous patient with a nonreassuring fetal heart rate pattern and poor progress is not a candidate for either an instrumental trial or oxytocin, and prompt cesarean delivery is best.

If normal progress ceases, or only desultory uterine activity is present, and the pelvis is adequate with the child appropriately positioned, the best treatment for poor labor progress (if membranes have been ruptured) is a trial of oxytocin stimulation under close observation. In the presence of reassuring fetal status (a normal and reactive EFM tracing, or a normal auscultated fetal heart rate in an

uncomplicated pregnancy) and with a clinically adequate pelvis, oxytocin stimulation should always be considered and usually attempted before resorting to either cesarean or instrumental delivery.

Judging the point of intervention is not always easy. Both flexibility and humility are necessary on the part of the clinician, since the course of labor is never entirely predictable. Many clinicians of exceptional competence and vast experience have confidently predicted either uncomplicated labor or inevitable dystocia for a particular case, only to subsequently have been proved wrong! The problem for the modern obstetrician in labor management is that of balance. The equation includes fetal and maternal interests, requirements of the profession, and the demands of society, third-party payers, the family, and the medicolegal environment. In often complex clinical settings, obstetricians are expected to arrive at management decisions that choose cesarean delivery sparingly, restricting interventions to clinical settings when benefits clearly exceed risks. At the same time, patients and their families expect painless labors, absolute safety, the absence of complications, and the certainty of no fetal/neonatal injuries.

Controversies concerning obstetric management of labor are inevitable and ultimately healthy for clinical practice. The current high rate of cesarean delivery remains both problematic and controversial. The experience of recent years has shown that the virtually unrestricted use of cesarean delivery is not invariably beneficial to either mother or child. However, a return to the period of heroic obstetric intervention aimed at achieving vaginal delivery at any cost is likewise inappropriate. Rethinking the standard obstetric responses to poor progress in labor, modification of techniques for epidural anesthesia/analgesia, reasonable protocols for instrumental vaginal delivery, less invasive forms of fetal/maternal monitoring, and the continued support for VBAC trials among other changes can all help to restore the appropriate balance between medical and surgical interventions in obstetric practice.

## REFERENCES

- Hannah ME, Ohlsson A, Farine D, Hewson SA, Hodnett ED, Myhr TL, Wang EE, Weston JA, Willan AR: Induction of labor compared with expectant management for prelabor rupture of membranes at term: The Term PROM study group. *N Engl J Med* 1996 Apr;334(16):1005–1010.
- O'Driscoll K, Meagher D, Boylan P: Active management of labor. Aylesbury, England: Mosby Year Book Europe, Ltd., 1993.
- Eltzschig HK, Lieberman ES, Camann WR: Medical Progress: Regional anesthesia and analgesia for labor and delivery. *N Engl J Med* 2003 Jan;348(4):319–332.
- Wong CA, Scavone BM, Peaceman AM, McCarthy RJ, Sullivan JT, Diaz NT, Yaghmour E, Marcus RJ, Sherwani SS, Sproviero MT, Yilmaz M, Patel R, Robles C, Grouper S: The risk of cesarean delivery with neuraxial analgesia given early versus late in labor. *N Engl J Med* 2005 Feb;352(7):655–365.
- Lee BB, Ngan Kee WD, Lau WM, Wong AS: Epidural infusions for labor analgesia: A comparison of 0.2% ropivacaine and 0.1% ropivacaine with fentanyl. *Reg Anesth Pain Med* 2002 Jan–Feb;27(1):31–36.
- Paech MJ, Pavy TJ, Sims C, Westmore MD, Storey JM, White C: Clinical experience with patient-controlled and staff-administered intermittent bolus epidural anesthesia in labour. *Anaesth Intensive Care* 1995 Aug;23(4):459–463.
- Halpern SH, Leighton BL, Ohlsson A, Barrett JF, Rice A: Effect of epidural vs. parenteral opioid analgesia on the progress of labor: A meta-analysis. *JAMA* 1998 Dec;280(24):2105–2110.
- Peisner DB, Rosen MG: Latent phase of labor in normal patients: A reassessment. *Obstet Gynecol* 1985 Nov;66(5):644–648.
- Peisner DB, Rosen MG: Transition from latent to active labor. *Obstet Gynecol* 1985 Oct;68(4):448–451.
- Zhang J, Troendle JF: Reassessing the labor curve in nulliparous women. *Am J Obstet Gynecol* 2002 Oct;187(4):824–828.
- Lavender T, Alfrevic Z, Walkinshaw S: Effect of different partogram action lines on birth outcomes: A randomized controlled trial. *Obstet Gynecol*. 2006 Aug;108(2):295–302.
- Gurewitsch ED, Diament P, Fong J, Huang GH, Popovtzer A, Weinstein D, Chervenak FA: The labor curve of the grand multipara: Does progress of labor continue to improve with additional childbearing? *Am J Obstet Gynecol* 2002 June;186(6):1331–1338.
- Friedman EA: Labor: Clinical Evaluation and Management, 2nd ed. New York: Appleton-Century-Crofts, 1978.

14. Norwitz ER, Robinson JN: Current concepts of labor. *N Engl J Med* 1999;341:660-666.
15. Mancuso PJ, Alexander JM, McIntire DD, Davis E, Burke G, Leveno KJ: Timing of birth after spontaneous onset of labor. *Obstet Gynecol* 2004 Apr;103(4):653-656.
16. American College of Obstetricians and Gynecologists. Committee on Obstetrics: Maternal and Fetal Medicine: Obstetric forceps. Technical Bulletin No. 59. Washington, DC: American College of Obstetricians and Gynecologists, 1988.
17. O'Grady JP: *Modern Instrumental Delivery*. Baltimore: Williams & Wilkins, 1988.
18. Alexander JM, Sharma SK, McIntire DD, Leveno KJ: Epidural analgesia lengthens the Friedman active phase of labor. *Obstet Gynecol* 2002 Jul;100(1):46-50.
19. Dupuis O, Silveira R, Zentner A, Dittmar A, Gaucherand P, Cucherat M, Redarce T, Rudigoz RC: Birth simulator: Reliability of transvaginal assessment of fetal head station as defined by the ACOG classification. *Am J Obstet Gynecol* 2005 Mar;192(3):868-874.
20. Cohen WR: Normal and abnormal labor. In: Hobbins JC, Mahoney MJ, Petrie RH (eds): *Medicine of the Fetus and Mother*. Philadelphia: JB Lippincott, 1992.
21. Hellman LM, Prystowsky H: The duration of the second stage of labor. *Am J Obstet Gynecol* 1952 Jun;63(6):1223-1233.
22. Fraser WD, Cayer M: Risk factors for difficult delivery in nulliparas with epidural analgesia in second stage of labor. *Obstet Gynecol* 2002, Mar;99(3):409-418.
23. Cohen WR: Influences of the duration of second stage of labor on perinatal outcome and puerperal morbidity. *Obstet Gynecol* 1977 Mar;49(3):266-269.
24. Kadar N: The second stage. In: Studd J (ed): *The Management of Labour*. Oxford: Blackwell Scientific Publications, 1985; pp. 271.
25. Vahratian A, Troendle JF: Methodological challenges in studying labour progression in contemporary practice. *Paediatr Perinat Epidemiol* 2006 Jan;20(1):72-78.
26. Plunkett BA, Peaceman AM: Management of the second stage of labor in nulliparas with continuous epidural analgesia. *Obstet Gynecol* 2003 Jul;102(1):109-114.
27. American College of Obstetricians and Gynecologists Practice Bulletin Number 49, December 2003: Dystocia and augmentation of labor. *Obstet Gynecol* 2003 Dec;102(6):1445-1454.
28. Piper JM, Bolling DR: The second stage of labor: Factors influencing duration. *Am J Obstet Gynecol* 1991 Oct;165(4 Pt 1):976-979.
29. Hauth JC, Hankins GD, Gilstrap LC 3rd: Uterine contraction pressures with oxytocin induction/augmentation. *Obstet Gynecol* 1986 Sep;68(3 Pt 1):305-309.
30. Seitchik J, Castillo M: Oxytocin augmentation of dysfunctional labor. II. Uterine activity data. *Am J Obstet Gynecol* 1983 Mar;145(5):526-529.
31. Borell U, Fernstrom I: The movements at the sacroiliac joints and their importance to changes in the pelvic dimensions during parturition. *Acta Obstet Gynecol Scand* 1958;36(1):54-60.
32. Gardosi J, Hutson N, B-Lynch C: Randomised, controlled trial of squatting in the second stage of labour. *Lancet* 1989 Jul;2(8654):74-77.
33. Chen SZ, Aisaka K, Mori H, Kigawa T: Effects of sitting position on uterine activity in labor. *Obstet Gynecol* 1987 Jan;69(1):67-73.
34. Gupta JK, Hofmeyr GJ: Position of women during second stage of labour. *Cochrane Database Syst Rev* 2004;(1):CD002006.
35. Terry RR, Westcott J: Postpartum outcomes in supine delivery by physicians vs. nonsupine delivery by midwives. *Am J Osteopath Assoc* 2006 Apr;106(4):199-202.
36. Caldwell WE, Moloy HC: Anatomic variations in the female pelvis and their effect in labor with a suggested classification. *Am J Obstet Gynecol* 1933;26:479-483.
37. Steer CM: *Moloy's Evaluation of the Pelvis in Obstetrics*, 3rd ed. New York: Plenum Medical Book Company, 1975.
38. Klapholz H: Evaluation of fetopelvic relationships. In: Cohen WR, Friedman EA (eds): *Management of Labor*. Baltimore: University Park Press, 1983.
39. Sandmire HF: Whither ultrasonic prediction of fetal macrosomia? *Obstet Gynecol* 1993 Nov;82(5):860-862.
40. American College of Obstetricians and Gynecologists. Practice Bulletin Number 22: Fetal Macrosomia. Washington, D.C: American College of Obstetricians and Gynecologists, 2000.
41. Gimovsky ML, Willard K, Neglio M, Howard T, Zerme S: X-ray pelvimetry in a breech protocol - a comparison of digital radiography and conventional method. *Am J Obstet Gynecol* 1985 Dec 15;153(8):887-888.

42. Chauhan SP, Grobman WA, Gherman RA, Chauhan VB, Chang G, Magann EF, Hendrix NW: Suspicion and treatment of the macrosomic fetus: A review. *Am J Obstet Gynecol* 2005 Aug;193(2):332–346.
43. McNamara H, Johnson N, Lilford R: The effect on fetal arteriolar oxygen saturation resulting from giving oxygen to the mother measured by pulse oximetry. *Br J Obstet Gynaecol* 1993 May;100(5):446–449.
44. Carbonne B, Cudeville C, Maillard F, Goffinet F, French Study Group on Pulse Oximetry: Predictive value of pulse oximetry and fetal scalp blood pH in the case of meconium-stained amniotic fluid. *Eur J Obstet Gynecol Reprod Biol* 2003 Jul;109(1):27–32.
45. Goodfellow CF, Hull MG, Swaab DF, Dogterom J, Buijs RM: Oxytocin deficiency at delivery with epidural analgesia. *Br J Obstet Gynaecol* 1983 Mar;90(3):214–219.
46. O'Grady JP, Youngstrom P: Must epidurals always imply instrumental delivery? *Contemp Obs/Gyn* 1990;35:19–27.
47. Thorp JA, Parisi VM, Boylan PC, Johnston DA: The effect of continuous epidural analgesia on cesarean section for dystocia in nulliparous women. *Am J Obstet Gynecol* 1989 Sep;161(3):670–675.
48. Hoult IJ, MacLennan AH, Carrie LE: Lumbar epidural analgesia in labour: Relation to fetal malposition and instrumental delivery. *Br Med J* 1977 Jan;1(6052):14–16.
49. Newton ER, Schroeder BC, Knape KG, Bennett BL: Epidural analgesia and uterine function. *Obstet Gynecol* 1995 May;85(5 Pt 1):749–755.
50. Sleep J, Roberts J, Chalmers I: Care during the second stage of labour. In Chalmers I, Enkin M, Kierse M (Eds.). *Effective Care in Pregnancy and Childbirth*. Oxford University Press, 1989; pp. 1129–1199.
51. Russell JGB: Moulding of the pelvic outlet. *J Obstet Gynaecol Br Commonw* 1969;76:817–820.
52. Jeffcoate TNA, Martin RHL: Inefficient uterine action. *Surg Gynecol Obstet* 1952;95:257–273.
53. Weiniger CF, Ginosar Y. Changes in fetal position during labor and their association with epidural analgesia. *Obstet Gynecol* 2005 Sep;106(3):642.
54. Hillis DS: Diagnosis of contracted pelvis. *Ill Med J* 1938;74:131–134.
55. Compton AA: Avoiding difficult vaginal deliveries. In: Dilts PV, Sciarra JJ (eds): *Gynecology and Obstetrics*, Vol 2. Philadelphia: JB Lippincott, 1990;74; pp. 1–8.
56. Crichton D: A reliable method of establishing the level of the foetal head in obstetrics. *S Afr Med J* 1974 Apr;48(18):784–787.
57. Vacca A: *Handbook of Vacuum Extraction in Obstetric Practice*. London: Edward Arnold, 1992.
58. Philpott RH: Obstructed labour. *Clin Obstet Gynaecol* 1982;9:663–683.
59. Rayburn WF, Siemers KH, Legino LJ, Nabity MR, Anderson JC, Patil KD: Dystocia in late labor: Determining fetal position by clinical and ultrasonic techniques. *Am J Perinatol* 1989 Jul;6(3):316–319.
60. Rosen MG: *Management of Labor*. New York: Elsevier, 1990.
61. Cohen WR, Acker DB, Friedman EA (Eds): *Management of Labor*, 2nd ed. Rockville, MD: Aspen Publishers, 1989.
62. Cardoza L, Pearce JM: Oxytocin in active-phase abnormalities of labor: A randomized study. *Obstet Gynecol* 1990;75:152–157.
63. Bidgood KA, Steer PJ: A randomized control study of oxytocin augmentation of labour. I. Obstetric outcome. *Br J Obstet Gynaecol* 1987 Jun;94(6):512–517.
64. Seitchik J: The management of functional dystocia in the first stage of labor. *Clin Obstet Gynaecol* 1987 Mar;30(1):42–49.
65. Studd JWW, Cardozo LD, Gibb DMF: The management of spontaneous labour. In: Studd JWW (ed): *Progress in Obstetrics and Gynaecology*, Vol 2. Edinburgh: Churchill-Livingstone, 1982; p. 60.
66. Bottoms SF, Hirsch VJ, Sokol RJ: Medical management of arrest disorders of labor: A current overview. *Am J Obstet Gynecol* 1987 Apr;156(4):935–939.
67. Huszar G (ed): *The Physiology and Biochemistry of the Uterus in Pregnancy and Labor*. Boca Raton, FL: CRC Press, 1986.
68. Myers DA, Nathaniesz PW: Controversies in perinatal care. II. Biologic basis of term and preterm labor. *Clin Perinatol* 1993;20:9–28.
69. Cohen WR: The pelvic division of labor. In: Cohen WR, Friedman EA (eds): *Management of Labor*. Baltimore: University Park Press, 1983; pp. 41–64.
70. Rath W, Adelman-Grill BC, Peiper U, Kuhn W: Collagen degradation in the pregnant human cervix at term and after prostaglandin-induced cervical ripening. *Arch Gynecol* 1987;240(3):177–184.
71. MacKenzie IZ: The therapeutic roles of prostaglandins in obstetrics. In: Studd J (ed): *Progress in Obstetrics and Gynaecology*, vol 8. Edinburgh: Churchill-Livingstone, 1990; pp. 149–174.



72. Rayburn WF: Prostaglandin E2 gel for cervical ripening and induction of labor: A critical analysis. *Am J Obstet Gynecol* 1989 Mar;160(3):529-534.
73. Cochrane Database of Syst Rev: 2003 Birth Statistics Reports 54(2)2005:1-116.
74. MacDorman MF, Mathews TJ, Martin JA: Trends and characteristics of induced labor in the United States 1989-98. *Paediatr Perinat Epidemiol*. 2002 Jul;16(3):263-73.
75. Boulvain M, Kelly A, Lohse C, Stn C, Irion O: Mechanical methods for induction of labour. *Cochrane Database Syst Rev*: 2001.
76. Bricker L, Luckas M: Amniotomy alone for induction of labour. *Cochrane Database of Syst Rev* 2000; (4):CD002862. Review.
77. Bujold E, Blackwell S, Gauthier RJ: Cervical ripening with transcervical Foley catheter and the risk of uterine rupture. *Obstet Gynecol* 2004 Jan;103(1):18-23.
78. Karjane, NW, Brock EL, Walsh SW: Induction of labor using a Foley balloon, with and without extra-amniotic saline infusion. *Obstet Gynecol* 2006 Feb;107(2 Pt 2):234-239.
79. Gilson GJ, Russell DJ, Izquierdo LA, Qualls CR, Curet LB: A prospective randomized evaluation of a hygroscopic cervical dilator, Dilapan, in the preinduction ripening of patients undergoing induction of labor. *Am J Obstet Gynecol* 1996 Jul;175(1):145-149.
80. Dodd JM, Crowther CA, Robinson JS: Oral misoprostol for induction of labour at term: Randomized controlled trial. *Br Med J* 2006 Mar 4;332(7450):509-513.
81. Rayburn WF, Powers BL, Plasse TF, Carr D, Di Spirito M: Pharmacokinetics of a controlled-release misoprostol vaginal insert at term. *J Soc Gynecol Investig* 2006 Feb;13(2):112-117.
82. Bishop EH: Pelvic scoring for elective induction. *Obstet Gynecol* 1964 Aug;24:266-268.
83. Boylan PC, Parisi VM: Effect of active management on latent phase labor. *Am J Perinatol* 1990 Oct;7(4):363-365.
84. Seitchik J: The management of functional dystocia in the first stage of labor. *Clin Obstet Gynecol* 1987 Mar;30(1):42-49.
85. Wein P: Efficacy of different starting doses of oxytocin for induction of labor. *Obstet Gynecol* 1989 Dec;74(6):863-868.
86. Satin AJ, Hankins GDV, Yeomans ER: A prospective study of two dosing regimens of oxytocin for the induction of labor in patients with unfavorable cervixes. *Am J Obstet Gynecol* 1991 Oct;165(4 Pt 1): 980-984.
87. Gee H, Olah KS: Failure to progress in labour. In: Studd J (ed): *Progress in Obstetrics and Gynecology*, Vol 10. Edinburgh: Churchill-Livingstone, 1992; pp. 159-181.
88. Dodd JM, Crowther CA, Robinson JS: Morning compared with evening induction of labor: A nested, randomized controlled trial. *Obstet Gynecol* 2006 Aug;108(2):350-360.
89. Rogers RG, Gardner MO, Tool KJ, Ainsley J, Gilson G: Active management of labor: A cost analysis of a randomized controlled trial. *West J Med* 2000 Apr;172 (4):240-243.

## Lucy A. Bayer-Zwirello

*This indeed is the unforgiving stage of labor, and in there lurks more unheralded treachery than in both the other stages combined. The normal case can, within a minute, become abnormal, and successful delivery can turn swiftly to disaster.*

I. Donald (1910–1987)

*Practical Obstetric Problems*

London: Lloyd-Luke, 1979, 5th edition, p 748.

The process of placental delivery and the subsequent involution of the uterus during the puerperium are often described as the third and fourth stages of labor, respectively. Obstetric complications during these periods are common and occasionally serious. This chapter presents a brief historical review concerning third- and fourth-stage events, followed by a discussion of the physiology of placental separation and uterine involution. The management of common complications and techniques for the repair of superficial and deep perineal injuries are also reviewed. The diagnosis and treatment of retained placenta and membranes (secundines), uterine inversion, postpartum hemorrhage and atony, and hematomas are also considered. Finally, specific recommendations for best practice are made.

### HISTORY

The same issues and controversies concerning third- and fourth-stage management that exist in modern practice were faced in the past by practitioners from all cultures. Although contemporary approaches employ drugs and surgical procedures that are more effective than those used by our predecessors, the clarity of the descriptions and the sensible clinical management of the best of these earlier practitioners remain unrivaled. Reading their original descriptions impresses the reviewer with both their clinical competence and how well they succeeded in many dire situations despite the severe limitations imposed by the medical science and pharmacology of their times.

In the 17th century, the renowned French accoucheur, François Mauriçeau (1637–1709), in his textbook of clinical cases entitled *Observations sur la Grossesse et L'Accouchement des Femmes*, reported no less than 45 cases of postpartum hemorrhage caused by retained placenta (*arrière-faix retenus*) or retained membranes (*membranes retenues*) [1]. He observed that these complications were associated with early fetal demise (at 5–6 months of gestation) and reported that some led to death of the mother from catastrophic hemorrhage

or infection. He also discussed other problems, including uterine inversion, which he felt was due to incompetent midwifery and overzealous intervention.

In his review of postpartum hemorrhage and atony, Mauriçeau astutely related these complications to macrosomia, multiple gestations, post-datism, intrauterine fetal demise, uterine inversion, and uterine rupture. To better appreciate the difficulties that Mauriçeau and his contemporaries worked, it is important to remember that these practitioners lacked anesthesia, effective uterine relaxants, potent uterotonics, atraumatic delivery instruments, or the ability to transfuse. As treatment for hemorrhage, Mauriçeau recommended the judicious use of version and extraction, and prompt manual removal of the placenta, as required. Probably less effectively, he relied on "quinquina" and the use of leeches.

Mauriçeau had tragic personal experience with obstetric hemorrhage. He was called to attend his own sister, who had sustained sudden and serious bleeding from a placenta previa. When the hemorrhage did not abate and the other birth attendants refused to act, Mauriçeau delivered her by manual cervical dilatation followed by version and extraction. This was the accepted method of treatment at the time and a procedure in which Mauriçeau was an acknowledged master. Unfortunately, in this case despite his best efforts, his sister died.

Jean-Louis Baudelocque (1746–1810) combined the best of classic French obstetric teaching with new ideas derived from the developing English school led by William Smellie (1697–1763) and his contemporaries. In discussing management of third-stage complications, Baudelocque reported a case in which an accoucheur vainly tried to stem a postpartum hemorrhage. Failing in his quest for a suitable tampon, in desperation this practitioner tore off his wig and stuffed it into the unfortunate woman! This wigless and unnamed clinician was temporarily successful in arresting the observed hemorrhage; however, he could not prevent the eventual death of the woman from exsanguination. Thus, as Baudelocque tartly observed, the wig was "vainly sacrificed" [2]. His unfortunate colleague had treated only the symptom of the problem, rather than the cause.

The importance of the third stage of labor was also well recognized by the major 18th and 19th

century English practitioners, including William Smellie, John Bard (1716–1799), and the prominent American physician, William Potts Dewees (1768–1841). These clinicians believed that delay in the delivery of the placenta led to most postpartum complications; thus they taught that early intervention to ensure prompt placental delivery was the best management [3]. This encouraged routine intervention when placental delivery was not immediate, an approach that was likely not in the best interests of many women.

Important cultural and historical events in world history have been directly influenced by complications of involving the third stage of labor. The existence of the Taj Mahal (Crown Palace) in Agra, India is one example. The Taj Mahal is a remarkably beautiful white marble edifice, built over a nearly 20-year period. Reputedly, the construction required the efforts of 20,000 workers at the then remarkable cost of 32 million rupees. The Taj was constructed in honor of Mumtaz Mahal, wife and a grand multipara, who died in the year 1631 at age 39. The queen of the Mughal Emperor Shah Jahan (? –1666), Mumtaz Mahal died of a postpartum hemorrhage that occurred during her fifteenth pregnancy. Her mausoleum, the Taj, was situated in a riverside garden on a bend in the Jamuna River at the direction of her grieving spouse, so it could be easily seen from Emperor Jahan's personal palace at Agra Fort.

Postpartum hemorrhage and the failure of birth attendants to intervene when necessary have also played an important role in the history of the British royal family [4]. In 1817, Princess Charlotte, the only legitimate child of George III, died several hours postpartum after a long and difficult labor. The princess was attended by a prominent practitioner, Sir Richard Croft (1762–1818), a firm believer in nonintervention in the process of labor. After a more than 50-hour labor and the painfully slow delivery of a normal-appearing but stillborn male infant, the princess succumbed to postpartum hemorrhage, exhaustion, and dehydration. Croft was severely criticized for failing to intervene earlier with forceps, which were available, and to provide supportive care. Under the weight of this disapproval, he subsequently committed suicide.

With the death of the princess the English throne was suddenly without an immediate and legitimate

heir. Eventually, Edward, Duke of Kent, a 54-year-old bachelor then living with his mistress of 20 years was identified as the most likely candidate to sire an appropriate heir. He was forced to throw over his paramour and seek another, socially acceptable partner, Princess Victoria, widow of the Prince of Loiningen. Through this somewhat improbable union, in May of 1819 the new couple produced a daughter who in 1837 ascended the throne as Queen Victoria, the longest reigning of the English monarchs. (For additional information concerning the background of basic obstetric interventions, see Chapter 1, A History: Operative Delivery.)

## NORMAL THIRD-STAGE PHYSIOLOGY

### **Placental Separation and Physiology**

Complications of placental separation and delivery are frequent and responsible for important and potentially serious maternal morbidity and, rarely, mortality. Normal uterine physiology both expels the placenta and limits blood loss following delivery of the infant. The normal postpartum uterine contractions serve to promote placental separation, progressively occlude the major myometrial blood vessels, and autotransfuse the mother by expelling pooled blood into her general circulation. The fibrin that is subsequently deposited on the endometrial surface activates the clotting mechanism. These effects, the normal hypercoagulability of pregnancy combined with the direct occlusion of intramyometrial vessels by uterine contractions collectively result in local hemostasis and the restriction of postpartum blood loss.

The mechanism of placental separation is imperfectly understood. Most of the current knowledge comes from cases of hemorrhage that progressed to hysterectomy; however, a description of separation has been reported, using real-time ultrasound to visualize the activity of the myometrium and the changing uterine contour [5].

In response to the initial postpartum contractions, the size of the uterine cavity decreases rapidly within minutes of the delivery of the infant. The noncontractile placenta is thus progressively sheared from its attachment on the uterine wall and propelled into the lower uterine segment [5,6,7]. Beyond the simple change in the shape of the uterus, the formation of a retroplacental hematoma also promotes

normal placental separation. The hemotoma develops as the placenta is detached and spiral arteries are avulsed, leading to retroplacental bleeding. Control of this bleeding from the placental bed is caused by the unique anatomy of the myometrium. The progressive shortening of the intertwining fibers of the myometrium progressively pinch off and occlude arterialized feeding vessels underlying the placental site, thus limiting blood loss. These physiologic vessel ligations fail if the myometrium cannot or does not contract firmly, a condition that occurs with postpartum atony and subsequent hemorrhage. These observations emphasize the importance of both emptying the uterus so it can contract and ensuring its firmness in the control of primary atony, the most common type of postpartum bleeding.

It was previously but incorrectly believed that placental separation occurs at the basal layer along Nitabuch's stria; however, separation actually occurs in a layer deeper to the basal plate. Apparently, Nitabuch's stria remains mostly adherent to the placenta. The basal layer consists almost entirely of maternal cells, decidual glands, and other components of endometrial stroma. Some fetal cells are also found in this layer, mostly X cells – so-called because their origin was initially unknown [7–11].

In addition to the processes previously discussed, normal placental separation also depends on the normality of the underlying decidua at the implantation site. Animal studies reveal progressive histologic changes in the decidual spongy zone, commencing several days prior to delivery or labor. In humans, a comparable finding is seen in premature delivery, when the spongy zone decreases from 4 mm to 0.5 mm prior to the onset of labor [8]. Neither the mechanism for this change nor its role in normal separation is understood. When studied ultrasonographically, separation is also heralded by decreased blood flow to the placental base [5,6,7]. If this blood flow decrease is not observed, it could be a sign of abnormal placentation, such as a placenta accreta.

### **Physiology of Uterine Involution**

*Postpartum uterine involution* (the fourth stage) is another little-understood physiologic process. Most information concerning involution comes from histopathologic studies of lochial fluid and

lochial-decidual remnants, or from examination of hysterectomy specimens. Because of the usual postpartum myometrial contractions, the uterus rapidly decreases in size, and the uterine cavity is deformed, causing deep furrowing in its inner surface. Following placental separation, the uterine cavity is rapidly covered by a fibrin layer. The fibrin that is deposited forms a thick mesh in which deformed erythrocytes are trapped [8,13]. This process presumably enhances local hemostasis, complementing the "physiologic ligations" of the placental perfusing vessels due to uterine contractions.

Postpartum, most of the residual endometrial/decidual lining undergoes necrosis. Regeneration subsequently occurs from the residual glands and stroma. Although nonplacental site endometrium appears grossly intact by the 16th postpartum day, this process requires 28 days or more for completion. The placental site can take several additional weeks to completely return to normal. This delay is presumed to be caused by the slow resolution of thrombosed vessels at the implantation site. Anderson and Davis [14] studied the placenta site prospectively and demonstrated that it is still identifiable up to 11 weeks postpartum, although much reduced in size. The placental implantation site decreases from 9 to 10 cm in size at delivery, to approximately 1 to 2 cm at 11 weeks postpartum [8,14–16].

The myometrial cells that occlude rapidly shrink in size in the puerperium. Within several weeks, the uterus decreases in weight from 1,000 g to a mere 100 g. In the poorly understood clinical condition of *postpartum subinvolution*, the uterus remains enlarged, and episodes of intermittent but limited bleeding are common. For unknown reasons, in subinvolution the normal regression of the myometrial cells does not occur, and the endometrium stops regenerating. When subinvolution exists, the clinical history is commonly that of recurrent episodes of moderate bleeding. On physical examination, the uterus feels excessively large, is often described as "boggy," and can be slightly tender to palpation.

Occasionally during the process of involution, small areas of retained placental tissue coalesce to form combinations of placenta, fibrin, and clot, termed a *placental polyp*. Such polyps can be a source of delayed postpartum bleeding [17–20]. Histologic evidence of inflammation, marked by superficial plasma cell infiltrates, phlebothrombosis, and the

presence of bacteria, is also common in subinvolution specimens.

## MANAGEMENT OF THE THIRD STAGE

### Routine Technique

#### *Delivery of the Placenta*

Immediately after the infant is expelled, the uterus initially relaxes. Contractions then resume several minutes later, and as has been discussed, acute changes in uterine shape results in the separation of the placenta from its insertion site. Clinically, separation is usually heralded by a sudden gush of blood as the retroplacental hematoma escapes, an event accompanied by observed lengthening of the cord. Palpation of the uterine fundus can also reveal when separation occurs owing to the rapid change in uterine contour, from ovoid to round. In addition, the uterus usually rises in the abdomen as well. Placental separation can be easily confirmed by pelvic examination, even in a woman lacking anesthesia [21]. The operator's index finger is gently inserted into the introitus, passed into the vagina, and through the open cervix. If separation has occurred, the placental edge is easily palpable. If a partial or incomplete separation has occurred, the uterus might have contracted around the placenta, partially entrapping it. If this has happened, the surgeon feels the bulk of the placental mass in the vagina, whereas the cervix remains high and difficult to palpate. To relieve this condition, the accessible placental mass is simply grasped in the operator's hand and removed with moderate but continuous traction, with or without a twisting motion. Pharmacologic uterine relaxation with a parenteral betamimetic or nitroglycerine is infrequently required to facilitate this process.

To assist normal placental delivery, a constant but not forceful tension on the cord in the axis of the birth canal is performed while the uterus is pressed upward above the pubic symphysis (Brandt-Andrews maneuver) [24,25–26]. Direct cord traction should not be conducted without this concomitant upward manual countertraction. Excessive cord tension should be avoided because it can cause umbilical cord avulsion or possibly contribute to uterine inversion [21–23]. The problem of uterine inversion is discussed later. With active traction, avulsion of the cord occurs in approximately 3% of deliveries [23]. Avulsion is not a serious

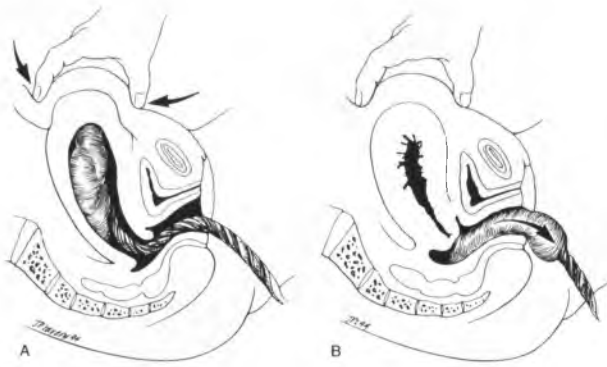


FIGURE 11.1.  
*Placental delivery I. (A) Brandt-Andrews maneuver; (B) vaginal placental delivery, Schultz mechanism.*

misadventure, but it leaves the surgeon without a point of leverage and predisposes to manual removal of the placenta. Cord avulsion can also herald a placenta accreta.

After separation, the final delivery of the placenta is usually performed by gently elevating the uterus out of the pelvis with the abdominal hand while providing gentle umbilical cord traction to lift the placenta out of the birth canal (Figure 11.1) [24–27].

Some attendants complete the delivery of the placenta and membranes by rapidly twisting the placenta to roll up the membranes at the last moment as the placenta is removed from the vagina. Although this technique is popular, it is not necessarily an improvement on simply lifting the placenta out, and it can spread rectal contaminants into the perineal or introital area. It therefore is not recommended. Retained membranes can be easily removed by grasping them with a ring forceps or Kelly clamp and pulling gently.

When expressing the placenta, the author does not recommend continuously kneading the fundus (Credé method) to promote separation, because this can predispose to hemorrhage, inversion, or trauma. Limited massage is acceptable, however. The placenta usually delivers inverted with maternal side on the inside (Schultz mechanism; see Figure 11.1B). Sometimes this does not occur, however, and the maternal side appears first (Duncan mechanism). There is no specific clinical significance to either delivery method of observation.

After delivery of the infant, if minimal bleeding occurs, the fundal examination is normal, and maternal vital signs are stable, some physicians choose to

repair the episiotomy or other lacerations before the placental delivery. With a delayed placental delivery, or especially if manual removal becomes necessary, a completed or partial perineal repair can be disrupted, however. Nevertheless, early repair of episiotomy or perineal lacerations reduces blood loss, and a subsequent spontaneous placental delivery usually does not disrupt the repair as long as a manual extraction is not required. Therefore, because a retained placenta is uncommon, many clinicians favor proceeding with any necessary repairs while awaiting separation. Either approach is acceptable. Episiotomy and episiotomy repair are discussed in greater detail later.

### RETAINED PLACENTA

The median time of placental delivery is 6 minutes. Fully 95% of spontaneous placental deliveries occur within 30 minutes of delivery of the infant. The author's practice is to infuse 10 IU to 20 IU of oxytocin in 1L of lactated Ringer's or a similar balanced salt solution immediately after the delivery of the infant, to prompt uterine contractions and accelerate placental separation. Oxytocin is preferred to ergot derivatives, because the drug is safer and fewer cases of retained placenta result [31–37].

If the placental delivery is tardy or if bleeding develops, manual removal is indicated (Figure 11.2). Before the attempt, it is prudent for the operator to change gloves to reduce the risk of contamination. The procedure should be briefly explained to the parturient, and the clinician must ensure that an acceptable degree of anesthesia/analgesic is necessary. The maternal vital signs are checked and a secure, large-bore intravenous line inserted if one is not already in place. The clinician should consider moving the parturient to an operating suite. A general, low spinal or epidural anesthesia is usually required for this procedure. In cooperative patients, uncomplicated manual removal of the placenta can be performed under intravenous analgesia or conscious sedation, but this is not possible in all cases. Before administering the anesthetic, the uterus and cervix should be examined for a simple cervical closure or for a constriction ring that could have entrapped the placenta (Figure 11.3). If either is present, the administration of 150  $\mu\text{g}$  to 350  $\mu\text{g}$  of nitroglycerine IV or 250  $\mu\text{g}$  of terbutaline SC assists removal. If terbutaline is chosen, uterine relaxation

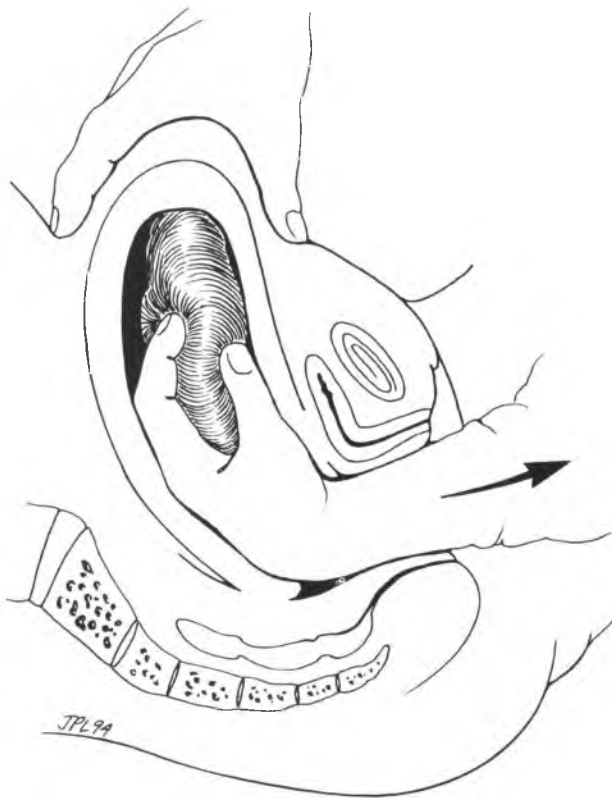


FIGURE 11.2.  
*Placental delivery II. Manual removal.*

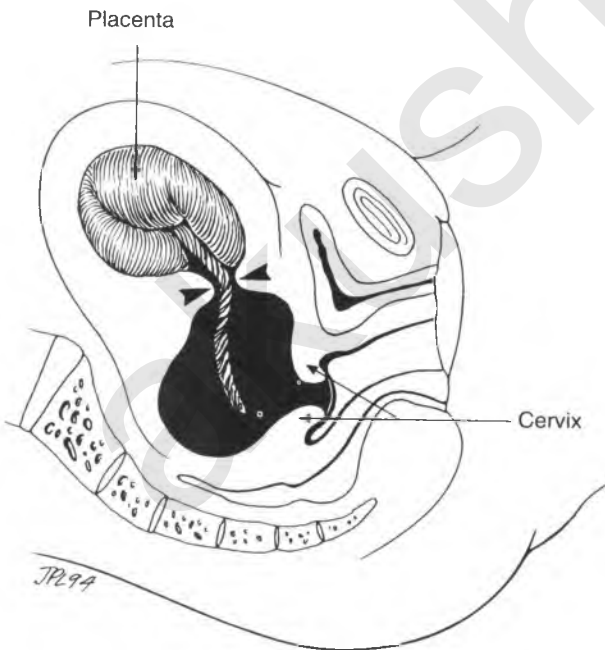


FIGURE 11.3.  
*Placental retention associated with Bandl's ring (arrows indicate constriction site).*

usually occurs within 2 to 3 minutes of the administration of the drug. When the placenta is detached but entrapped, myometrial relaxation normally permits an easy manual removal.

In terms of technique, the surgeon's hand passes through the cervix and up into the uterine cavity. If the placenta remains partially or completely adherent, it is finger dissected away from the uterine wall. The mass of placenta is then grasped and removed from the uterus. A slow and steady pressure is best to help avoid placental fragmentation. After removal, a uterotonic is administered parenterally. Close attention to the possibility of secundines is necessary, as some degree of placental disruption is common with a manual removal. Failure of easy placental separation can be due to incomplete cervical dilation, inadequate analgesics or anesthesia, partial or complete placenta accreta or, very rarely, to the more advanced forms of placental adherence such as placenta increta or percreta. If the cervix is not widely dilated, precluding a complete examination, or if the placenta is difficult to remove, the procedure is terminated. The parturient should then be moved to an operating room and an anesthesiologist summoned, because additional procedures will be required. Management of the abnormally adherent placenta is discussed in a latter section.

### POSTDELIVERY EXAMINATION

Once the stability of the mother and baby are ensured, the placenta, membranes, and cord should be routinely examined. Gross placental examination is best performed by picking up the placenta with both hands on the fetal side in the same manner as passing a dish. Curling the operator's fingers upward allows the placenta to assume a bowl shape, fetal side up, facilitating the examination. The placenta is first examined for intactness. The accoucheur should note torn or incomplete edges, or ruptured peripheral vessels, suggesting missing or fragmented cotyledons. The placenta should then be turned over and the edge again closely scrutinized. Vessels passing to the periphery of the disk with ragged or avulsed edges suggest a missing or succenturiate lobe or a velamentous insertion. The cord length, gross appearance, and weight of the placenta should be estimated [28–30]. If abnormalities are suspected, the cord is examined for obvious knots, hematomas, or other lesions. Membrane abnormalities such as a circumvallate placenta, opacity, staining

with meconium, or a furcated insertion should also be reported in the medical record. The cut end of the cord is then examined, and the number of vessels noted. The observation of a two-vessel cord is important because it has a variable association with fetal anomalies; if noted, the pediatrician should be notified [8,29]. In complicated cases, if the immediate condition of the neonate is poor or uncertain, or if the cord is very long (>70 cm) or short ( $\leq 35$  cm) or there are other anomalies noted, the placenta should be submitted for examination by the pathologist [30]. As is discussed later, much information concerning events that could have affected fetal growth and development can be derived from gross and microscopic placental examination.

The intentional placental drainage of fetal blood can reduce the length of the third stage, but the effect is not marked [38,39]. If drainage is contemplated, be certain that a twin gestation is not present. In theory, drainage of the cord of one twin might result in at least partial exsanguination of the second fetus if vascular connections exist between the two fetal circulations (monochorionic twinning). Beyond the potential effects of drainage, there are data to suggest that the injection of an oxytocin solution (e.g., 10 IU diluted in saline) directly into the umbilical vein *might* accelerate placental delivery in cases of retention [40–45]. Small studies of cord injection have suggested that blood loss is significantly reduced in normal term patients if such cord injection is performed [44]; however the evidence for this effect was not found compelling in the recent Cochrane review [45]. Because the supporting data for these practices are quite limited, the author does not recommend either routine drainage or injection. In the setting of placental retention for 30 minutes or more without significant bleeding, when the alternative is administration of an anesthetic and manual removal, it is reasonable to attempt either injection or drainage while preparations are made for operative placental removal. The maternal risk is minimal, and success can avoid potentially complex obstetric manipulations. Also, in the absence of another specific indication the author does not routinely administer antibiotics following manual extraction.

### **ACTIVE MANAGEMENT OF THE THIRD AND FOURTH STAGE**

Active management of the third stage of labor consists of the immediate administration of oxytocin

after delivery of the infant, early cord clamping, and gentle traction on the cord, combined with gentle uterine massage to prompt placental separation. The basic components of this technique have been adopted in many centers. There are good data that show that active third-stage management shortens the process of placental delivery and significantly reduces the risk of postpartum hemorrhage [32,33]. Five clinical trials have documented an approximate 60% reduction in the incidence of postpartum hemorrhage (defined as estimated loss of greater than 500 ml) when active management is performed. In these studies, active management also reduced the need for the subsequent administration of additional therapeutic uterotonics by 80%. Declines in maternal hemoglobin values to less than 9 g and the requirement for transfusion were similarly reduced. Thus, 1 of every 67 parturients undergoing active management avoids possible transfusion. Furthermore, for every 12 deliveries following the protocol, one potential case of PPH is prevented. Active management does not alter the risk of placental retention, however [35].

Although the routine use of intravenous oxytocin postdelivery is recommended, this is not the only possible treatment protocol. Several studies reported through the Cochrane Database confirm that the postpartum administration of oxytocin with the drug syntometrine versus dilute oxytocin alone results in a small but statistically significant reduction in the rate of PPH [36]. This positive effect does extend to blood losses exceeding 1000 ml, for which these agents are apparently of equal efficacy. Because syntometrine, a fixed oxytocin (5 IU) and ergometrine (0.5 mg) is a combination drug and is not available in the United States, it is difficult to translate these data into clinical practice.

Uterotonics are potent pharmacologic agents and must be administered with care. Intravenous bolus (nondilute) administration of oxytocin is not recommended; this dosing can result in rapid alterations in maternal blood pressure, with episodes of severe hypotension possible. Oxytocin is best administered in dilute intravenous infusions only. For routine postpartum administration, the author favors the addition of 10 IU to 20 IU of oxytocin per 1000 ml of Ringer's lactate, normal saline, or another balanced salt solution. It is best to begin with a rapid infusion of the dilute solution over 10 to 15 minutes until the uterus firms to palpation, then reduce the rate to 125 ml/hr to 150 ml/hr.



Intramuscular ergot derivatives such as methergonovine maleate (Methergine) can be administered *after* the delivery of the placenta, as an alternative to dilute intravenous oxytocin. For this indication ergots offer no specific advantage and have other potentially important side effects, however. Ergot preparation can predispose to placental retention and can cause nausea, vomiting, and elevations in arterial pressure, side effects largely absent with oxytocin [36,37]. The ergots are, however, an excellent adjuvant therapy for maintenance oral treatment after a postpartum hemorrhage is controlled. Because of their potent effects, these compounds should *never* be administered to known hypertensive or preeclamptic women.

For these reasons, despite their efficacy, the administration of the ergot derivatives is best limited to selected postpartum cases when oxytocin has failed and the bleeding is excessive. Although ergot has been used in medicine since the nineteenth century [46], newer uterotonics, such as the prostaglandin derivatives, have been readily available for clinical use only since the 1980s. Some of these new compounds have been used in treating postpartum atony; however, few controlled studies have employed them in active management of the normal third stage. The prostaglandins have been found to be effective in shortening the third stage and preventing hemorrhage but have not offered any specific advantage over oxytocin in *routine* management [47–50]. Several of the prostaglandin compounds, carboprost (Hemabate, 15-methylprostaglandin F2 alpha IM), and prostaglandin E2 by suppository (Prostin) are restricted in use to cases of serious postpartum hemorrhage/atony or in the induction of abortion. Potentially dangerous complications, including bronchospasm or anaphylaxis, are more common with these prostaglandin derivatives than with the other major uterotonics, oxytocin [51] or misoprostol [48]. In recent years, misoprostol (PGE1, Cytotec) has become the most popular of the prostaglandin derivatives. Misoprostol has been administered for labor induction and as well as a substitute for methergonovine maleate in the acute treatment of postpartum atony [47]. Misoprostol has the advantage that it does not promote bronchospasm as it is a bronchial muscle relaxant. Given in doses of 800 µg to 1,000 µg rectally, misoprostol can be effective in the prevention of postpartum

hemorrhage and result in reduced blood loss. In randomized trial, however, its efficacy versus placebo has questioned. Further, side effects such as shivering were more common in comparison to placebo [48–50].

Other controversies in third-stage management are the benefits or risks associated with early versus later cord clamping and placental drainage. Draining the placenta after delivery can decrease the risk of fetomaternal blood transfer (from 10.2% to 7.9%), but as noted previously, the effects on separation are less clear [55,56]. Early cord clamping leads to heavier placentas (higher mean residual blood volume) but has no significant clinical importance for the mother. For the infant, a lower incidence of respiratory distress syndrome, possibly lower levels of childhood anemia and greater iron stores, are potential benefits reported with delayed clamping [52–54]. The clinical importance of these claimed benefits in otherwise normal cases is unclear and probably limited. A normal child has a sufficient red cell mass and increasing it iatrogenically is of no benefit and can be of some potential harm. The problem is that delayed cord clamping or cord stripping transfers a significant volume of unneeded blood to the fetus. In otherwise normal neonates, forced transfusion can result in polycythemia and increase the risk for hyperbilirubinemia by increasing the amount of hemoglobin in the neonate's circulation.

This is a situation when the paucity of data indicating significant harm should permit flexibility. In counseling families anticipating uncomplicated term deliveries, clinicians should try to dissuade the parents from cord stripping. Because the effects of the timing of cord clamping have not been subjected to extensive study, neither the risks nor the benefits should be exaggerated, and the rules of reasonable behavior should apply. Stripping or milking the cord to increase blood transfer in otherwise normal deliveries of term infants should be discouraged. These effects are of questionable efficacy, and this procedure is specifically not recommended as routine. Holding the newborn below the level of the placenta for "autotransfusion" is effectively the same as cord stripping and is also not advisable routinely.

If the parents strongly desire to position the child in some manner that they believe to be appropriate, or to delay cord clamping until pulsations cease, these requests can be followed at little if any real risk

to mother or infant, unless contraindicated by specific clinical circumstances.

There is a situation when it is best to clamp the cord promptly after delivery. Specifically, this is when cord blood is electively collected for banking. In this situation, the cord is clamped promptly and the blood is subsequently collected by simple drainage via needle tubing leading to a blood collection bag. This will normally permit up to 150 ml to be withdrawn from the placenta. For an otherwise normal neonate this early cord clamping is essentially risk free. When cord blood collection is planned in a multiple gestation, blood removal must wait until all the infants are delivered. Because vascular connections between twins or greater multiples are reasonably common, the removal of blood from one cord has at least the theoretical potential to drawn volume from the undelivered infant(s). Thus, the delay in moving to cord drainage is prudent until after the delivery of the last infant.

## Episiotomy

### *Episiotomy Technique*

The role of episiotomy in routine practice has been hotly debated, especially in recent decades. It is now generally accepted that the routine episiotomy increases the risk of third- and fourth-degree perineal tears, without demonstrated benefit in protecting the integrity or function of the muscles and connective tissues of pelvic support [57,59]. In the United States, when an episiotomy is performed, the median incision is favored, whereas in Europe the mediolateral is preferred. The median incision has a better cosmetic result and generally results in less pain. Unfortunately, median incisions predispose to extensions posteriorly into the rectal sphincter and rectum. In contrast, the mediolateral incision is more painful, heals with more difficulty, and is more likely than the median to result in permanent distortion of the perineum and long-term dyspareunia. Although the mediolateral incision reduces the risk of anal sphincter injury, it does not entirely exclude it. (See Chapter 23, Birth Injury, for additional discussion.)

Episiotomy incisions are traditionally performed with scissors, although an occasional practitioner favors the use of a scalpel. The use of bandage scissors is discouraged. A Mayo scissors is usually eas-

ier to manipulate and offers greater flexibility in extending the vaginal epithelium cephalad. In the usual technique, a local anesthetic agent such as lidocaine is administered into the perineum, unless another form of anesthesia is already present. To perform the incision, one blade of the scissors is placed between the presenting part and vaginal epithelium, with the other blade resting on the perineal skin. The presenting part is protected by the surgeon's finger while the internal blade is guided to the correct depth and angle to avoid inadvertent extension into the anal sphincter. After the initial cut is made, the surgeon's guiding finger protects and directs subsequent small midline cuts toward the vaginal apex, as required.

After delivery, a careful inspection of the entire birth canal is mandatory, including close observation of the episiotomy site for occult lacerations of the vagina and cervix. The integrity of the rectal mucosa and sphincter must also be routinely evaluated by a digital rectal examination. This examination carefully explores for hidden "buttonhole" defects in the rectal wall, which might not be detected by visual examination alone.

If tears or lacerations are present, their extent and extension are gauged and the parturient evaluated for the extent and acceptability of anesthesia. If an extensive repair is necessary or adequate light or exposure is a problem, transfer to an operating suite is best. During routine repairs, small sponges should not be used because they are all too easily forgotten. Instead, only vaginal obstetric tampons or laparotomy sponges with an attached tie or tape clamped to a Kelly or similar small clamp are appropriate for insertion into the birth canal. In the author's institution, all sponges must be counted by the clinician at the end of the delivery. This requirement and the avoidance of the use of small sponges have essentially eliminated our prior difficulties with the occasional retained vaginal sponge and unhappy parturients.

## **SURGICAL REPAIRS OF PERINEAL AND PERIURETHRAL INJURIES**

### **Overview**

Common birth canal injuries following instrumental or spontaneous delivery include superficial soft-tissue abrasions, ecchymoses, and minor lacerations.

Midline episiotomy increases the risk for posterior extensions into the rectum (fourth-degree laceration) or rectal sphincter (third-degree laceration). In multiparous women or in the occasional nullipara, slow and gentle fetal extraction, with attention to control of the fetal head and maternal coaching, can often avoid both episiotomy and laceration. Postpartum ultrasound examination of the rectal sphincter suggests that occult tears occur spontaneously in 15% to 25% of parturients with otherwise normal vaginal deliveries. Most of these parturients are asymptomatic. The long-term effects of such injuries remain to be elucidated [61].

In the literature of birth management, the importance of avoiding periurethral and anterior vaginal vault lacerations is underemphasized [62]. Failure to apply traction in the correct pelvic curve, faulty application of Ritgen's maneuver, and in some cases no episiotomy with rapid delivery over a firm or unyielding perineum predispose to anterior or periurethral lacerations. When timed correctly, episiotomy does reduce injury to these periurethral tissues, although there is risk of an extension into the sphincter or rectum. Periurethral lacerations, which often bleed freely, appear in the thin tissues on either side of the clitoris or urethra. Although repair is usually not difficult, suturing in this area commonly leads to a temporary inability to void and, uncommonly to long-term dyspareunia after healing. If bleeding occurs, prompt anatomic closure of the periurethral or paraclitoral lacerations with the minimal possible number of fine, absorbable interrupted stitches is best. Nonbleeding tears that do not gape can be left to spontaneous healing. Sitz baths and intermittent catheterization, as required, are additional appropriate therapies, but avoidance is the best management.

### Vaginal Lacerations

Most vaginal lacerations are small, superficial, and relatively easy to repair. If necessary, in cases involving jagged tears, the edges are best freshened with scissors prior to resuturing. Specific bleeding sites are either clamped for a few moments or suture ligated. Superficial oozing usually does not require specific suturing beyond tissue reapproximation. The normal anatomy is reconstructed employing the finest uninterrupted or continuous-suture material that will reapproximate the tissue (see Figures 11.4

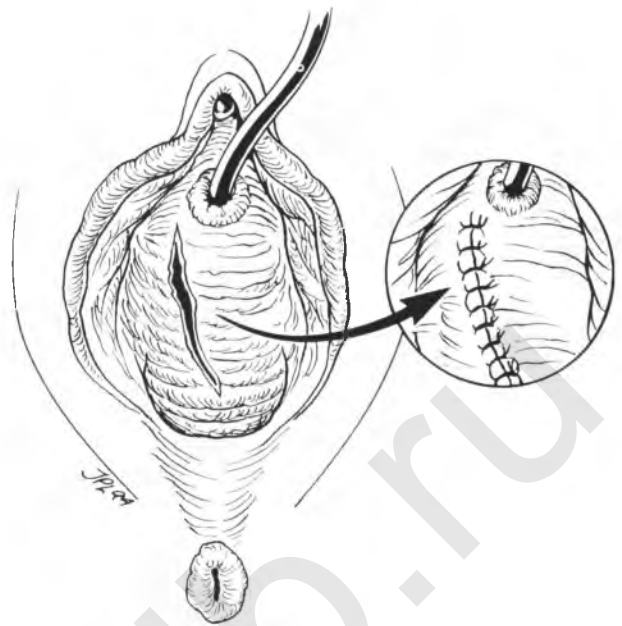


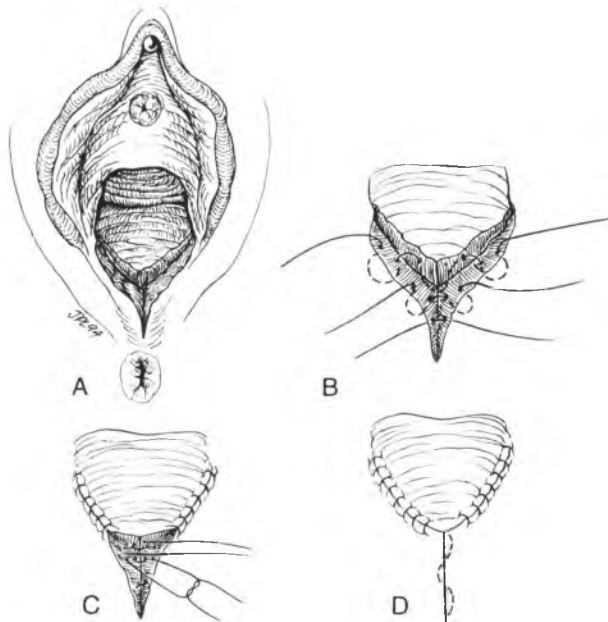
FIGURE 11.4.

*Repair of superficial vaginal laceration. A catheter is passed to ensure integrity of the urethra. The laceration is then reapproximated with fine interrupted sutures. Sitz baths and intermittent catheterization are frequently necessary postpartum. See text for additional discussion.*

and 11.5). If vaginal lacerations are extensive or are near the urethral orifice, urinary retention is common. The postpartum use of baths is recommended, and intermittent or even indwelling catheterization is sometimes required for relief, until the edema resolves and pain abates.

### Routine Episiotomy Repair

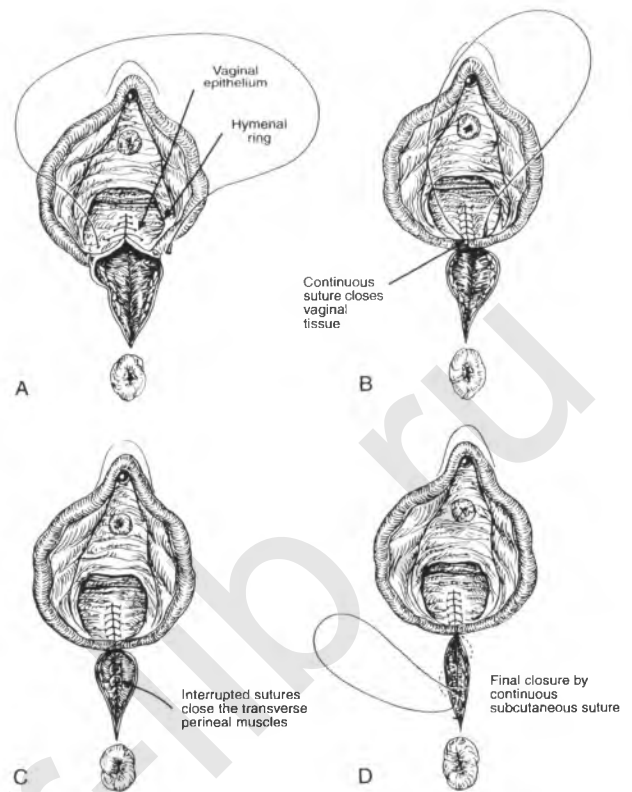
After an examination and the administration of appropriate anesthesia, the vaginal epithelium is closed [59,60] (Figure 11.6). A running, locking chromic, or a synthetic absorbable suture (preferred) of 000 or 0000 starting, approximately 1 cm above the apex, reapproximates the tissue. A polyglycolic suture or one of its newer, more rapidly reabsorbed derivatives is the author's recommendation. This closure reapproximates the anatomy and controls bleeding from the subepithelium. This suture continues to the level of the hymenal ring. In the most popular technique, the needle is then grasped by a Kelly clamp and temporarily put aside. Alternatively, this initial suture may be tied at this point after the operator pulls the suture through the



**FIGURE 11.5.**  
*Repair of vaginal/perineal laceration. After the integrity of the sphincter and rectal mucosa are verified, the laceration is closed with a combination of interrupted and running sutures to reapproximate normal anatomy and control bleeding (B–D).*

epithelium and tying so as to bury the knot. The perineal closure as described later is then performed.

Depending on the depth of the episiotomy and the distinctiveness of the anatomy, the pubococcygeus as well as the deep and superficial transverse perineal muscles can be individually reapproximated by the placement of one or more sutures. Placing two fingers in the vagina to push these muscles forward can improve repair technique. As formation of edema is inevitable, a snug but not tight closure is appropriate, and simple, uninterrupted sutures only should be used for this repair. At this point, the bulbocavernosus muscles, if avulsed and retracted, are reapproximated. In this repair, a stitch transfixes one bulbocavernosus, including some of superficial transverse perinei, and attaches it back to the normal position on the central perineal raphe. The original vaginal epithelial suture, or a new suture if the original were tied, is then passed under the mucosal dermal junction or started at this location and continued toward the anal orifice, closing subepithelial tissues. Usually, the same stitch is returned ventrally as a subcuticular closure. Interrupted single sutures can also be used at the sur-



**FIGURE 11.6.**  
*Repair of routine episiotomy. The vaginal epithelium is initially closed by a running suture (A) to the hymenal ring (B). This suture is usually tied, and the transverse perineal muscles are reapproximated by interrupted sutures (C). A final closure by a continuous subcuticular technique follows.*

geon's discretion to reapproximate deeper tissues of the perineum. In all of these repairs, the surgeon's aim is to arrest bleeding while accomplishing gentle and not overly tight reapproximation of normal perineal anatomy, closing dead space at the same time and leaving the smallest amount of suture material in the wound [60].

If "buttonhole" defects are detected in the rectal mucosa, they should be repaired in layers without tension. When such mucosal rents are detected, a complete and careful examination of the entire birth canal under good light and direct vision is mandatory. Rectal mucosa lacerations are often associated with other injuries either to the internal or external sphincter mechanism or both, and these must be sought. As long as the entire injury can be visualized and reapproximated in layers without undue tension, the original tear or incision does not necessarily have to be lengthened. All rectal mucosal

repairs should include two or more layers of tissue closed without tension above the site of the original rent.

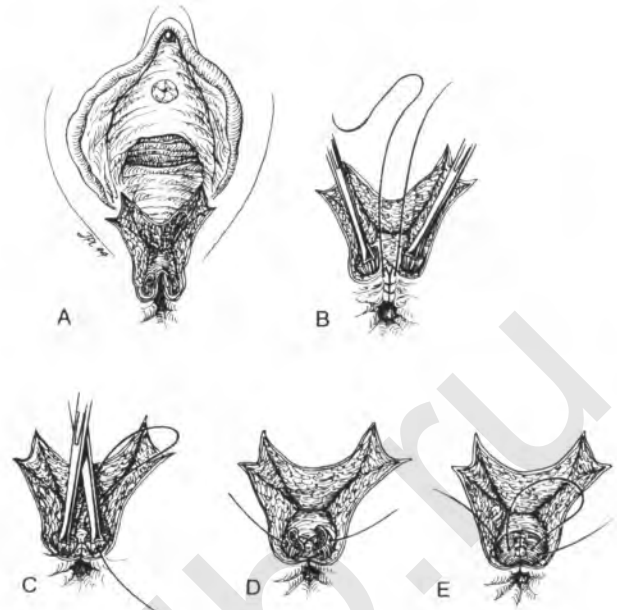
### Third- and Fourth-degree Lacerations

Repair of fourth-degree lacerations with proper identification of the various tissue layers can prove difficult owing to poor light, exposure, localized bleeding, or retraction of the various tissue planes. A sphincter injury must always be considered an important surgical issue. Such injuries are closed in layers using meticulous technique under the best light and retraction possible.

To repair a rectal mucosa tear, the apex is first identified. The mucosa is then reapproximated using a fine (000) absorbable suture, everting the tissue edges together. Through-and-through suturing of the mucosa is best avoided. This repair can be performed with the operator's gloved finger in the rectum to ensure that the suture does not transfix the mucosa.

This closure is followed by a second, imbricating layer of the same suture material. If bleeding is a problem, continuous irrigation assists in delineating tissue planes. It is usually best to simply press ahead with the repair rather than stop and attempt to control bleeding, unless specific bleeding vessels are identified. Closure of the appropriate tissue planes is usually rapidly hemostatic.

When the doughnut-shaped external sphincter (ES) has been severed (i.e., fourth-degree laceration), there is virtually always a laceration of the higher internal sphincter (IS) as well (Figures 11.7 and 11.8). It is now recognized that when the ES is repaired, whenever possible, reapproximation of the IS should also be performed. The IS is a less distinct, musculofascial tissue layer that lies above the ES. Usually identified by its thin white fascia that accompanies the muscle, this layer should be reapproximated by either an interrupted or a running nonlocking fine suture *before* the repair of the ES is begun. The IS layer sometimes retracts laterally but can usually be easily located, grasped, and drawn to the midline with an Allis clamp. Whether layered closure of both the IS and ES will improve healing and ensure retention of sphincter function better than the conventional technique, in which this layer was often not specifically identified or closed, is not known. The author favors the technique of IS iden-

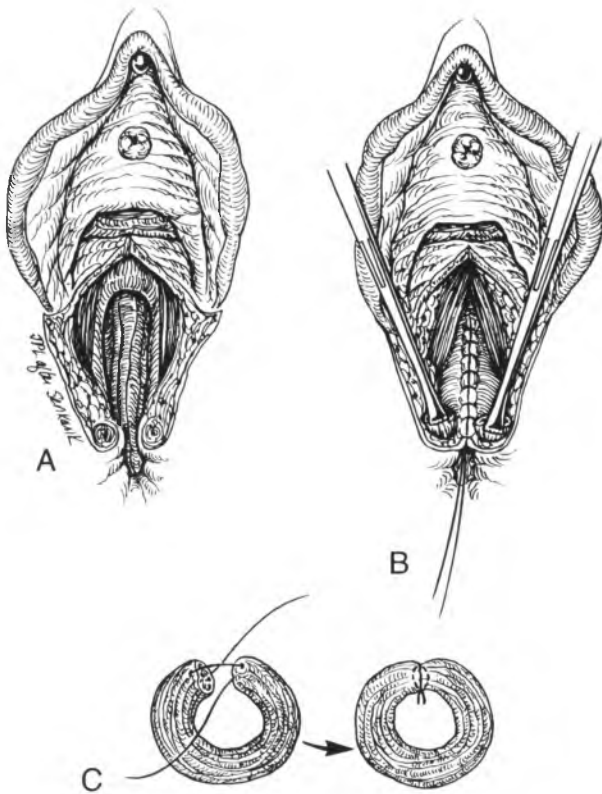


**FIGURE 11.7.** Repair of rectal sphincter (third-degree) laceration. The retracted ends of the external sphincter are grasped (A and B) and reapproximated by interrupted sutures (C and D). Adjacent fascia is closed, completing the sphincter repair. Overlapping repair, external rectal sphincter (E). Repair of episiotomy or any vaginal lacerations follows. See text for details.

tification and closure if possible because it seems to reconstruct normal rectal/perirectal anatomy better. (See Chapter 23, Birth Injury, for additional discussion.)

In the traditional repair of the ES, the fascial edge of the muscle is grasped, and simple, interrupted sutures of 00 or 000 polyglycolic acid (Vicryl) or PDS sutures are placed in the posterior, inferior, and superior aspects of the muscle bundle, taking care to incorporate the fascia. The free ends of the sutures are initially clamped and not tied, because immediate reapproximation obscures visualization of and access to the remaining muscle body and fascia. Thereafter, two or more additional simple sutures are placed anteriorly to complete the closure of the (ES) fascia of the sphincter muscle. The best technique for repair of laceration of the ES is currently unclear. Although most clinicians were instructed in an end-to-end closure technique, as outlined, overlapping techniques are becoming popular (see Figure 11.8).

Regardless of the method of ES closure, during the process of suture tying the operator's finger is



**FIGURE 11.8.** (A) Repair of fourth-degree laceration. The rectal mucosa is reapproximated by a running suture (B). The tissues for the internal sphincter are also closed as a separate Payer. Then, the retracted sphincter edges of the internal and external sphincter are identified, grasped, and reapproximated by interrupted sutures (C). See text for details.

inserted into the rectum to verify the circumferential tightening of the orifice as the defect is closed. Once placed, the tension of these sutures is adjusted during sequential tying to achieve reapproximate of the severed tissues without undue tension or strangulation. In the puerperium, antibiotics are administered at the clinician's discretion. In most cases stool softeners or bulk laxatives are also ordered. Rectal surgeons in Europe favor routine antibiotic use. In the United States, traditionally, antibiotics have not administered for a routine obstetric rectal sphincter repair but practice is changing. This is another technical point that awaits additional clinical investigation.

Current methods for the repair of third- and fourth-degree perineal lacerations are recognized as both inconsistent and inadequate. As long-term outcome studies verify, the traditional techniques

for repair of these injuries still result in a substantial number of long-term complications. There are two reasonable approaches to this problem. First, the focus must remain on the *avoidance* of posterior perineal injuries whenever possible. Second, we must learn from current prospective studies the best methods for repair of third- and fourth-degree lacerations as well as critically review the possible benefits of ancillary therapies, including the administration of antibiotics. Finally, the potential role for mediolateral episiotomy in selected cases, when the risk of rectal injury is high, requires additional investigation.

### Suture Material

The choice of suture material to reapproximate vaginal or cervical tears or to repair an episiotomy or rectal injury is at the surgeon's discretion. Despite theoretical considerations, infection of episiotomy or birth canal lacerations is uncommon and cannot be ascribed to the choice of suture material. Because of data concerning tissue reactivity and reports of perineal pain, the author prefers to use a polyglycolic acid or one of the new more rapidly dissolving derivative sutures for routine perineal repairs. Over the years, we have favored 3-0 polyglycolic acid (Vicryl) for most repairs and usually but not invariably employed 2-0 sutures for the reapproximation of the ES. Some practitioners now favor the use of fine PDS suture for sphincter repairs, believing that its longer tissue retention time better ensures complete healing. There are no reliable data on this point, however. The use of chromic suture material in the perineum is not recommended due to its high degree of tissue reaction. As always, control of bleeding, closing of dead space, leaving minimal residual suture material in the wound, avoiding tissue strangulation, and correct anatomic reconstruction are the surgeon's primary goals. These factors are more important to the final result than the choice of suture material.

### Issues Concerning Episiotomy

Episiotomy during vaginal childbirth was once routine and is still a common procedure in American obstetric practice. Perineal incision to assist delivery was apparently first described by Fielding Ould in his treatise of midwifery in 1742 [63]. The

term *episiotomy* was coined by Carl Braun in 1857 and specifies a surgical procedure for incising and thus enlarging the vaginal introitus during childbirth [64]. Anna Broomall brought the technique of median episiotomy to America from Austria in the late 19th century [65].

With the shift to hospital delivery in the early 20th century and the popularization of episiotomy and prophylactic routine forceps delivery, the concept of medical management of birth changed radically from that of simple observation to one of active intervention. In 1918, Pomeroy advocated routine episiotomy for all nulliparas to limit the second stage and reduce pressure to the fetal head [66]. In conjunction with this idea, DeLee introduced the concept of prophylactic outlet forceps with episiotomy in 1920 to shorten the second stage, and, thus, it was believed, to better protect the infant from intracranial injury [67]. Thereafter, and virtually without scientific study, episiotomy became a standard American obstetric procedure. In retrospect, it is difficult to understand the near-universal acceptance of episiotomy for so many years. In later decades, it became enshrined as a belief that episiotomy had an even more important role in the avoidance of third- and fourth-degree lacerations and other injuries to the pelvic support tissues that were thought to predispose to long-term complications. In previous decades, the discussion in the medical literature concerning episiotomy addressed only alternative techniques for the performance or repair of the incision, not the need for the operation. The literature of recent decades has focused instead on scientific inquiry into the benefits, risks, efficacy, and safety of episiotomy, along with follow-up studies of the effects of childbirth and common complications of obstetric procedures on rectal sphincter function and pelvic support [68].

Several confounding factors affect the occurrence of perineal lacerations resulting from childbirth. These factors include previous vaginal delivery, fetal size and presentation, inherent tissue elasticity, operative vaginal delivery, type of anesthesia, duration of the second stage of labor, and, as noted, the type of episiotomy (midline versus mediolateral) performed [65–66,70,72,74–76].

Traditionally, and in the education of many older practitioners, the prevention of long-term pelvic floor dysfunction and uterine prolapse were cited as reasons for episiotomy. Labor and delivery were

understood to place a tremendous strain on the pelvic diaphragm and other pelvic support tissues. Clinicians had long associated obstetric trauma with both subsequent pelvic relaxation and rectal dysfunction. The evidence usually forwarded to support this contention includes claims of higher rates of pelvic relaxation among women of high parity than among women of low parity and associations between demonstrable anatomic pelvic floor abnormalities, parity, and symptoms such as urinary and rectal incontinence. Part of the motivation for recommending routine episiotomy was to limit the “physiologic” insult to the muscles and connective tissue of the pelvis from vaginal delivery and thus, in theory, to reduce the long-term sequelae of birth trauma [67,68,76,77].

In 1935, Aldridge and Watson studied 2,800 primigravidas and concluded that injuries to the pelvic floor were substantially decreased when midline episiotomy and prophylactic forceps were used [78]. The definitions of pelvic floor injury were not clearly defined, however, and the episiotomy rate in the group studied was 20%.

In 1955, Gainey [79] reviewed examination data on 2,000 women for trauma sustained during parturition. In his initial series of 1,000 patients, the deliveries were made without forceps or episiotomy, except for maternal or fetal indications. In a separate group of 1,000 patients, all deliveries occurred using routine outlet or low forceps with a right mediolateral episiotomy. Anatomic studies included evaluation of the urogenital diaphragm; the levators, vaginal wall attachments, including detachment of the urethra; cystocele, rectocele, and enterocele detachment; prolapse of the vaginal walls; and internal as well as external sphincter tone. Gainey concluded that with the exception of urethral detachment, pelvic damage was greater in the group delivered spontaneously without episiotomy. He claimed that each succeeding labor increased soft-tissue trauma, and that for multiparous women, if operative intervention did not occur, they showed significant increases in damage. In contrast, the patients who delivered operatively were observed to sustain less damage. He believed that the vagina was most vulnerable to injury and that detachment of the vagina from its retropelvic attachments and subsequent descent of the urethra and bladder neck were the most critical injuries. Thus, significant protection of the vagina and endopelvic fascia

attachment was claimed as a benefit of episiotomy. Gainey did not discuss third- and fourth-degree perineal lacerations because he routinely employed a mediolateral incision. This was by no means a randomized study, and the influence of observer bias is difficult to ascertain.

In 1946, Power [74] discussed the anatomic sequence of events and the mechanism of changes in the pelvic floor during parturition and defined trauma arising from childbirth as the principal cause of pelvic floor injury. He claimed that once the fetus advanced to the level of the ischial spines, the plane of origin of the pelvic floor, the levator and muscular segments were already stressed. Levator funneling having occurred early in nulliparous labor, he stated that obstetric management (i.e., episiotomy), at best might prevent trauma to tissue distal to the ischial spines, including the vagina and the endopelvic fascia. Power argued that an episiotomy that extended up into the vaginal canal, rather than down toward the perineal body, before the fetal calvarium distended the perineum, would decrease trauma to both the external anal sphincters as well as to the vagina and endopelvic fascia. This theory lacks supporting data, however. Study by endorectal ultrasound and 3D transperineal ultrasound reveals subtle tears in perirectal and other tissues after normal vaginal deliveries without evidence of unusual trauma [61]. Data connecting these occult injuries and long-term anal dysfunction are lacking, and there is no information to support the theory that episiotomy would prevent these lesions.

A continuing controversy with episiotomy is timing. Depending on how the extant data are weighted, early episiotomy might reduce injury to perivaginal and paravesical fascia, whereas late or outlet episiotomy results in reduced blood loss. Unfortunately, late episiotomy also predisposes to third- or fourth-degree lacerations [68].

In sum, the data claiming protection of pelvic fascia by episiotomy are difficult to interpret and in general methodologically unsound. Anal but not urinary incontinence seems largely limited to women who have experienced direct third- or fourth-degree tears. Labor is an important variable in injury to perineal supports. Recently, studies investigating pudendal nerve and external and internal anal sphincter damage suggest that most perineal damage is secondary to vaginal delivery and associated with macrosomic infants and instrumentation but

not necessary to episiotomy, unless there is an overt rectal tear.

Unfortunately, for traditionalists, the benefits classically ascribed to episiotomy – a reduced risk of perineal injury and easier repair, prevention of fetal cranial trauma, and protection of the pelvic floor muscle – are either poorly documented or undocumented in the medical literature [68]. None of these is currently accepted as a valid indication for the procedure.

The issue of the relationship between episiotomy and lacerations of the perineum was long debated but is now settled. Early reports claimed benefit for episiotomy in the reduction of third- and fourth-degree lacerations during delivery in nulliparas as well as in forceps-assisted deliveries [64]. Recent reports have yielded strikingly different data, however, with the near-universal observation of an increased incidence of third- and fourth-degree lacerations following performance of an episiotomy [68,69]. As an example, Shiono and coworkers [71] reported on 24,114 deliveries from The Collaborative Perinatal Project. Women who had midline episiotomies were nearly 50 times more likely to experience perineal lacerations than were women who had no episiotomy. In this same study, mediolateral episiotomies and use of forceps were associated with an eightfold increase in the incidence of perineal laceration. Finally, nulliparous women were ten times more likely than multiparous women to have an episiotomy, and the use of forceps in the absence of an episiotomy was rare.

Mediolateral incisions do reduce the risk of third- or fourth-degree lacerations but do not entirely exclude these injuries. Mediolateral episiotomy incisions have distinct limitations. They result in more postpartum pain, are technically more difficult to repair, provide a less satisfactory cosmetic result, and are associated more often with dyspareunia and distortion of perineal anatomy than are midline incisions [72]. How to best employ episiotomy and which type of incision is best if elective division of the perineum is indicated have not been established; both are topics are subjects of ongoing investigation.

Is there a correct answer concerning episiotomy? The traditional claims for episiotomy are not supported by the best recent data [68–71,75]. It appears that long-term adverse effects (specifically pelvic relaxation and incontinence) of pregnancy,



labor and vaginal delivery are more important, and the benefits of episiotomy are much less than previously believed [68,71,75,81]. The author believes that the obstetric surgeon should attempt to avoid episiotomy and episiotomy extensions whenever spontaneous and instrumental deliveries are performed. Despite previously held beliefs, no convincing data support the various protective claims long made for *routine* episiotomy; however, it is also safe to say that the last word on this issue is far from being written.

## COMPLICATIONS OF THE THIRD STAGE

### Postpartum Hemorrhage

Hemorrhage is a common complication of pregnancy and a leading cause of maternal morbidity and mortality [84–89]. The incidence of postpartum hemorrhage (PPH) is estimated to range from 5% to 10% of all deliveries, depending on definition. Approximately 5% of vaginal births are associated with a 1000-ml or greater blood loss [90]. Approximately 10% of maternal deaths in Western industrialized countries are due to hemorrhage. Maternal deaths from PPH are much more frequent in the Third World, and World Health Organization statistics suggest that as much as 25% of all maternal mortalities can be ascribed to this cause [86]. The goals of management during a hemorrhage are rapid control of blood loss, restoration of circulating volume, and the prevention of maternal cardiovascular collapse. As previously discussed, active management of the third stage with the routine administration of parenteral uterotonics can avoid many but not all cases of PPH.

*Early PPH* is defined as an episode of hemorrhage occurring within the first 24 hours following delivery. These episodes are largely due to uterine atony or retained products of conception [83] (Table 11.1) [87]. *Late PPHs*, defined as those occurring more than 24 hours after delivery but usually prior to 6 weeks after the parturition, are principally due to placental site subinvolution, a poorly understood condition that is usually associated with chronic inflammation, or from retained products (secundines) or placental polyps. There are well-recognized difficulties in the clinical estimation of the volume of hemorrhage, and the range for normal is wide. It is therefore best to define PPH based

TABLE 11.1 Potential Causes of Postpartum Hemorrhage

#### Early

- Placental:
  - Secundines
  - Placenta previa
  - Abruptio placentae/marginal sinus separation
  - Placenta accreta/increta/percreta
- Uterine:
  - Postpartum atony
  - Rupture
  - Inversion
- Birth canal injuries:
  - Uterine lacerations/rupture
  - Cervical lacerations
  - Vaginal or vulvar lacerations
- Uncommon causes:
  - Intrauterine fetal demise syndrome
  - Amniotic fluid embolism
  - Coagulopathies
  - Administration of heparin/warfarin (Coumadin)

#### Late

- Uterine
  - Subinvolution of placental site/placental polyps
  - Chronic endometritis
  - Secundines
  - Gestational trophoblastic disease

on clinical parameters, combining observations of maternal signs and symptoms with visual estimations of total blood loss.

Although every postpartum patient has some potential for puerperal hemorrhage, high-risk cases are identified based on events of labor and delivery, prior history, or preexisting medical condition. Women experiencing cesarean delivery, receiving general anesthesia, or with pregnancy complicated by amnionitis, preeclampsia, and protracted active phase or second-stage arrest disorders are at an increased risk for bleeding. In vaginal deliveries, multiparity, amnionitis, and overdistension of the uterus from multiple gestation, hydramnios, or the presence of placental abnormalities such as abruptio placentae or accreta are additional risk factors (Table 11.2). In selected high-risk patients with strong histories of prior atony or those in whom heavy blood loss is anticipated because of known coagulation or placental abnormalities, autologous antepartum

**TABLE 11.2 Clinical Associations: Postpartum Hemorrhage**

- 
- **Uterine atony:**
    - Tocolytic/anesthetic agents
    - Multiple gestations
    - High parity
    - Hydramnios
    - Fetal macrosomia/shoulder dystocia
    - Prolonged labor
    - Precipitate labor
    - Chorioamnionitis
  - **Uterine inversion:**
    - Complete
    - Partial
  - **Birth canal lacerations:**
    - Prolonged/precipitate delivery
    - Operative vaginal delivery
    - Episiotomy
    - Fetal macrosomia/shoulder dystocia
    - Breech extraction
  - **Placental complications:**
    - Antepartum hemorrhage
    - High parity
    - Prior cesarean delivery
    - Uterine (Müllerian) anomalies
  - **Uterine dehiscence/rupture:**
    - High parity
    - Prolonged, obstructed labor
    - Trauma
    - Operative vaginal delivery
    - Previous hysterotomy scar
    - Breech extraction/internal podalic version
  - **Coagulopathy:**
    - Administration of heparin/warfarin(Coumadin)
    - Abruptio placentae
    - Amniotic fluid embolism
    - Septic shock
    - Prolonged intrauterine fetal demise
    - Hereditary coagulation defects
- 

blood donation for potential delayed transfusion is appropriate.

Healthy women with normal vascular volume and red cell mass and good prior nutritional status can tolerate substantial blood losses surprisingly well. In contrast, women of poor nutritional status, marked anemia, or who have serious preexisting medical or obstetric conditions (e.g., severe

preeclampsia, advanced insulin-requiring diabetes mellitus, or chronic hypertension) can develop serious difficulties despite much less extensive blood losses. It is estimated that in some parts of the Third World blood loss exceeding as little as 250 ml can be life threatening [85].

There are other uncommon but nonetheless important causes of peripartum bleeding. Coagulation defects secondary to abruptio placentae, unusual placenta adherence, amniotic fluid embolism, or severe preeclampsia can result in excessive blood loss. Women with previously undiagnosed coagulopathies such as von Willebrand's disease or who are receiving anticoagulants occasionally experience postpartum bleeding. Beyond the special cases, the most common obstetric cause for an acquired postpartum coagulopathy is simply prolonged bleeding. Severe hemorrhage progressively depletes clotting factors beyond the ability of the body to replace these substances, resulting in both hemodynamic problems and a coagulopathy. Fortunately, most significant chronic medical conditions are recognized prior to parturition and thus are managed prospectively. Nonetheless, even given a previously normal prenatal course, in every delivery there is a small but definite possibility for an event that can result in sudden, unanticipated, and even life-threatening hemorrhage [84,91–92]

### DIAGNOSIS

Vaginal bleeding is the most common sign of hemorrhage; however, bleeding can be occult, and in most cases of active hemorrhage, blood loss is underestimated. Occasionally, however, anxious or inexperienced attendants can actually overestimate blood loss, leading to unnecessary concern or unwarranted treatment.

The initial maternal response to hemorrhage varies and can be confusing. The usual indicators of circulatory function, including arterial pressure and pulse rate, are often normal in pregnant women despite substantial blood loss. In late pregnancy, the usual orthostatic measurements, such as the tilt test, often are either inaccurate or difficult to interpret. Thus, even with a substantial hemorrhage, orthostatic hypotension is an inconsistent sign and can be confused by the presence of supine hypotension or anesthesia. More important signs for clinical attention include persisting hypotension despite

fluid administration, delayed capillary filling at the periphery, oliguria, patient complaints of sudden severe abdominal or pelvic pain, and persisting tachycardia with or without dyspnea. These signs and symptoms require prompt investigation, regardless of the visual estimate of blood loss.

Routine blood pressure determinations are an imperfect means of clinical evaluation. Cuff position, maternal arm size, and the biophysical technique of measurement easily alter results. Apparently normotensive arterial pressure readings in a patient with prior hypertension but blood loss can be confusing, as are elevated pressures when a too small a cuff is applied around the arm. Sympathetic blockade from conduction anesthesia and medical treatment with tocolytics, sedatives, or other drugs can also confuse the interpretation of arterial pressure data.

The most objective and least invasive of organ perfusion measures is hourly urinary output. In the absence of pharmacologic manipulation, urine output of  $\geq 30$  ml/hr from an indwelling catheter indicates adequate renal perfusion. In a previously normal patient, persisting oliguria in the face of observed hemorrhage strongly suggests compromised renal blood and an inadequate circulating volume [90,92]. Unfortunately, if the hemorrhage is sudden and severe, this parameter is not useful in judging immediate losses or in estimating the extent of the acute fluid replacement required for resuscitation.

If the initial hemorrhage is promptly arrested by obstetric maneuvers, and the maternal signs and symptoms improve to normal following fluid infusion and uterotonics alone, no additional treatment might be necessary (Table 11.3). The need for more aggressive therapy is best gauged by combining blood loss estimates with clinical data such as heart rate, arterial pressure, and evaluation of peripheral perfusion. In terms of patient evaluation, the author prefers the following simple four-stage classification scheme proposed by Benedetti (Table 11.4) [88]. *Class 1* hemorrhage patients with blood losses  $\leq 900$  ml (15% of blood volume) have minimal signs and symptoms. A *Class 2* hemorrhage corresponds to a 20% to 25% loss of total blood volume. These patients normally have orthostatic changes, delayed peripheral capillary filling, and a narrowed pulse pressure. The pulse pressure narrows when there is a slight decline in the observed systolic pressure com-

**TABLE 11.3 Management of Volume Replacement in Postpartum Hemorrhage**

**Insert:**

Two large-bore intravenous lines

Foley catheter

In selected cases: an arterial line

**Initially infuse:**

1 or more liters Ringer's lactate or normal saline containing 20–40 IU of oxytocin

Thereafter, administer 3 ml of crystalloid/ml of estimated blood loss. Aim to maintain urine output of  $\geq 30$  ml/hr while sustaining maternal arterial pressure

**Administer as uterotonics:**

Ergonovine maleate (Methergine, 200  $\mu$ g IM), or

Prostaglandin 15-methyl-F<sub>2</sub> (Hemobate, 250  $\mu$ g IM, or intramyometrial), as clinically required

**Transfuse:**

Blood or blood products, as required: packed cells, fresh-frozen plasma, platelets, or cryoprecipitate

combined with a rise in diastolic pressure. These findings reflect diminished cardiac output owing to reduced diastolic filling combined with increased sympathetic tone. Women with *Class 3* hemorrhage have lost more than 25% of their blood volume. These women are tachycardic and tachypneic, frequently have cool extremities, and are overtly hypotensive. Urgent treatment of these cases is required to avoid additional deterioration. Finally, *Class 4* patients are those whose intravascular losses exceed 40% of total blood volume. These women are usually in profound

**TABLE 11.4 Classification of Puerperal Hemorrhage**

Class of Hemorrhage	Approximate or Estimated Blood Loss*	
	Volume (ml)	Percentage of Total Blood Volume
1	$\leq 900$	15
2	1200–1500	20–25
3	1800–2100	30–35
4	$>2400$	40

\*Clinical estimates of blood loss are notoriously inaccurate. These data must be combined with observations of pulse rate, arterial pressure, capillary filling, and other signs and symptoms. See text for details.

Modified from Benedetti TJ: Obstetric hemorrhage. In: Gabbe SG (ed): *Obstetrics: Normal and Problem Pregnancies*. New York: Churchill Livingstone, 1991:485–515, with permission.

**TABLE 11.5 Initial Laboratory Tests for Acute Postpartum Hemorrhage**

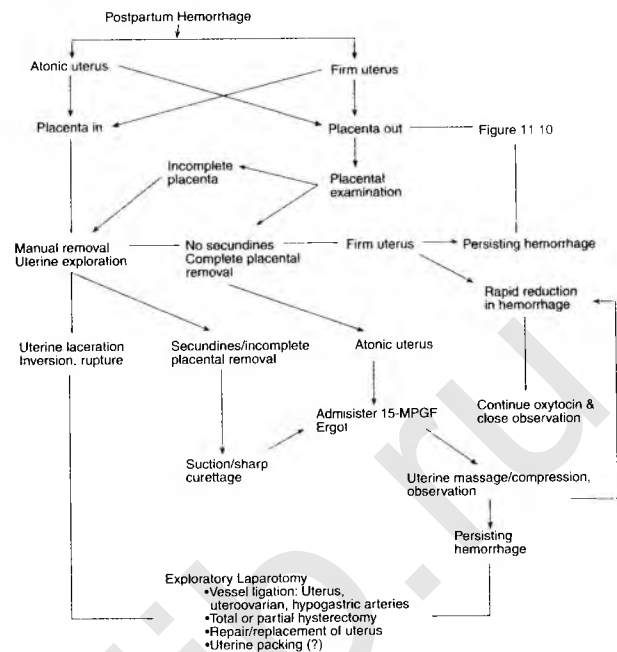
- Hemoglobin/hematocrit/platelet count
- Blood type, antibody screen, cross match
- Fibrinogen concentration
- Fibrin degradation product concentration
- Prothrombin time
- Partial thromboplastin time

shock, with markedly depressed or nonobtainable blood pressure and might or might not be lucid. Prompt and aggressive treatment for these women is mandatory to avoid permanent injury or death.

Appropriate laboratory investigations for hemorrhage include determination of hemoglobin/hematocrit and performance of basic coagulation studies. A blood sample must promptly be sent to the blood bank for crossmatching for blood and blood products (Table 11.5). Clinicians must recognize that the demand for definitive therapy might not permit waiting for the return of laboratory data. Deciding to administer blood or blood products acutely or to perform surgery in a case of serious and acute obstetric hemorrhage depends principally on clinical observations and not the results of laboratory tests.

## MANAGEMENT

Hemorrhage observed immediately after delivery warrants a prompt assessment. If the problem is suspected to be atony and the placenta is retained, manual removal is indicated. A complete inspection of the birth canal for lacerations and the placenta for intactness follows. If the placenta is thought to be incomplete, even if the hemorrhage has apparently abated, either an intrauterine manual exploration or a real-time ultrasound scan of the uterus should be performed. If the cervix is not widely dilated or there is no anesthesia, it is best to proceed first with the ultrasound scan. If the ultrasound study is suspicious for secundines, a manual uterine exploration or, if the patient is under anesthesia, a curettage is indicated for atony, best initial treatment is often bimanual compression (see Figures 11.9 and 11.10). Rarely, a manual exploration uncovers an occult uterine rupture or other pathology, emphasizing the importance of this basic examination.



**FIGURE 11.9.** General management scheme for postpartum hemorrhage. (Modified from Beydoun SN: *Postpartum hemorrhage and hypovolemic shock*. In: Hassam F (ed): *Diagnosis and Management of Obstetric Emergency*. Menlo Park, CA: Addison-Wesley, 1982:193–213, with permission).

If the uterus remains atonic, an intravenous infusion containing 20 to 40 units of oxytocin in 1000 ml of an isotonic salt solution such as Ringer's lactate or normal saline is administered rapidly. Volumes of 500 ml of fluid or more per 10 minutes might be required to stabilize maternal vital signs, depending upon the extent of the blood loss.

Close reevaluation of vital signs and symptoms after rapid volume expansion helps to gauge the need for the administration of blood or blood products. The goal of the initial supportive therapy is to maintain uterine tonus and maternal pressure and sustain a urinary output of 30 ml/hr. Ongoing blood losses are replaced with crystalloid at an approximate 3-to-1 ratio.

If the uterus responds poorly to the administration of uterotonics and massage, other methods of treatment are necessary. Colloidal solutions as volume expanders have a limited role in fluid resuscitation as they are associated with more complications than crystalloids. These solutions should not be routinely administered. If the uterus does not firm promptly after the initial brisk infusion and

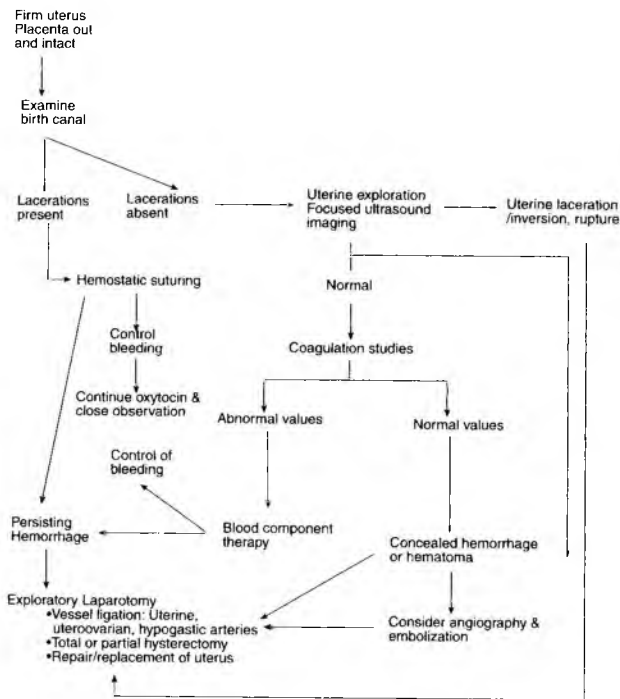


FIGURE 11.10.

*Management of postpartum hemorrhage with firm uterus following removal of intact placenta.* (Modified from Beydoun SN: *Postpartum hemorrhage and hypovolemic shock*. In: Hassam F (ed): *Diagnosis and Management of Obstetric Emergency*. Menlo Park, CA: Addison-Wesley, 1982:193–213, with permission).

bleeding persists, methylergonovine maleate 200  $\mu\text{g}$  (Methergine) or, in the absence of hypertension, 250  $\mu\text{g}$  of 15-methylprostaglandin-F2-alpha (Hemabate), is administered intramuscularly. In patients known or suspected to have reactive airway disease, misoprostol (PGE<sub>1</sub>, Cytotec) can be administered per rectum at the dose of 0.8 mg to 1.0 mg as an alternative but might not be as effective as the other uterotonics. Continued atony might require the administration of additional doses of 15-methylprostaglandin-F2-alpha, misoprostol, or methylgonovine every 20 to 30 minutes for four or more doses. At cesarean delivery, 15-methylprostaglandin-F2-alpha is commonly administered intramyometrially in cases of hemorrhage, but there are no data to suggest that this form of administration is more rapid or effective than the usual intramuscular technique. Intravenous bolus injections of *undiluted* oxytocin, methylgonovine, or 15-methylprostaglandin-F2-alpha are contraindicated. The failure to control the hemorrhage after

three or perhaps four doses of F2-alpha, an ergot derivative, or misoprostol indicates that medical management alone will probably fail, and alternative methods of treatment are necessary (Figure 11.10).

In severe hemorrhage due to unresponsive atony, techniques such as bimanual uterine compression, gauze packing, or the use of an intramyometrial balloons can reduce blood loss until blood or blood products are obtained or preparations for surgical intervention or embolization are completed. (See Chapter 18, Cesarean Delivery and Surgical Sterilization.)

*Gauze packing* of the uterus, although popular previously, is now rarely performed except by practitioners trained in prior decades. There is continued interest in this procedure, however [93–98]. Packing should be performed by experienced clinicians only, while potent uterotonics are administered concomitantly. Packing has a limited but occasionally important role in management and is still useful as a temporizing measure to reduce blood loss while blood is being obtained, assistance is summoned, or until the patient can be transferred to an operating suite or the radiology service for embolization. Before packing is attempted, uterine rupture, genital tract lacerations, and retained secundines are to be excluded by examination and ultrasound scanning.

If packing is chosen, it may be performed with a specialized instrument such as the Torpin packer or more simply by using a vaginal speculum and ring forceps. To achieve an effective tamponade, it is necessary to firmly pack as much of the uterine cavity as possible without leaving voids. As usually practiced, as many yards as necessary of 1- or 2-inch plain gauze with or without initial soaking in a vasopressin (Pitressin)/saline solution (10–20 units/250–500 ml normal saline) are firmly packed into the atonic uterus using a long ring forceps while another instrument grasps the cervix for counter traction. Packing can be performed blindly or under real-time ultrasound guidance. Traditionally, plain gauze has been used as the packing material, but iodine-impregnated gauze can be substituted. All gauze strips must be securely knotted together. Some clinicians pack the vagina as well as the uterus. There is no consistency in approach, nor are there any data favoring one method over another. The theory of vaginal packing is presumably that it better retains the uterine pack, helping to avoid voids and areas of incomplete compression. The problem of

vaginal packing is that it provides a large area for the sequestration of blood if hemorrhage from the uterus continues, potentially misleading the clinician into the assumption that the hemorrhage is controlled. When a packing is inserted, a Foley catheter is required because spontaneous voiding will not be possible. The author usually places a suture in the last portion of the packing, removes the needle and then ties a knot, leaving the suture ends long. The remaining ends are then loosely tied around the Foley catheter, and the end of the pack is tucked into the vagina. When the time for removal comes, the suture loop around the Foley catheter is located, the knot severed, and the pack end is then easily withdrawn.

In terms of its physiology, a uterine pack directly compresses the wall of the myometrium, thus mimicking uterine contractions. This compresses or occludes myometrial vessels and arrests the bleeding. An intrauterine compression balloon works in a similar fashion [102–105]. If it is elected to attempt a balloon, either one or more Foley catheters with large bulbs can be inserted into the uterus [104] or a Sengstaken-Blakemore tube [105] or a commercially available balloon can be substituted. (Figure 11.11) [103]. Effective compression devices have even been constructed on site from intravenous tubing and a rubber glove or a condom [105]. The commercial balloon looks like a large Foley catheter [90g]. It is inflated with up to 500 ml of normal saline, as required. Intrauterine balloon insertion may be easier than traditional packing if the equip-



FIGURE 11.11.  
*Cook balloon for ultrauterine tamponade.*

ment is immediately available. Although balloons are potentially more convenient than gauze packing, there are no comparison data concerning efficacy. In theory at least, a balloon should be less likely to hide blood loss than a pack, the intrauterine pressure can be modulated as required, and the entire uterine cavity is simultaneously compressed without the risk of voids. If either packing is performed or a compression balloon is inserted and the technique proves successful in arresting the hemorrhage, the pack or balloon is left in place for at least 12 hours, and broad-spectrum antibiotics are administered.

The usual complaint against packing – which could also be leveled against balloon tamponade – is that the procedure is nonphysiologic because it prevents uterine contractions, hides hemorrhage, or introduces infection. These arguments are not supported by clinical experience, however. Packing and other forms of internal uterine compression, including intrauterine balloon or Foley use, should remain in the repertoire of obstetric surgeons. One or more of these techniques could well prove useful and even lifesaving in a specific clinical circumstance.

Persisting hemorrhage in the face of an intact placenta and a firm uterus demands other considerations. An occult uterine inversion, uterine rupture, or a cervical or vaginal laceration must be promptly excluded. The entire birth canal should be immediately examined under good light and retraction, with the uterus carefully palpated. As lacerations or hematomas are identified they should be sutured, evacuated, or, if necessary, packed. If an incomplete inversion is diagnosed during the manual exploration, the uterus must be promptly returned to the anatomic position, as discussed later. Perineal hematomas are usually obvious and present as an acute, painful swelling involving the vulva, perineum, ischiorectal fossa, or paravaginal tissues. Hematomas developing high in the pelvis or extending upward into the broad ligament or other retroperitoneal areas are difficult to identify, despite careful examination. Often but not invariably in these cases, palpation of the upper vagina and the inguinal region either identifies a mass or notes persisting lateral deviation of the uterus. In difficult cases, prompt real-time ultrasound examination or other imaging studies are helpful in establishing the correct diagnosis.

If small vaginal or perineal hematomas are identified, the parturient is hemodynamically stable, and

the mass is not observed to increase in size, these are best managed expectantly. Enlarging hematomas resulting in severe pain or associated with signs or symptoms of cardiovascular compromise require surgical exploration, however. At surgery, ligation of any bleeding points, obliteration of the hematoma cavity (usually by packing), and drainage are performed. If a high hematoma is present, laparotomy and vessel ligation or embolization are sometimes necessary for control of bleeding. (See Chapter 18, Cesarean Delivery and Surgical Sterilization, for additional discussion.) If hemorrhage persists despite a normal intrauterine exploration, careful evaluation of the birth canal, administration of uterotonics, and the use of intrauterine compression pack or balloon, selective angiography can be performed. At angiography, bleeding vessels are identified by the injection of radiopaque dye and then directly embolized [99–101,106]. This procedure has a high efficacy rate and an acceptably low incidence of complications. In many institutions, although embolization is available, it might not be immediately available. Embolization can be of great assistance when there is some but not complete control of bleeding and immediate laparotomy is not mandated by the patient's condition. In these circumstances, if administration of blood, blood products and crystalloids can maintain the mother's cardiovascular status, there is sufficient time to assemble a team and attempt an embolization procedure.

Temporizing measures such as balloon insertion, uterine massage, administration of uterotonics, and even embolization can fail or in some instances are not available or appropriate. In these circumstances, exploratory surgery is performed. When the cause for the persistent uterine bleeding arises from atony or laceration, bilateral ligation of the uterine and utero-ovarian arteries can be quickly performed (modified O'Leary technique) to either control or reduce the hemorrhage. In cases of atony, other types of surgical control of hemorrhage such as the B-Lynch (or another type of compression sutures) are also appropriate. Ultimately, hysterectomy might be required to control bleeding, depending on the patient's condition and her response to prior therapy [85,108,109].

In the O'Leary technique, for direct ligation of the uterine artery, the uterus is elevated by an assistant and deviated laterally [107]. An area close

to the uterine isthmus is exposed, and a No. 1 absorbable suture (chromic or polyglycolic acid) is then passed through 1 cm of the myometrium, at approximately the level of the endocervix. The suture is next passed through an avascular segment of the broad ligament, with the appropriate site chosen by transillumination. This suture is then firmly tied either anteriorly or posteriorly, with attention to not inadvertently incorporating neither the bowel nor the omentum. When the body of the uterus is the source of hemorrhage, uterine artery ligations (O'Leary) are much easier and safer to perform and more likely to be effective than ligating the hypogastric arteries, as no retroperitoneal dissection is required, and the course of the ureter is not of concern and surgical access is difficult at best [108].

When the problem is atony, either the B-Lynch or one of the other types of compression sutures, or direct oversewing of the placental site can be effective in controlling postpartum hemorrhage unless there are other contributing factors (e.g., placenta accreta or percreta) [85,110]. The B-Lynch suture (brace suture) is usually performed using a 1 chromic or polyglycolic suture. The original report described its placement through a transverse myometrial cesarean incision. We find this to be unnecessarily complex. We favor the use of one of the variations of this procedure, employing simple through-and-through sutures placed in the myometrium and passing across the fundus. In our experience this technique is successful and much less difficult to conduct. Placement of any compression suture requires that attention be given to ensure drainage of the endometrial cavity, as a hematometrium or a pyometrium are potential complications.

In the modified B-Lynch technique that we recommend, an assistant supports the uterus while the primary surgeon passes a suture (No. 1) through the myometrium anteriorly to posteriorly, at the level where a low transverse uterine incision is normally placed (i.e., approximately 2 cm medial to the edge of the uterine wall). The suture is then passed over the fundus. A knot is made and subsequently slowly drawn tight and then secured. This compresses the myometrium, resulting in an unusual M-shaped appearance, mimicking the effects of bimanual compression. (See Chapter 18, Cesarean Delivery and Surgical Sterilization.)

Other clinical situations require the use of different approaches. Rarely, in the case of a retroperitoneal hematoma, persistent bleeding after hysterectomy, or a high paracervical laceration, a unilateral or bilateral hypogastric artery ligation is required to control bleeding. Occasionally, the feeding vessels arise directly from the hypogastrics or their branches and not simply from the uterine artery [108]. (Chapter 18, Cesarean Delivery and Surgical Sterilization.)

If a vessel ligation or compression sutures do not control the hemorrhage, a rapid supracervical or complete hysterectomy becomes necessary. In extreme instances, manual compression of the aorta above the bifurcation assists in acute patient stabilization. The reason for progressive vessel ligations and the use of compression sutures is because an emergency hysterectomy for exsanguinating obstetric hemorrhage is a potentially morbid event. Ureteral injury, cardiac arrest, septic pelvic thrombophlebitis, and maternal death are possible sequelae [92,109].

If unfamiliar with any of these specialized techniques for vessel ligation or placement of compression sutures, the treating physician should request assistance from a gynecologic surgeon or another experienced obstetrician. It is well to remember the potential benefits of radiographic embolization because when possible, this technique is highly effective in controlling hemorrhage and avoids the myriad complications of major abdominal surgery [99,106].

### Late Postpartum Hemorrhage

Late postpartum hemorrhage is usually attributed to the poorly understood condition termed *subinvolution*. Retained products (placental polyps), chronic endometritis, or previously undiagnosed uterine or cervical tumors are possible additional causes [87]. Rarely, gestational trophoblastic disease presents in this fashion. In the usual case of subinvolution, the uterus is enlarged, boggy, and occasionally slightly tender to palpation. An endometrial biopsy will reveal plasma cell infiltrates or other histologic evidence suggesting chronic inflammation. Real-time ultrasound is useful in identifying candidates for curettage or other surgery, because occasionally occult secundines or other masses such as placental or endometrial polyps are identified within

the uterus. If the diagnosis is simple subinvolution, scanning usually does not identify much beyond a nonspecific enlargement of the uterus and the presence of scant echogenic material within the cavity. Large amounts of retained products are normally easily identified. As ultrasound scanning cannot distinguish between intrauterine clots versus small amounts of decidual debris, judgment is necessary in determining which cases should go immediately to curettage versus those in which a less aggressive approach is possible [14]. If retained products of conception are not identified or suspected, and prompt control of bleeding follows the administration of a uterotonic (e.g., intravenous oxytocin, an ergot derivative, or a combined prostaglandin with a broad-spectrum antibiotic), expectant management is usually best. The usual treatment for subinvolution is to administer a broad-spectrum antibiotic such as doxycycline (100 mg bid for 5 to 7 days, if the patient is not nursing a broad-spectrum cephalosporin if she is) combined with a potent uterotonic such as methylergonovine maleate (Methergine; 200 µg PO, 96 hours for 4 to 6 doses). A curettage is required, however, if secundines are suspected or if the bleeding persists or recurs after a trial of expectant management. In cases requiring curettage, real-time ultrasound in the operating suite can assist the surgeon both in the safe placement of surgical instruments and in ensuring that the uterus is empty.

### Uterine Atony/Inversion

Both uterine atony and inversion can result in exsanguinating hemorrhage. As mentioned previously, the risk of atony is substantially reduced but not eliminated by active management of the third stage and routine use of uterotonics. Atony has several important clinical associations [32]. Atony is more common when the uterus is overdistended, especially after delivery of a macrosomic infant or a multiple gestation. Infection and abruptio placentae also predispose to atony, as does prolonged oxytocin stimulation, precipitate labor, and the use of halogenated anesthetic agents, although the later are rarely used.

Treatment for atony initially includes the administration of uterotonics, uterine massage, and, occasionally, direct uterine compression (Figure 11.12). As discussed earlier, uterine packing, placement of an intrauterine balloon, vessel ligations (O'Leary),





FIGURE 11.12.  
*Bimanual uterine compression for atony/hemorrhage.*

a B-Lynch or other surgical compression procedure, uterine artery embolization, or hysterectomy might be required for control if a true hemorrhage ensues [85,87,92]. Parenteral administration of uterotonics combined with uterine massage, to prompt myometrial contractions, is the initial therapy. A dilute solution of 20 IU to 40 IU of oxytocin in a non-glucose-containing balanced salt solution is administered intravenously at a brisk rate. If bleeding continues despite oxytocin, or if the uterus relaxes after massage is stopped, then an ergot derivative or one of the prostaglandins is administered. If medical management fails, an endometrial balloon, packing, arterial embolization, selective vessel ligation, brace/compression suture placement, or hysterectomy should be considered.

### **Uterine Inversion**

*Uterine inversion* is an uncommon postpartum complication that occurs in from 1/2,000 to 1/20,000 deliveries [22,112,116-125]. Uterine inversions are usually described as either *partial* or *complete*, with or without placental attachment, and either acute or, very unusually, chronic. Incomplete inversion

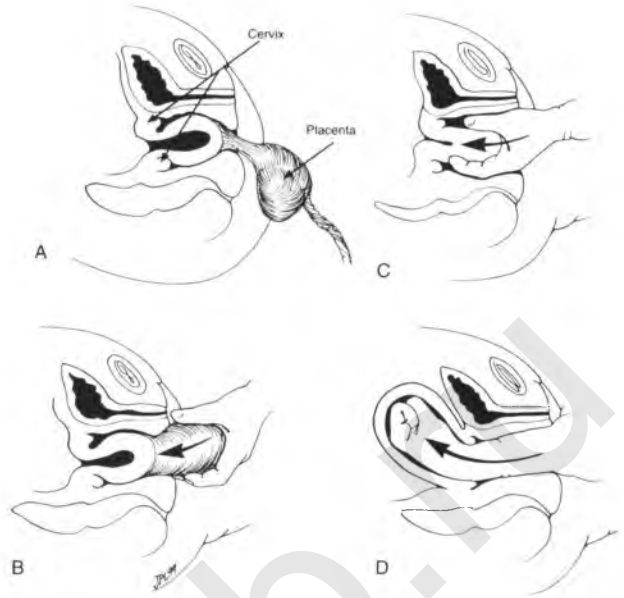


FIGURE 11.13.  
*Manual reduction of uterine inversion. (A) Depicts a complete uterine inversion with spontaneous placental separation. Vaginal replacement involves administration of a tocolytic and gentle but steady upward pressure (B, C) to reduce the inversion (D). Uterotonics are then administered, and the patient is observed closely for possible reinversion.*

occurs when the fundus of the uterus partially inverts, but the uterus does not entirely evert. This type of partial inversion is difficult to diagnose until a uterine exploration is performed. Several variations of partial inversion are possible, but in most the cervix is usually palpable as a distinct anatomic structure (Figure 11.13). The principal risk factors for inversion are a flaccid lower uterine segment combined with a fundal placental implantation, occasionally but not invariably assisted by cord traction or fundal pressure.

Acute uterine inversion requires prompt diagnosis and restoration of the usual uterine contour as rapidly as possible by either physical manipulation or surgery, because blood loss is characteristically both sudden and severe [22,114,116,119]. In the absence of an accreta, early diagnosis with prompt uterine replacement will often avoid the need for a surgical exploration.

The presumed predisposing factors for inversion are so common but the actual event so rare that which concatenation of events is necessary to predispose the uterus to invert in a given case remains

unknown. In general, inversion occurs in association with fundal implantation of the placenta, unusual placental adherence (i.e., accreta, increta, percreta), and Müllerian abnormalities such as a bicornuate uterus [112,114,121].

Incompetent midwifery with inappropriate cord traction has long been taught as the cause for inversion; however, this explanation does not explain all instances. This association remains valid if excessive cord traction is performed when the placenta is not separated and the uterus remains flaccid, however [22,119]. An unpublished retrospective study of 16 cases (of 26,000 deliveries) in Toronto by the author found cord traction to be an important factor in 11 of 16 cases, with fundal implantation present in 60%. Forty percent of the inversions occurred without a history of cord traction or fundal massage/pressure, however. In fact, spontaneous inversion is occasionally observed at cesarean delivery. In a recent review of 40 uterine inversions, one half occurred with cesareans, with an overall five times higher rate compared with vaginal deliveries [22].

Historically, in either acute or chronic uterine inversion, maternal mortality rates were high. The mortal risk of nearly 18% for inversion was reported as recently as 1953 [121]. In recent decades, however, fatalities from this condition have become rare, except in neglected cases. With better understanding of this disorder and more aggressive obstetric management, the risk to the mother's life from an inversion is now less than 1%.

If the uterus inverts externally, the correct diagnosis is usually immediately apparent and frequently dramatic. A large, regular, and erythematous mass suddenly presents at the introitus, often with the placenta still attached. A hemorrhage of rapid onset commonly accompanies the pelvic/vaginal mass, and the uterine fundus is usually not palpable. A partially prolapsed or incomplete inversion is a more subtle condition, at times presenting only with sudden postpartum hemorrhage and shock. Incomplete cases are often misdiagnosed initially as a prolapsing leiomyoma, the expulsion of a retained placental fragment, or a succenturiate lobe [118]. Infrequently, chronic partial inversion occurs. These most unusual cases present as late as several days postpartum, with patients having signs and symptoms that include complaints of chronic bleeding, vaginal discharge, and pelvic pressure. On physi-

cal examination, the uterus feels unusually globular and enlarged (although the fundus cannot be felt rounded as usual) and secundines are commonly suspected as the principal diagnosis. If a chronic inversion is diagnosed and there are no acute symptoms, some suggest waiting for complete involution of the uterus (6 weeks) prior to repair or restoration. The reason for this waiting period is unclear, however. There is at least one case report of a patient with chronic incomplete inversion and infarction of the uterine fundus necessitating hysterectomy [122]. The prompt restoration of the uterus to its normal position once the diagnosis of any degree of inversion is made is strongly recommended.

Treatment of an inversion must be prompt, because delay results in the formation of a constriction ring, excessive blood loss, and tissue edema, all of which progressively render uterine restoration more complicated and more difficult. There are three important features to proper management. First, blood losses commonly are heavy and exceed the clinical estimates. Second, returning the uterus promptly to its normal position avoids the development of a constriction ring, which renders the process of restoration much more difficult. Third, administration of uterotonics is contraindicated until the uterus has been replaced; then aggressive treatment is needed.

## SURGICAL TREATMENT

Once the diagnosis is established, immediate replacement should be attempted while active hydration is administered. Prompt replacement is successful approximately 40% of the time or more. The technique for replacement is discussed later. If immediate replacement fails, active fluid resuscitation is continued, an intravenous tocolytic is administered, and a more extensive procedure is required in the operating suite. In modern practice, most parturients will have had epidural anesthesia, which provides analgesia for vaginal manipulations but not the profound uterine relaxation required for replacement.

The parturient is next transferred to the operating suite, and experienced help summoned, including senior obstetric staff, an anesthesiologist, and surgical assistants. If not already in place, large-bore intravenous needles are inserted for fluid resuscitation, and blood should be drawn immediately for

cross matching, because hemorrhage accompanies virtually all cases of inversion, and shock appears in up to 40% of cases [22,118,119]. Many of these women require aggressive fluid and blood transfusion to stabilize their vital signs and restore losses.

Most cases of uterine inversion are easily treated by prompt vaginal replacement of the prolapse by manual pressure (Johnson maneuver) performed per vagina either with parenteral tocolysis under epidural anesthesia or, if necessary, under general inhalational anesthesia (see Figure 11.13) [22,119,124,125,127–130,133,137]. The classic clinical rule for treatment of inversion is “last out, first in.” Working with finger pressure, the surgeon begins lateral to the central mass and progressively presses the prolapsed tissue upward in a circular pattern until the complete mass is returned to its normal contour within the pelvis/lower abdomen. Usually, with tocolysis, this procedure is relatively easy. Once the fundus (with or without placenta) is replaced, uterotonics are administered while the surgeon maintains his or her hand within the uterus until myometrial tone returns. Immediately after replacement, attention to the position of the uterus is necessary, because prompt reinversion is common. If the inversion is complete and the placenta remains intact, manual replacement is best performed first, before attempting to remove the placenta. Placenta accreta occasionally accompanies inversions. If the placenta does not separate entirely while the uterus remains inverted, additional and usually severe blood loss is likely. This loss can compromise the chances for the mother’s recovery. Best practice is to first restore the uterus to its anatomic position, and then support the maternal cardiovascular function by restoring circulating volume and red cell mass. Once the uterus is replaced and contracts, the placenta should separate spontaneously. If not, then manual removal is required. If an accreta is encountered, it will need management in the usual fashion for unusual placental adherence.

Occasionally in older but in some recent literature concerning inversion, the comment was made that the degree of shock seen in women with inversion was out of proportion to the estimated blood loss [126,127,130]. It was presumed that there was a neurogenic mechanism responsible, owing to intense parasympathetic stimulation resulting from stretch to the uterus and its adjacent structures. The

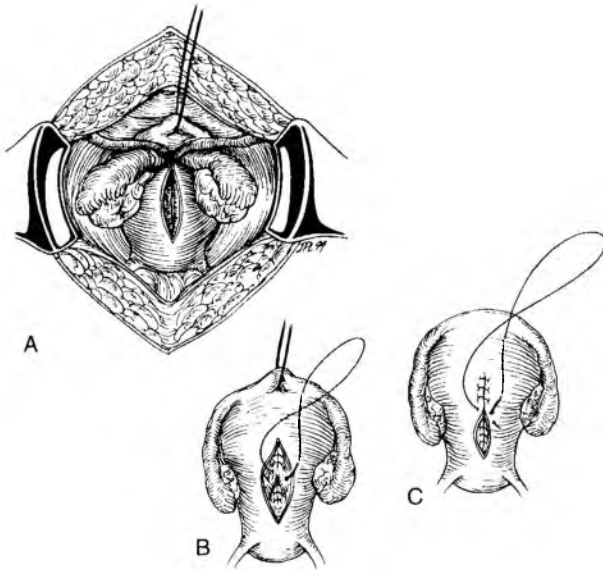
principal clinical markers of this condition included evidence of shock accompanied by bradycardia or peripheral vasodilatation [130]. Most literature does not support this hypothesis. It seems more likely that actual blood losses from inversion are simply more severe than clinicians estimate and that this hemorrhage is sufficient to explain the observed shock state [105c].

In terms of technique, uterine relaxation is often needed to restore a complete inversion. The author’s preference for relaxation is the administration of intravenous nitroglycerin [129,132,134]. With nitroglycerine, each case is an individual titration. Initially, a dose of 150  $\mu\text{g}$  to 200  $\mu\text{g}$  is administered. Thereafter, if relaxation is insufficient, additional boluses of 100  $\mu\text{g}$  to 150  $\mu\text{g}$  are administered several minutes apart, as required until the desired effect or a total dose of 500  $\mu\text{g}$  is reached. The most important maternal side effect of nitroglycerine is transient hypotension. Nitroglycerine should be used with caution in patients already compromised by low vascular volumes from prior hemorrhage, especially in cases complicated by preeclampsia or chronic hypertension. Other possible tocolytic agents include intravenous terbutaline given in doses of 150  $\mu\text{g}$  to 250  $\mu\text{g}$  or intravenous magnesium sulfate in a dose of 4 g to 6 g [123,124]. Because of their delayed onset of action and potential side effects, there is no reason to favor these agents over nitroglycerine.

An unusual technique for replacement is hydrostatic [135]. In this unique method, originally described by O’Sullivan, the introitus is tamponaded either by the surgeon’s forearm, or a plastic vacuum extractor is inserted into the vagina for the same purpose. Warmed sterile saline or water is then introduced into the vagina via an intravenous line or the vacuum port of the vacuum extractor. The progressive filling of the vaginal vault exerts sufficient pressure to slowly return the uterus to its normal position. The success rate or the efficacy of this technique in comparison to manual replacement is unknown, but successes of this unusual method have been reported.

### Abdominal Approach

If replacement under tocolysis fails, an abdominal exploration or a combined vaginal/abdominal procedure is required to replace the uterus surgically



**FIGURE 11.14.**  
***Surgical reduction of uterine inversion. Uterine inversion that cannot be reduced manually requires prompt surgical exploration and correction. A relieving myometrial incision (A) permits reduction. Then the myometrial defect is subsequently repaired in layers, as required (B, C). See text for details***

[136–138]. There are several abdominal approaches described for uterine inversion. These procedures are discussed in additional detail in Chapter 18, Cesarean Delivery and Surgical Sterilization, and only a brief outline is given below.

In the *Huntington procedure*, the abdomen is first entered by a low, transverse Pfannenstiel-type incision [136]. Visualization of the bizarre-appearing, classic inverted uterine “funnel,” with the round, broad, and utero-ovarian ligaments disappearing into the vagina, confirms the diagnosis (Figure 11.14). Either an inhalational agent that relaxes the uterus or a parenteral tocolytic is administered, and a gloved assistant is stationed at the perineum. From above, the wall of the uterus or the round ligament is grasped approximately 2 cm below the constriction ring with Allis or similar clamps. Alternatively, a No. 1 suture of Vicryl or chromic in a figure-of-8 stitch can be placed in a midportion of the fundus, if it can be visualized. The inverted organ is slowly pulled upward by progressively grasping the uterine tissue as it advances, aided by constant, upward pressure provided by the vaginal assistant on the traction suture. With the uterus restored to its normal contour, a uterotonic such as

15 methylprostaglandin-F<sub>2</sub>-alpha is administered. Prior to closing the abdomen, the surgeon should observe the uterus closely for several minutes to be certain that it firms normally and does not reinvert.

The second technique for surgical correction of inversion is the *Haultain operation* [138]. This differs from the Huntington procedure in that the ring of the inverted uterus is incised posteriorly to relax the opening of the funnel, thus easing the reinversion. This technique is best for chronic or silent inversions when the uterus has been inverted for a prolonged period, a situation that usually precludes a simple mechanical replacement procedure due to the formation of a dense retraction ring.

The *Spinelli operation* is a vaginal surgical procedure for inversion. In this rarely attempted operation, an anterior vaginal colpotomy is first performed, followed by an incision in the cervix and then the lower uterine segment. The uterus is then replaced by simple upward pressure, and the surgical incisions are then closed. A second possible vaginal approach is the *Kustner procedure*. In this operation, a posterior colpotomy is made. A posterior incision through the cervix and lower uterine segment is also performed, and uterine repositioning is then conducted followed by the usual repair of the incisions. Neither of these vaginal procedures is recommended because of the risks of the incision entering or extending into the bladder, ureter, or major vessels. An additional problem is the potential risk of cervical insufficiency in these women in subsequent pregnancies.

### Abnormal Placental Adherence

As previously discussed, in uncomplicated cases, if the placenta has not delivered by 30 minutes after delivery of the infant, the placenta is considered retained and intervention is indicated [140,141]. Usually 90% or more of placental deliveries occur within this period of time. Hemorrhage associated with a retained placenta requires immediate evaluation and treatment, however, regardless of the time elapsed. In general, placental retention is associated with prematurity, placenta accreta/increta/percreta, and cervical entrapment or for unknown causes [141]. Whenever the placenta does not separate normally, there is a finite possibility of severe hemorrhage or placental fragmentation, possibly requiring

a curettage, transfusion, or even hysterectomy. Management depends on the clinical circumstances.

To differentiate an entrapped but separated placenta from a partially adherent placenta requires a manual uterine exploration. If a placenta has been retained for a short time and bleeding is minimal, dilute intravenous oxytocin is administered to speed separation [142]. As previously discussed, if the placenta is retained, before proceeding to a manual removal, either drainage of placental blood or umbilical venous injection of a dilute oxytocin solution can be attempted. There is no risk to these maneuvers and some, albeit limited, data to suggest efficacy. If treatment is successful, further difficulty is avoided, and if the effort fails, nothing has been lost. In the somewhat unusual situation that the placenta has separated but remains entrapped within the uterus, blood can collect inside the cavity, resulting in an expanding fundal height. Under these circumstances, if the placental mass tamponades the cervix, the observed bleeding can be minimal.

Placenta accreta and the other, more severe types of abnormal placental adherence result from abnormal trophoblast invasion of either the myometrium (increta) or the myometrium and adjacent tissues (percreta). In placenta accreta, the villi are adherent to the myometrium, and normal separation cannot occur. An accreta can be complete, involving the entire placenta, or only partial. With complete accreta, no plane of cleavage is found when manual placental extraction is attempted; the placenta will come away in fragments, usually accompanied by a sudden and substantial hemorrhage. If the accreta is partial, a plane of cleavage can be found, but it does not continue throughout the placental disc. While the diagnosis of accreta is histologic, as a practical matter, the clinical findings at the time of attempted placental removal are so characteristic that the clinician is rarely in doubt concerning the correct diagnosis.

Placenta accreta/increta/percreta is associated with advanced parity, low-lying presentation (placenta previa), Müllerian anomalies, or preexisting uterine scars, especially prior cesarean delivery scars [144,145]. With the rising cesarean delivery rate, the incidence of placenta accreta has also increased substantially. Endometrial damage from any source, including a prior cesarean delivery or Asherman's syndrome, increases the risk of unusual placental adherence by several-fold [8,146]. When there has

been a prior cesarean, the risk of accreta increases if the placenta in the current gestation implants over the prior cesarean scar. Women with accreta often but not invariably also give a history of mid-trimester bleeding. Such a history of mid-trimester bleeding or the rotation of an elevated AFP in the mid-trimester combined with a review of the woman's prior surgical background should prompt ultrasound scanning which can often identify suspect cases in advance of labor.

The etiology of placenta accreta or percreta is unknown but is likely associated with an abnormal maternal-fetal immunologic relationship at the cellular level, leading to abnormal trophoblastic invasion of the myometrium [147]. The definitive diagnosis of an abnormally adherent placenta is histologic and requires that the pathologist directly study either the uterus or review uterine curettings that include the myometrium. Direct villous invasion into myometrial cells must be histologically confirmed to secure the diagnosis [144–146]. Placenta increta and percreta are differentiated histologically by the extent of the myometrial invasion. In placenta increta, the trophoblast invades the myometrium deeply, whereas in percreta it passes entirely through the myometrium to appear at the serosal surface [8]. Dysfunction of maternal leukocytes and various immunologic abnormalities have been suggested as etiologies for such abnormal placentation [143,147]. It is fair to say, however, that the pathophysiology leading to placenta accreta/increta/percreta has yet to be convincingly established. Other abnormal findings in these cases are common. In Fox's comprehensive study of accreta cases, only 8% of patients with an adherent placenta had no identified pathology or abnormality to explain the abnormal placentation, and 35% of the women with placenta previa also had placenta accreta [28].

Rarely, a patient with a placenta percreta that invades entirely through the uterus presents with exsanguinating intraabdominal hemorrhage. More frequently, the placenta percreta invades adjacent tissues, notably the bladder [161–163]. Because of this, unexplained hematuria can be an early sign. Although adenomyosis has been proposed as a predisposing factor to placenta percreta, this pathology is rarely identified in surgical specimens. Müllerian anomalies such as a bicornuate uterus increase the risk, for unknown reasons. In this situation, the

abnormal placentation usually invades the septum. The risk for the infant remains high [163]. The incidence in the Third World could be 20 times higher than that in industrialized nations because of a higher likelihood of predisposing factors such as multiparity, prior missed abortions, and severe postpartum endometritis, all of which result in endometrial injury [148].

Placenta accreta has been diagnosed in all trimesters of pregnancy and has complicated first-trimester abortions [149,150]. Several authors report that at least 50% of patients with placenta accreta have otherwise unexplained increased maternal serum  $\alpha$ -fetoprotein (MSAFP) [151–154]. In some instances, the diagnosis of abnormal placental invasion is suspected antenatally by ultrasound examination. The ultrasound criteria for suspecting placental adherence include 1) loss of the normal hypoechoic retroplacental fetal-maternal interface 2) thinning or disruption of the hyperechoic uterine serosa-bladder interface, 3) the observation of focal exophytic masses invading into the maternal bladder; and 4) the presence of large or abnormal placental venous lakes [155–157]. Doppler ultrasonography may document arterial vessels crossing from the placenta to adjacent tissues, the loss of venous flow in the peripheral placental margin, or intraplacental lacunae with apparent arterial flow [157]. It should be noted that most patients in these ultrasound studies were already considered at high risk for various reasons, including combinations of placenta previa, known previous cesarean scar, unexpected vaginal bleeding, or high mid-trimester MSAFP levels. The diagnosis of a placenta accreta before parturition in an asymptomatic pregnancy is not always possible. Because of the limitations of current methods of surveillance, caution in diagnosing placenta accreta based on ultrasonic data alone is prudent. MRI scans can prove useful in confirming abnormal placenta adherence in suspect cases, but experience is necessary to provide accurate diagnoses [158,159].

#### **TREATMENT OF PLACENTA ACCRETA/INCRETA/PERCRETA**

Placenta accreta is implicated in at least one half of all emergency postpartum hysterectomies [132]. In cases involving only small areas of abnormal adherence, however, hysterectomy can on occasion

be avoided. In the focal type of placenta accreta, the combination of sharp uterine curettage and the administration of uterotonics can prove successful in avoiding hysterectomy. Despite the occasional success, hysterectomy is still required in most placenta accreta cases when a substantial portion of the placenta is involved. Nonoperative management is rarely a reasonable choice owing to the high incidence of serious complications. Up to 95% of cases of true placenta accreta/increta/percreta eventually required hysterectomy. In the rare instance when the abnormal placentation is diagnosed prior to delivery, and clinical circumstances make retention of fertility a major issue, it can be possible to manage an occasional case conservatively. This includes leaving the placenta undisturbed at delivery and, possibly, administering methotrexate to hasten placental resorption. If the parturient is hemodynamically stable after a vaginal delivery when the presumptive diagnosis of accreta is made and there is no vaginal bleeding, the cord is simply cut as short as possible and the woman subsequently observed. If bleeding resumes as the uterus contracts, however, immediate intervention is required. Most often bleeding recurs approximately 6 to 10 days postpartum as the process of endometrial regeneration begins. Conservative treatment leaving the placenta in situ is a more reasonable choice when the abnormal placentation is associated with either a cervical or an abdominal pregnancy. In these instances, however, the complication rate remains high. Such unusual circumstances require a careful and detailed discussion with the parturient. Conservative treatment should not be attempted unless the woman is aware of the associated risks.

If the diagnosis of placenta percreta is strongly suspected antepartum, special measures should be taken at the time of the planned delivery to ensure the immediate availability of appropriate equipment and personnel. When the laparotomy has been performed, the infant has been successfully removed, and placental invasion is confirmed, if the findings are more extensive than originally anticipated and conditions are not optimal for immediate surgical removal, alternative management needs consideration. In highly selected instances, assuming that the placenta has not been disrupted and there is no unusual bleeding, the abdomen can simply be closed. Other procedures, such as prophylactic embolization of the hypogastric arteries, are

considered at the same time and the parturient may electively be administered methotrexate. Definitive surgery is then scheduled after a delay when it is presumed that vessel involution and trophoblast necrosis will render the placental removal less difficult and dangerous. Alternatively, in these unusual circumstances, the woman can be transferred to a referral institution where special equipment and more experienced surgeons are available [163].

Uterine or vaginal vault packing has a limited role in the control of bleeding after the diagnosis of placenta accreta in a vaginal delivery [164,165]. After successful manual removal and curettage of a partial placenta accreta, gauze packing or the insertion of an intrauterine balloon accompanied by the aggressive use of uterotonics often will initially control hemorrhage. Such treatment might prevent hysterectomy in some cases, but the risk of complication is very high. More importantly, this approach permits time for maternal stabilization. The surgeon plans either a subsequent move to the operating suite or, possibly, transfer of the mother to another institution if appropriate personnel and facilities are not available at the site of delivery.

In the occasional case, tamponade vaginal vault pelvic packing can be lifesaving. The most commonly used pelvic pack consists of a mass of Kerlix gauze that is placed in a mesh or plastic bag and introduced into the pelvis after laparotomy. The ties securing the bag are brought out through the vagina. Continuous traction on this pack provides compression and thus mechanical hemostasis. If the pack is successful in controlling the immediate hemorrhage, the patient can subsequently be treated by arterial embolization or, if bleeding is secondary to a coagulopathy, by correcting the deficiency. Obviously, such complex cases are rare in obstetric practice and should be managed in conjunction with an experienced gynecologic consultant or surgeon.

## LACERATIONS OF THE BIRTH CANAL

### **Uterine Lacerations**

Uterine laceration or rupture can follow several obstetric misadventures such as an instrumental vaginal delivery, extraction of the second of twins, a vaginal breech extraction, a severe shoulder dystocia, or a trial of vaginal birth after cesarean delivery. Spontaneous rupture of the previously normal and

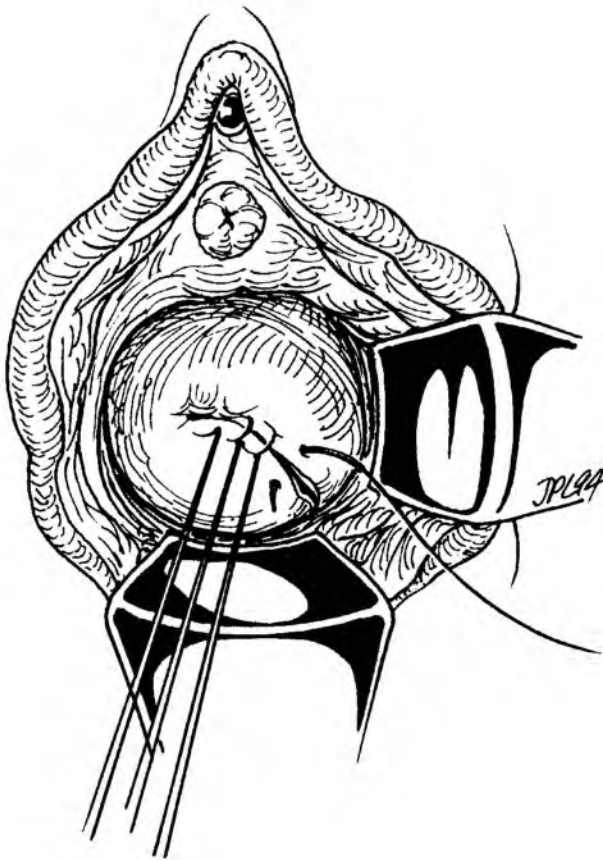
non-scarred uterus of a nullipara in a normal pregnancy is rare. In multiparas, however, spontaneous uterine rupture is much more likely. At present, many uterine ruptures are associated with vaginal birth after cesarean (VBAC) trials. Abnormal placentation (e.g., placenta percreta) or other problems (e.g., occult Müllerian anomalies or obstructed or dystotic labor) also can predispose a patient to uterine rupture. Otherwise, unexplained cardiovascular collapse, vaginal hemorrhage, loss of station, or rapid-onset fetal distress in a high-risk patient should alert the physician to consider the diagnosis of a uterine rupture or laceration. (See Chapter 18, Cesarean Delivery and Surgical Sterilization.)

### **Cervical Lacerations**

After any complicated delivery, the cervix must be carefully examined. If significant or bleeding lacerations are discovered, they should be reapproximated with interrupted sutures of absorbable suture material (Figure 11.15). Tears that extend upward beyond the fornix can require exploratory laparotomy if injury to the lower uterine segment or the urethra or bladder is suspected, or if there is a possibility of hematoma formation. Aggressive blind lateral suturing for laceration repair or for hemostasis is inappropriate owing to the proximity of the ureters. In considering the repair of cervical lesions, the rule of reason must apply. After complete dilatation, the cervix can appear torn, but suturing apparent nonbleeding tears of less than 2 cm is usually inappropriate since these are inconsequential and this unnecessary intervention may predispose to cervical stenosis. Only tears greater than 2 cm in length or those that are bleeding briskly or do not respond to simple tamponade should be reapproximated. The long-term outcome is the issue. A woman sustaining a major cervical injury is probably at risk for subsequent cervical insufficiency. Reexamination of such patients prior to subsequent attempts at conception, and serial examinations and cervical ultrasound studies for cervical length (beginning in the early second trimester once a subsequent pregnancy is established) are prudent.

### **Vaginal Lacerations**

Vaginal lacerations occur commonly after both spontaneous and instrumental delivery but are



**FIGURE 11.15.**  
*Repair of a cervical laceration. Interrupted sutures are inserted to reapproximate normal anatomy and control bleeding.*

clearly more common after obstetric interventions. Vaginal vault lacerations are usually easy to repair, but some extend into the lateral fornix or dissect deeply into the ischioanal fossa, leading to hemorrhage or the formation of hematomas. The most serious of these injuries involve either spontaneous or induced lateral hematomas occurring from vaginal wall vessels. The pudendal artery can be ruptured or avulsed during delivery without a history of pudendal nerve block or a lateral wall tear. Regardless of the vessels injured, the resulting hematomas can rapidly reach a surprisingly large size, dissect into the retroperitoneal space, and even threaten maternal cardiovascular stability.

The usual presenting complaint for a pelvic hematoma is severe perineal/vaginal pain accompanied by acute, progressive, unilateral swelling of the labia. The hematoma can remain entirely intravaginal. It is also possible for the mass to dissect

upward into the retroperitoneal space and be correctly identified only after a vaginal examination with the patient under anesthesia or at laparotomy. Treatment consists of surgical exploration, with the ligation of any observed bleeding vessels followed by drainage and vaginal packing. Usually a single, distinct bleeding site is not found, and the surgeon must be content to evacuate the hematoma, ligate or cauterize as many bleeding vessels as can be identified, and finally pack the vagina firmly to compress the site. When a hematoma is surgically explored, a suction drain should be inserted and broad-spectrum antibiotics administered. With a vaginal pack in place, spontaneous voiding is not possible and a Foley catheter is required. The packs are progressively removed after 12 to 24 hours. Rarely, blood losses from vaginal wall hematomas can be extensive enough to require transfusion.

## INFECTION

### **Superficial Perineal Infection**

Infections of vaginal lacerations or episiotomy sites are usually superficial and minor, although rarely, serious problems ensue [166,167]. If there is infection, the usual outcome is the disruption of episiotomy or laceration repair. For superficial infections, the classic treatment is wound exploration, débridement, and closure by secondary intention. Antibiotics are administered if signs of cellulitis, induration, or gross infection are present. Sitz baths and analgesics usually provide symptomatic relief. In the past, simple wound breakdown at the episiotomy site or laceration was reapproximated following débridement, but only after a variable waiting period of up to 3 months. At present, in uncomplicated cases, waiting is not believed to be necessary, and repeat suturing can be performed once the wound is clean, with the high likelihood for a successful repair.

### **Necrotizing Fasciitis**

Necrotizing fasciitis (NF) is a rare and potentially fatal disorder with several clinical variants [167–170]. NF is caused by an infection tracking along fascial planes, which results in progressive tissue necrosis. The three most important forms of NF are Type I, polymicrobial; Type II, streptococcal,



and Type III, clostridial (gas gangrene/myonecrotic). Historically, NF has received many names including Meleney ulcer, acute dermal gangrene, hospital gangrene, suppurative fasciitis, synergistic necrotizing cellulitis, or hemolytic streptococcal gangrene [168].

NF developing in the perineal area is sometimes termed *Fournier's gangrene*. Although this term was originally used to describe a variant of scrotal NF, this condition is part of the same general infectious disease process.

In obstetric cases, the causative organism for NF is usually Group A  $\beta$ -hemolytic *Streptococcus* alone or in combination with various anaerobic bacteria. The latter are most often *Bacteroides* species. Less frequently, a *Streptococcus* species combined with bacteria other than anaerobes or *Enterobacteriaceae* is responsible. Obstetric patients who develop NF usually but not inevitably experience extensive perineal lacerations with substantial blood loss. In some cases, however, the only surgical injury is a routine episiotomy with repair [167]. NF can involve superficial tissues only (Camper's and Colles' fascia) – superficial fasciitis – or progress to involve deep perineal fascia or muscles. As the infecting microorganisms invade fascial planes, localized ischemia, vascular occlusion, and tissue necrosis occurs. In this process, superficial nerves are damaged, leading to the characteristic but not invariable anesthesia of the wound.

The serious complications of NF result from the pathophysiology of the infecting organisms. The combined release of pyrogenic bacterial exotoxins and streptococcal antigens leads to the elaboration of cytokines, resulting in various additional clinical signs and symptoms including hypotension. Hydrogen, nitrogen, hydrogen sulfide, and methane gases produced by bacterial action can result in gas forming in infected areas, leading to the classic finding of wound crepitation. The most common presenting symptom for NF is the sudden onset of severe perineal/vaginal pain, accompanied by characteristic "dishwater" serous wound discharge. Localized tenderness is often but not always present. As the disease progresses, the affected area usually becomes progressively anesthetic, although hyperesthesia can occur earlier. Complaints of severe pain, the characteristic discharge, and crepitus, erythema, edema, or bullae beyond the immediate episiotomy site or area of the laceration repair collectively suggest the cor-

rect diagnosis. Skin changes overlying the involved area vary, especially on the perineum. In addition, labial edema is not a reliable sign of this infection, unless it is unilateral and extreme.

Associated laboratory findings can include evidence of hemoconcentration, anemia, and occasionally hypocalcemia. The last is believed to occur because of saponification of fatty acids within tissue spaces [168]. The white blood count is also usually but not invariably elevated above 14,000/ml. The suspicion of progression, combined with complaints of severe pain and symptoms of systemic toxicity, differentiate NF from simple cellulitis.

If NF is suspected, visual observation alone is insufficient to establish the correct diagnosis. A biopsy of the suspected area must be performed. Standard radiographs are not helpful; MRI or CT studies can identify gas in tissues and could be of assistance in delineating the extent of the NF, but these studies remain ancillary to direct surgical exploration and are not considered confirmatory. Indications for surgical exploration of a suspect episiotomy or perineal laceration site include extension of an infection beyond the labia, severe unilateral labial edema, systemic signs/symptoms of toxicity, deterioration in clinical status, and persistence of apparent infection beyond 24 to 48 hours despite routine antibiotic therapy or wound drainage [170–173]. When the characteristic clinical picture is present, prompt open biopsy of suspected areas with immediate frozen-section study is mandatory. Characteristic histologic findings include gram-positive coccobacilli in tissue planes, polymorphonuclear cell infiltration of the deep dermis and fascia, vessel inflammation with fibroid necrosis, the presence of venous and arterial thrombi, and fascial necrosis with absence of muscle involvement. Frozen-section data must be interpreted in light of the overall clinical picture, because falsely reassuring results are possible. A high degree of clinical suspicion and selective rebiopsy might be required to establish the correct diagnosis [172,173]. If NF is strongly suspected, with or without a supportive biopsy report, prompt surgical exploration with aggressive débridement is indicated, because this condition is potentially life threatening and has the potential for explosive advancement.

At surgery, as the wound is probed, the finding of a characteristic watery discharge, easy

separation of tissue from deeper fascia, yellowish-green necrotic fascia, and failure of tissues to bleed following incision are consistent with the presumptive diagnosis. Surgical treatment must be prompt and aggressive, because the area of necrosis is typically more advanced than anticipated and the infection advances rapidly. Radical débridement of all devitalized tissues until active bleeding is encountered is required. Extensive dissection into the buttocks, anterior abdominal wall, or thigh is possible, because any infected muscle, fascia, or connective tissue must be entirely extirpated. The surgical wound should be copiously irrigated and left open. Periodic reevaluations and repeat débridement might be required, at times on a daily basis. General supportive measures include ample intravenous hydration, the administration of broad-spectrum antibiotics, and close cardiovascular monitoring. The principal treatment is aggressive surgical removal of devitalized tissue, however. Medical management is ancillary. If *Streptococcus* is believed to be the primary organism, high-dose penicillin or ampicillin, combined with anaerobic coverage such as provided by gentamicin combined with clindamycin, vancomycin, or even chloramphenicol are suggested [168]. The benefit from hyperbaric oxygen or the administration of intravenous immunoglobulin is unclear. Because these treatments can reduce the mortality rate, their use as ancillary techniques is favored by some.

Progressive synergistic bacterial gangrene (Meleney ulcer) is an indolent variant of fascial necrosis, rarely encountered in obstetric practice. Characteristically, in this condition there is a central, necrotic ulcer with two surrounding zones. The inner zone is dark red to purple in appearance; the outer is erythematous. This slowly progressive, painful ulcerative lesion is associated with the same mixed bacterial flora characteristic of the more rapidly advancing forms of necrotizing fasciitis. Treatment consists of surgical débridement and the administration of broad-spectrum antibiotics.

Infection reaching deep tissues can result in the rarest and most extreme form of NF (Type III), classic gas gangrene or myonecrosis [168]. The organism most frequently associated with obstetric infections of this type is *Clostridium perfringens*. This infection can result as an extension of previously occurring superficial fasciitis or develop de novo. Severe pain, systemic signs of sepsis, rapid-to-explosive progres-

sion, cutaneous gangrene, and wound crepitation are the classic signs. If *C. perfringens* is the cause, rapid and massive intravascular hemolysis, severe vascular volume constriction, and marked renal dysfunction are common, accompanying rapid cardiovascular collapse and other signs of extreme toxicity. Shock with renal failure is the usual cause of death. In terms of diagnosis, a smear or frozen-section biopsy of the deep wound tissue reveals plump gram-positive rods. Treatment includes immediate and aggressive surgical wound débridement, high-dose penicillin therapy, and general supportive treatment. The concomitant use of polyvalent antitoxin and hyperbaric oxygen are ancillary measures to aggressive surgery, and in this setting they are of uncertain benefit. Mortality remains high (67%–100%). Fortunately, many clinicians will never experience these rare cases.

## Special Issues

### *Histologic Placental Examination*

Close and critical review of obstetric management is never more intense than when a neurologically damaged or “bad” baby results from a delivery [174,179,181]. Such cases are often complex and difficult to defend legally. A complete histologic examination of the placenta by an experienced pathologist can provide important data concerning the etiology of an infant’s injury and should never be omitted when fetal injuries are observed or suspected at birth. Potential benefits from placental examination are several. If a pathologic condition involving the fetus is present, it might be possible to determine if the problem was acute or chronic. Furthermore, the etiology of specific clinical entities such as premature/preterm delivery, intrauterine growth retardation (IUGR), stillbirth, or neurologic injury might be revealed by combined gross, histologic, and specialized laboratory study of the placenta and the membranes [174,178–179,182]. The placental findings of nucleated red blood cells, chronic ischemia, intimal cushions, intervillous fibrin, and acute and chronic meconium staining, among other findings, can help to determine whether acute or chronic fetal disorder were a factor in the etiology of a child’s observed deficits. Veteran pathologists emphasize that both experience and humility are necessary to evaluate

TABLE 11.6 Conditions for which Placental Examination Is Suggested

Fetal Conditions	Maternal Conditions	Placental Conditions
Perinatal death/stillborn	IDDM/GDM	Gross abnormality of the placenta, cord, or membranes
Multiple gestations	Pregnancy induced hypertension (PIH)	Placenta accreta/percreta
Congenital anomalies	Post term pregnancy (>42 weeks)	Chorioangioma or masses in the placenta
Growth restriction	Fever and/or infection	
Hydrops/polyhydramnios/ oligohydramnios	Drug or substance abuse	
Thick meconium	Repetitive bleeding	
Admission to NICU	Abnormal serum screening/ thrombophilia	
Apgar score <3 at 5 minutes	History of reproductive failure	
Suspected infection	IVF pregnancy	
Seizures	Previous caesarean section	
Gestation <32 weeks	Premature labor/premature rupture of membranes	
	Preterm premature rupture of membranes	

placental specimens appropriately. At present, many institutions follow the recommendations of the College of American Pathologists Consensus Committee in determining which placentas to study [180] (Table 11.6). Some institutions have implemented various programs for routine gross placental examination, with preparation and permanent storage of microscopic blocks should subsequent histologic examination be required, even years later.

## REFERENCES

- Mauriceau F: Observations sur la Grossesse et l'Accouchement des Femmes, et sur Leurs Maladies et celles des Enfants nouveau-nes, Edition Deuxième. Paris: La Compagnie des Libraires Associez, 1740.
- Speert H: Obstetrics and Gynecology in America: A History. Washington, DC: American College of Obstetricians and Gynecologists, 1980.
- Dewees WP: Compendious System of Midwifery. Philadelphia: H. C. Carey and I. Lea, 1826.
- Dewhurst J. Royal Confinements. New York: St Martin's Press, 1980.
- Herman A, Weinraub Z, Bukovsky I, Arielis S: Dynamic ultrasonographic imaging of the third stage of labor: New perspectives into third-stage mechanism. *Am J Obstet Gynecol* 1993;168:1496.
- Herman A: Complicated third stage of labor: Time to switch on the scanner. *Ultrasound Obstet Gynecol* 2000;15:89.
- Deyer TW, Ashton-Miller JA, Van Baren PM, Pearlman MD: Myometrial contractile strain at uteroplacental separation during parturition. *Am J Obstet Gynecol* 2000 Jul; 183(1):156-159.
- Benirschke K, Kaufman P: Pathology of the Human Placenta, 2nd ed. New York: Springer-Verlag, 1990; pp. 275-297.
- Khudr G, Soma H, Benirschke K: Trophoblastic origin of the X cell and the placental giant cell. *Am J Obstet Gynecol* 1973;115:530-533.
- Kim CK, Benirschke K: Autoradiographic study of the "X-cells" in the human placenta. *Am J Obstet Gynecol* 1971;109:96-102.
- Kim CK, Altshuler GP, Benirschke K: Karyotypic analysis of the X-cell in the human placenta. *Am J Obstet Gynecol* 1971;37:72-82.
- Kim CK, Najtolin T, Benirschke K: Immunohistochemical studies of the X-cell in the human placenta with anti-human chorionic gonadotropin and anti-human placental lactogen. *Am J Obstet Gynecol* 1971;111:672-676.
- Ludwig H: Surface structure of the human term placenta and the uterine wall postpartum in the screen scan electron microscope. *Am J Obstet Gynecol* 1971 Oct;111(3):328-344.
- Anderson WR, Davis J: Placental site involution. *Am J Obstet Gynecol* 1968;102:23-33.
- Mukaida T, Yoshida K, Soma H: A surface ultrastructural study of placental separation site. *Journal of Clinical Electron Microscopy* 1975;8:5-6.
- Anderson WR, Davis J: Placental site involution. *Am J Obstet Gynecol* 1968;102:23-33.

17. Lester WM, Bartholomew RA, Calvin ED, Grimes WH, Fish JS, Galloway WH: Role of retained placental fragments in immediate and delayed postpartum hemorrhage. *Am J Obstet Gynecol* 1956;72:1214–1226.
18. Hoberman LK, Hawkinson JA, Beechman CT: Placental polyps: Report of 3 cases. *Obstet Gynecol* 1963;22:25–29.
19. Dyer I, Bradburn DM: An inquiry into the etiology of placental polyps. *Am J Obstet Gynecol* 1971;109:858–857.
20. Thoreinsson VT, Kempers RD: Delayed postpartum bleeding. *Am J Obstet Gynecol* 1970;107:565–571.
21. Levy V, Moore J: The midwife's management of the third stage of labour. *Nurs Times* 1985;81:47–50.
22. Baskett TF: Acute uterine inversion: A review of 40 cases. *J Obstet Gynaecol Can.* 2002;24(12):953–956.
23. Bonham DG: Intramuscular oxytocics and cord traction in the third stage of labour. *Br Med J* 1963;2:1620–1623.
24. Kimbell N: Brandt-Andrews technique of delivery of the placenta. *Br Med J* 1954;1:130–132.
25. Andrews CJ: The third stage of labor with an evaluation of the Brandt method of expression of the placenta. *South Med Surg* 1940;102:605–608.
26. Kemp J: A review of cord traction in the third stage of labour from 1963–1969. *Med J Aust* 1971;1:899–903.
27. Brandt ML: The mechanism and management of the third stage of labor. *Am J Obstet Gynecol* 1933;25:662–665.
28. Fox H: *Pathology of the Placenta*. Philadelphia: W. B. Saunders, 1978.
29. Naeye RL: *Disorders of the Placenta, Fetus and Neonate*. St. Louis: Mosby-Year Book, 1992; pp. 92–115.
30. Rayburn WF, Beynen A, Brinkman DL: Umbilical cord length and intrapartum complications. *Obstet Gynecol* 1981;57:450–452.
31. Hammar M, Bostrom K, Borgvall B: Comparison between the influence of methylergometrine and oxytocin on the incidence of retained placenta in the third stage of labour. *Gynecol Obstet Invest* 1990;30(2):91–93.
32. Prendiville W, Elbourne D, McDonald S: Active versus expectant management in the third stage of labour. *Cochrane Database Syst Rev.* 2000; 3.
33. Cotter A, Ness A, Tolosa J: Prophylactic oxytocin for the third stage of labour. *Cochrane Database Syst Rev* 2001;4.
34. Sorbe B: Active pharmacologic management of the third stage of labor. *Obstet Gynecol* 1978;52:694–697.
35. Pierre F, Mesnard L, Body G: For a systematic policy of i.v. oxytocin-induced placenta deliveries in a unit where a fairly active management of the third stage of labor is yet applied: Results of a controlled trial. *Eur J Obstet Gynecol Reprod Biol.* 1992 Jan;43(2):131–135.
36. McDonald S, Abbott JM, Higgins SP: Prophylactic ergometrine-oxytocin versus oxytocin for the third stage of labour. *Cochrane Database Syst Rev.* 2004; 1.
37. Elbourne DR, Prendiville WJ, Carroli G, Wood J, McDonald S: Prophylactic use of oxytocin in the third stage of labour. *Cochrane Database Syst Rev.* 2001; 4.
38. Thomas IL, Jeffers TM, Brazier JM, Burt CL, Barr KE: Does cord drainage of placental blood facilitate delivery of the placenta? *Aust NZ J Obstet Gynecol* 1990;30:314–318.
39. Soltani H, Dickinson F, Symonds I: Placental cord drainage after spontaneous vaginal delivery as part of the management of the third stage of labour. *Cochrane Database of Syst Rev* 2005; 4.
40. Wingeier R, Griggs R: Management of retained placenta using intraumbilical oxytocin injection. *J Nurse Midwifery* 1991;36:240–244.
41. Bivins HA, Cope DA, Darrell A, Newman RB, Eller DP: Randomized trial of intraumbilical vein oxytocin in midtrimester pregnancy losses. *Am J Obstet Gynecol* 1993;169:1070–1073.
42. Huber MGP, Wildschut HJJ, Boer K, Kesiverda G, Hoek FJ: Umbilical vein administration of oxytocin for the management of retained placenta: Is it effective? *Am J Obstet Gynecol* 1991;164:1216–1219.
43. Bider D, Zolti M, Menashe Y, Dulitzky M, Mashiach S, Ben-Rafael Z: Oxytocin or saline injected intra-umbilically did not influence the third stage of labor. *Acta Obstet Gynecol Scand* 1991; 70:321–323.
44. Reddy VV, Carey JC: Effect of umbilical vein oxytocin on puerperal blood loss and length of the 3rd stage. *Am J Obstet Gynecol* 1989;160:206–208.
45. Carroli G, Bergel E: Umbilical vein injection for management of retained placenta. *Cochrane Database Syst Rev.* 2001; 4.

46. Stearns J: Letter J. Stearns Saratoga County, New York to Mr. S. Akerly, dated Waterford, January 25, 1807. An account of the therapeutic use of pulvis parturiens. *Medical Repository*, V, 1807.
47. Chong YS, Su L, L Arulkumaran S: Misoprostol: A quarter century of use, abuse, and creative misuse. *Obstet Gynecol Sur*. 2004 Feb;59(2):128–140.
48. Joy SP, Sanchez-Ramos L, Kaunitz AM: Misoprostol use during the third stage of labor. *Intern J Gynecol Obstet*. 2003; 82:143–152.
49. Sharma S, El Refaeyy H: Prostaglandins in the prevention and management of postpartum haemorrhage. *Best Pract Res Clin Obstet Gynaecol* 2003 Oct;17(5):811–23.
50. Gülmezoglu AM, Forna F, Villar J, Hofmeyr GJ: Prostaglandins for prevention of postpartum haemorrhage. *Cochrane Database Syst Rev*. 2004; 1.
51. Poeschmann RP, Doesburg WH, Eskes TK: A randomized comparison of oxytocin, sulprostone and placebo in the management of the third stage of labour. *Br J Obstet Gynaecol* 1991 Jun;98(6):528–530.
52. Spears RL, Anderson GV, Brotman S, Farrier J, Kwan J, Masto A: The effect of early versus late cord clamping on signs of RDS. *Am J Obstet Gynecol* 1966;95:564–568.
53. Frank DJ, Gabriel M: Timing of cord ligation and newborn RDS. *Am J Obstet Gynecol* 1967; 97:1142–1144.
54. Gupta R, Ramji S: Effect of delayed cord clamping on iron stores in infants born to anemic mothers: A randomized controlled trial. *Indian Pediatr* 2002 Feb;39(2):130–135.
55. Ladipo OA: Management of the third stage of labour, with particular reference to reduction of feto-maternal transfusion. *Br Med J* 1972 Mar; 1(5802):721–723.
56. Thomas IL, Jeffers TM, Brazier JM, Burt CL, Barr KE: Does cord drainage of placental blood facilitate delivery of the placenta? *Aust N Z J Obstet Gynaecol* 1990 Nov;30(4):314–318.
57. Scott JR: Episiotomy and vaginal trauma. *Obstet Gynecol Clin North Am* 2005 Jun;32(2):307–321.
58. Hartmann K, Viswanathan M, Palmieri R, Gartlehner G, Thorp J: Outcomes of routine episiotomy: A systematic review. *JAMA* 2005 May; 293(7):2141–2148.
59. Barter RH, Parks J, Tyndal P: Median episiotomies and complete perineal lacerations. *Am J Obstet Gynecol* 1960;80:654–662.
60. Dallas DA: The routine use of episiotomy with a description of the continuous knotless repair. *West J Surg Obstet Gynecol* 1953;61:24–35.
61. Valsky D, Rozenak D, Patkova R, Savchev S, Messing B, Yagel D, Hochner-Celnikier D: Postpartum evaluation of anal sphincter tears, clinically unrecognized and immediately following surgical repair, by transperineal 3D US in primiparous women. *Am J Obstet Gynecol* 2005;193(6):S47–S47.
62. Sleep J, Roberts J, Chalmers I: Care in the second stage of labour. In: Chalmers I, Enkin M, Kierse MJNC (eds): *Effective Care in Pregnancy and Childbirth*. Oxford: Oxford University Press, 1989; pp. 1129–1144.
63. Ould F: *A Treatise of Midwifery in Three Parts*. Dublin: Nelson, 1742.
64. Nugent FB: The primiparous perineum after forceps delivery. *Am J Obstet Gynecol* 1935;30:249–256.
65. Broomall AE: The operation of episiotomy as a prevention of perineal ruptures during labor. *Am J Obstet Dis Woman Child* 1878;11:517–527.
66. Pomeroy R: Shall we cut and reconstruct the perineum for every primipara? *Am J Obstet Gynecol Dis Woman Child* 1918;78:211–220.
67. DeLee JB: The prophylactic forceps operation. *Am J Obstet Gynecol* 1920;1:34–44.
68. Thacker SB, Banta HD: Benefits and risks of episiotomy: An interpretive review of the English language literature 1860–1980. *Obstet Gynecol Surv* 1983;38:322–338.
69. Wilcox LS, Strobino DM, Baruffi G, Dellinger WS: Episiotomy and its role in the incidence of perineal lacerations in a maternity center and a tertiary hospital obstetric practice. *Am J Obstet Gynecol* 1986;160:1047–1052.
70. Green JR, Soohoo SL: Factors associated with rectal injury in spontaneous deliveries. *Obstet Gynecol* 1989;73:732–738.
71. Shiono P, Klebanoff MA, Carey CJ: Midline episiotomies: More harm than good? *Obstet Gynecol* 1990;75:765–770.
72. Coats PM, Chan KK, Wilkins M, Beard RJ: A comparison between midline and mediolateral episiotomies. *Br J Obstet Gynaecol* 1980 May;87(5): 408–412.
73. Sleep JM, Grant A, Garcia J, Elbourne D, Spencer J, Chalmers I: West Berkshire perineal management trial. *Br Med J* 1984;289:587–590.

74. Thorp JM, Bowes WA Jr, Brame RG, Cefalo R: Selected use of midline episiotomy: Effect on perineal trauma. *Obstet Gynecol* 1987 Aug;70(2):260-262.
75. Thorp JM, Bowes WA Jr. Episiotomy: Can its routine use be defended? *Am J Obstet Gynecol* 1989; 160:1027-1030.
76. Power RMH: The pelvic floor in parturition. *Surg Gynecol Obstet* 1946;83:296-311.
77. Gainey HL: Postpartum observation of pelvic tissue drainage. *Am J Obstet Gynecol* 1943;45:457-466.
78. Aldridge AH, Watson P: Analysis of end results of labor in primiparas after spontaneous versus prophylactic methods of delivery. *Am J Obstet Gynecol* 1935;30:554-565.
79. Gainey HL: Postpartum observation of pelvic tissue damage: Further studies. *Obstet Gynecol* 1955;70:800-807.
80. Cohen WR: The pelvic division of labor. In: Cohen WR, Friedman EA (eds): *Management of Labor*. Baltimore: University Park Press, 1983.
81. Klein MC, Gauthier RJ, Robbins JM, Kaczorowski J, Jorgensen SH, Franco ED, Johnson B, Waghorn K, Gelfand MM, Guralnick MS: Relationship of episiotomy to perineal trauma and morbidity, sexual dysfunction, and pelvic floor relaxation. *Am J Obstet Gynecol* 1994 Sep;171(3):591-598.
82. Eogan M, O'Brien C, Daly L, Byrne P, O'Connell C, O'Herlihy C: Monitoring pudendal nerve conduction during labor and early puerperium. *Am J Obstet Gynecol* 2005;193(6):S41.
83. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI: Anal sphincter disruption during vaginal delivery. *N Engl J Med* 1993;329:1905-1911.
84. American College of Obstetricians and Gynecologists: Postpartum hemorrhage. Technical Bulletin No. 76. October, 2006. Washington D.C.: The American College of Obstetricians and Gynecologists.
85. B-Lynch C, Keith LG, Lalonde Andre B, Karoshi M: *A Textbook of Postpartum Hemorrhage*. Duncow, Kirkmahoe, Dumfriesshire UK, Sapiens Publishing, 2006.
86. AbouZahr C: Global burden of maternal death and disability. *Br Med Bull* 2003;67:1-11.
87. Varner M: Postpartum hemorrhage. *Crit Care Clin* 1991;7:883-897.
88. Kaunitz AM, Hughes JM, Grimes DA, Smith JC, Rochat RW, Kafrissen ME: Causes of maternal mortality in the United States. *Obstet Gynecol* 1985 May;65(5):605-612.
89. Pritchard JA, Baldwin RM, Dickey JC: Blood volume changes in pregnancy and the puerperium, II: Red blood cell loss and changes in apparent blood volume during and following vaginal delivery, cesarean section, and cesarean section plus total hysterectomy. *Am J Obstet Gynecol* 1962;84:1271-1273.
90. El-Refaey S, Rodeck C: Postpartum haemorrhage: Definitions, medical and surgical management: A time for change. *Br Med J* 2003;67:205-217.
91. Beydoun SN: Postpartum hemorrhage and hypovolemic shock. In: Hassam F (ed): *Diagnosis and Management of Obstetric Emergencies*. Menlo Park, CA: Addison-Wesley, 1982; pp. 193-213.
92. Benedetti TJ: Obstetric hemorrhage. In: Gabbe SG (ed): *Obstetrics: Normal and Problem Pregnancies*. New York: Churchill Livingstone, 1991; pp. 485-515.
93. Bagga R, Jain V, Kalra J, Chopra S, Gopalan S: Uterovaginal packing with rolled gauze in postpartum hemorrhage. *Med Gyn Med* 2004 Feb; 6(1):50.
94. Haq G, Tayyab S: Control of postpartum and postabortal haemorrhage with uterine packing. *J Pak Med Assoc* 2005 Sep;55(9):369-371.
95. Hsu S, Rodgers B, Lele A, Yeh J: Use of packing in obstetric hemorrhage of uterine origin. *J Reprod Med* 2003 Feb;48(2):69-71.
96. Druzin ML: Packing of lower uterine segment for control of postcesarean bleeding in instances of placenta previa. *Surg Gynecol Obstet* 1989 Dec;169(6):543-545.
97. Wittich AC, Salminen ER, Hardin EL, Desantis RA: Uterine packing in the combined management of obstetrical hemorrhage. *Mil Med* 1996 Mar;161(3):180-182.
98. Maier RC: Control of postpartum hemorrhage with uterine packing. *Am J Obstet Gynecol* 1993; 169:317-323.
99. Chung JW, Jeong HJ, Joh JH, Park JS, Jun KJ, Kim SH: Percutaneous transcatheter angiographic embolization in the management of obstetric hemorrhage. *J Reprod Med* 2003 Apr;48(4):268-276.
100. Ornan D, White R, Pollak J, Tal M: Pelvic embolization for intractable postpartum hemorrhage: Long-term follow-up and implications for fertility. *Obstet Gynecol* 2003;102(5 Pt 1):904-910.

101. Deux JF, Bazot M, Le Blanche AF, Tassart M, Khalik A, Berkane N, Uzan S, Boudghene F: Uncontrollable postpartum bleeding. *AJR Am J Roentgenol* 2001;177(1):145–149.
102. Akhter S, Begum MR, Kabir Z, Rashid M, Laila TR, Zabeen F: Use of a condom to control massive postpartum hemorrhage. *Med Gen Med* 2003 Sep 11;5(3):38.
103. Bakri YN, Amri A, Abdul Jabbar F: Tamponade-balloon for obstetrical bleeding. *Int J Gynaecol Obstet.* 2001 Aug;74(2):139–142.
104. De Loor JA, van Dam PA: Foley catheters for uncontrollable obstetric or gynecologic hemorrhage. *Obstet Gynecol* 1996 Oct;88(4 Pt 2):737.
105. Seror J, Allouche C, Elhaik S: Use of Sengstaken-Blakemore tube in massive postpartum hemorrhage: A series of 17 cases. *Acta Obstet Gynecol Scand* 2005 Jul;84(7):660–664.
106. Ojala K, Perälä J, Kariniemi J, Ranta P, Raudaskoski T, Tekay A: Arterial embolization and prophylactic catheterization for the treatment for severe obstetric hemorrhage. *Acta Obstet Gynecol Scand* 2005 Nov;84(11):1075–1080.
107. O'Leary JL, O'Leary JA: Uterine artery ligation for control of postcesarean section hemorrhage. *Obstet Gynecol* 1974;43:849–853.
108. Clark SL, Phelan JP, Yeh SY, Bruce SR, Paul RH: Hypogastric artery ligation for obstetric hemorrhage. *Obstet Gynecol* 1985;66:353–356.
109. Clark SL, Yeh SY, Phelan JP, Bruce SR, Paul RH: Emergency hysterectomy for obstetric hemorrhage. *Obstet Gynecol* 1984;64:376–380.
110. Allam MS, B-Lynch C: The B-Lynch and other uterine compression suture techniques. *Int J Gynaecol Obstet* 2005 Jun;89(3):236–241.
111. Lee CY, Madrazo B, Drukker BH: Ultrasonic evaluation of the postpartum uterus in the management of postpartum bleeding. *Obstet Gynecol* 1981;58:227–232.
112. Achanna S, Mohamed Z, Krishnan M. Puerperal uterine inversion: A report of four cases. *J Obstet Gynaecol Res* 2006 Jun;32(3):341–345.
113. Miras T, Collet F, Seffert P. Acute puerperal uterine inversion: Two cases. *J Gynecol Obstet Biol Reprod (Paris)* 2002 Nov;31(7):668–671.
114. Platt LD, Druzin ML. Acute puerperal inversion of the uterus. *Am J Obstet Gynecol* 1981;141:187–190.
115. Cunningham FG, MacDonald PC, Leveno KJ, et al: Conduct of normal labor and delivery. In: Cunningham FG, MacDonald PC, Leveno KJ (eds): *Williams Obstetrics*, 19th ed. Norwalk, CT: Appleton & Lange, 1993; pp. 371–394.
116. Watson P, Besch N, Bowes WA Jr: Management of acute and subacute puerperal inversion of the uterus. *Obstet Gynecol* 1980 Jan;55(1):12–16.
117. Bowes WA Jr, Watson PT: Inversion of the uterus. In: Iffy L, Apuzzio JJ, Vintzeleos AM (eds): *Operative Obstetrics*, 2nd ed. New York: McGraw-Hill, 1992; pp. 298–303.
118. Kochenour NK: Intrapartum obstetric emergencies. *Crit Care Clin* 1991 Oct;7(4):851–864.
119. Shah-Hoseini R, Evrard JR: Puerperal uterine inversion. *Obstet Gynecol* 1989;73:567–570.
120. Bellucci M, DiOrio J, Moubayed S: Uterine inversion secondary to placenta accreta in a diethylstilbestrol-exposed parturient: A case report. *J Reprod Med* 1987;32:236–237.
121. Bell JE Jr, Wilson F, Wilson L: Puerperal inversion of the uterus. *Am J Obstet Gynecol* 1953;66:767–769.
122. Romo MS, Grimes DA, Strassle PO: Infarction of the uterus from subacute incomplete inversion. *Am J Obstet Gynecol* 1992 Mar;166(3):878–879.
123. Kovacs BW, DeVore GR: Management of acute and subacute puerperal uterine inversion with terbutaline sulfate. *Am J Obstet Gynecol* 1984;150:784–786.
124. Thierry M, Delbeke L: Acute uterine inversion: Two-step management with a beta-mimetic and a prostaglandin. *Am J Obstet Gynecol* 1985;153:891–892.
125. Catanzarite VA, Moffitt KD, Baker ML, Awadalla SG, Argubright KF, Perkins RP: New approaches to the management of acute puerperal uterine inversion. *Obstet Gynecol* 1986;68:7s–10s.
126. Thomas AJ, Greer IA: Non-haemorrhagic obstetric shock. *Baillieres Best Pract Res Clin Obstet Gynaecol* 2000 Feb;14(1):19–441.
127. Beringer RM, Patteril M: Puerperal uterine inversion and shock. *Br J Anaesth* 2004;92(3):439–441.
128. Watson P, Besch N, Bowes WA Jr. Management of acute and subacute puerperal inversion of the uterus. *Obstet Gynecol* 1980 Jan;55(1):12–16.
129. DeSimone CA, Norris MC, Leighton BL: Intravenous nitroglycerin aids manual extraction of a retained placenta. *Anesthesiology* 1990;73:787.
130. Beringer RM, Patteril M: Puerperal uterine inversion and shock. *Br J Anaesth* 2004;92(3):439–441.

131. Wendel PJ, Cox SM: Emergency obstetric management of uterine inversion. *Obstet Gynecol Clin North Am* 1986;68.
132. Dayan SS, Schwalbe SS: The use of small-dose intravenous nitroglycerine in a case of uterine inversion. *Anesth Analg* 1996;82:1091–1093.
133. Hostetler DR, Bosworth MF. Uterine inversion: A life-threatening obstetric emergency. *J Am Board Fam Pract* 2000;13:120–123.
134. Altabef KM, Spencer JT, Zinberg S: Intravenous nitroglycerin for uterine relaxation of an inverted uterus. *Am J Obstet Gynecol* 1992;166:1237–1238.
135. Ward HR: O'Sullivan's hydrostatic reduction of an inverted uterus: Sonar sequence recorded. *Ultrasound Obstet Gynecol* 1998 Oct;12(4):283–286.
136. Huntington JL: Acute inversion of the uterus. *Boston Med Surg J* 1921;15:376–381.
137. Brar HS, Greenspoon JS, Platt LD, Paul RH: Acute puerperal inversion: New approaches to management. *J Reprod Med* 1989;34:173–177.
138. Haultain FWN: The treatment of chronic uterine inversion by abdominal hysterectomy with a successful case. *Br Med J* 1901;2:974–980.
139. Wilson SB: Retained placenta. In: Friedman EA, Acker DB, Sachs BP (eds): *Obstetrical Decision Making*, 2nd ed. Philadelphia: BC Decker, 1987; pp. 264–265.
140. Combs CA, Laros RK: Prolonged third stage of labor: Morbidity and risk factors. *Obstet Gynecol* 1991;77:863–867.
141. Romero R, Hsu YC, Athanassiadis AP, Hagay Z, Avila C, Nores J, Roberts A, Mazor M, Hobbins JC: Preterm delivery: A risk factor for retained placenta. *Am J Obstet Gynecol* 1990;163:823–825.
142. Wilkin-Jensen C, Strom V, Nielsen MD, Rosenkilde-Gram B: Removing a retained placenta by oxytocin – a controlled study. *Am J Obstet Gynecol* 1989;161:155–156.
143. Earl V, Bulmer JN, Briones A: Placenta accreta: A immunohistological study of trophoblast populations. *Placenta* 1987;8:273–282.
144. Khong TY, Robertson WB: Placenta accreta and placenta praevia accreta. *Placenta* 1987;8:399–409.
145. Sumawong V, Nondasuta A, Thanapath S, Budthimedhee V: Placenta accreta: A review of the literature and a summary of 10 cases. *Obstet Gynecol* 1966 Apr;27(4):511–516.
146. Friedman A, DeFazio J, DeCherney A: Severe obstetric complications after aggressive treatment of Asherman syndrome. *Obstet Gynecol* 1986; 67:864–867.
147. Bulmer JN: Immune aspects of pathology of the placental bed contributing to pregnancy pathology. *Baillière Clin Obstet Gynaecol* 1992;6:461–488.
148. Barss P, Misch KA: Endemic placenta accreta in a population of remote villagers in Papua New Guinea. *Br J Obstet Gynaecol* 1990 Feb;97(2): 167–174.
149. Ecker JL, Sorem KA, Soodak L, Roberts DJ, Safon LE, Osathanondh R: Placenta increta complicating a first-trimester abortion: A case report. *J Reprod Med* 1992;37:893–895.
150. Harden MA, Walters MD, Valente PT: Postabortal hemorrhage due to placenta increta: A case report. *Obstet Gynecol* 1990;75:523–526.
151. Kupfermirc MJ, Tamura RK, Wigton TR, Glassenberg R, Socol ML: Placenta accreta is associated with elevated maternal serum alpha-fetoprotein. *Obstet Gynecol* 1993 Aug;82(2):266–269.
152. McCool RA, Bombard AT, Bartholomew DA, Calhoun BC: Unexplained positive/elevated maternal serum alpha-fetoprotein associated with placenta increta: A case report. *J Reprod Med* 1992 Sep;37(9):826–828.
153. Ginsberg NA, Fausone ME, Gerbie M, Applebaum M, Verlinsky Y: Elevated maternal serum alpha-fetoprotein associated with placenta accreta. *J Assist Reprod Genet* 1992:497–500.
154. Zelop C, Nadel A, Frigoletto FD Jr, Pauker S, MacMillan M, Benacerraf BR: Placenta accreta/percreta/increta: A cause of elevated maternal serum alpha-fetoprotein. *Obstet Gynecol* 1992; 80:693–694.
155. Finberg HJ, Williams JW: Placenta accreta: prospective sonographic diagnosis in patients with placenta previa and prior cesarean section. *J Ultrasound Med* 1992;11:333–343.
156. Hoffman-Tretin JC, Koeningsberg M, Rabin A, Anyaegbunam A: Placenta accreta. Additional sonographic observations. *J Ultrasound Med* 1992; 11:29–34.
157. Rosemond RL, Kepple DM: Transvaginal color Doppler sonography in the prenatal diagnosis of placental accreta. *Obstet Gynecol* 1992;80:508–510.
158. Thorp JM Jr, Councell RB, Sandridge DA, Wiest HH: Antepartum diagnosis of placenta previa percreta by magnetic resonance imaging. *Obstet Gynecol* 1992;80:506–508.



159. Maldjian C, Adam R, Pelosi M, Pelosi M 3rd, Rudelli RD, Maldjian J: MRI appearance of placenta percreta and placenta accreta. *Magn Reson Imaging* 1999 Sep;17(7):965-971.
160. Stanco LM, Schrimmer DB, Paul RH, Mishell DR Jr: Emergency peripartum hysterectomy and associated risk factors. *Am J Obstet Gynecol* 1993; 168:879-883.
161. Ramin SM, Gilstrap LC II: Placental abnormalities: Previa, abruption and accreta. In: Plauch WC, Morrison JC, O'Sullivan M (eds): *Surgical Obstetrics*. Philadelphia: WB Saunders, 1992; pp. 203-216.
162. Sanders RR. Placenta praevia percreta invading the urinary bladder. *Aust N Z J Obstet Gynaecol* 1992;32:375-376.
163. Leaphart WL, Schapiro H, Broome J, Welander CJ, Bernstein IM: Placenta percreta with bladder invasion. *Obstet Gynecol* 1997;89:834-835.
164. Robie GF, Morgan MA, Payne GG Jr, Wasemiller-Smith L: Logothetopoulos pack for the management of uncontrollable postpartum hemorrhage. *Am J Perinatol* 1990 Oct;7(4):327-328.
165. Hallak M, Dildy GA 3d, Hurley TJ, Moise KJ Jr: Transvaginal pressure pack for life-threatening pelvic hemorrhage secondary to placenta accreta. *Obstet Gynecol* 1991;78:938-940.
166. Sweet RL, Gibbs RS: Postpartum infection. In: Sweet RI, Gibbs RS: *Infectious Diseases of the Female Genital Tract*. Baltimore: Williams & Wilkins, 1990; pp. 356-373.
167. Shy KK, Eschenbach DA: Fatal perineal cellulitis from an episiotomy site. *Obstet Gynecol* 1979; 54:292-298.
168. Maynor M: Necrotizing fasciitis. eMedicine, 2005 <http://www.emedicine.com/emerg/topic332.htm>.
169. Golde S, Ledger WJ: Necrotizing fasciitis in postpartum patients: A report of four cases. *Obstet Gynecol* 1977; 50:670-673.
170. Meltzer RM: Necrotizing fasciitis and progressive bacterial synergistic gangrene of the vulva. *Obstet Gynecol* 1983;61:757-760.
171. Ewing TL, Smale LE, Elliot FA: Maternal deaths associated with postpartum vulvar edema. *Am J Obstet Gynecol* 1979;134:173-179.
172. Pruitt BA: Biopsy diagnosis of surgical infection [editorial]. *N Engl J Med* 1984;310:1737-1738.
173. Stamenkovic L, Lew PD: Early recognition of potentially fatal necrotizing fasciitis: The use of frozen-section biopsy. *N Engl J Med* 1984;310: 1689-1693.
174. Aetshuler G: Placenta within the medicolegal imperative. *Arch Pathol Lab Med* 1991;115:688-695.
175. Hargitai B, Marton T, Cox PM: Examination of the human placenta. *J Clin Pathol* 2004;57:785-792.
176. Yetter JF: Examination of the placenta. *Am Fam Physician* 1998 Mar; 57(5):1045-1054.
177. Salafia CM, Vintzileos AM: Why all placentas should be examined by a pathologist in 1990. *Am J Obstet Gynecol* 1990 Oct;163(4 Pt 1):1282-1293.
178. Roberts DK: A guest editorial: Medical-legal aspects of placental examination. *Obstet Gynecol Surv* 1993;48:777-778.
179. Schindler NR: Importance of the placenta and cord in the defense of neurologically impaired infant claims. *Arch Pathol Lab Med* 1991;115:685-687.
180. Altshuler G, Deppisch LM: College of American Pathologists Conference XIX on the examination of placenta: Report of the working group on indications for placental examinations. *Arch Pathol Lab Med* 1991;115:701-703.
181. Benirschke K: The placenta in the litigation process. *Am J Obstet Gynecol* 1990;163:1445-1450.
182. Naeye RL: Functionally important disorders of the placenta, umbilical cord, and fetal membranes. *Hum Pathol* 1987;18:680-691.

## Martin L. Gimovsky

*It is an undoubted truth . . . that the several unnatural Postures, wherein Children present themselves at their Birth, are the cause of most of the bad Labours and ill Accidents for which usually recourse is had to Chirurgeons.*

F. Mauriçeau (1637–1709)

*The Diseases of Women and Child, and in Child-bed,*

H. Chamberlen (trans.)

London: Darby, 2nd edition, 1683, xiv, p 218.

Clinicians have long recognized the excessive perinatal morbidity and mortality associated with the breech-presenting fetus [1–6]. Breech presentation complicates 3% to 4% of all deliveries. In premature labor and delivery, breech presentation is a coincident finding in as many as 25% of pregnancies. As term approaches, however, this incidence falls dramatically (Table 12.1).

Multiple factors are responsible for the well-documented three- to fourfold increase in poor outcome seen with breech presentation compared with cephalic presentation [6–14]. The major problems are *congenital malformations* (Table 12.2), *prematurity*, and *traumatic birth injury*. These risks can occur separately or in conjunction.

In the past, severe birth injury and stillbirth were commonly associated with breech labor and delivery [15]. Umbilical cord prolapse and entrapment of the aftercoming head contributed to adverse outcome, as did potentially traumatic delivery techniques, such as total breech extraction. In addition, other obstetric risk factors (e.g., placenta previa, abruptio placentae, and multiple gestation) also contributed to a poor outcome.

Before the development of blood banking, antibiotics, and modern anesthetic techniques, routine vaginal delivery was the clear mode of choice for delivery of the breech-presenting fetus. A cesarean delivery was not a reasonable alternative for improving outcome because of its attendant risks of maternal morbidity and mortality.

The potential maternal risks from cesareans demanded the demonstration of clear benefits before this method of delivery could be routinely recommended. Historically, operative abdominal delivery was reserved for clinical situations in which the life of the mother was clearly in jeopardy. The well-being of the fetus was generally considered to be of secondary importance. (See Chapter 1, Operative Delivery: A History.)

The liberalization of indications for cesareans followed the development of increasingly safer operative procedures and better means to evaluate fetal condition. As cesarean delivery became an accepted routine, its use in breech labor and delivery

TABLE 12.1 Breech Presentation by Gestational Age\*

Gestational Age (wks)	Total Deliveries	Breech Fetus	% Breech
37–42	21,241	531	2.5
33–36	3,117	214	6.9
29–32	787	153	19.4
25–28	221	82	37.1
Total	25,366	980	3.9

\*Pooled data from refs. 2, 11, 22, 27, and 55.

TABLE 12.2 Anomalies Frequently Diagnosed in Breech Fetuses

CNS	Hydrocephaly Anencephaly Meningomyelocele Dysautonomia
Genitourinary	Potter's syndrome
Musculoskeletal	Myotonic dystrophy Congenital dislocation of the hips
Multiple anomalies	Prader-Willi syndrome Trisomy 13 Trisomy 18 Trisomy 21 De Lange syndrome Zollinger-Ellison syndrome Smith-Lemli-Opitz syndrome Fetal alcohol syndrome

CNS = central nervous system.

From Brenner WE, Bruce RS, Hendricks CH: The characteristics and perils of breech presentation. *Am J Obstet Gynecol* 1974 Mar 1; 118(5):700–12; with permission.

increased sharply (Figure 12.1). In a landmark study of 1,456 breech presentations delivered in the early 1950s, reported by Hall and Kohl [2], the authors concluded that cesarean delivery resulted in the lowest perinatal mortality. Among their most important observations was reflected in the statement, "... the perinatal mortality associated with *breech presentation* is not the same as for *breech delivery*..." [2]. In 1959, Ralph Wright went so far as to write that "... any patient of more than 35 weeks' gestation who entered labor with a living baby in breech presentation should be born by cesarean section, provided there was no maternal disease that contraindicated abdominal delivery..." [14].

In recent decades, techniques of medical intervention have evolved, improving the safety of child-

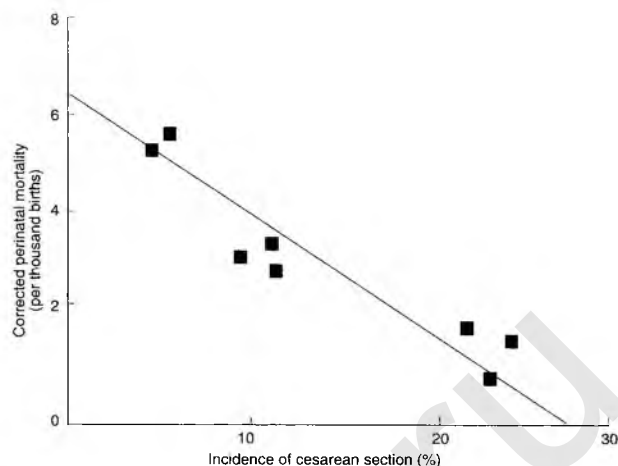


FIGURE 12.1.

*Relationship between death from labor and delivery in breech presentation and cesarean delivery rate. (From Gimovsky ML, Petrie RH: Strategy for choosing the best delivery route for the breech baby. Contemporary OB/GYN 1983;21:201–15; with permission.)*

birth [16–18]. Modern methods of fetal monitoring, the ability to promptly perform an emergency cesarean, and the widespread availability of safe anesthesia have reduced the maternal risks of abdominal delivery. Owing to these and other changes in both surgical practices and the attitudes of clinicians, most breech fetuses are now delivered by a cesarean.

Concurrent obstetric problems such as intrauterine growth restriction (IUGR), fetal distress in labor, cord prolapse, placenta previa, abruptio placentae, and difficulties in extracting the fetus are features of breech presentation that continue to complicate delivery. An international trial reported in 2000 attempted to evaluate these issues prospectively [19]. The reports issued by The Term Breech Trial are the subject of much debate [13,78,89–91]. Interpreted by some to be the definitive word on management of the term breech presentation, others have suggested that the results of this international trial seem more to provide support to the argument that an emergent cesarean delivery in labor is the major risk factor, regardless of presentation. Although the interpretation of this randomized trial is controversial, practitioners are well advised to consider an elective cesarean for their patients at term when the diagnosis of breech presentation is made prior to the onset of labor. The American College of Obstetricians and

Gynecologists (ACOG) Committee on Obstetric Practice recommends that “great caution . . . be exercised” in women undergoing an attempt at vaginal breech delivery [20]. It should be noted that their opinion specifically excludes women presenting in “. . . advanced labor . . . or patients whose second twin is in a nonvertex presentation.” This leaves open the possibility of a vaginal breech delivery under selected circumstances.

The controversy that remains is whether a cesarean should be performed routinely on all breech presentations when clinically possible, or whether specific groups of breech fetuses can be reasonably allowed a trial of labor (TOL). There is little controversy that the groups best served by cesarean delivery include fetuses that are between 26 and 34 weeks of gestation, those in incomplete or footling presentations, or any breech presentation in which the fetal head is extended at the neck. The presence of lethal congenital anomalies also affects the decision to perform a cesarean delivery [7,8]. In contrast, other risks seen in conjunction with breech presentation including the presence of nonlethal congenital anomalies (see Table 12.2) and extreme prematurity (26 weeks or less) might be minimally impacted by the route of delivery [10–13].

There are additional issues for consideration in the management of the low-birthweight breech fetus. Prior to the development of neonatal intensive care facilities, a cesarean to save a premature infant posed an unreasonable risk to maternal well-being. Clearly, that has changed dramatically with the availability of the neonatal intensive care unit (NICU) and contemporary management of very premature infants with the concomitant decline in morbidity and mortality.

With the change from routine vaginal delivery to virtually routine cesarean delivery for the breech-presenting fetus, a shift has occurred from the acceptance of a predominant risk of fetal/neonatal injury or death, to that of maternal surgical risk. The exact balance of decrease in fetal risk and increase in maternal risk associated with cesarean delivery is subject to debate. An additional consideration is the decreasing number of practitioners and institutions willing to conduct any trial of vaginal birth after a cesarean (VBAC). This has resulted in an ever-increasing number of women requiring cesarean delivery, with a concomitant increase in overall maternal morbidity.

A contemporary policy of routine cesarean delivery for the breech fetus is easy to understand. With a cesarean, the risks to the fetus are minimized, although they are not eliminated [21–22]. The resultant increase in maternal risk is difficult to quantify for an individual patient. In actual practice, a cesarean does not eliminate all preventable fetal morbidity and mortality [13]. Infants who succumb following surgical birth are usually either congenitally malformed or premature. Rarely, a normal infant is compromised during the delivery process by complications such as cord prolapse, difficult extractions, or unrecognized hyperextension of the aftercoming head. Because many cesarean deliveries must be performed to avoid even a single rare but severe injury, a policy of routine surgical delivery is not effective in the avoidance of risk unless virtually all breech fetuses are delivered by the abdominal route [14]. The clinical reality is that this goal is unobtainable.

What cesarean rate is appropriate for breech presentation? An analysis of retrospective series from the 1940s and 1950s demonstrates that deaths attributed to factors unique to breech presentation decrease as more cesarean deliveries are performed (see Figure 12.1). The data allow an estimation of an ideal operative delivery rate by extrapolation. To avoid virtually all breech-presentation–related complications that result in neonatal death, an overall cesarean rate of approximately 30% for breeches is required [24]. The challenge for the clinician is to select surgery for these cases which are at greatest risk and most likely to benefit. This observation is central to the use of clinical algorithms designed to select the route of delivery best suited to an individual patient [19,24–28].

Many clinicians believe that selected breech fetuses can safely be allowed a labor trial. A method to select those breech presentations that might be at lower risk and therefore safely be allowed a TOL deserves consideration. Selection of cases for a TOL is not complex but requires careful and thorough attention to detail. The use of external cephalic version (ECV) antepartum or in early labor to avoid breech labor and delivery also has advocates [29]. If selected cesarean delivery is possible, the resulting decrease in the frequency of surgical delivery can potentially benefit both mother and infant. Management of breech presentation is challenging. Experience in the conduct of breech labor and delivery,

whether during a cesarean or at a vaginal delivery, is vital to a safe outcome [13]. Cesarean breech delivery demands a clear knowledge of the same maneuvers used in a vaginal breech delivery. In fact, a poorly performed breech delivery during a cesarean results in injuries similar to those sustained at vaginal delivery. These include, among others, brachial plexus injuries, bony fractures, spinal cord injury, and crush injuries to the viscera [1,5,30].

This chapter reviews the fundamentals of the techniques for breech delivery and the evaluative process required for appropriate management. Also reviewed are external cephalic version (ECV) and internal podalic version (IPV) and the special needs of the premature breech fetus at delivery. These concepts and approaches are applicable in all breech presentations, independent of the route of delivery. This discussion is illustrated first by describing the basic precepts of vaginal breech delivery. As previously emphasized, when a breech fetus is delivered by cesarean, the basic principles of safe management are the same as those for a vaginal delivery. A basic problem exists in properly training new practitioners to manage breech presentation. In current practice, resident education programs simply cannot provide a sufficient number of vaginal breech delivery cases to permit students to learn the techniques and to develop the manual dexterity necessary to conduct safe breech delivery. *Approaching every cesarean breech delivery with the same precepts as employed at vaginal delivery can, at least in part, address these issues.*

## DEFINITIONS

### Breech Presentation

*Breech presentation* refers to the longitudinal lie of a fetus, with the breech or buttocks as the presenting part. The *position* in a breech presentation is designated by the orientation of the sacrum with respect to the maternal pelvic outlet. *Station* in breech presentation refers to the location in the pelvis of the lowermost portion of the buttocks in relationship to the plane of the maternal ischial spines. Station is expressed in centimeters ( $\pm 5$  cm) in the same manner as for a cephalic presentation.

There are several types of breech presentation that are defined by the anatomic relationships of the lower extremities at the hip and knee joints

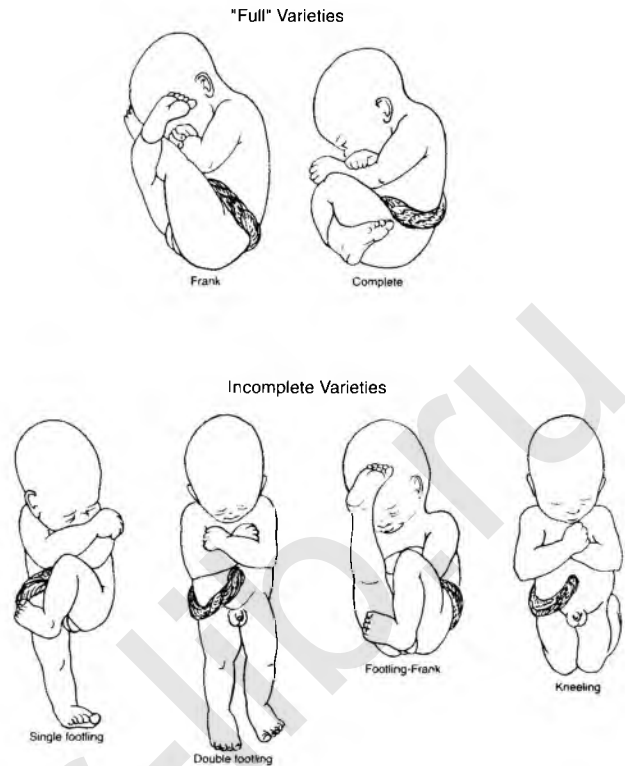


FIGURE 12.2.  
*Variations of breech presentations.*

(Figure 12.2). In *frank breech* presentation, both hip joints are flexed and both knee joints are extended. This is the most common type of breech and is seen in more than 50% of breech fetuses during labor at term. In the *complete breech* presentation, both hip joints and both knee joints are flexed. In the *incomplete breech*, there can be incomplete flexion at either the hip or knee joints. Thus the incomplete breech can present as either a *single* or *double footling*. Although pelvic examination is helpful in determining which type of breech fetus is present, confusion is possible between double footling and complete breeches, or between complete and incomplete breeches. Bedside real-time ultrasound scanning clarifies the exact type of presentation. Ultrasound scan also permits evaluation of the fetal head and its relationship to the cervical spine and can discover a fetal anomaly or another problem that was not previously diagnosed.

## MECHANISM OF LABOR

Labor for breech presentation is influenced by the mechanical disadvantages inherent in this

malpresentation. In comparison to the flexion that occurs at the cervical vertebrae in cephalic deliveries, in breech presentation flexion occurs at the lumbar vertebrae. Also significant is the difficulty encountered in the lateral flexion of the trunk necessary for delivery. Flexion of the trunk is especially problematic in frank breech presentation because of the tendency of the fetal legs to splint the body.

A breech fetus does not fill the birth canal as completely as a cephalic fetus. The proximity of the umbilical cord to the presenting part results in an increased risk of umbilical cord prolapse, a complication seen primarily during the expulsive maneuvers of the second stage of labor.

During the second stage of labor as the fetal body descends in the birth canal, a breech presentation delivers successively larger diameters of the fetal body to the bony pelvis, culminating with the largest bony diameter, the aftercoming head. Where a cephalic-presenting fetus might have hours for cranial molding to allow for adaptation and safe navigation through the bony pelvis, the aftercoming head in breech presentation has only minutes to accomplish the same task. Thus, any significant disproportion between the pelvis and the aftercoming head could result in a delay in delivery or fetal trauma resulting in significant fetal injury or rarely even death [6,15,21].

As labor with a breech enters the second stage, and after lateral flexion of the trunk occurs, the anterior hip is forced against and underneath the symphysis. The expulsive phase that follows delivers first the anterior and then subsequently the posterior buttock. As the back rotates anteriorly, the shoulders usually enter the pelvic inlet transversely. The shoulders then rotate and generally deliver in the oblique diameter where the pelvic size is greatest. Delivery maneuvers by the clinician are performed only in the final stages of expulsion. When the anterior scapula is delivering, the operator sweeps the infant's right humerus across the chest. Rotation of the fetal body then delivers the posterior scapula. The left humerus is then delivered in a similar fashion. It is important not to attempt delivery of the fetal arm until the scapula is visualized. At this point, the aftercoming head has entered the pelvic inlet slightly, and internal rotation and cranial flexion rapidly occur. Subsequently, the back of the fetal neck extends slightly against the symphysis pubis as

the chin passes over the perineum, completing the delivery.

## TECHNIQUES FOR DELIVERING THE BREECH FETUS

The approaches available for breech delivery include a *cesarean*, *spontaneous breech delivery*, *assisted breech delivery*, and *partial* and *total breech extraction*. External cephalic version and internal podalic version are additional obstetric techniques commonly associated with the delivery of the breech fetus.

### **Assisted Breech Delivery**

The preferred approach to the vaginal delivery of a breech fetus is by *assisted breech delivery*. In this process, delivery occurs with a minimal of operator interference. First, in preparation for the delivery assistants are identified and the maternal bladder is emptied. As previously noted, the infant is allowed to deliver spontaneously to the umbilicus with minimal intervention (Figure 12.4). A frank breech presentation can require concentrated maternal expulsive effort to permit the legs to be spontaneously expelled and patience on the part of the accoucheur is necessary. Whenever possible the fetus should be positioned so that the back is anterior. Delivery with the abdomen anterior is problematic and is best avoided because of its inherent difficulty. Once the infant is delivered to the umbilicus, several different maneuvers can be used to assist the delivery of the fetal body.

A combination of the *Løvset* and the *Bracht maneuvers* are favored for delivery of the fetus (Figure 12.3) [31]. With the infant delivered spontaneously to the umbilicus, the obstetrician electively wraps the fetal body in a warm, wet towel. In theory, this was originally done to inhibit fetal breathing efforts and maintain fetal warmth. Neither of these reasons is especially compelling, thus, the use of the towel is optional. Both of the surgeon's hands are placed, thumbs together, over the fetal sacrum. The hands need to be positioned on the pelvis/thighs, avoiding pressure over the kidneys and adrenal glands. This grasp must be sufficient to guide the body while at the same time care is used to avoid excessive force. The trunk is guided downward with gentle traction as the body is progressively delivered. As this occurs, if necessary, the

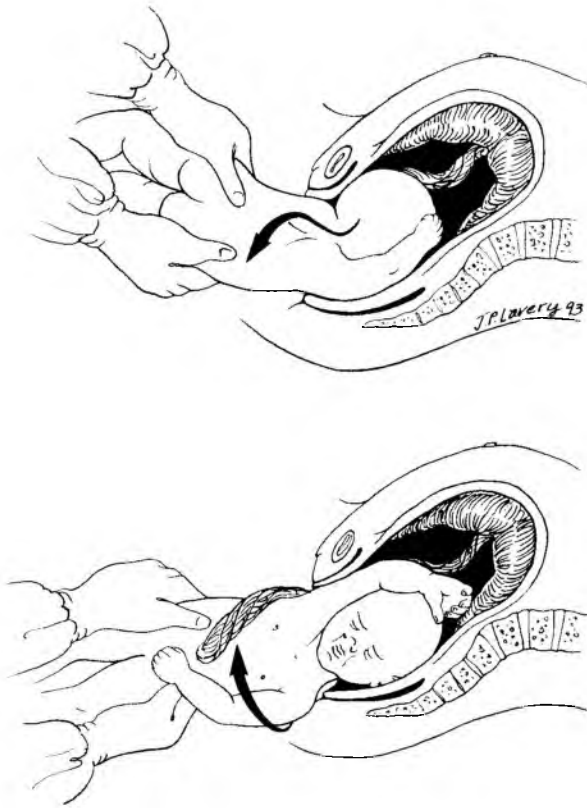


FIGURE 12.3.  
*Lovset maneuver. See text for details.*

body is rotated to bring the spine anteriorly. In performing a rotation of the fetal trunk while the head remains undelivered, the operator must also remember that the vertebral arteries and the cervical spine are susceptible to injury from excessive rotation or traction. While rare, serious spinal cord injuries are possible with manipulation of the fetal body unless such maneuvers are performed slowly and expertly. (See Chapter 23, Birth Injuries.)

As the scapula appears (wings), the fetal body is rotated first to one side and then to the other to permit delivery of the upper extremity by simply sweeping the hand downward across the chest. This extraction of the fetal arms by clockwise and counterclockwise rotation of the thorax/abdomen with minimal torque minimizes the risk of injury (Figure 12.3). This process is usually easy, and with rotation the fetal arms can spontaneously deliver without other manipulations. The head is then expelled by gentle but firm suprapubic pressure and the delivery completed. An episiotomy is usually performed to reduce the extent of traction necessary for cranial delivery unless the perineum is flaccid.

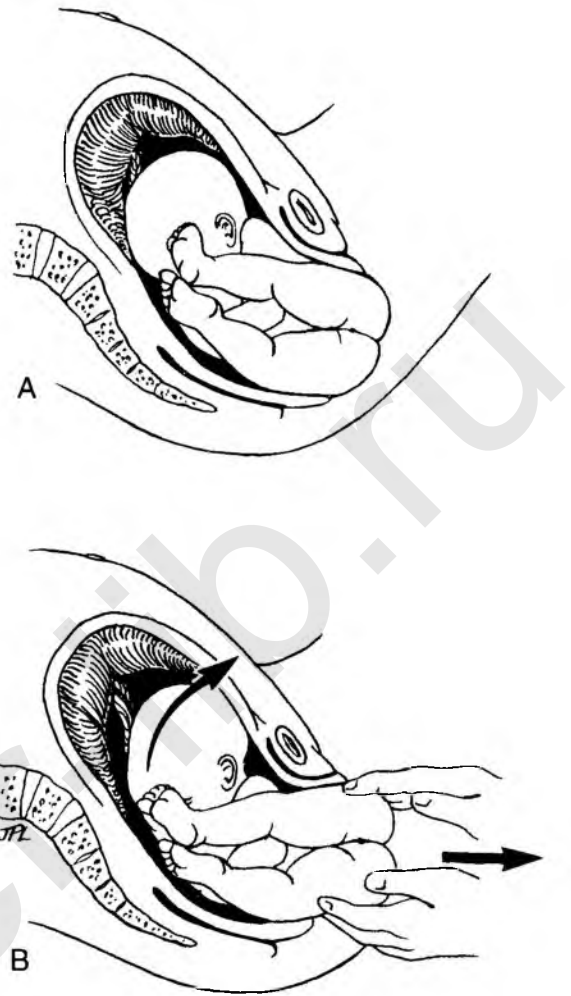


FIGURE 12.4.  
*A, Maintaining flexion is crucial to achieving safety in breech delivery. No interference with the birth is indicated until the umbilicus delivers. B, Early traction simply risks unnecessary cranial deflection, as indicated by arrow. (See also Figure 12.7.)*

A common problem is *nuchal arms*. When the descent of the body stops before the delivery of the shoulder girdle, nuchal arms are always suspect. Nuchal arms are especially a risk during breech extractions. In this setting the arms frequently pass upward as the fetal body rapidly descends due to tissue pressure from the birth canal. If one or both of the fetal arms has passed behind the head descent is blocked. Either traction from below or the use of fundal pressure only compounds the obstruction. The malpositioned arms must be located and drawn down across the chest and delivered before further descent of the fetal body is possible.

The manipulations to release the nuchal arms are usually easy when properly performed. These procedures are conducted only between contractions while the mother is specifically instructed not to push. In some cases it is best to push the fetal body slightly upward as the procedure is performed. The upward displacement helps to free the arms and provides working room for the surgeon. To release the obstruction, the surgeon first inserts two fingers across the fetal back toward the occiput, verifying the location of the malpositioned arm(s). Next, the fetal body is rotated 45 degrees or more until the scapula wings. At this point, the operator's fingers are advanced into the antecubital fossa and the extremity is then simply swept downward and across the fetal chest. The infant is then rotated in the opposite direction and a similar maneuver frees the other arm, permitting its extraction. Care must be taken during the extraction of the nuchal arms as it is easy to pull downward and deflex the fetal head. After the arms have been freed, the surgeon should recheck for cranial deflection. Loss of cranial flexion is a potentially serious complication as a deflexed head requires extra force for extraction. If cranial deflection is diagnosed, the fetal body is pushed slightly upward while gentle suprapubic pressure is applied. Uncommonly in this process of extraction of the nuchal arms, relaxation of the uterus is required. For acute tocolysis, the author's approach is to administer 150  $\mu\text{g}$  to 350  $\mu\text{g}$  IV of nitroglycerin, in bolus doses as needed, to a total of 500  $\mu\text{g}$ .

### Delivering the Aftercoming Head

The principles for the various delivery techniques for an aftercoming head are the same regardless of whether the delivery is a cesarean or vaginal: maintenance of cranial flexion and the avoidance of excessive force. As in the cephalic-presenting fetus, the loss of flexion presents the larger diameter of the fetal head (occipitofrontal for suboccipitobregmatic) to the pelvis, making delivery more difficult and thus more hazardous (Figure 12.7). By placing one hand on the mother's abdomen over the fetal head, an assistant helps maintain flexion of the aftercoming head (*Naujok's maneuver*) as it descends during the delivery (Figure 12.5). This simple maneuver should be routinely performed.

The *Mauriceau-Smellie-Viet (MSV) maneuver* (Figure 12.6) is useful for assisting delivery when the

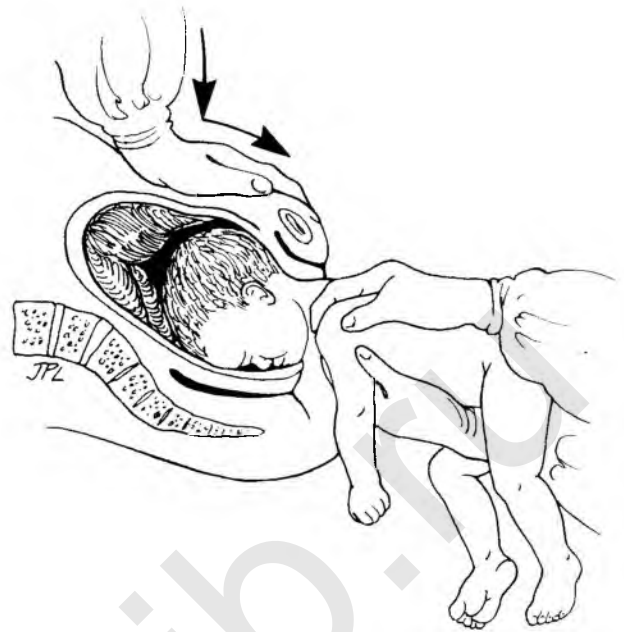


FIGURE 12.5.  
*Naujok's maneuver.*

head of the infant is in the occiput anterior position, with the fetal back toward the pubic symphysis. In the MSV maneuver, the body of the infant is supported on the operator's arm. The surgeon's middle finger is inserted into the baby's mouth while the index and ring fingers are applied to the face to maintain flexion. The head is then delivered by downward traction until the back of the neck extends under the pubic arch. A modification of the MSV maneuver is the *Wigand-Martin-Winkle maneuver*. When the aftercoming head is higher than the operator had anticipated, the first tractive efforts made to accomplish delivery must complete flexion and internal rotation. Suprapubic pressure is therefore applied with one hand, while the other keeps the fetal head flexed and helps to guide it out the birth canal [33].

The aftercoming head can be successfully delivered by the judicious use of suprapubic pressure alone (*Kristellar maneuver*). In fact, most clinicians combine the MSV maneuver within suprapubic pressure. Most often, the suprapubic pressure is provided by an assistant surgeon under the immediate direction of the senior obstetrician conducting the delivery.

Rarely, the fetus must be delivered with the abdomen anterior and the occiput posterior. The *Prague maneuver* is the technique used for



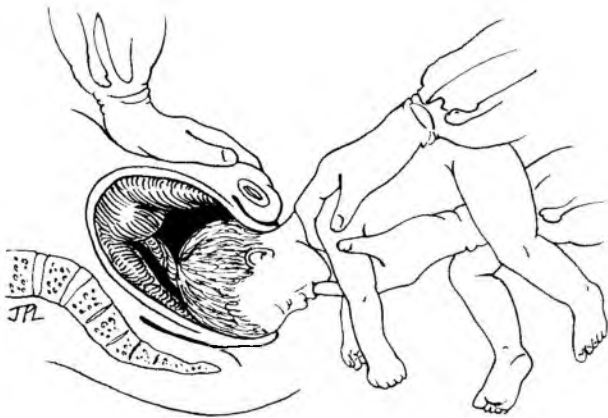


FIGURE 12.6.  
*Mauriceau-Smellie-Viet/Wigand-Martin-Winkle maneuver. See text for details.*

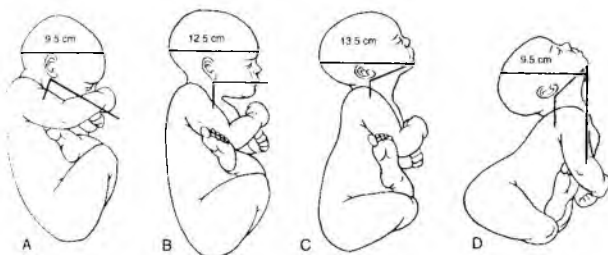


FIGURE 12.7.  
*Importance of cranial flexion is emphasized by noting the increased diameters presented to the birth canal with progressive deflection. A, flexed head; B, military position; C and D, progressive deflection.*

delivery in this situation (Figure 12.8). The infant is laid with his/her back on the operator's arm [34]. The index and middle fingers of the hand hook around each side of the fetal neck from behind. The infant is delivered in a large arc while flexion is maintained. If the occiput is directed posteriorly and the head is deflexed, delivery can be achieved as follows: the baby's back is positioned on the attendant's forearm, with the attendant's other hand grasping the legs above the ankles. This results in the occiput passing forward over the sacral concavity. The fetal larynx serves as a fulcrum in this type of maneuver.

### Breech Extraction

Extraction of the breech fetus can be total or partial. In *total breech extraction*, the entire body of the fetus is extracted by the obstetrician prior to engagement of the presenting part. This technique



FIGURE 12.8.  
*The Prague maneuver for delivery of the aftercoming head. See text for details.*

is still employed in the delivery of a second twin. In the past, breech extraction was infrequently used to deliver a singleton breech fetus when fetal distress or another important complications such as a cord prolapse developed prior to full engagement but with full cervical dilation. Such procedures require considerable skill and should never be attempted by an inexperienced surgeon without immediate expert instruction. Uterine relaxation facilitates any extraction procedure and is recommended. A breech extraction should be a smooth and slow operation, with an assistant providing continuous fundal pressure to retain cranial flexion. Judiciously applied force exerted from behind the head, *vis a tergo*, tends to flex both the fetal head and the arms, reducing risk.

In total breech extraction, the fetal body is entirely within the uterus prior to effecting delivery. When the legs do not present as in a frank breech presentation, the operator must first flex the fetal legs at the hip by digital pressure in the popliteal fossa to bring down one and, preferably, both legs, thus converting the position to a footling breech (*Pinard maneuver*) [39].

A *partial breech extraction* refers to the extraction of the fetal body after spontaneous delivery of

fetal parts but prior to engagement of the head. For example, during the vaginal delivery of a preterm breech, the parturient can spontaneously deliver the fetal legs through the cervix prior to becoming fully dilated and prior to engagement. By extracting the remainder of the fetal body, the obstetrician hopes to avoid entrapment of the fetal neck by the cervix. This differs from an assisted breech delivery. In the latter procedure, the obstetrician performs maneuvers to achieve delivery *after* the spontaneous expulsion of the fetal body to the umbilicus, as a result of maternal expulsive efforts that occur after engagement.

To deliver the child in a total breech extraction, the surgeon grasps both feet and applies traction while gentle force is applied to the uterine fundus by an assistant to maintain cranial flexion. With downward pressure, the fetal body is drawn into the pelvis until the sacrum has delivered. The surgeon's hands are then applied over the sacrum, thumbs together, encircling the fetal thighs. The fetal body is next extracted, rotating the trunk to release each arm in sequence. Nuchal arms are anticipated and managed as previously described. If the fetal body begins to enter the pelvis as a sacrum posterior, the legs are simply crossed. With subsequent downward traction the fetal body is progressively rotated until the spine is anterior. Normally, this is an easily performed and atraumatic maneuver, but the rotation should neither be sudden nor forced.

### FORCEPS IN BREECH DELIVERY

In 1924, Edmond Piper of Philadelphia introduced a special forceps design (*Piper forceps*) for delivery of the aftercoming head in breech delivery [35,36]. The principal advantage of the Piper instrument was the long shanks, permitting a more appropriate pelvic curve. With this modification the handles are lower than the blades when the forceps is properly applied. Although these forceps are longer than average, they are easy to apply from below to the aftercoming head. Traction and elevating the handles delivers the head in a wide arc (Figure 12.9). Before an application is attempted, the aftercoming head must have descended to fill the pelvis and must be in the direct occiput anterior position. A pelvic application of the blades is then made. Delivery of the fetus is accomplished using the forceps to elevate the aftercoming head about a point formed by

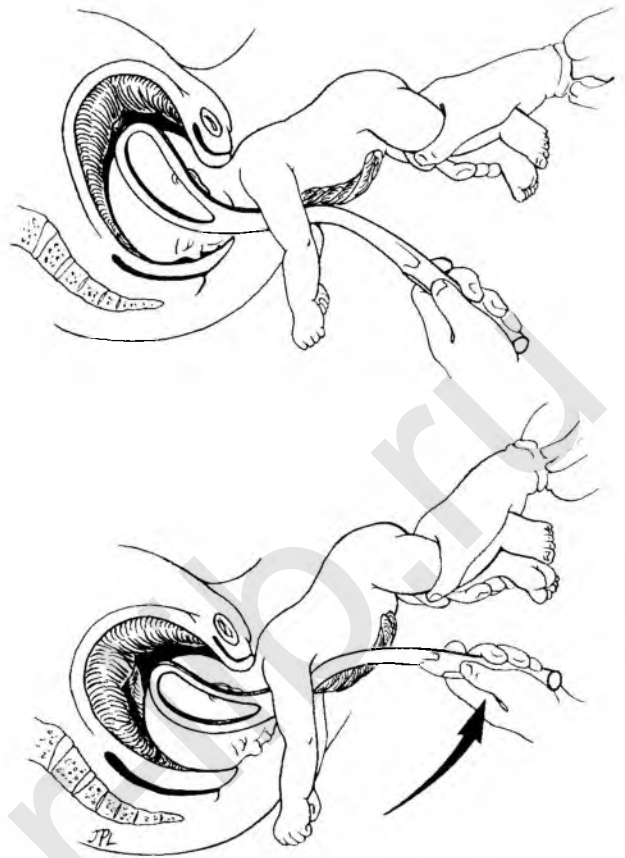


FIGURE 12.9.  
*Piper forceps for the delivery of the aftercoming head.*

the posterior aspect of the fetal neck where it meets the symphysis pubis. Very little traction is necessary to effect delivery with Piper forceps; they allow for delivery primarily by acting as a first-class lever while maintaining cranial flexion. In a similar fashion either the *Simpson* and *Kielland* forceps can also be used to assist delivery of the aftercoming head [36]. (See Chapter 17, Instrumental Delivery.)

### **Spontaneous Breech Delivery**

In spontaneous breech delivery, the patient delivers the aftercoming head entirely by her own expulsive efforts. This has theoretic advantages over the extraction procedures. By means of uterine contraction and accompanying maternal bearing down (Valsalva) efforts, the fetal head retains its flexed position relative to the neck. With spontaneous breech delivery, however, it is possible that the aftercoming head might deliver precipitously, which in theory could subject the fetus to an increased risk of

intracranial hemorrhage [33]. In the analysis of vaginal breech deliveries involving both multiparas and nulliparas, the lower 5-minute Apgar scores seen with multiparous patients has been ascribed to these presumed rapid fluctuations in intracranial pressure [21]. Because of this, close monitoring of the delivery process is recommended with special care in the coaching of the bearing-down efforts. Note that the provision of a regional anesthetic such as an epidural or a saddle block is strongly recommended to assist in the control of the delivery during this final expulsion phase.

### Cesarean Delivery

During a cesarean for a breech-presenting fetus, an extraction is commonly required. Even during an abdominal delivery, this is an inherently more dangerous procedure than partial breech extraction because of the increased risk of extending the fetal head [37–38]. An adequate incision of both the uterus and the abdominal wall are required to minimize the risk of fetal trauma. Inadequate incisions can lead to traumatic injury to the extremities or the cervical spinal cord as the surgeon struggles to free an entrapped infant. Thus, when the abdominal incision is performed, the surgeon should err on the side of a generous wound to insure adequate space for the delivery. This same consideration holds for the uterine incision. In all cases where a breech extraction might be required, the anesthesiologist should be requested to prepare a parenteral tocolytic, should uterine relaxation become necessary. In situations of a transverse lie when an extraction is highly likely, and in most instances of surgery for breech presentations, we routinely administer 150–350 µg of nitroglycerine intravenously just as the vesicouterine fold is incised. This usually results in adequate uterine relaxation at the time that the extraction actually occurs. There are minimal maternal risks to nitroglycerine since the drug has a very short half-life and the commonly observed maternal hypotension is transient [40] (see Chapter 9, Obstetric Anesthesia).

After the uterus is opened and the fetal hips are in view, they are drawn upward into the wound and gentle force is exerted at the fundus to deliver the fetal legs and body. Partial breech extraction can then be performed to complete the delivery. In this process, the aftercoming head is delivered primarily by force exerted at the fundus. As noted previ-

ously, regardless of the type of breech encountered, any extraction must be performed *slowly* to prevent deflection of either the head or arms. When the surgeon moves deliberately with the extraction, in conjunction with continuous pressure on the fundus, the risk of cranial or extremity extension is minimized.

The same general maneuvers used in assisted vaginal breech delivery are employed to complete the cesarean birth. Extraction of the hips through the incision is largely accomplished by force provided by the surgeon's hand directed downward at the uterine fundus, assisted by pressure exerted by hooking the fingers at the thigh. As the buttocks appear, the surgeon grasps the fetal pelvis, fingers along the sacrum, and applies downward traction to complete the delivery. As the child delivers, the surgical assistant maintains pressure on the fundus, following the fetal head downward until its eventual extraction.

If nuchal arms are encountered, rotation of the trunk and shoulders is required and implemented in the same fashion as during a vaginal delivery. The fetal body is simply rotated in one direction until the scapula wings. The arm is then flexed on the chest and swept free. Rotation in the other direction with a similar set of maneuvers frees the second arm. Upward displacement of the body can ease this maneuver. Correction of nuchal arms is, however, normally an easy procedure. Force that displaces the head laterally should be avoided during these manipulations, because this could predispose to brachial plexus injury. Next the aftercoming head is brought into the operative field by pressure directly on the fundus. Delivery of the head follows with a MSV maneuver to maintain cranial flexion. Paralleling a vaginal delivery, pressure by an assistant on the fundus or lower uterine segment will assist the cranial delivery. Because Müllerian anomalies are more common with malpresenting fetuses, the uterine cavity should be explored after delivery. A septate uterus, as well as leiomyomas and other minor uterine anomalies, can be found associated with breech-presenting fetuses [1,9].

### Internal Podalic Version

*Internal podalic version* is the conversion of a cephalic presentation to a breech presentation. This procedure is occasionally indicated with the delivery of a second twin (twin B). When ultrasonic scan or

pelvic examination after delivery of the first twin reveals twin B to be in a longitudinal lie and cephalic presentation but not engaged, and clinical features of the case require an expedited delivery, an IPV can be performed to extract twin B as a breech. This same approach can also be applied when twin B is compound in presentation, with the fetal arm/hand or umbilical cord prolapsed in front of the fetal head.

Adequate anesthesia and uterine relaxation are required prior to attempting the extraction. To perform the extraction, the surgeon places one hand on the uterine fundus to support the uterus. The other hand is passed through the cervix into the uterine cavity. The fetal head is gently but firmly displaced upward. A Pinard maneuver can be necessary first to secure the feet. With the fetal feet firmly grasped, they are pulled slowly downward toward the pelvic inlet. As the downward traction on the feet is performed, the operator's external hand gently pushes the fetal head upward towards the uterine fundus. The fetal body is next rotated as is necessary to guide the fetal spine anteriorly. When the fetal legs reach the introitus, a total breech extraction follows. In this type of extraction, nuchal arms should be anticipated and are managed in the usual manner. Occasionally but infrequently, once the fetus is correctly positioned, the parturient may be able to perform a Valsalva maneuver, leading to an assisted breech delivery [41]. The use of real-time ultrasound scan to locate fetal parts and the help of an assistant surgeon facilitates this sometimes complicated procedure. *It is to be emphasized that these procedures are not for the neophyte surgeon and should never be attempted without uterine relaxation and immediate expert assistance.*

### External Cephalic Version

ECV is the technique employed to convert a breech to a cephalic presentation. Although not strictly a procedure for breech delivery (i.e., it is a procedure used to *avoid* breech delivery), the technical aspects are pertinent to the overall approach to the problem of breech presentation.

ECV is usually performed before the onset of labor. Even without an ECV, up to 20% of breech presentations diagnosed as late as 37 to 39 weeks of gestation spontaneously convert from breech to vertex prior to labor [29]. Thus, the recognition of breech presentation near term but prior to labor can

allow for this approach to the overall management of breech presentation. Routine administration of a tocolytic agent such as terbutaline (0.250 mg SC) 15 to 20 minutes before the attempt eases the procedure and can increase the likelihood of success [42].

During an ECV procedure, the administration of either anesthesia or potent analgesia should be avoided. Pain or major discomfort during the procedure is a potential safeguard in limiting the force employed [43,44]. ECV should also be performed in reasonable proximity to the labor and delivery suite, in case of an emergency that requires expedited delivery.

The procedure begins with an ultrasound scan to document fetal presentation. This step should not be omitted, because spontaneous conversion of a breech to a cephalic presentation is not uncommon. Under real-time scanning the fetal position, cranial orientation, amniotic fluid volume and placental location should be noted. Fetal testing with a non-stress test (NST) is also performed. If the fetal testing is not reassuring or the ultrasound examination detects abnormalities, a version should not be attempted and additional evaluations are necessary.

After the fetal evaluations are complete and an informed consent has been obtained, the operator places one hand over the breech for elevation and the other over the head, and rotates the infant through the shortest arc from breech to cephalic (Figure 12.10). Rotation is accomplished slowly, avoiding undue force that might compromise the cord or placental circulation. Fetal-maternal hemorrhage is a risk with ECV, as is the rare occurrence of a fetal cervical spine injury. The risks for either are most likely related to the force with which the ECV is attempted. Avoiding analgesia and excessive force, and remaining attentive to the mother's discomforts minimizes risk.

This procedure has a success rate of about 60% at 37 to 39 weeks of gestation. Comparison of the success of ECV for breech and transverse presentations demonstrates a significant decrease in success with transverse presentations. This decrease has been ascribed to the greater incidence of unfavorable placental locations. Even with a successful ECV, the infants converted to cephalic presentations have higher rate of cesarean delivery than predicted [45]. The higher incidence of eventual cesareans relates to dysfunctional labors and, presumably, subtle forms of relative disproportion responsible for the initial malpresentation.

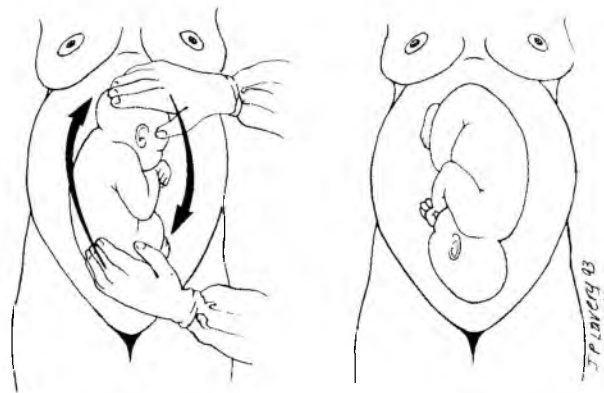


FIGURE 12.10.  
External cephalic version. A "head-over-heels" version is depicted. See text for details.

### Selective Trial of Labor

Selective labor trials in women with known breech presentations is another approach to delivery. Although a cesarean is performed for most women with a breech fetus, there are advocates for a TOL on a highly selective and individual basis [21–28]. If an attempt at version is either contraindicated or simply refused by the patient, then a next step to consider is a TOL. For example, a TOL might be reasonable choice for a multiparous woman first diagnosed as breech in early labor and at or about term with a frank or complete breech presentation.

A protocol for the management of term breech presentation is described in Figure 12.11. With the onset of labor, patient choice, type of breech, pelvimetry and estimated fetal weight become important management issues (Figures 12.14, 12.15). The protocol for intrapartum management has been studied prospectively [22,28,46,47]. Such a selective plan for TOL addresses the risks of cord prolapse, fetal distress in labor, and prolapse of the fetus through an incompletely dilated cervix. Management includes the use of continuous electronic fetal monitoring, frequent clinical evaluation, and the availability of emergency cesarean delivery, if required.

The decision process begins with a discussion with the woman about the fetal and maternal risks and benefits of a vaginal delivery versus an elective cesarean. One school of thought is that the risk to the breech fetus of a vaginal delivery is acceptable in selected circumstances with strict adherence to protocol. It has been shown that immediate neonatal outcome is similar when comparing

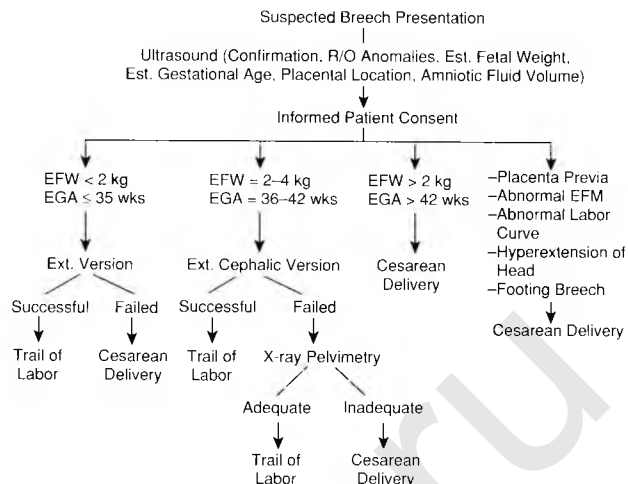


FIGURE 12.11.  
Flow chart for general management of breech presentation. See also Figure 12.15.

groups undergoing either carefully monitored labor and assisted breech delivery, or a routine cesarean [1,22,28,46,47]. Maternal morbidity increases with abdominal delivery, although the magnitude of this effect is usually minimal unless emergency procedures are performed in labor [18,19,22,28]. The risk of fetal injury or demise is not entirely eliminated by any one strategy, and there is a small risk of fetal/neonatal death associated with breech labor and vaginal delivery even with strict adherence to TOL protocols [22]. Therefore, patients must be counseled carefully and the management strategy chosen carefully.

Any woman laboring with a breech presentation should be prepared for abdominal delivery. Both anesthesia and nursing must be informed, and appropriate surgical assistants need to be identified. Regional anesthesia is best as, if required, a cesarean can be performed during labor on an expedited but nonemergent basis under epidural anesthesia [48]. Prior to a decision about mode of delivery, bedside ultrasound scan is performed to exclude obvious anomalies, confirm the gestational age, estimate fetal size, and note the degree of cranial deflection or hyperextension. Exclusion of a borderline pelvis is also part of the evaluation and is discussed later.

### Computed Tomography Pelvimetry and Ultrasonography in a Selective Trial of Labor

Radiographic evaluation has been used in the past to exclude a borderline pelvis for both cephalic and

breech fetuses. A combination of ultrasound scan and CT studies to devise a fetopelvic index has been advocated to assess risk of injury from shoulder dystocia in cephalic deliveries. This approach also could help to avoid trauma during breech delivery. Based on earlier studies, the consensus is that women with a borderline pelvis should be excluded from a breech TOL [6,22,28].

Todd and Steer promoted the advantage of radiographic evaluation of the maternal pelvis in selective delivery protocols [6]. These authors reported on the delivery of over 1,000 term breech-presenting fetuses during the 1950s and 1960s. They determined that the immediate neonatal outcome of vaginal breech delivery was associated with the pelvic diameters as determined by radiographic (x-ray) pelvimetry. With an inlet of the pelvis that measured 11 cm or greater in the anteroposterior diameter, and 12 cm or greater in the transverse diameters, the majority (85%) of infants delivered vaginally with acceptable perinatal mortality for that era. When either of these critical measurements was not achieved, the majority of infants ultimately required cesarean delivery (60%), and the perinatal mortality rate among those infants who delivered vaginally was determined to be 12 times greater than in the group with adequate measurements. The additional requirement of a 10-cm or greater diameter at the midpelvis followed, as did the use of CT studies for improved pelvimetry measurement (Figures 12.12 and 12.13, Table 12.3) [24–28,46,47,49,50].

A CT study not only evaluates pelvic anatomy but also permits reliable evaluation of the relationship between the head and the cervical spine vis-à-vis hyperextension of the fetal head. Hyperextension complicates about 5% of breech presentations at term [51]. To judge the degree of flexion or extension of the fetal head with respect to the cervical spine, the anterior angle between the mandible and the cervical spine is estimated. Hyperextension is diagnosed when this angle exceeds 90 degrees (Figures 12.7 and 12.13). In experienced hands, bedside real-time ultrasound scanning replaces radiographic studies for the evaluation of cranial hyperextension. Hyperextension must always be excluded regardless of the mode of delivery, because cranial deflection is strongly correlated with spinal cord injury from birth trauma [51]. The fetus with hyperextension is problematic to deliver. Even at a cesarean infants with this presentation require careful extraction to avoid injury. The potential causes for hyperexten-

sion include, among others, multiple loops of nuchal cord, fetal neck masses, torticollis, and fetal neurologic abnormalities. In many cases, however, the problem is idiopathic.

## **MANAGEMENT OF LABOR AND DELIVERY OF THE BREECH FETUS**

### **Management by Trial of Labor**

If ECV is unsuccessful or unacceptable to the patient, and the decision is made to conduct a labor trial, the woman should be instructed to present herself for evaluation at the earliest suggestion of labor or at the time of rupture of membranes. A multipara with a frank or complete breech presentation at term is a potential candidate for a TOL. With the exclusion of a borderline pelvis, the estimation of a fetus of average size, and an exclusion of cranial hyperextension, a TOL can be undertaken following an informed consent. These trial criteria are quite strict and when CT pelvimetry is used to evaluate women for a TOL, approximately 50% are excluded because of inadequate measurements of the bony pelvis. These women and those with unacceptable ultrasound examinations are then delivered by a cesarean [22,28].

In the remaining group of women, the evaluation of fetal status and progress of labor is managed in the same manner as with a fetus in a cephalic presentation [22,28,52–55]. In breech presentation, the EFM strips are interpreted in the same fashion as tracings from cephalic-presenting fetuses. Presumed fetal jeopardy/fetal distress during a breech trial is evaluated and managed in the usual manner. Lateral positioning, supplemental oxygen, increase in intravenous fluids, and administration of tocolytics all provide for in-utero resuscitation, as required.

The risk that the breech fetus might become acidotic during labor and delivery is marginally greater than for its cephalic counterpart [48,53]. This acidosis is usually respiratory and transient. As with a cephalic presentation, a suspicion of metabolic acidosis remote from the expected time of delivery generally results in an expedited delivery by cesarean.

The use of oxytocin was quite limited even in the era when breech labor trial were common. The rates of cervical dilation and descent of the presenting part in nulliparous women with a breech presentation are comparable to those observed with cephalic

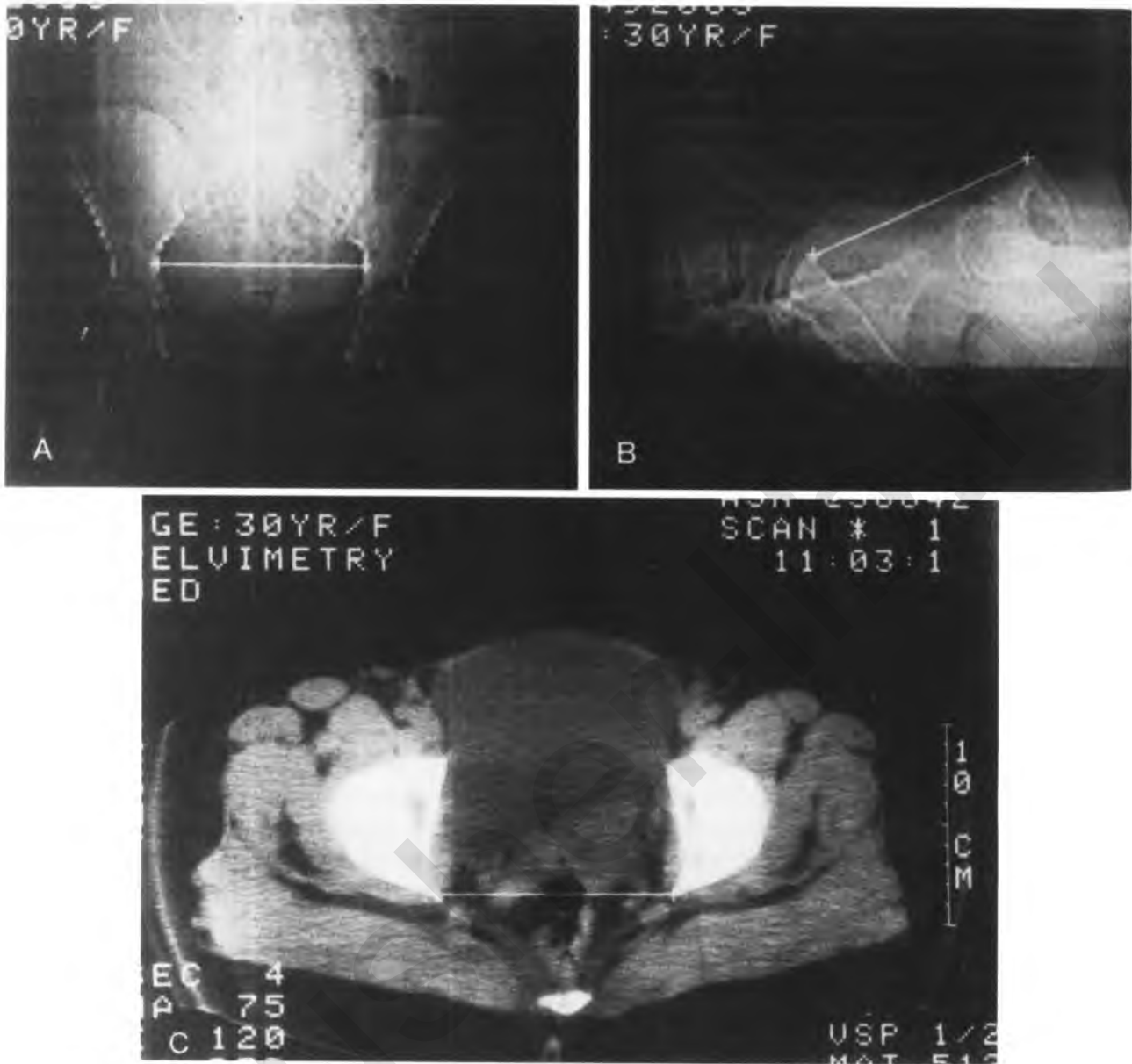


FIGURE 12.12.

*CT evaluation of the maternal bony pelvis. A, The widest transverse distance at the pelvic inlet. B, The anteroposterior distance at the pelvic inlet. C, The interspinous distance, measured at the midpelvis.*

presentations [56]. Among multiparous women, the maximal slopes of both dilation and descent have been reported as uniformly greater for breech labor than for cephalic labor. Breech fetuses with arrest of dilation or descent should be delivered by a cesarean.

In current practice, women with breech fetuses occasionally present to the labor and delivery suite with delivery imminent, and they frequently have

had no or little prenatal care. The decision about how best to proceed might not be easy. There is often little time for reflection. If the presenting part is truly crowning, a cesarean is often impossible and a vaginal delivery should be performed. It is better to conduct a well-controlled vaginal delivery than a poorly performed cesarean with improper technique or inadequate anesthesia. If circumstances are not so pressing, a tocolytic is administered and the

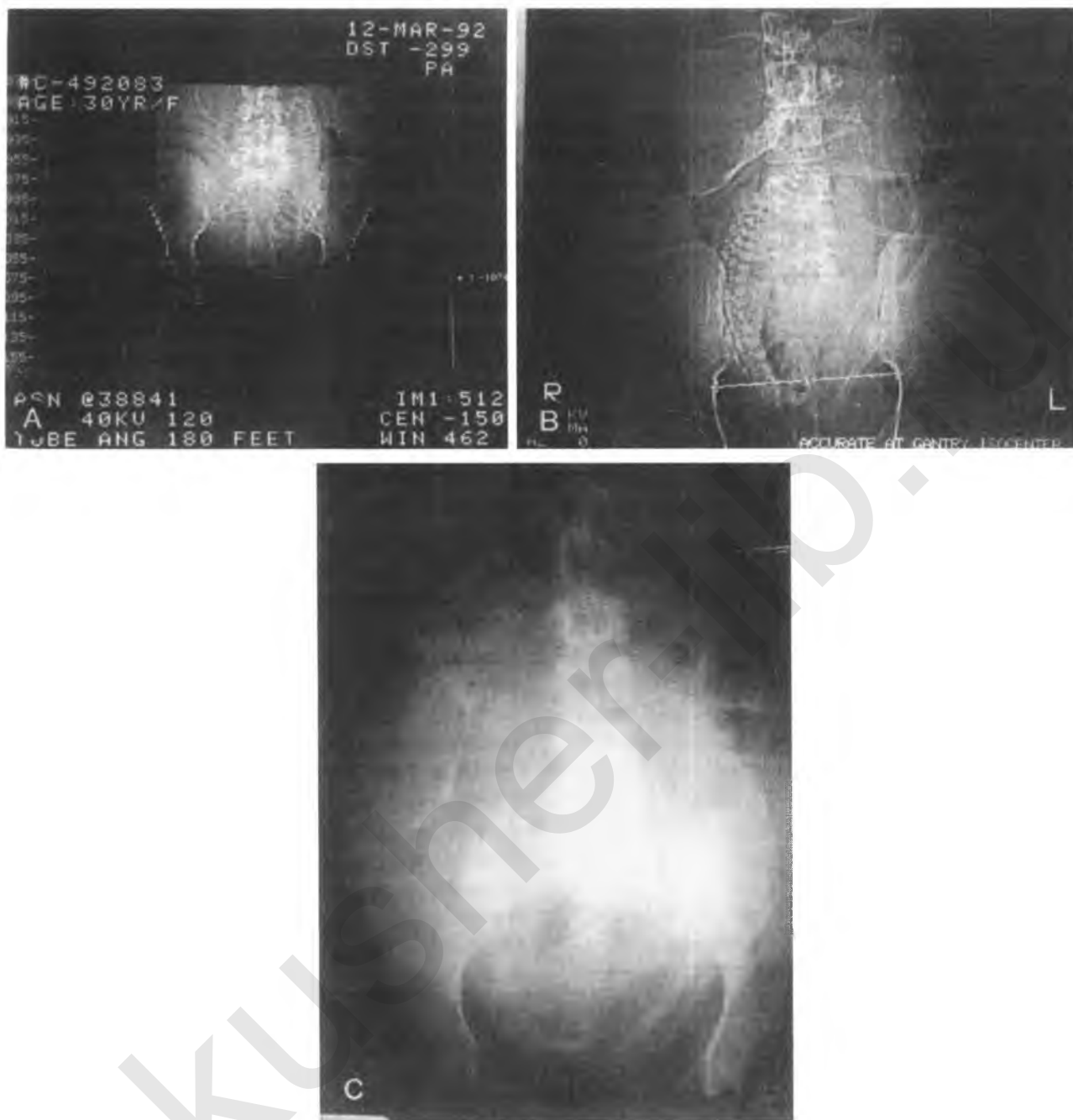


FIGURE 12.13.

*CT evaluation of the aftercoming head in breech presentation. A, The normal relationship between the head and neck is one of flexion. B, The head of this infant is hyperextended with reference to the cervical spine. C, In this fetus, the head is extremely hyperextended; this was classically referred to as a "star-gazing" fetus.*

woman is transported to the operating suite. If no studies of fetal size, cranial flexion, or the maternal pelvis have been performed, a cesarean is best, independent of the patient's parity, unless one is faced with the unusual case in which medical reasons

preclude the safe administration of an anesthetic agent.

When vaginal delivery is imminent, the bladder is catheterized. A generous episiotomy is performed as the buttocks crown. The membranes should be



**TABLE 12.3 Pelvimetry Criteria for a Trial of Labor for the Term Breech Fetus**

Dimension	Minimum Measurement (cm)
Pelvic inlet	
Anteroposterior	11
Transverse	12
Midpelvis	
Interspinous	10

From Gimovsky ML, Wallace RL, Schifrin BS, Paul RH: Randomized management of the nonfrank breech presentation at term: a preliminary report. *Am J Obstet Gynecol* 1983 May 1;146(1):34-40; with permission. See also Figures 12.12 and 12.13.

left intact as long as possible. The infant should be allowed to deliver spontaneously and to progress as far as possible. As noted, most infants deliver to the umbilicus with minimal assistance. Then, if the membranes are intact, they are then ruptured and a loop of umbilical cord is freed and pulled gently down. The Mauriceau-Smellie-Viet or Wigand-Martin-Winkel maneuvers are then used to complete delivery. Piper forceps (or alternatively, Simpson or Keilland forceps) can be used for delivering the aftercoming head at the clinician's discretion [57].

In the case of the vaginal delivery of a very-low-birthweight breech fetus (<750 g, <26 weeks), the membranes should be left intact, if possible, an epidural anesthetic provided, and fundal pressure judiciously used to affect delivery *en caul*. Hydrostatic force as distributed by intact membranes facilitates the delivery and reduces the risk of fetal entrapment and resultant injury or death.

Once the umbilical cord appears, clinicians often erroneously believe that there is only a short interval before severe neurologic injury or fetal demise occurs. In fact, when the interval between the appearance of the umbilicus and completion of delivery is 6 minutes or less, the 5-minute Apgar score usually indicates the need for only minimal support of the newborn [1,4,22,28]. In this setting, undue haste by the obstetrician could result in more traumatic injury than the putative benefits of a rushed delivery. In the past it was supposed that the cord was totally occluded during a delivery. Although this is the worst possible situation,

its occurrence is a distinct rarity. The median time for completing delivery after the cord appears is about 60 seconds in a vaginal breech delivery [27]. Although this might seem an eternity in the delivery suite, it is vital that the obstetrician use deliberate speed in effecting delivery. As previously reviewed in detail, haste can result in nuchal arms or, more dangerously, inadvertent extension of the fetal head and extremities, unnecessarily increasing the risk of delivery trauma.

Overall, the differences in outcome between breech-presenting infants delivered by an elective cesarean or vaginally following the described protocol are minimal for infants who are of average size and at term. Average size is defined as infants between 2,500 g and 3,500 g at 36 to 40 weeks of gestation [4,22,28,53,55]. The fetal outcome is not fundamentally affected by the specific type of breech presentation or by maternal parity [22].

#### Management by Elective Cesarean Delivery

At present, most practitioners elect to deliver the breech fetus by a cesarean regardless of other considerations [14,16,19,20,59]. This decision is multifactorial and involves both medical and nonmedical issues, but the overwhelming reason for deciding to proceed with a cesarean is the attempt to avoid traumatic birth injury. The risk of asphyxia during both labor and delivery is also minimized. Both of these adverse events are rarely seen after a cesarean delivery. Nonmedical decision parameters include patient and clinician preference as well as concerns about liability [59,60].

Routine cesarean delivery in breech presentation is most beneficial for 1) patients delivering in facilities not equipped with full-time support services in anesthesia and pediatrics; 2) women delivering breech-presenting fetuses less than 36 to 37 weeks of gestation, or estimated fetal weights less than 2,000 g; 3) women delivering fetuses at 42 or more weeks' gestation or with estimated fetal weights greater than 4,000 g; 4) any breech-presenting fetus having hyperextension of the head; or 5) a patient who desires a cesarean.

#### Management by External Cephalic Version

ECV is most successful when the breech is not engaged and spontaneous uterine activity is

minimal [61,62]. Multiparous women with complete or footling breech presentation prior to engagement are good choices for an attempt at ECV. Sufficient amniotic fluid volume is necessary. The location of the placenta is important because an anterior location makes the procedure more difficult. Maternal obesity and parity, frank breech presentation, low station, and advanced cervical dilation are factors predictive of ECV failure. Fetuses that are at excessive risk, such as those with premature rupture of membranes, placenta previa, abruptio placentae, or who have abnormal fetal testing, should not undergo ECV.

The decision to attempt ECV can be made either antepartum or in early labor [61]. With the use of a tocolytic agent and appropriate surveillance methods, ECV at 37 to 39 weeks of gestation is safe and an effective means to resolve the problem of breech presentation [62]. Amnioinfusion as an adjunct to facilitate ECV has also been described, and ECV has been performed in some cases of preterm labor.

### Team Approach to Breech Delivery

In effecting breech delivery by vaginal delivery or by cesarean, experienced personnel are necessary for optimal outcome. Minimally, the obstetrician must have a gowned and gloved assistant present at a vaginal delivery or a cesarean.

A delivery room in which either type of delivery can be performed should be prepared in advance. Warm, wet towels and forceps should be available in addition to the regular delivery instruments. Equipment should include a fetal monitor and a real-time ultrasound scanner to permit continuous monitoring.

Both anesthesia and pediatric support are crucial to the successful outcome of breech delivery. Regional anesthesia has become the norm for both breech and cephalic deliveries. Once avoided for vaginal breech delivery, epidural anesthesia is now recognized as particularly helpful in special situations, such as with a premature breech or with a non-frank breech presentation at term [37]. On occasion, breech deliveries also benefit from the administration of uterine relaxants such as nitroglycerine when manipulations are necessary.

Because the first few minutes after breech delivery are invariably associated with some degree of neonatal depression, immediate pediatric support is

key to the good outcome for the breech infant [55]. This is evidenced by the high incidence of low 1-minute Apgar scores seen at term breech delivery, independent of delivery mode [17]. Five-minute Apgar scores demonstrate no significant differences between vaginally delivered breech infants and those delivered by cesarean when a selective protocol has been used for managing labor [1,5,13,55]. The rare occurrence of a seriously depressed infant mandates that a skilled pediatrician be present at any breech delivery, however. Umbilical cord gases frequently yield evidence of mild transient respiratory acidosis. This finding could result from transient, partial cord compression as the fetal body passes through the birth canal or be secondary to flow compromise from a nuchal cord, a feature that frequently accompanies breech presentation.

An experienced clinician should monitor all breech labors. Certain problems are more common with a breech presentation during labor, and expert assistance can be required because special manipulations might become necessary for an atraumatic delivery. As an example, prolapse of either the umbilical cord or the fetal body through a partially dilated cervix is a potential problem that requires immediate intervention. Use of regional anesthesia, avoiding early and inappropriate intervention, and not rupturing the membranes for as long as possible minimizes the risk of this complication.

### Breech Delivery in Everyday Practice

A major difficulty in managing breech delivery is obtaining sufficient experience, because the number of both planned and unanticipated breech deliveries has declined. Many obstetricians are concerned when faced with the imminent delivery of a breech fetus, especially in a facility that lacks the necessary equipment and personnel required to perform an emergency cesarean. It therefore behooves the clinician to learn about the manual maneuvers required to assist safely at breech vaginal delivery and to practice these same techniques at the time of cesarean delivery. Conducting partial or total breech extractions at cesarean delivery when there is an appropriate indication provides the clinician with an opportunity to safely enhance his/her skill and confidence with the techniques of breech delivery.

In a prospective series at Los Angeles County Hospital in the 1980s, 12% of patients with breech

presentation randomized to a cesarean delivered vaginally before the operation could be performed [22]. In current practice, a smaller percentage of advanced cases present in this manner. With the goal of providing the best possible care for the parturient and her breech fetus, several other issues have a significant impact on the choice of method of delivery including

- The local patterns of practice
- The need to obtain informed consent
- The desires and preferences of the patient
- The ability of the facility to support an emergency cesarean
- The availability of anesthesia and pediatric assistants.

## OUTCOMES

### **Term Breech Infant**

Infants delivered from breech presentation, regardless of management strategy, are at greater risk for adverse outcomes than are those with a cephalic presentation. Overall, there is a threefold increase in perinatal mortality with breech presentation [1]. Congenital anomalies, IUGR, prematurity, and birth-related traumatic injuries are contributing factors. The spectrum of possible fetal injuries, including cerebral palsy, direct birth injury, and neonatal depression, is addressed later.

Although the ability to recognize many congenital anomalies has increased dramatically, effective obstetric interventions that have a significant impact on outcomes are limited. Furthermore, accurate early recognition of severe growth disturbances (e.g., IUGR) and the ability to correct those problems prior to birth are still at best limited.

There is enormous pressure to make significant inroads in reducing adverse outcome caused by birth trauma from labor and delivery. This pressure is complemented by a desire to practice medicine consistent with current societal expectations [60].

The potential for traumatic injury during breech labor and delivery has long been appreciated. Possible problems include umbilical cord prolapse; brachial plexus injury; trauma to intraabdominal

organs; fracture of the clavicle, long bones, or skull; and various injuries to the spinal cord.

The fetal head and neck are at particular risk in breech labor and delivery, regardless of the chosen delivery mode. When hyperextension of the fetal head complicates delivery, intracranial hemorrhage from intracerebral bleeding, tentorial tears, subdural hematomas, and cervical cord trauma can result in permanent neurologic injury [64]. Fetal hypoxemia and ischemia can also result in severe neurologic impairment. Similar injuries can be caused by entrapment of the aftercoming head, whether at the pelvic inlet or by a partially dilated cervix. The possible relationship of breech presentation at term and cerebral palsy has also been cited as a reason for a routine cesarean for breech delivery. This conclusion might have arisen from the observed association between breech presentation at term and low Apgar scores [65]. In a more thorough analysis, however, differences between breech and cephalic infants in rates of cerebral palsy are best attributed to the higher incidence of severe growth abnormalities (IUGR) among breech fetuses [66,67].

Breech presentation can be a marker for another problem, such as IUGR, itself a risk factor for cerebral palsy. Breech delivery complicated by anoxia or birth trauma has the potential to result in cerebral palsy. Studies in which the mode of delivery was assessed as to the risk of development of cerebral palsy fail to demonstrate a decreased incidence of cerebral palsy following cesarean delivery, however [65,66,67].

Brachial plexus injuries, which can occur following vertex or breech vaginal deliveries, are a special concern. Geutjens and coworkers studied differences in the pattern of brachial plexus injury sustained at both breech and cephalic vaginal deliveries [68]. In breech deliveries, 81% of the brachial plexus injuries were avulsions at the upper roots, C5, C6, ± C7. Low-birthweight infants constituted 30% of the infants with brachial plexus injury, a threefold increase in risk compared with the general population. Brachial plexus injuries associated with cephalic presentations demonstrated post-ganglionic rupture of nerve roots C5, C6, ± C7 in 75% of infants. Shoulder function, as demonstrated by Mallet's score, indicates a much worse injury for breech infants. Additionally, 22% of the breech infants were initially diagnosed with bilateral

brachial plexus injuries. Similar findings about the patterns of brachial plexus injury were also described by Al-Qattan [69]. Both groups concluded that the more severe injuries sustained by breech infants are specific to the presentation and are less likely to resolve spontaneously than are similar injuries occurring at cephalic deliveries. The authors also concluded that brachial plexus injury sustained by a breech fetus requires surgical correction earlier and more frequently. Although these authors' studies refer specifically to vaginal deliveries, brachial plexus injuries are also possible during cesarean delivery.

The need for resuscitation at delivery, as assessed by Apgar score, is greater for breech than cephalic infants. Low 1-minute Apgar scores ( $\leq 3$ ) occur three to five times more frequently in breech as opposed to cephalic deliveries [4,55]. As noted previously, umbilical cord gases demonstrate a mild respiratory acidosis more frequently at vaginal breech delivery [52,53].

Bony injury and peripheral nerve damage are increased in vaginal breech delivery [5,9,55]. Not generally appreciated is the fact that these injuries also occur at cesareans, although at a lesser rate.

Intracranial hemorrhage, spinal cord trauma, bony fractures, and visceral injury are rare but potentially serious problems that can occur during delivery of the breech fetus [1,2,9,55]. Earlier prospective studies suggest that the use of a selective management protocol in conjunction with an experienced operator reduces the frequency of these injuries [22,28,55].

Although ECV is a routine procedure, related injuries can include vaginal bleeding, fetal-maternal bleeding, cesarean scar rupture, and umbilical cord prolapse [62,70–74]. An emergency cesarean in conjunction with FHR abnormalities to avoid fetal hypoxemia is also an uncommon ECV procedure-related risk.

The additional increase in cesareans for breech delivery (to 90% or more at present) has not significantly decreased perinatal losses. Indeed, the almost universal use of cesarean delivery has generated birth injuries attributable to the procedure. The breech fetus born at a cesarean can suffer the same range of injuries as one born at vaginal delivery, albeit at a lower rate. Iatrogenic fetal birth trauma at cesarean delivery has been estimated to be in excess of 1% [75–76]. Care with surgical and anesthesia

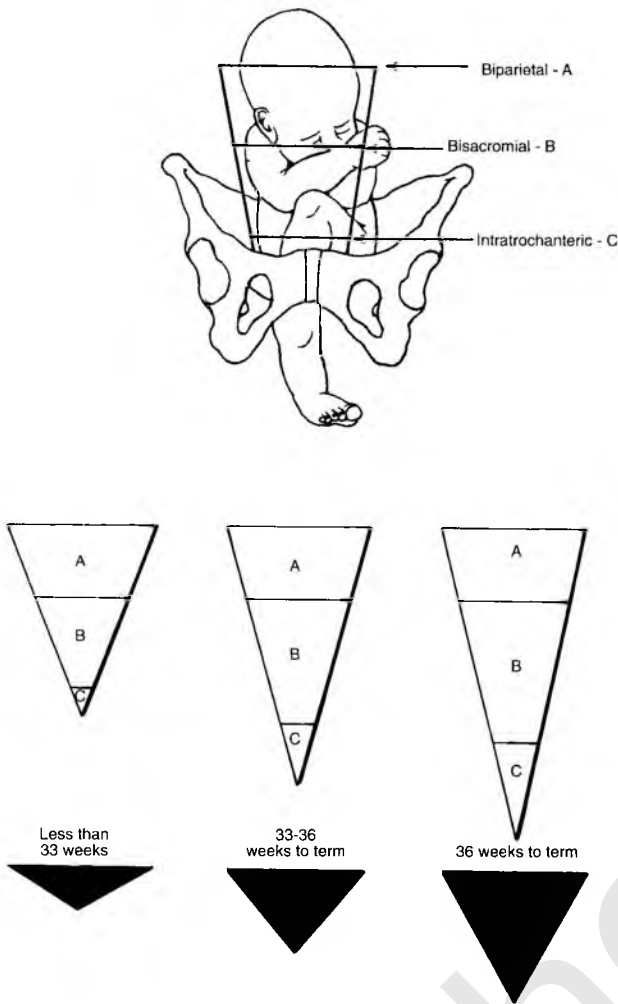
techniques and better understanding of the mechanism of breech delivery are important to minimize these types of injuries.

The Term Breech Trial, conducted with enormous effort by the researchers involved, reported on worldwide clinical practices in breech delivery [19,77,90,91]. Differing practices and attitudes by both clinicians and patients led these authors to the conclusion that the immediate neonatal outcome was better for the infants born by a planned cesarean than by planned vaginal delivery. In a follow-up report in 2004, however, the same group reported no significant differences in breech infants born by planned vaginal delivery or planned cesarean at age 2 years [77].

### Preterm Breech Infant

About 25% of all breech-presenting fetuses are premature at delivery. Most premature breech fetuses are delivered by a cesarean. The volume of amniotic fluid after spontaneous rupture of membranes and the relative size of the fetus are factors that predispose the premature breech fetus to an increased risk of traumatic delivery. The greater disparity between head and abdomen dimensions results in a significant disadvantage to the preterm fetus in negotiating the pelvis, making vaginal delivery inherently riskier than for the term fetus (Figure 12.14). The partially dilated cervix, which more often allows for delivery of the smaller fetal diameters of a low-birthweight fetus, increases the risk of entrapment of the fetal body or the aftercoming head. Other potential delivery risk factors seen with preterm breech fetuses include 1) increased incidence of non-frank presentations with attendant increased risk of umbilical cord and/or body prolapse [3,9–11]; 2) increased incidence of premature rupture of membranes, which compromises both umbilical cord and placental blood flow and results in less tolerance to the stress of labor [78–83]; and 3) the absence of an effective dilating wedge.

ECV has also been employed with the preterm breech fetus. Chervenak and coworkers reported their experience with ECV with tocolysis for low-birthweight breech infants in preterm twin gestations [63]. These investigators reported one neonatal death among the group of low-birthweight breech fetuses in a 1,000-g infant, suggesting a serious potential risk of ECV in this group.



**FIGURE 12.14.**  
*The shape of the fetus depends greatly on gestational age. The relationship of the three diameters that approximate shape (biparietal, bisacromial, and intratrochanteric) becomes more favorable as gestational age reaches term. This is commonly reflected in the ratio of the head and abdominal circumference as determined by ultrasound scanning reaching unity.*

Selected frank and complete breech-presenting fetuses between 32 and 35 weeks of gestational age still await study to determine the safety of ECV, however.

Breech-presenting infants weighing more than 700 g but less than 2,000 g (27 to 36 weeks) at birth are at great risk for significant injury at birth [10,11,13]. The incidence of low Apgar scores (less than 7 at 5 minutes), difficulty with the aftercoming head, peripheral nerve injury, and perinatal mortality (corrected) is greater when breech-presenting

infants weighing less than 1,500 g are delivered vaginally.

Among preterm breech-presenting infants followed for 1 to 5 years after birth, the incidence of developmental or neurologic abnormalities was tenfold greater in premature breech-presenting infants (less than 35 weeks) delivered vaginally as compared with those born by a cesarean. The incidence of intracranial bleeding was four times greater among vaginally delivered infants. One third of the breech infants in whom significant neurologic sequelae occurred were small for gestational age [80]. The frequency of neonatal death among vaginally delivered preterm breech-presenting infants is increased compared with those delivered by a cesarean, even with a mean gestational age of 34 weeks [78,79]. Death occurs secondary to asphyxia, respiratory insufficiency, or intracranial hemorrhage.

The very-low-birthweight fetus (less than 700 g, less than 27 weeks) must be given separate consideration. Among these very-low-birthweight infants, injuries sustained at delivery are similar regardless of mode of delivery; therefore, a cesarean delivery does not, on balance, improve survival for these groups [12,79]. In these extremely young and small fetuses, multiple factors relating to prematurity appear to be more significant in determining the neonatal outcome than the mode of delivery [3,10–12].

The many causes of compromised outcome among premature infants, vertex or breech, confuse the issue of the benefits that can be derived from a cesarean. The nonasphyxiated infant born with a minimum of birth trauma is the highest-quality survivor. A cesarean delivery performed when there has been little or no labor seems to result in the best outcome.

## CONCLUSION

Once a breech presentation has been diagnosed, the patient and her family can be counseled and instructed about the potential problems that might be encountered. Of course, this is most easily accomplished when the diagnosis is made prior to labor.

Most women understand that breech presentation involves risks to their unborn child, but they should also understand that the risks are not limited to the route of delivery. Pregnant women are

also influenced by the comments that are made by family, friends, their own on-line investigations, and the media. It is thus crucial that physicians continue to be well informed and available to answer questions. Physicians must also be prepared to explain to patients the nature of the risks and benefits that are associated with alternatives to a routine cesarean, including ECV and a selective TOL [88].

In an era in which decreasing family size and increasing expectations for a perfect baby following an atraumatic birth coexist, it must be decided how to integrate these expectations and their subsequent sequelae into clinical obstetric practice. This is another reason to encourage patients to participate actively in their understanding and choices about childbirth.

It is anticipated that the reliance on cesarean delivery for breech infants will continue to increase. With the rise in the number of repeat procedures, surgical complications will also rise. A more selective approach to the overall use of cesarean delivery could help to limit this increased risk.

Another consideration is the inadequate experience in breech management in residency education programs. The profession is currently well on the way to producing a generation of obstetricians trained to rely exclusively on cesareans for breech delivery. This inexperience with the management of breech vaginal delivery could compromise the advantages of cesarean delivery for the safe delivery of the breech fetus. Reports of birth injuries suffered at cesarean delivery of breech infants confirm this potential. The importance of learning all aspects of breech delivery technique therefore cannot be overstated. Clinical teaching of operative obstetrics using advanced simulators offers promise in this area [86,88].

Breech presentation during labor remains a high-risk situation and requires careful management to achieve the optimal outcome. It is an oversimplification to make the assumption that cesarean section is the complete and final answer [87–89].

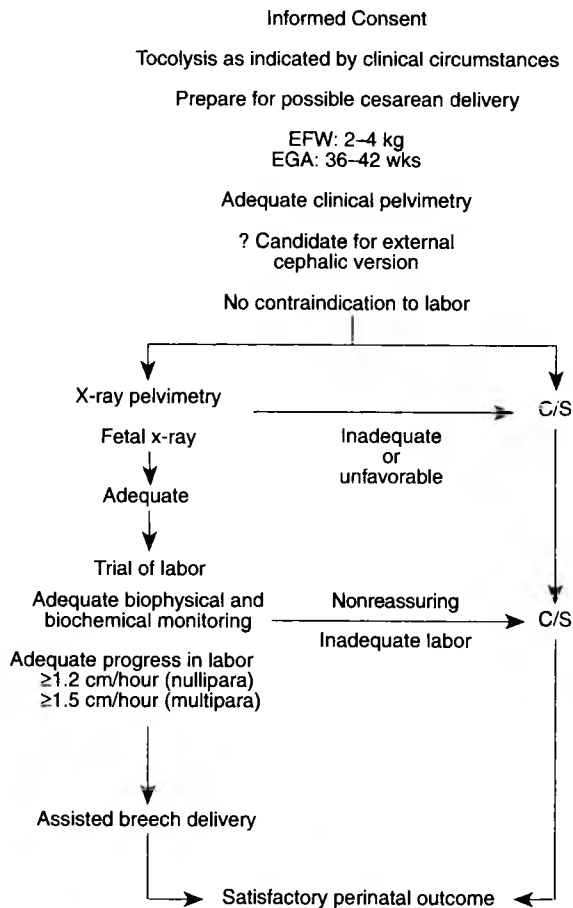
## SUMMARY

Management of breech presentation is complex. Although a role remains for selective breech vaginal trials, the number of such cases is now quite limited. It is important to recognize that when used properly,

cesarean delivery for breech-presenting infants has the potential to dramatically decrease the incidence of intrapartum fetal/neonatal injury and death. As we have seen, however, cesarean delivery does not solve all the problems inherent in breech presentation or ensure the delivery of a normal child. Further, the techniques required for vaginal breech delivery are in many ways the same as those necessary at a cesarean. Due to the problems inherent in clinical medicine and its unpredictability, physicians in training should develop the skills necessary to perform both version for breech presentation as well as both vaginal and cesarean breech delivery. Moreover, it is necessary to understand all the clinical options available, as well as the controversy in the management of the breech-presenting fetus, in order to permit selection of the safest and most optimal delivery route for each patient in accord with each woman's preferences.

## RECOMMENDATIONS FOR PRACTICE

- Breech-presenting infants in frank or complete presentation at 36 to 42 weeks are potential candidates for a TOL if strict criteria are satisfied (Figures 12.11 and 12.15). The double-footling breech, likely to be at greater risk for cord prolapse and prolapse of the fetal body through an incompletely dilated cervix, is the most difficult term breech-presenting fetus to manage effectively through labor and delivery and should be delivered by a cesarean. Breech presentations require evaluation by real-time ultrasound scan once the diagnosis is established, and if a vaginal trial is considered, radiographic evaluation of the maternal pelvis is necessary.
- Breech-presenting fetuses at a transitional gestational age, 32 to 35 weeks, in frank or complete presentation, are borderline by size and by shape (head-to-abdomen ratio) for a TOL. Except in highly selected cases, cesarean delivery is best for this group.
- Breech-presenting fetuses at 26 to 32 weeks of gestation are best managed by cesarean delivery.
- For breech-presenting fetuses when delivery is required prior to 26 weeks of gestational age, cesarean delivery is not routinely indicated.



**FIGURE 12.15.**  
*Overview and flow diagram of the management of breech presentation in early labor for estimated gestational ages 36 to 42 weeks. See text for details.*

Vaginal delivery, particularly when membranes are intact, remains a reasonable alternative for these very premature infants, under specific circumstances.

## REFERENCES

1. Kauppila O: The perinatal mortality in breech deliveries and observation on affecting factors. *Acta Obstet Gynecol Scand Suppl* 1975;44:13–9.
2. Hall JE, Kohl S: Breech presentation: A study of 1,456 cases. *Am J Obstet Gynecol* 1956 Nov;72(5):977–90.
3. Karp LE, Doney JR, McCarthy T, Meis PJ, Hall M: The premature breech: Trial of labor or cesarean section? *Obstet Gynecol* 1979 Jan;53(1):88–92.
4. Sibony O, Luton D, Oury JF, Blot P: Six hundred and ten breech versus 12,405 cephalic deliveries at term: Is there any difference in the neonatal outcome? *Eur J Obstet Gynecol Reprod Biol* 2003 Apr;107(2):140–4.
5. Gimovsky ML, Paul RH: Singleton breech presentation in labor: Experience in 1980. *Am J Obstet Gynecol* 1982 Aug; 143(7):733–9.
6. Todd WD, Steer CM: Term breech: Review of 1006 term breech deliveries. *Obstet Gynecol* 1963 Nov;22:583–95.
7. Braun F, Jones KL, Smith DW: Breech presentation as an indicator of fetal abnormality. *J Pediatr* 1975 Mar;86(3):419–21.
8. Axelrod FB, Leistner HL, Porges RF: Breech presentation among infants with familial dysautonomia. *J Pediatr* 1974 Jan;84(1):107–9.
9. Brenner WE, Bruce RD, Hendricks CH: The characteristics and perils of breech presentation. *Am J Obstet Gynecol* 1974 Mar;118(5):700–12.
10. Cruikshank DP, Pitkin RM: Delivery of the premature breech. *Obstet Gynecol* 1977 Sep;50(3):367–9.
11. Gimovsky ML, Petrie RH: The intrapartum and neonatal performance of the low-birth-weight vaginal breech delivery. *J Reprod Med* 1982 Aug; 27(8):451–4.
12. Green JE, McLean F, Smith LP, Usher R: Has an increased cesarean section rate for term breech delivery reduced the incidence of birth asphyxia, trauma, and death? *Am J Obstet Gynecol* 1982 Mar;142(6 Pt 1):643–8.
13. Gimovsky ML, Breech Delivery. in Queenan JT, Hobbins JC, Sprong CY: *Protocols for High-Risk Pregnancies*, 4th ed. Oxford UK: Blackwell Publishing, 2005; pp. 55–8.
14. Wright RC: Reduction of perinatal mortality and morbidity in breech delivery through routine use of cesarean section. *Obstet Gynecol* 1959;14:758–63.
15. Potter MG Jr., Heaton CE, Douglas GW: Intrinsic fetal risk in breech delivery. *Obstet Gynecol* 1960 Feb;15:158–62.
16. NIH Consensus Developmental Task Force statement on cesarean childbirth. *Am J Obstet Gynecol* 1981 Apr;139(8):902–9.
17. White PC, Cibils LA: Clinical significance of FHR patterns during labor. VIII. Breech presentations. *J Reprod Med* 1984 Jan;29(1):45–51.
18. Papiernik E: The role of emergency obstetric care in preventing maternal deaths: An historical perspective on European figures since 1751. *Int J Gynaecol Obstet* 1995;50(2):73–7.

19. Hannah ME, Hannah WJ, Hewson SA, Hodnett ED, Saigal S, Willan AR: Planned caesarean section versus planned vaginal birth for breech presentation at term: A randomised multicentre trial. Term Breech Trial Collaborative Group. *Lancet* 2000 Oct;356(9239):1375–83.
20. Committee on Obstetric Practice. American College of Obstetricians and Gynecologists Committee Opinion: number 265, Dec 2001: Mode of term singleton breech delivery. *Obstet Gynecol* 2001 Dec;98(6):1189–90.
21. Aburezq H, Chakrabarty KH, Zuker RM: Iatrogenic fetal injury. *Obstet Gynecol* 2005 Nov;106 (5 Pt 2):1172–4.
22. Gimovsky ML, Wallace RL, Schifrin BS, Paul RH: Randomized management of the nonfrank breech presentation at term: A preliminary report. *Am J Obstet Gynecol* 1983 May;146(1):34–40.
23. Daw E: Hyperextension of the head in breech presentation. *Am J Obstet Gynecol* 1974 Jun;119 (4):564–5.
24. Gimovsky ML, Petrie RH: Strategy for choosing the best delivery route for the breech baby. *Contemporary OB/GYN* 1983;21:210–5.
25. Gimovsky ML, Petrie RH, Todd WD: Neonatal performance of the selected term vaginal breech delivery. *Obstet Gynecol* 1980 Dec;56(6):687–91.
26. O'Leary J: Vaginal delivery of the term breech. *Obstet Gynecol* 1979 Mar;53(3):341–3.
27. Watson WJ, Benson WL: Vaginal delivery for the selected frank breech infant at term. A preliminary report. *Obstet Gynecol* 1984 Nov;64(5):638–40.
28. Collea JV, Chein C, Quilligan EJ: The randomized management of term frank breech presentation: A study of 208 cases. *Am J Obstet Gynecol* 1980 May;137(2):235–44.
29. Van Dorsten JP, Schifrin BS, Wallace RL: Randomized control trial of external cephalic version with tocolysis in late pregnancy. *Am J Obstet Gynecol* 1981 Oct;141(4):417–24.
30. Vasa R, Kim MR: Fracture of the femur at cesarean section: Case report and review of literature. *Am J Perinatol* 1990 Jan;7(1):46–8.
31. Dunn PM: Erich Bracht (1882–1969) of Berlin and his "breech" manoeuvre. *Arch Dis Child Fetal Neonatal Ed* 2003 Jan;88(1):F76–77.
32. Taylor ES, ed: *Beck's Obstetrical Practice*, 9th ed. Baltimore: Williams & Wilkins, 1972; p. 252.
33. Danforth DN, ed: *Obstetrics and Gynecology*, 3rd Ed. Philadelphia, Harper & Row, 1977.
34. Kiwisch FA: Zur lehré von der extraction des kindes auf den füßen [A lecture on fetal extraction by the feet]. *Beitr Geburtsk Wurzb* 1846; i:62–71.
35. Piper EB, Bachman C: The prevention of fetal injuries in breech delivery. *JAMA* 1929;92:217–21.
36. O'Grady JP: *Modern Instrumental Delivery*. Baltimore, MD: Williams & Wilkins, 1988.
37. Kerr M: Breech presentation. In: Myciscoyh P, Mori J, eds: *Munro Kerr's Operative Obstetrics*, 8th ed. Baltimore: Williams & Wilkins, 1971; pp. 139–87.
38. Douglas RG, Stomme WB, eds: *Operative Obstetrics*, 3rd ed. New York: Appleton-Century-Crofts, 1976; p. 600.
39. Pinard A: *Traité du palper abdominal au point de vue obstetrical et de la version par manoeuvres externes, précédé d'une préface de M. Pajot* [Treatise concerning abdominal palpation from the obstetrical point of view and external version, preceded by comments by Dr. Pajot]. Deuxième Ed. Paris, 1889.
40. Altabef KM, Spencer JT, Zinberg S: Intravenous nitroglycerine for uterine relaxation of an inverted uterus. *Am J Obstet Gynecol* 1992 Apr; 166(4):1237–8.
41. Oxborn H, Foote WR: *Human Labor and Birth*, 3rd ed. New York: Appleton-Century Crofts, 1975.
42. Fernandez CO, Bloom SL, Smulian JC, Ananth CV, Wendel GD Jr: A randomized placebo-controlled evaluation of terbutaline for external cephalic version. *Obstet Gynecol* 1997 Nov; 90 (5):775–9.
43. Ranney B: The gentle art of external cephalic version. *Am J Obstet Gynecol* 1973 May;116 (2): 239–51.
44. Eglinton GS: The role of external version in modern obstetrics. In: Clark S, Phelan J, eds: *Cesarean Delivery*. Philadelphia: W.B. Saunders, 1991.
45. Zhang J, Bowes WA Jr, Fortney JA: Efficacy of external cephalic version: A review. *Obstet Gynecol* 1993 Aug;82(2):306–12.
46. Christian SS, Brady K, Read JA, Kopelman JN: Vaginal breech delivery: A five-year prospective evaluation of a protocol using computed tomographic pelvimetry. *Am J Obstet Gynecol* 1990 Sep;163(3):848–55.



47. Gimovsky ML, O'Grady JP, Morris B: Assessment of computed topographic pelvimetry within a selective breech management protocol. *J Reprod Med* 1994 Jul;39(7):489-91.
48. Crawford JS: An appraisal of lumbar epidural blockade in patients with singleton fetus presentation by the breech. *J Obstet Gynecol Br Commonw* 1974 Nov;81(11):867-72.
49. Fine EA, Brackers M, Berkowitz RL: An evaluation of the usefulness of x-ray pelvimetry: Comparison of the Thoms and modified ball methods with manual pelvimetry. *Am J Obstet Gynecol* 1980 May;137(1):15-20.
50. Federle MP, Cohen HA, Rosenwein MF, Brant-Zawadzki MN, Cann CE: Pelvimetry by digital radiography: A low-dose examination. *Radiology* 1982 Jun;143(3):733-5.
51. Caterini H, Langer A, Sama JC, Devanesan M, Pelosi MA: Fetal risk in hyperextension of the fetal head in breech presentation. *Am J Obstet Gynecol* 1975 Nov;123(6):632-6.
52. Eilen B, Fleischer A, Schulman H, Jagani N: Fetal acidosis and the abnormal fetal heart rate tracing: The term breech fetus. *Obstet Gynecol* 1984 Feb; 63(2):233-6.
53. Christan SS, Brady K: Cord blood acid-base values in breech-presenting infants born vaginally. *Obstet Gynecol* 1991 Nov;78(5 Pt 1):778-81.
54. Clark SL, Gimovsky ML, Miller FC: The scalp stimulation test: A clinical alternative to fetal scalp sampling. *Am J Obstet Gynecol* 1984 Feb;148 (3): 274-8.
55. Gimovsky ML, Petrie RH: The intrapartum management of the breech presentation. *Clin Perinatol* 1989 Dec;16(4):975-86.
56. Cohen WR, Friedman EA, eds: *Management of Labor*. Rockville, Maryland: Aspen Systems Corporation, 1983.
57. Milner RD: Neonatal mortality of breech deliveries with and without forceps to the aftercoming head. *Br J Obstet Gynaecol* 1975 Oct;82(10): 783-5.
58. Huchcroft S, Wearing PM, Buck CW: Late results of cesarean and vaginal delivery in cases of breech presentation. *Can Med Assoc J* 1981 Oct;125(7): 726-31.
59. Vimercati A, Greco P, Kardashi A, Rossi C, Loizzi V, Scioscia M, Loverro G: Choice of cesarean section and perception of leg pressure. *J Perinat Med* 2000;28(2):111-7.
60. Gimovsky ML: A breach with a breech. *Obstet Gynecol* 2002;100:613-4.
61. Kaneti H, Rosen D, Markov S, Beyth Y, Fejgin MD: Intrapartum external cephalic version of footling-breech presentation. *Acta Obstet Gynecol Scand* 2000 Dec;79(12):1083-5.
62. Collaris RJ, Oei SG: External cephalic version: A safe procedure? A systematic review of version-related risks. *Acta Obstet Gynecol Scand* 2004 Jun;83(6):511-8.
63. Chervanek FA, Johnson RE, Berkowitz RL, Grannum P, Hobbins JC: Is routine cesarean section necessary for vertex-breech and vertex-transverse twin gestations? *Am J Obstet Gynecol* 1984 Jan;148(1):1-5.
64. Medlock MD, Hanigan WC: Neurologic birth trauma. *Clin Perinatol* 1997 Dec;24(4):845-57.
65. Krebs L, Langhoff-Roos J: Breech delivery at term in Denmark 1982-1992: A population-based case-control study. *Paediatr Perinat Epidemiol* 1999 Oct;13(4): 431-41.
66. Krebs L, Topp M, Langhoff-Roos J: The relation of breech presentation at term to cerebral palsy. *Br J Obstet Gynaecol* 1999 Sep;106(9): 943-7.
67. Danielian PJ, Wang J, Hall MH: Long-term outcome by method of delivery of fetuses in breech presentation at term: Population-based follow up. *Br Med J* 1996 Jun;312(7044):1451-3.
68. Geutjens G, Gilbert A, Helsen K: Obstetric brachial plexus palsy associated with breech delivery: A different pattern of injury. *J Bone Joint Surg Br* 1996 Mar;78(2):303-6.
69. Al-Qattan MM: Obstetric brachial plexus palsy associated with breech delivery. *Ann Plast Surg* 2003 Sep;51(3):257-64.
70. Papp S, Dhaliwal G, Davies G, Borschneck D: Fetal femur fracture and external cephalic version. *Obstet Gynecol* 2004 Nov;104(5 Pt 2):1154-6.
71. Ghidini A, Korcker V: Fetal complication after external cephalic version at term: Case report and literature review. *J Matern Fetal Med* 1999 Jul-Aug;8(4):190-2.
72. Mayank S, Kriplani A: Fetal demise following external cephalic version. *Int J Gynaecol Obstet* 1997 Feb;56(2):177-8.
73. Gimovsky ML, Boyd C: Funic presentation as a complication of external cephalic version: A case report. *J Reprod Med* 1999 Oct;44(10):897-8.
74. Lau TK, Leung TY, Lo KW, Fok WY, Rogers MS: Effect of external cephalic version at term

- on fetal circulation. *Am J Obstet Gynecol* 2000 May;182(5):1239–42.
75. Smith JF, Hernandez C, Wax JR: Fetal laceration injury at cesarean delivery. *Obstet Gynecol* 1997 Sep;90(3):344–6.
76. Gimovsky ML, Vourlos D, Baldemero RE: A way to minimize C/S trauma. *Contemporary OB/GYN* 2005;(5):32–3.
77. Whyte H, Hannah M, Saigal S: Outcomes of children at 2 years of age in the term breech trial. *Am J Obstet Gynecol* 2004;189(Suppl):S57.
78. Granati B, Rondinelli M, Capoti C, Carnielli V, Bottos M, Rubaltelli FF: The premature breech presentation: Outcome of newborn infants born by vaginal or abdominal delivery. *Am J Perinatol* 1984 Jan;1(2):145–7.
79. Duehoelter JH, Wells CE, Reisch JS, Santos-Ramos R, Jimenez JM: A paired controlled study of vaginal and abdominal delivery of the low birth weight breech fetus. *Obstet Gynecol* 1979 Sep;54(3):310–3.
80. Westgren M, Ingemarsson I, Svenningsen NW: Long-term follow up of pre-term infants in breech presentation delivered by cesarean section. *Dan Med Bull* 1979 May; 26(3):141–2.
81. Gimovsky ML, Petrie RH: Optimal method of delivery of the low birthweight breech fetus: An unresolved issue. *Perinatol* 1988 Spring;8(2): 141–4.
82. Zlatnik F: The Iowa premature breech trial. *Am J Perinatal* 1993 Jan;10(1):60–3.
83. Myers SA, Gleiden N: Breech delivery: Why the dilemma? *Am J Obstet Gynecol* 1987 Jan;156(1): 6–10.
84. Deering S, Brown J, Hodor J, Satin AJ: Simulation training and resident performance of singleton vaginal breech delivery. *Obstet Gynecol* 2006 Jan;107(1):86–90.
85. Kotaska A: Inappropriate use of randomized trials to evaluate complex phenomena: Case study of vaginal breech delivery. *Br Med J* 2004 Oct; 329(7443):1039–42.
86. Lavin JP Jr, Eaton J, Hopkins M: Teaching vaginal breech delivery and external cephalic version: A survey of faculty attitudes. *J Reprod Med* 2000 Oct;45(10):808–12.
87. Hauth JC, Cunningham FG: Vaginal breech delivery is still justified. *Obstet Gynecol* 2002 Jun; 99(6):1115–6.
88. Gimovsky ML: Singleton vaginal breech delivery at term: Still a safe option. *Obstet Gynecol* 2004 Jul;104(1):191.
89. Glezerman M: Five years to the term breech trial: The rise and fall of a randomized controlled trial. *Am J Obstet Gynecol* 2006 Jan;194(1):20–5.
90. Ross S, Hannah M, Term Breech Trial Steering Committee: Interpretation of the Term Breech Trial findings. *Am J Obstet Gynecol* 2006 Dec; 195(6):1873; author reply 1873–4.
91. Hannah ME, Whyte H, Hannah WJ, Hewson S, Amankwah K, Cheng M, Gafni A, Guselle P, Helewa M, Hodnett ED, Hutton E, Kung R, McKay D, Ross S, Saigal S, Willan A, Term Breech Trial Collaborative Group: Maternal outcomes at 2 years after planned cesarean section versus planned vaginal birth for breech presentation at term: The international randomized Term Breech Trial. *Am J Obstet Gynecol* 2004 Sep;191(3):917–27.

V. Ravishankar

J. Gerald Quirk

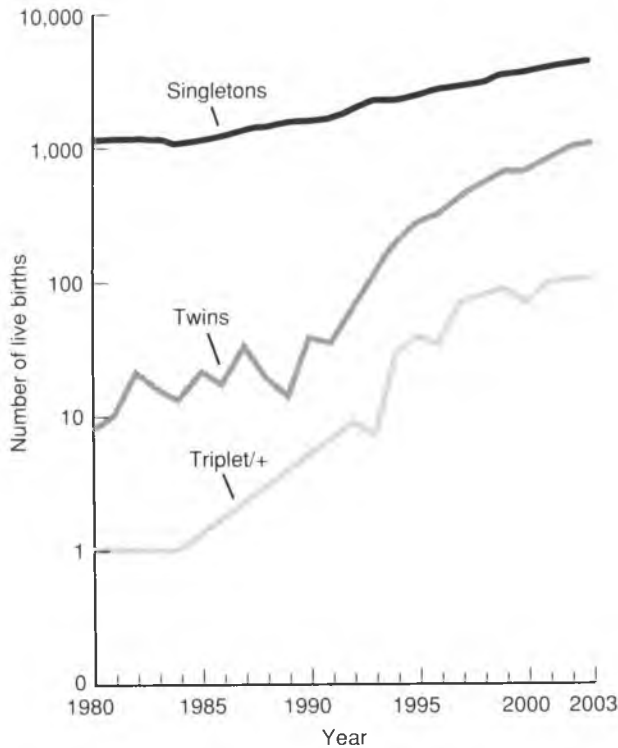
*By her he had two children at one birth.*

William Shakespeare (1564–1616)

*Henry VI, Part II (IV, ii)*

Since its inception, more than 1 million children have been born through assisted reproductive technologies (ART). Fertility treatments account for the explosive increase in twins and in high-order multiple (HOM) births seen in the last two decades. Specifically, twin births increased by 66% from 1980 to 1998, and triplets increased by more than 400% during the same period (against an increase of 6% in singleton births) [1]. In 2003 in the United States, the twin birth rate was 31.5 per 1,000 total live births, and the triplet birth rate was 187.4 per 100,000 live births [1]. Reynolds and colleagues analyzed the trends in multiple births from ART in the United States between 1997 and 2000 and reported that more than 50% of live births from ART were multiples [2]. Besides ART, ovulation-inducing drugs and intrauterine inseminations account for the increase in multiple births. The other major factor contributing to this surge is the older maternal age at childbearing. Delayed childbearing and increasing reliance on ART to achieve pregnancy have led to an increase in multiple births. Between 1990 and 2003, the number of twin and HOM births born to women in the age group 45 to 49 years increased notably (Figure 13.1) [1].

The disturbing rise in multiple births imposes a great burden on medical care, mainly because of an increase in preterm births. Multiple births account for 10% of all preterm births. The last two decades witnessed a considerable decrease in perinatal mortality rate in singletons principally because of advances in neonatal management that include the use of surfactants and antenatal corticosteroids [3]. Although multiple gestations account for only 3.3% of live births [1], they contribute significantly to the increase in very-low-birthweight (VLBW) infants. About 11% of all multiple births have birthweights less than 1,500 g (VLBW by definition; Figure 13.2). Although the overall perinatal mortality rates have decreased in multiples in the last decade, they are still high compared with that of singletons. Twins have a fourfold increase and triplets have a ninefold increase in the perinatal mortality rates



NOTE: Triplet/+ includes births in greater than twin deliveries. Rates are plotted on a log scale.

FIGURE 13.1.

*Multiple births to women 45 to 49 years of age: United States 1990–2003. (From Martin JA, Hamilton BE, Sutton PD et al. Births: Final Data for 2003. National Vital Statistics Reports 2005;54; with permission.)*

compared with that in singletons [4]. Multiple factors such as race, maternal age, smoking, and socioeconomic status influence the perinatal outcomes. Rydhstroem and coworkers [5] analyzed, from the Swedish Medical birth registry, the fetal and infant mortality rates of singletons and twins born between 1973 and 1996 and found that twins born before 34 weeks had six- to eightfold mortality compared with singletons. The fetal mortality rate in twins was consistently higher at all gestational ages and was higher in same-sex twins.

### CEREBRAL PALSY IN MULTIPLES

The last two decades witnessed a great surge in multiple births from ART, and the long-term neurologic consequences are now only beginning to unfold. Pharoah and Cooke reported a higher prevalence of cerebral palsy in survivors of multiples (44.8 in triplets, 12.6 in twins, and 2.3 per 1,000 births) [6]. Scher and coworkers collected data from more than 1 million singleton births and from 26,000 twin births from five populations in Australia and the United States [7]. They reported a higher prevalence of cerebral palsy in twin survivors than in singleton survivors at 1 year of age (0.59% vs. 0.14%,  $p \leq 0.0001$ ). The cerebral palsy rates were also higher in twins born after 38 weeks' gestation (or above 2,500-g birthweight). Overall, the higher prevalence of cerebral palsy seems to be gestational age dependent up to 36 or 37 weeks. After 37 weeks, twins seem to be at a disadvantage [8]. Intrauterine

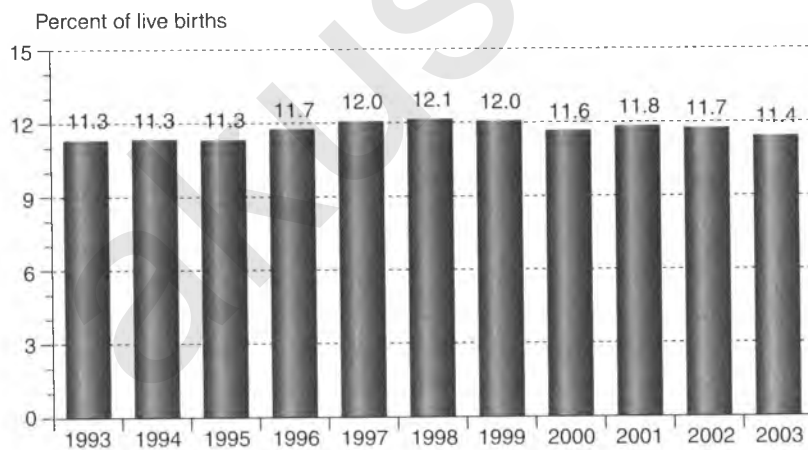


FIGURE 13.2.

*Prevalence of VLBW infants among multiples. (From [www.marchofdimes.com/peristats](http://www.marchofdimes.com/peristats); with permission.)*

death of a monochorionic twin also increases the cerebral palsy rate in the surviving twin.

Maternal and fetal complications also are increased in multiple gestations. Hypertensive disease, anemia, pyelonephritis, and postpartum hemorrhage are more common, as are preterm deliveries, fetal malpresentations, and placenta previa, which increase the likelihood of cesarean sections. Congenital malformations, growth restriction, polyhydramnios, and fetal losses are more common in multiple gestations.

This chapter explores the maternal and fetal complications, advances in prenatal diagnosis, and management of complications unique to multiple gestations.

## EPIDEMIOLOGY

An accurate determination of the incidence of multiple gestations is difficult. The spontaneous twinning rate in Caucasians is about 12.5 per 1,000 births [9]. The lowest incidence in twinning is seen in the Japanese population (6–7/1,000 births [10]) and the highest incidence of spontaneous twinning occurs in Nigerians (35/1,000 births [11]). *Monozygotic (MZ) twins*, which result from splitting of a fertilized ovum, occur at a constant rate of 4 per 1,000 births in all populations, and this is not influenced by age, parity, or race. *Dizygotic (DZ) twins*, which result from the fertilization of two separate ova, are more common and account for about two thirds of all *spontaneous twin* conceptions.

Spontaneous triplet gestation is very rare – about 1 per 10,000 births. Spontaneous conceptions of HOM gestations are extremely rare. The causes of twinning are not well known. Maternal characteristics influence DZ twinning rates. Taller and heavier women have an approximately 30% increased rate of DZ twinning against shorter and thinner women [12]. Increased maternal age and parity also account for increased twinning. Maternal age increases DZ twinning and, to a lesser extent, MZ twins [13]. Twinning rates increase with maternal age up to the mid- to late thirties and then decline [14]. Again, rising gonadotropin levels with increasing age and their decline before menopause could account for the increase in multiple gestations in the older age group [12].

Extreme nutritional deprivation decreases the twinning rates in humans. In animal studies, improv-

ing nutrition produced the opposite effect by increasing the size of litters. Maternal familial and genetic factors influence the incidence of DZ but not MZ twins. Paternal history does not seem to have an effect on the twinning rate. Mothers of DZ twins are more likely to have subsequent twin gestations than are women with previous singleton births [14]. Women who were born of a twin gestation are more likely than the general population to give birth to twins.

Fertility treatment is the most significant contributor to the increase in multiple gestations, because all ovulation-inducing drugs cause multiple ovulations. Women treated with clomiphene have a twinning rate between 6.8% and 17%. Gonadotropin stimulation has a twinning rate between 18% and 53.5% [15].

## PHYSIOLOGY OF MULTIPLE GESTATIONS

Twins can result either from two zygotes (*dizygotic twin*) or from splitting of one zygote (*monozygotic twin*). DZ twins occur in two thirds of spontaneous twin conceptions, and MZ twins represent the rest. DZ twins, resulting from fertilization of two separate ova, can be different or the same sex, whereas MZ twins are always the same sex and have identical genetic material.

In assisted reproduction, DZ twins account for almost all births, with a few exceptions of MZ twins resulting from the splitting of the inner cell mass of the blastocyst. Older textbooks went to great lengths to explain the phenomena of superfecundation and superfetation. *Superfecundation* is the term used for fertilization of ova at different times by separate events of coitus but in the same ovulatory cycle, and *superfetation* occurs when the fertilization of the ova occurs at different cycles. Neither has ever been demonstrated in human reproduction.

MZ twinning results from the splitting of the zygote at various times after fertilization. The cause of this splitting is not well known. Fertility treatments also seem to increase the incidence of MZ twinning by two- to fourfold [16]. ART, especially embryo transfers at the blastocyst stage, is associated with an increase in the MZ twinning rates up to 5% [17,18]. It has been proposed that MZ twinning could result from exposure to teratogens. MZ twinning reportedly increased in mice treated with vincristine sulfate [19]. Recently, Bamforth

TABLE 13.1 Diagnosis of Multiple Gestations

Time of Zygote Splitting	Type of Placentation
0–3 days	Dichorionic-diamniotic
4–7 days	Monochorionic-diamniotic
8–11 days	Monochorionic-monoamniotic
>11 days	Conjoined twins

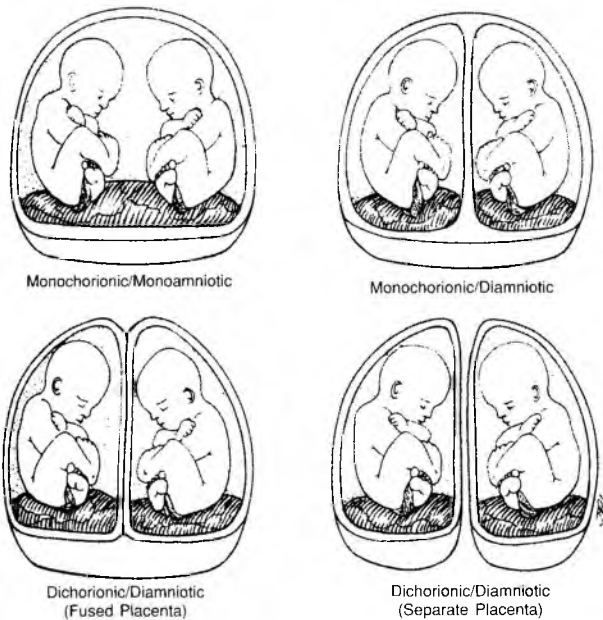


FIGURE 13.3.  
Possible placental types in twin gestations.

and coworkers explored the role of a cell adhesion molecule E-cadherin in MZ twinning. In their study of both DZ and MZ placentas, they found slight overrepresentation of polymorphism in MZ twins, which did not reach statistical significance [20].

### Placentation in Twin Gestations

The possible placental types in twin gestation are 1) *dichorionic-diamniotic* placenta, which contains two chorions and two amnions; 2) *monochorionic-diamniotic* placenta, having one chorion and two amnions; and 3) *monochorionic-monoamniotic* placenta, in which the twin fetuses are enveloped by a single amnion surrounded by one chorion (Figure 13.3).

It is important to understand the difference between zygosity and chorionicity. All DZ twins have dichorionic-diamniotic placentas. In dichorionic placentas, the implantation of the blastocysts can be at different sites. This is easily seen on ultrasound scan when the placentation occurs on the anterior and posterior walls of the uterus. If two implanting blastocysts are proximal to each other, the placentas might fuse, but the placental type remains dichorionic-diamniotic. When fusion of dichorionic placentas occurs, a distinctive firm, raised, yellow to yellow-white chorionic ridge normally forms at the site where the two chorions meet. Despite the proximity, placental vascular anasto-

moses rarely occur. Histologic examination of the placenta shows four layers – composed of two chorions and two amnions – separating the fetuses.

In monozygotic twins, depending on the time during which the zygote splits, three forms of placentations can occur (Table 13.1). Splitting of the zygote in the first three days after fertilization results in a dichorionic-diamniotic placentation. These twins are “identical” but likely to suffer fewer complications because the chorion has not differentiated, and the vascular communications between the placentas are rare. Approximately 30% of monozygotic twins have this form of placentation [21]. When the blastocyst divides between the fourth and seventh day after fertilization, the chorion and not the amnion has already differentiated to form a monochorionic-diamniotic placenta. This is the most common type and occurs in about 70% of monozygotic twin gestations. Vascular anastomoses between the fetuses commonly occur in the placenta, and this type of placentation is beset with unique complications, the chief of them being twin-to-twin transfusion syndrome (TTTS). Rarely, the zygote splits at the bilaminar germ disc stage before the appearance of the primitive streak (between 8 and 11 days after fertilization) to form a monochorionic-monoamniotic placenta. Division of the embryo after the formation of the primitive streak and node results in *conjoined twins*, which are further classified according to the union of the fetal parts. It can involve sharing of internal organs like the liver and of major circulation. Fortunately, this is very rare.

Prior to introduction of ultrasound scan as part of routine prenatal care, twin gestations were detected often later in pregnancy or sometimes only during delivery. The chorionicity can now be established by



FIGURE 13.4.  
*Ultrasound scan showing “twin peak” or “lambda” sign.*

ultrasound scan very early in the pregnancy, and this helps in management and parental counseling.

As early as by fifth postmenstrual week, the chorionic sacs can be identified on transvaginal ultrasonography by their sonolucency, surrounded by a bright rim of chorion [22]. This requires expertise in the use of transvaginal ultrasonography, since the yolk sac might be mistaken for a separate gestational sac. The fetal heartbeats are evident very early in the sixth postmenstrual week, and identification of two or more fetal heartbeats confirms the presence of a multiple gestation. Because the amnion does not differentiate before the eighth week, it is not possible to see it separately from the chorion in very early pregnancy.

With high-resolution ultrasonography, it is now possible to determine chorionicity by 10 weeks. In a dichorionic placenta, the junction between placentas appears thick and has the characteristic ultrasound appearance called the *lambda* or *twin peak* sign (Figure 13.4). The intervening membrane measures at least 2 mm and is made up of two chorions and two amnions. In monozygotic twin gestation, the junction has an attenuated look and is T-shaped where it meets the uterine wall (Figure 13.5). Similar diagnostic criteria are applied in HOMs. Carroll and coworkers accurately diagnosed chorionicity in 149 out of 150 twins between 10 and 14 weeks by ultrasound scan, which was later confirmed by histologic examination of the placentas after delivery [23]. The fetal positions should be described in relation to the maternal side. In good practice, a diagrammatic representation can help in identifying the fetus with problems, especially with advancing gestation.



FIGURE 13.5.  
*Monochorionic twin gestation showing thin amniotic membrane between the sacs (T-sign).*

In the mid-trimester scan, there are other features that can help in determining chorionicity. Fetuses with discordant sexes have dichorionic placentas, and about 45% of the fetuses with the same sex have dichorionic placentas. One should look for the placental positions, and in about one third of the pregnancies, the placentas are seen to be clearly separated and can be on the opposite uterine walls. The sensitivity of this finding in diagnosing dichorionic placentas is 97% [24].

Monozygotic placentas are seen in 20% of all twins, and perinatal complications are about three to five times higher than in those with a dichorionic placenta [25]. In addition, monozygotic twins are more likely to have preterm deliveries, congenital anomalies, low birthweights, and growth restrictions, and to suffer from neurologic complications, including cerebral palsy. TTTS occurs in 10% to 15% of monozygotic twins.

Monoamniotic twins constitute 1% of MZ twins [21]. Because these fetuses share a common amniotic cavity, cord entanglement can occur, with disastrous consequences. A perinatal mortality rate of 40% or higher has been reported [26].

Because complications depend on the type of placentation, establishing chorionicity early in pregnancy is important. The couple should be counseled on the type of placentation and its significance. The use of terms *identical* and *fraternal* can be misleading and should be avoided. About one third of monozygotic twins have dichorionic placentations and might be erroneously labeled as fraternal twins.

Although ultrasonography establishes an accurate diagnosis of twins, there are some clinical features that indicate the diagnosis. The clinician should have a high index of suspicion of multiple gestation when a patient 1) on examination has a fundal height measurement greater than normal for the gestational age, 2) has unexplained anemia or excessive nausea early in pregnancy, 3) has two separate fetal heart beats on Doppler examination, 4) develops early or unanticipated carbohydrate intolerance, or 5) has an elevated maternal serum  $\alpha$ -fetoprotein (MSAFP) levels on mid-trimester screening.

### MATERNAL PHYSIOLOGIC CHANGES

Pregnancy imposes demands on the mother, and the physiologic adaptations occur in almost every organ system. Maternal physiologic changes in pregnancy are exaggerated in multiple gestations.

In a singleton pregnancy, the plasma volume increases by 40% to 45% and reaches its peak at 32 weeks [27]. In late gestation, the blood volume increases by an average of 1,570 ml in singletons against an increase of 1,980 ml in twin gestations [28]. The cardiac output also increases owing to increases in both stroke volume and heart rate. Echocardiographic studies have confirmed the increase in cardiac output in twin gestations over that in singleton pregnancies [29]. The normal fall in diastolic blood pressure seen in mid-trimester is exaggerated in multiple gestations. The venous flow in the lower extremity is further impeded by a greater degree of uterine distension in multiple gestations.

As would be expected, uterine growth is marked in multiple gestations. By 18 weeks, the 50th percentile for the uterine height in twin pregnancies exceeds the 90th percentile for that of singleton pregnancies. By 28 weeks of gestation, the 10th percentile for the uterine size in twin gestations is greater than the 90th percentile for singleton pregnancies [30]. Additional burden can be imposed by polyhydramnios, a possible complication of twin gestations.

Respiratory symptoms such as shortness of breath are exaggerated as a consequence of uterine overdistension. Tidal volume and oxygen consumption in multiple gestations are increased, as is the normal alkalosis seen in singleton pregnancy.

Hematologic changes are more marked, and the additional blood volume increase exaggerates the physiologic anemia. The red cell expansion lags behind the plasma volume increase, and the mean hematocrit is lower in multiple gestations [28]. In multiple gestations, as in singleton pregnancies, coagulation factors VII, VIII, IX, X, and XII, and fibrinogen and von Willebrand's factor are increased. Prothrombin (factor II), factor V, antithrombin III, and protein C are unchanged. There is a decrease in factor V and protein S levels in pregnancy. There is a gradual decrease in the fibrinolytic activity, as reflected by marked increases in plasminogen activator inhibitors (PAI) PAI-1 and PAI-2 [31]. These changes make pregnancy a hypercoagulable state and predispose to venous thromboembolism.

Serum protein and electrolyte levels can be reduced. The total protein content, sodium, potassium, and chloride levels, and serum osmolality are unchanged, as in singleton gestations [32]. Higher incidence of iron-deficiency anemia and folate deficiency are seen in twin gestations, and they should be corrected by supplementation. More women with twin gestations suffer from nausea and vomiting in early pregnancy, and gastric reflux symptoms and constipation are more common. The frequency of intrahepatic cholestasis is doubled, and the dreadful complication of acute fatty liver of pregnancy is increased in multiple gestations. Davidson and coworkers reported three cases of acute fatty liver confirmed by liver biopsies, and their resolution after delivery [33]. They hypothesized that in triplet gestations, an increased production of free fatty acids and decreased free fatty acid oxidation and increased fatty acid metabolism by the fetuses could lead to acute fatty liver. Carbohydrate metabolism is altered to a greater degree in twin gestations. Falls in blood glucose and insulin levels are more marked especially in late gestation, and ketonuria is more common [34]. MacGillivray [32] reported an increase in gestational diabetes mellitus (GDM) in twin pregnancies, which was not supported by another clinical study [9]. Recently, Schwartz and others reported the frequency of GDM (using a screening cut-off value of 135 mg at 1 hour) in twins against that in singletons [35]. The levels of MSAFP and human chorionic gonadotropin (hCG), both serum markers in screening for fetal aneuploidies, are elevated and are about twice the mean value for singleton pregnancies [36]. Human placental lactogen, estradiol,



and progesterone levels are also significantly higher than those seen in singleton pregnancies.

### WEIGHT GAIN IN MULTIPLE PREGNANCIES

Poor maternal weight gain is associated with fetal growth restriction and preterm deliveries [37,38]. The Institute of Medicine suggested that a weight gain of 35 lbs to 45 lbs (18.2 kg–20.5 kg) is adequate for a term twin gestation [39]. Adequate weight gain should be achieved by 24 weeks' gestation to have a sufficient impact on fetal birthweight. Before 20 weeks, only monozygosity and smoking seem to affect fetal growth negatively [40]. Inadequate maternal weight gain (i.e., less than 24 pounds by 24 weeks) is associated with lower birthweights ( $\leq 2,500$  g).

Luke and coworkers, in their extended study of 1,564 pregnancies, noted the impact of maternal weight gains at different stages of pregnancy on the rates of fetal growth [41]. They showed that early maternal weight gain (before 20 weeks) and mid-pregnancy weight gain (between 20 and 28 weeks) had a significant effect on the fetal growth between 20 and 28 weeks, and 28 weeks to birth respectively. This ripple effect underscores the importance of early maternal weight gain. Furthermore, they showed that a majority of women who were counseled by registered dietitians had the highest weight gains and lowest percentage of very-low-birth-weight infants (2% versus 12%).

### MATERNAL COMPLICATIONS

Ideally, all women undergoing fertility treatment should have preconception counseling on the possible complications of multiple gestations. Overall, pregnancy complications are increased (Table 13.2) in multiple gestations. Of these, the most serious complication is preterm delivery, which affects more than one half of all twins [1]. The mean gestational age at delivery of twins is 35.3 weeks and that of triplets is 32.2 weeks [42].

### HYPERTENSIVE DISORDERS

Hypertensive disorders increase by at least twofold in twin gestations. In their secondary analysis of data from the trial of low-dose aspirin for prevention of preeclampsia, Sibai and coworkers con-

TABLE 13.2 Maternal Complications

Spontaneous miscarriages
Preterm labor
Anemia
Hypertension
Intrauterine death of one twin
Hypertensive disorders
Pyelonephritis
Gestational diabetes
Fatty liver of pregnancy
Placental and cord complications
Postpartum hemorrhage
Increased cesarean delivery

cluded that women with twin gestations have higher rates of gestational hypertension (RR 2.04; 95% CI, 1.60–2.59) and preeclampsia (RR 2.62, 95% CI, 2.03–3.38) [43]. In addition, severe preeclampsia and hemolysis, elevated liver enzymes, low platelet count syndrome (HELLP) occurred more often in twin gestations. Management of hypertensive disorders in twin pregnancy should follow the same guidelines as that in singleton pregnancies. Twin gestations are more susceptible to fluid overload, and patients receiving magnesium sulfate prophylaxis should be closely monitored for pulmonary edema. Resolution of preeclampsia has been seen with the death of one fetus (usually the growth-restricted fetus). Heyborne and Porreco reported two cases of severe preeclampsia associated with growth restriction of one twin and one case of Ballentyne syndrome with developing preeclampsia [44]. Selective fetocides of the affected twins were performed in all the pregnancies, which resulted in the resolution of preeclampsia and prolongation of pregnancies by several weeks. It is possible that preeclampsia resolved following the involution of placenta of the affected twin.

Hemorrhagic complications occur more frequently with twins. Placenta previa and placental abruption are more common [45,46]; the latter can also result from sudden decompression of the uterus following delivery of the first twin. Monochorionic twins have a higher incidence of velamentous cord insertions and of vasa previa. Higher cesarean delivery rates from malpresentations entail greater blood loss, and postpartum hemorrhage from uterine atony occurs more frequently in multiple gestations.

Maternal anemia is four times as common in multiple gestations [47]. Multiple gestation places a greater demand on iron and folate requirements, so that these should be supplemented during pregnancy. Pyelonephritis and cholestasis are also observed more frequently [48].

### **PRETERM LABOR**

Preterm deliveries (less than 37 weeks' gestation) occurred in 10.6% of singleton pregnancies against a phenomenal rate of 61.2% of live births in multiple gestations [1]. In addition, 13.2% of multiple births occur at less than 32 weeks' gestation [49]. Preterm delivery is the single most important cause of perinatal mortality and morbidity in multiple gestations. Although there is an increase in the preterm births in multiple gestations, the survival of the infants is gestational age dependent, and similar outcomes are expected in singletons as well as in multiples [50,51].

Ideally, it should be possible to predict the chances of preterm delivery by means of easily available and reproducible tests so that interventions can be instituted to prevent preterm births. Home uterine monitoring has not shown to be effective in early diagnosis of preterm labor, however, and is in fact likely to increase costs by frequent visits to the hospital and by increased and unnecessary tocolytic therapy [52]. For prediction of preterm births, fetal fibronectin and cervical length measurements by transvaginal ultrasonography have shown promising results. *Fetal fibronectin* is a glycoprotein found in amniotic fluid and placental extracts, and it probably helps in binding uteroplacental and fetal membrane-decidual surfaces. Although present normally in the cervix between 16 and 20 weeks, fetal fibronectin does not reappear until about 35 weeks. A commercially available assay can test for the presence of fetal fibronectin in the cervix (using a cut-off value of 50 ng/ml), and the results are usually available in 1 to 2 hours. The fetal fibronectin test has a very high negative predictive value (NPV, 99%) and a low positive predictive value (PPV, 13%) for delivery within 7 days [53]. This test could be used in twin gestations equally with probably greater PPV (55% versus 13%) [54].

Ultrasound measurement of cervical length is more frequently used to predict preterm deliveries. A cervical length of 25 mm or less is associated

**TABLE 13.3 Fetal Complications**

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"Vanishing twin"
Fetal anomalies
Fetal growth restriction
Intrauterine death of one or both twins
Twin-to-twin transfusion syndrome
Prematurity
Cerebral palsy

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with greater chances of delivery before 32 weeks (OR 6.9; 95% CI 2.0–24.2) [53]. Souka and coworkers obtained cervical length measurements at 23 weeks' gestation in 215 twin pregnancies and reported that the sensitivity of cervical length measurements in predicting spontaneous preterm deliveries is the same as that in singletons [55]. Rates of pregnancies with cervical lengths less than 25 mm were higher when pregnant with twins compared with singletons (11% vs. 8%). Neither fetal fibronectin levels nor cervical length measurements have been shown to influence obstetric interventions, however. Their usefulness in HOM gestations is not clear.

### **FETAL COMPLICATIONS**

As a consequence of preterm births, perinatal mortality rates are disproportionately higher in twins. The stillbirth rate is twice that seen in singletons [56]. Cord entanglements or prolapse, twin-to-twin transfusion syndrome, congenital anomalies, and birth trauma are some of the unique complications that contribute to the higher perinatal mortality seen in twins (Table 13.3).

### **"VANISHING TWIN" SYNDROME**

In the United States, 84% of women begin their prenatal care in the first trimester [1]. As ultrasonography is incorporated more frequently into early prenatal care, some of the multiple gestations that are diagnosed early in the pregnancy resorb subsequently ("vanishing twin" syndrome). Higher fetal loss rates occur with both spontaneous and artificial reproduction. In one study on the follow-up of pregnancies from fertility treatments, spontaneous reduction of one or more gestational sacs or of embryos before 12 weeks' gestation occurred in 36%

of twins, 53% of triplets, and in 65% of quadruplet pregnancies. In contrast, only 19.2% singleton pregnancy losses were seen [57]. Pregnancies following ART are more closely monitored from conception and therefore reflect the true incidence of fetal losses with greater accuracy. In an earlier study of 274 multiple pregnancies, Dickey and colleagues found that the presence of fetal heart beats at 7 weeks of gestation ensured twin births in 90% of cases [58]. The live birth rates were slightly lower in women aged 30 years or older. Contrarily, in the same study, the authors found that when the presence of two gestational sacs was the diagnostic feature of the twins, only 63% of patients had twin births. These observations should be taken into consideration when counseling the parents about the possibility of fetal losses. Pregnancy outcomes following spontaneous loss of one twin before 12 weeks are usually good.

### GROWTH RESTRICTION IN MULTIPLE GESTATIONS

Growth of singletons and twins is comparable until 27 weeks' gestation (Figure 13.6). The uterus accommodates the larger volume imposed by twins by overdistension, and beyond a certain limit, premature labor can result. The preferential restriction in fetal size favors the advancement in gestational age. At 31 weeks, the median combined weight of twins equals the median birthweight at 40 weeks' gestation of a singleton. In triplets, this is achieved as early as at 28 weeks [59]. Birthweight discordance is the difference between the larger and the smaller

twin (or between the largest and the smallest fetuses in triplets) and it is expressed as a percentage of the larger twin. Discordance (%) can be calculated on the estimated fetal weights (EFW) on ultrasound scan by the following formula:

$$\frac{\text{EFW of the larger twin} - \text{EFW of the smaller twin}}{\text{EFW of larger twin}} \times 100$$

About 75% of twins are less than 15% discordant, and only about 5% of twins are greater than 25% discordant [60]. Twins with less than 25% growth discordance usually have favorable outcomes. *Even if markedly discordant, the neonatal mortality rate of the smaller twin was higher only if he or she weighed less than the tenth birthweight percentile [61].*

Serial monitoring by ultrasonography should be undertaken to assess fetal growth reliably. Singleton growth curves usually are used, and prospective studies have not shown any differences in the fetal biometrics of singletons and twins [62,63]. If growth restriction of one twin is identified, surveillance by serial growths and Doppler studies should be instituted. Management of twin pregnancy with one severely growth-restricted fetus poses a challenge, since early delivery places the normally grown twin at risk for severe prematurity.

### INTRAUTERINE DEATH

The incidence of fetal demise of one twin is approximately 0.5% to 6.8% [64–66]. More recently, it is estimated that intrauterine death of at least one fetus

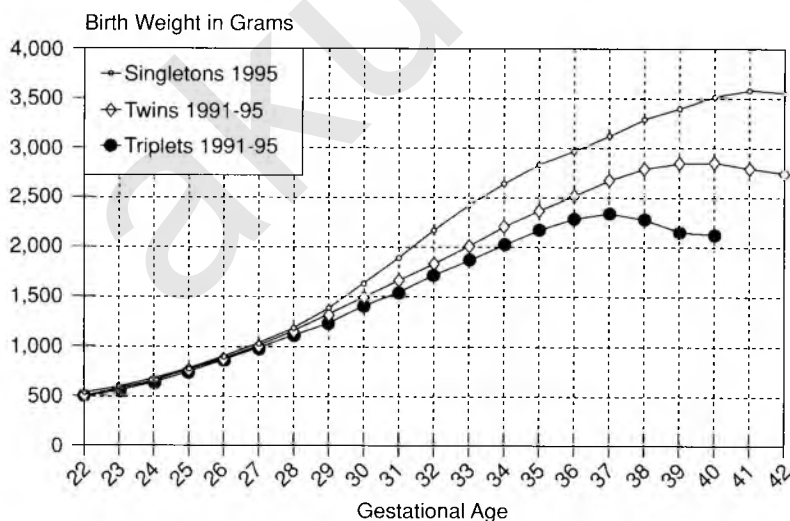


FIGURE 13.6. U.S. resident live births: 50th birthweight percentile by gestational age for singletons, twins, and triplets. (From Alexander GP, Kogan M, Martin J, Papiernik E: *What are the fetal growth patterns of singletons, twins, and triplets in the United States?* Clin Obstet Gynecol 1998;41:115; with permission.)



FIGURE 13.7.  
*Fetus compressus or papyraceous.*

occurred in 2.6% of twin and 4.3% of triplet gestations that survived to 20 weeks' gestation [67]. Early demise of one twin before 12 weeks is usually without complications. *Fetus papyraceus* or a *membranous twin* is the term applied when, after demise, the tissue fluid and the amniotic fluid are resorbed slowly and the fetus is compressed and incorporated into the membranes (Figure 13.7). Unless these are subjected to careful examination, this finding might be easily overlooked at delivery. The fetal parts might be absent or too small to be visible on gross inspection. Disseminated intravascular coagulation in the mother, following the death of a twin, is extremely rare.

Fetal death of one twin is more commonly seen in monochorionic twins. TTTS, cord accidents, velamentous insertion of the cord, and placental abruption are possible causes. Subsequently, the co-twin in a monochorionic pregnancy could suffer the consequences of fetal death or major neurologic mor-

bidity. Multicystic encephalomalacia was reported to occur in 12% of surviving twins [68]. It is possible that at the time of fetal death sudden hypotension or hypoxic injury occurs in the surviving twin [69,70]. Johnson and Zhang [67], in their analysis of data from the Matched Multiple Birth File from the U.S. National Center for Health Statistics, found an inverse relation between time at death of one fetus and outcome of the co-twin. The earlier the death of one twin occurred, the higher were the chances of demise of the co-twin, and same-sex twins had higher rates of subsequent demise of the other twin than did opposite-sex twins.

Pharoah and Adi obtained information on the surviving twins (following fetal demise of co-twin) by surveying the general practitioners, in the United Kingdom, taking care of these twins [71]. Surviving twins were between 4 and 6 years of age at the time of survey. Of the surviving twins, the prevalence of cerebral palsy in the same-sex twins was 106 per 1,000 (95% CI, 70–150) versus 29 per 1,000 (95% CI, 6–84) surviving infants in opposite-sex twins ( $p \leq 0.02$ ). They also noted higher neurologic complications like speech and language delays in the survivors of same-sex twins. The limitations of this study were that chorionicity could not be predicted with accuracy, and in some cases, the sex of the dead twin was assigned by the parents when it could not be determined accurately.

The management of monochorionic pregnancies complicated by in-utero death of one twin is challenging. Preterm delivery occurs more frequently [72,73]. The parents should be counseled about the possible death and neurologic morbidity of the surviving twin. Multicystic encephalomalacia is sometimes evident on MRI after some days or weeks after fetal demise of the co-twin. Early delivery is not warranted because the neurologic insult more likely occurred at the time of death of the co-twin. Contrary to former belief, disseminated intravascular coagulation in the mother is very rare [73]. Cesarean delivery does not confer additional benefits and should be undertaken for obstetric indications only.

### CONGENITAL ANOMALIES

Congenital anomalies are more common in multiple gestations. In one large study, incidences of congenital anomalies were 1.4% in singletons, 2.7% in

twins, and 6.1% in triplet gestations [74]. In addition to structural defects, especially open neural tube and cardiac defects, the incidence of chromosomal anomalies is also higher in twin gestations. Cleft lip and palate, abdominal wall defects, and limb reduction defects are also more common [75,76]. Anomalies are more common in monochorionic twin gestations and presumably result from the twinning process itself. The frequency of chromosomal anomalies in DZ twins is similar to that in singletons, but the pregnancy risk of carrying at least one fetus with a chromosomal anomaly is approximately twice that of singleton gestation [77]. From the embryology of MZ twinning, one would assume that all MZ twins are genetically and phenotypically identical, and this is normally the case. On rare occasions in MZ twins, however, chromosomal nondisjunction occurs just before or at the time of twinning, leading to chromosomal discordance. This phenomenon is called *heterokaryotypia*. Discordance, presumably from postzygotic error, has been reported for Turner syndrome [78]. Theoretically, the discordance can occur with any autosomal or X-linked disorders. The discordance with trisomy 21, although reported, is very rare. A discordant structural anomaly in MZ twins should prompt sampling of amniotic fluid for karyotype from both sacs [79].

When couples discover the presence of discordant anomaly, they are faced with difficult management choices. They can opt for continuation of the pregnancy, selective fetocide of the anomalous fetus, or termination of the entire pregnancy. Appropriate counseling should be provided to enable them to make an informed choice. Evans and coworkers reported the results of selective fetocide performed in 402 dichorionic pregnancies at various gestational ages [80]. Fetocide was performed by intracardiac or intrafunic injection of potassium chloride. They did not find any significant difference in the fetal loss rates up to 24 weeks. The overall fetal loss rate was 7.1% when the pregnancies were reduced to singletons.

Selective fetocide by intracardiac injection of potassium chloride in monochorionic pregnancies with discordant anomalies can cause the death of other twin by transplacental passage of the drug. Bipolar coagulation [81] or umbilical cord ligation by fetoscopy [82,83] of the affected twin in experienced hands is a safer method.

## ACARDIAC TWINS

Acardiac anomaly is a rare complication with an incidence of 1 in 35,000 births, and it occurs in 1% of monochorionic twin gestations [84]. In this anomaly, the cardiac structure in one twin is missing, and this twin receives its blood supply from the normal fetus ("pump twin"). The blood flow is directed toward the acardiac twin. It is also called *twin reversed arterial perfusion* (TRAP) sequence. This wasteful effort of feeding the parasitic twin leads to long-term morbidity in the normal twin. The size of the acardiac twin varies and can even be larger than the pumping twin. Frequently, the head, upper limbs, and internal organs, including liver, bowels, and pancreas, are absent. The spine can be present in rudimentary form [85]. The vascular anastomoses are usually on the placental surface, and, infrequently, there is a direct attachment of the umbilical cord to that of the pumping twin. The demonstration of retrograde flow toward the acardiac twin by Doppler ultrasound scan can establish the diagnosis. Management options include a conservative approach of nonintervention, or in experienced hands, by the use of bipolar coagulation of the cord of the acardiac twin.

## TWIN-TO-TWIN TRANSFUSION SYNDROME

TTTS is a unique complication that occurs in 15% of monochorionic pregnancies [25]. The type of vascular communication and the direction of blood flow determine the donor and the recipient status of the twins. In the absence of intervention, the donor suffers from hypotension, oliguria, growth restriction, oligohydramnios and ultimately fetal death. The volume overload in the recipient leads to polyhydramnios, polyuria, cardiomegaly, and cardiac failure, ultimately leading to hydrops.

### **Pathophysiology**

In a monochorionic placenta, vascular anastomoses between the twins are common. Superficial and deep anastomoses are usually seen. Three types of vascular anastomoses can occur in monochorionic pregnancies: 1) arterioarterial (AA), 2) arteriovenous (AV), or 3) venovenous (VV). Bajoria and coworkers perfused monochorionic placentas with and without TTTS and noted that in TTTS

placentas, there were fewer anastomoses of each type [86]. In addition, the presence of deep anastomoses was related to the development of TTTS. They concluded that in TTTS: 1) there are fewer placental vascular anastomoses; 2) there were more deep than superficial anastomoses; and 3) there was at least one arteriovenous communication, in which an unpaired artery of one twin anastomoses with the corresponding vein of the other twin, resulting in a unidirectional flow from one twin to the other. Bidirectional AV anastomoses maintain equilibrium in perfusion and are common in monochorionic placentas, which explains why TTTS occurs in only 15% of the cases. Denbow and associates [87] demonstrated that TTTS placentas have fewer superficial anastomoses, and that there is an absence or paucity of superficial arterioarterial anastomoses.

It is possible that other mechanisms are at work in the pathophysiology of TTTS. In one study, immunochemistry and in-situ hybridization of the donor kidneys showed an overexpression of renin. The upregulation of renin synthesis is a possible consequence of hypoperfusion, which leads to the synthesis of angiotensin II. Vasoconstriction of intrarenal vessels induced by angiotensin II can worsen the hypoperfusion and oligohydramnios in the donor. The recipient kidneys showed downregulation of renin synthesis. The hypertensive changes in the recipient kidneys were thought to result from the transfer of circulating renin from the donor [88]. It is still not clear whether transfer of small amounts of vasoactive substances could account for the renal changes seen in the recipient twin.

### Diagnosis

Diagnosis of TTTS is sometimes made earlier, by ultrasound scan, before the onset of symptoms of polyhydramnios or of preterm labor. Increased nuchal translucency (>95th percentile) in the recipient can be a forerunner of a full-blown TTTS in later gestations. Other ultrasound features include

- Folding of intertwin membrane can be seen at 16 weeks of gestation.
- Polyhydramnios in the recipient (maximal vertical pocket greater than 8 cm) and oligohydramnios in the donor (maximal vertical pocket of 2 cm or less; Figure 13.8). In severe oligohydramnios, the amniotic membrane is closely applied to the



FIGURE 13.8. *Ultrasound scan showing oligohydramnios in the recipient twin in TTTS.*



FIGURE 13.9. *Ultrasound scan showing oligohydramnios (stuck-twin appearance) in the donor twin in TTTS.*

fetus, which lies apposed to the uterine wall (*stuck twin*, Figure 13.9). An enlarged fetal bladder can be seen in the recipient, and the bladder can be barely visible in the donor twin.

- In severe cases, no end-diastolic or reversed end-diastolic flow in the umbilical artery of the donor, and reversed flow in the ductus venosus and pulsatile umbilical venous flow in the recipient can be seen.

Accordingly, TTTS is divided into five stages with escalating severity based on the ultrasound characteristics [89]; the staging forms the basis for the interventional management.

## STAGING OF TTTS

Stage I	Polyhydramnios/oligohydramnios. Donor bladder is visible.
Stage II	Polyhydramnios/oligohydramnios. Donor bladder not visible. Normal umbilical artery Doppler studies.
Stage III	Polyhydramnios/oligohydramnios. Donor bladder not visible. Abnormal Doppler studies of at least one of the following: 1) absent or reverse end-diastolic volume in the umbilical artery, 2) reverse flow in the ductus venosus, or 3) pulsatile umbilical venous flow.
Stage IV:	Hydrops in either twin.
Stage V:	Fetal demise of either twin.

Only one fourth of the TTTS fetuses exhibit a difference of more than 15% difference in the hematocrit levels [90], and fetal blood sampling is not required to make the diagnosis of TTTS. The hemodynamic changes lead to structural and functional alterations in the heart of the recipient twin. Ventricular hypertrophy predominates; echocardiographic changes and ventricular dilations are infrequently seen. The right heart is affected first, and with the progression of the disease, left ventricular hypertrophy can also be evident [91]. Biventricular diastolic dysfunction is seen in two thirds of recipients, and right ventricular systolic dysfunction and tricuspid regurgitation is seen in about one third of the recipients [92].

### **Management**

In the absence of intervention, most cases of TTTS are complicated by death or severe morbidity of one or both twins. Some cases resolve spontaneously with a favorable outcome, however. Treatment options include

#### 1. Serial amnioreduction.

*Amnioreduction* is performed by the introduction of an 18-gauge needle, under ultrasound guidance, into the polyhydramniotic sac. A large quantity of amniotic fluid can be drained by this method. The aim is to restore equilibrium in the fluid volume in the sacs, but the exact mechanism by which it improves the outcome is not clear.

Serial amnioreduction helps by reducing the chances of preterm delivery from polyhydramnios. Earlier studies reported survival rates varying from 37% to 83% [93,94]. These studies were limited by the small number of cases, recruitment at various gestational ages, and the technique employed. Mari and coworkers reported the results of amnioreduction on 223 twins with TTTS [95]. The procedure-related complication rate was 15%, with most cases complicated by premature rupture of membranes. The rate of overall perinatal survival to 4 weeks was 60%. The recipients had a slightly more favorable outcome than the donors (65% vs. 55%), which was attributed mainly to decreased intrauterine mortality of the recipients (18% vs. 26%). Of the surviving infants, about one fourth had abnormal cranial ultrasound scans at 4 weeks of age. There was no difference in the abnormal cranial scans between the donors and recipients. Although the long-term neurologic outcomes were not available, the severity is expected to be much less because infants having abnormal scans do not always have severe neurologic impairments. In another study, Mari and others found a cerebral palsy rate of 4.2% in the survivors of TTTS treated with serial amnioreduction [96]. Similarly, the Australian-New Zealand Twin-Twin Transfusion Registry reported an overall perinatal survival rate of 62.5% for the 112 pregnancies with TTTS treated with serial amnioreduction [97]. They also reported abnormal cranial ultrasound findings in 27.3% and periventricular leukomalacia in 10.8% of the survivors.

#### 2. Laser photocoagulation of placental vascular anastomoses.

Against the inexpensive and easily mastered skill of amnioreduction, laser photocoagulation requires expensive equipment and experienced personnel. The procedure is usually performed with the patient under sedation or anesthesia. An endoscope is introduced – avoiding the placenta – into the amniotic cavity. The anastomosing vessels are ablated using a laser (Nd:YAG). Selective photocoagulation, after mapping placental topography to ablate the arteriovenous communications, is more frequently used with improved outcome [98]. The Eurofetus trial randomized severe TTTS between 15 and 26 weeks for

selective laser photocoagulation or amniocentesis and showed a higher survival rate in the laser group of at least one twin to 28 days (76% versus 56%;  $p = 0.002$ ) [99]. The relative risk of death for both fetuses is 0.63% (95% CI, 0.25 to 0.93;  $p = 0.009$ ). The survival rate of at least one twin in this group was also higher at 6 months of age (76% vs. 51%;  $p = 0.002$ ). The laser group also had a later mean gestational age at delivery (33 vs. 29 weeks ( $p = 0.004$ )). In addition, a lower rate of neurologic morbidities, including cystic periventricular leukomalacia (6% versus 14%;  $p = 0.02$ ), was seen in the laser group. Criticism was expressed about the lower survival rate from amnioreduction group, which fared poorly in comparison with the previously published results [100–102]. Further studies are required to standardize the care, and long-term neurologic outcomes should be taken into account. In summary, the laser treatment seems to offer advantage at least in the short-term neurologic outcomes in these infants. The disadvantage of laser therapy is that it is available only in specialized centers, and to overcome the learning curve, clinicians must perform several procedures [103,104].

### 3. Septostomy.

*Septostomy* of the intertwin membrane is rarely performed; the goal is to create a communication between the sacs so that the amniotic fluid pressures can be equalized. In an international multicenter randomized trial of amnioreduction versus septostomy, Moise and coworkers [105], in their interim analysis, concluded that the survival rate of at least one infant in both groups is comparable (78% in the amnioreduction versus 80% in the septostomy group). Fewer procedures were required in the septostomy group. Criticism of this procedure rests on its assumption of unequal amniotic fluid pressures. Hartung and coworkers reported equally high pressures in the amniotic sacs in TTTS, and it is not clear how septostomy improves the outcome [106]. In addition, complications, including cord accidents and amniotic band syndrome, also have been reported [107].

### 4. Selective fetocide.

Selective fetocide involves the occlusion of the umbilical cord of the worse-affected twin to prevent exsanguination into the dead twin and placenta. Of several methods, cord coagulation with

bipolar cautery forceps is safer, but it requires experience and therefore is restricted to only a few centers.

## MONOAMNIOTIC TWINS

Monoamniotic twins are rare, occurring in only 1% of monozygotic twins [21]. In the absence of an intervening membrane, cord entanglement frequently occurs, and perinatal mortality rates ranging from 28% to 70% have been reported [108]. Prematurity contributes significantly to the increased perinatal mortality. Damaria and coworkers, in their review of 19 cases from a single institution, found an overall survival rate of 68% [109]. There were nine fetal deaths from five pregnancies, all occurring before 29 weeks. Roque and coworkers, in their Medline literature review of 133 cases of monoamniotic twins, found the perinatal losses to be constant at 2% to 4% between 15 and 32 weeks [110]. The perinatal mortality escalated to 11% and 22% between gestational ages 33 to 35 weeks and 36 to 38 weeks, respectively. Because cord accidents cannot be predicted by antenatal surveillance, the management of monoamniotic twins poses a challenge. Although there is no consensus for optimal delivery time, it seems reasonable to deliver at or about 32 weeks, after the administration of antenatal corticosteroids, to prevent the small increase in the perinatal mortality.

## PLACENTAL AND CORD COMPLICATIONS

Certain placental and umbilical cord abnormalities are more common in multiple gestations. Velamentous cord insertion occurs in 7% of twin pregnancies compared with 1% with singletons [111]. As a result, vasa previa, with its complication of fetal exsanguination, occurs more frequently. With malpresentations occurring more frequently in twins, cord presentation and prolapse are possible complications. A two-vessel cord is more frequently seen in twin gestations but is usually not related to other structural abnormalities.

## ANTEPARTUM CARE

Because of the increase in multiple gestations from ART, counseling should begin in the preconception period. Ideally, the couple should be seen by



maternal-fetal medicine (MFM) specialists before planning fertility treatment. HOM gestations and the possibility of fetal reduction should be discussed, so that the couple is better prepared to face these problems, which involve difficult ethical issues. Significant maternal and fetal complications can occur in multiple gestations, a situation that offers grounds for intensive prenatal care. Specific risks should be addressed at the outset, and the chief aim of prenatal care is to prevent preterm deliveries. Meyer and coworkers compared the clinical outcomes and financial costs of triplet gestations managed by MFM specialists with those managed by community physicians. The triplets born to women whose prenatal care was provided by the specialists weighed more at birth, and the incidence of extremely low birthweight ( $\leq 1,000$  g) was significantly less. The neonatal care costs were also significantly less ( $p = 0.01$ ).

#### **ANTEPARTUM VISITS AND NUTRITION**

Because most pregnant women seek prenatal care in the first trimester, this provides a great opportunity for directed counseling and planned prenatal care. Specifically, in multiple gestations, accurate determination of gestational age and chorionicity should be performed by ultrasound scan. Early prenatal visits are similar to those in singleton pregnancies. More frequent visits are planned after midgestation and tailored according to any problems identified. Iron and folate supplements should be given to match the increased requirements.

As mentioned previously, maternal weight gain in early pregnancy is essential to achieve normal birthweights for both infants. Ideally, trained nutritionists should provide counseling to achieve the desired goal.

#### **Ultrasound Evaluation**

Ultrasonography permits early diagnosis and dating, establishes chorionicity, and identifies congenital anomalies. Nuchal translucency (NT) screening and maternal blood screening can be performed between 11 and 13 weeks, and aneuploidy risks can be provided for each twin. Monitoring fetal growth by ultrasound scan is the standard of care now and is usually performed every 3 to 4 weeks. Monochorionic twins are monitored more frequently (every 2

to 3 weeks) to identify TTTS or selective growth restriction of one twin.

Giles and coworkers randomized twin pregnancies to be monitored by biometry with or without Doppler ultrasound scan of the fetal umbilical artery starting at 25 weeks' gestation [113]. They found no differences in the perinatal mortality rates between the no-Doppler group (11/1,000 live births) and Doppler group (9/1,000 live births). There were three fetal deaths in the Doppler group, which was not statistically significant.

Similarly, routine non-stress tests and fetal biophysical profiles are not indicated to assess fetal well-being. Close fetal surveillance is indicated in fetal growth restriction or severe discordance, and as with singleton gestations, in oligohydramnios or in maternal conditions such as diabetes and hypertension, or in any other high-risk conditions. The amniotic fluid should be assessed by the measurement of largest vertical pocket in each sac [114].

#### **Prenatal Screening**

In DZ twin gestation, risk for fetal aneuploidy is higher than in singleton pregnancies, because the greater fetal number increases the chances of at least one fetus being affected. This mathematical probability should be explained to the parents in genetic counseling. Prenatal screening for fetal aneuploidies includes NT measurement or serum screening in second trimester. The sensitivity of increased NT for Down syndrome in twins is the same as that for singletons [115]. It is also fetus specific, unlike maternal serum screening tests, and helps in selective invasive testing of the affected twin. The maternal serum markers used for aneuploidy screening are  $\alpha$ -fetoprotein, beta hCG, estriol, and inhibin. As would be expected, the mean MSAFP levels are almost doubled in twin pregnancies, and the adjusted multiples of median (MoM) are derived by using the twin cut-off levels of MSAFP. Similarly, MSAFP can be used to screen for open neural tube defects but with less accuracy.

Amniocentesis in a twin pregnancy is performed by two needle punctures, and there is no increased risk of miscarriage with this approach. Commonly, about 1 ml to 3 ml of indigo carmine dye is injected after sampling from one sac, and the subsequent aspiration of clear fluid from the other sac ensures that the operator has not sampled

from the same sac twice. Less commonly, some clinicians have used a single-puncture technique, advancing the needle through the intertwin membrane to sample the amniotic fluid from the other sac [116,117]. Difficulty in penetrating the intertwin membrane, potential contamination of the samples, and creating a pseudomonoamniotic sac are some of the reasons why this technique is not used that frequently. Chorionic villous sampling (CVS) in twin gestations depends on placental location; both transabdominal and transvaginal routes can be used.

### PREVENTION OF PRETERM BIRTHS

In the past, bedrest with hospitalization for twin pregnancies was freely advocated. A Cochrane review of the role of hospitalization and bedrest, however, showed that routine hospitalization for bedrest did not reduce the risk of preterm births or perinatal mortality in multiple gestations [118]. Actually, there was a significant increase in the preterm deliveries before 34 weeks' gestation (OR 1.84; 95% CI, 1.01–3.34). In addition, there were more low-birthweight infants born to women in the routinely hospitalized group (OR 1.93; 95% CI, 1.05–3.53).

As discussed previously, biochemical markers such as fetal fibronectin and cervical length measurements by transvaginal ultrasonography are frequently used in symptomatic patients for prediction of preterm birth; however, their value in asymptomatic twin gestations is not known. Prophylactic cervical cerclage has not conferred any advantage in twin or triplet gestations [119–121]. Recent study has shown that treatment with 17 alpha-hydroxyprogesterone treatment did not reduce the preterm birth rate in women with twin gestations [122].

### ANTENATAL CORTICOSTEROIDS AND TRIPLET GESTATIONS

Because the median age of delivery in triplet gestations is 33 weeks, some obstetricians prefer to give antenatal corticosteroids routinely to women carrying triplets. There are currently no recommendations for this approach, however. Blickstein and coworkers analyzed the incidence of respiratory

distress syndrome (RDS) from a cohort of 8,120 VLBW infants and found that antenatal corticosteroids reduced the incidence of RDS in all plurality groups compared with that in the partial or no treatment group [123]. In addition, antenatal corticosteroids reduced the incidence of grades III and IV intraventricular hemorrhage (IVH) in triplets in the complete and partial treatment group against that in the no-treatment group [124].

### MANAGEMENT OF PRETERM LABOR

Preterm labor is managed with tocolytic agents, as in singleton pregnancies. In the past, for acute tocolysis, magnesium sulfate was commonly used. Beta-sympathomimetic drugs like ritodrine have fallen out of favor because of cardiovascular complications in the mother. Indomethacin and calcium channel blockers can also be used. Terbutaline is occasionally administered for acute tocolysis. In multiple gestations, there is a significant risk for the development of pulmonary edema from tocolytic therapy. Pulmonary edema mainly occurs from volume overload rather than from tocolytic drugs. Patients receiving tocolytic therapy should be closely monitored, and pulmonary edema should be aggressively treated with diuretics and oxygen. After acute treatment, some clinicians have advocated maintenance tocolysis. Chronic treatment with beta-adrenergic drugs has been associated with a decrease in the number of uterine contractions, but not of preterm labor or delivery [125]. Elliott and colleagues reported very few side effects with continuous subcutaneous terbutaline infusions [126].

Antenatal corticosteroids should be given if preterm labor is diagnosed between 24 and 34 weeks of gestation. Repeat doses of steroids are not recommended [127].

### Preterm Rupture of Membranes

Management of preterm rupture of membranes (PROM) should be expectant, with prophylactic antibiotics and corticosteroids used to enhance lung maturity. Delivery should be considered at 34 to 35 weeks' gestation. Retrospective studies have shown that the latency period in twin gestations, especially after 30 weeks, was significantly shorter in twins compared with that in singletons. Perinatal and neonatal outcomes were similar [128,129].

### **Timing of Delivery**

The median gestational age at delivery in twins is around 35.2 weeks [1]. The perinatal mortality of twins reaches its lowest point at 37 to 38 weeks' gestation and increases slightly after that [130,131]. After 38 weeks, the perinatal mortality and cerebral palsy rates climb higher in twin gestations [7,132]. Whether in the absence of maternal and fetal complications, twin gestations should be electively delivered at 38 weeks is debatable, but delivery should be considered in the presence of maternal discomfort such as worsening dyspnea, difficulty in sleeping, painful varicose veins, and severe edema [133].

Assessment of fetal lung maturity is sometimes necessary if the gestational age is uncertain or if elective delivery is planned. Amniotic fluid assessment of lecithin/sphingomyelin (L/S) ratio or TDx fetal lung maturity assay (fluorescence polarization immunoassay) is commonly performed. Loveno and coworkers reported that an L/S ratio of 2:0 is reached earlier in twins (32 weeks vs. 36 weeks in singletons) [134]. McElrath and coworkers reported a higher TDx lung maturity values in twin gestations from 31 weeks onward compared with those of singletons [135]. Discordance in the amniotic value L/S ratio has also been reported [136]. It is reasonable to sample both gestational sacs unless access is difficult.

### **INTRAPARTUM MANAGEMENT**

Fetal presentations and weight, placental location, and the availability of experienced personnel influence the decision on the mode of delivery. In a study of 362 twin deliveries, Chevernak and coworkers found that vertex-vertex presentation occurs in 42.5%, vertex-nonvertex presentation occurs in 34.8%, and nonvertex-other presentation occurs in 19.1% of cases [137].

### **VERTEX-VERTEX PRESENTATIONS**

Successful vaginal delivery can be predicted in vertex-vertex deliveries, but counseling should take into account the possibility of cesarean section for the delivery of the second twin. Lack of adequate planning before vaginal delivery foretells disaster in some cases. An explanation about the number

of personnel involved in the delivery and care of the newborns can help to allay the fears of overwrought parents during labor. The pediatric team should include at least two experienced members well trained in the resuscitative efforts of the newborn. An anesthesiologist should be not only available but also present in the delivery suite. Delivery is usually undertaken in the operating suite so that a cesarean can be performed immediately if necessary. Ultrasound scan can help to monitor the presentations and fetal heart activity of the fetuses. Patients should have an intravenous line, and blood should be available for transfusion at short notice.

After the delivery of the first twin, the lie of the second fetus should be checked by ultrasound scan. Continuous fetal monitoring ensures fetal well-being. Vaginal examination is performed to confirm the engagement of head. Oxytocin infusion can be used if uterine contractions are not adequate, and the membranes are ruptured when the head is well engaged. If there are any maternal or fetal concerns, expedited delivery should occur.

### **INTERVAL BETWEEN DELIVERIES**

Traditional teaching stated that the second twin should be delivered within 15 minutes of the birth of the first twin. This is not supported by several studies, however. Rayburn and colleagues did not find any difference in the Apgar scores of second twins delivered later than 15 minutes after the first twin [138]. They noted an increase in the cesarean delivery associated with delay of more than 15 minutes, however. The umbilical cord gas values are not affected by route of delivery or by time interval [139]. Rydhstrom and Ingemarsson analyzed the data of 7,533 second twins from the Swedish Medical Birth Registry and found that the interdelivery interval did not influence the perinatal mortality of second twins [140]. Recently, in their study of 118 twin gestations over 34 weeks, Leung and coworkers reported a correlation with lower arterial pH values with increasing delivery intervals [141]. In summary, with continuous assessment of the fetal heart by electronic monitors and by ultrasound examination, the delivery interval delay seems to have little impact on the outcome of the second twin.

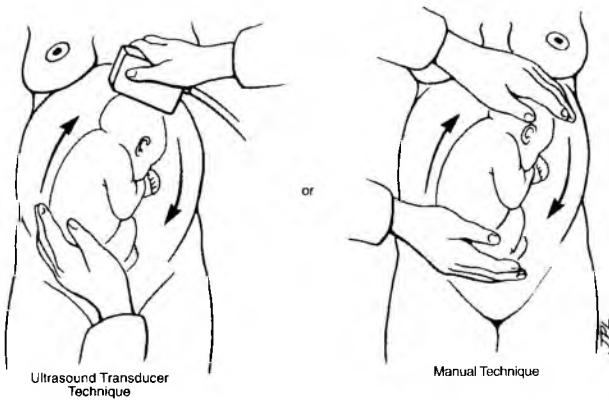


FIGURE 13.10.  
*Delivery of second nonvertex twin fetus; external version.*

### VERTEX-NONVERTEX PRESENTATIONS

In vertex-nonvertex presentations, after delivery of the first twin, the presentation of the second twin should be checked by ultrasound scan. Options for delivery then include 1) external cephalic version (ECV), 2) assisted breech delivery, and 3) breech extraction. Several studies have not found any difference in the neonatal outcomes of twins delivered by cesarean or vaginally [137,142]. Operator experience, and local practice patterns parental wish influence the decision concerning delivery mode.

ECV (Figure 13.10) can be accomplished easily in many instances, and vaginal delivery is successful most of these cases. Chervenak and coworkers reported a successful ECV in 73% of the cases and a successful vaginal delivery in 90% of the cases that had undergone ECV [143]. The safety of breech extraction of a second twin has been addressed, and infants weighing less than 1,500 g have a better neonatal outcome when delivered by a cesarean. Allowing for a 20% error in the estimation of fetal weight by ultrasound scan, one might wish to counsel vaginal delivery of the second twin (nonvertex) if the fetal weight is estimated at  $\geq 2000$  g [143]. After the delivery of the first twin, ultrasound scan should be performed to confirm the lie. Delivery should be expedited by breech extraction if footling breech presentation or transverse lie is seen. Breech extraction can be performed with or without ultrasound guidance and should be undertaken only by experienced operators.

*Assisted vaginal delivery* is also possible, but with a longer interval the cervix can reconstitute and pose

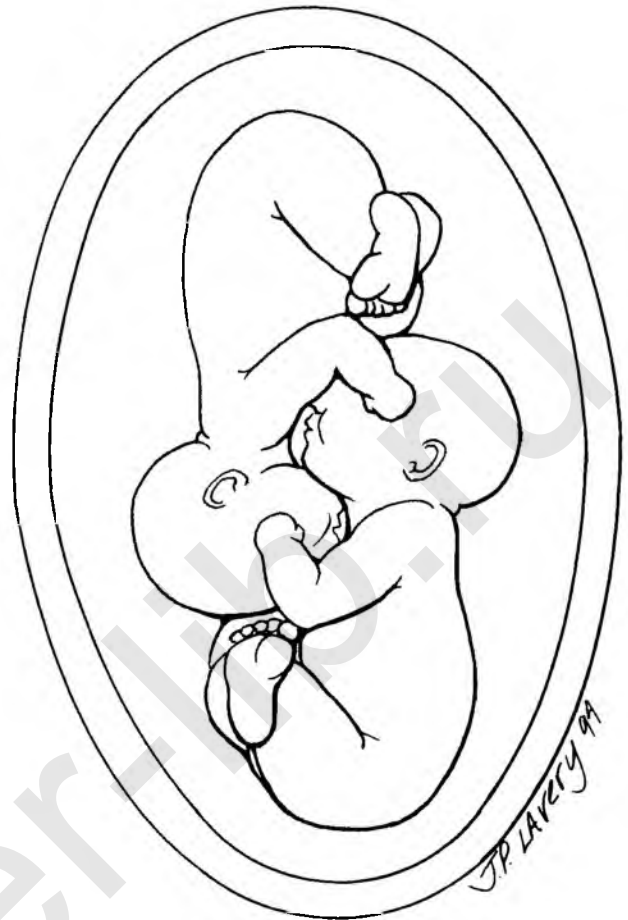


FIGURE 13.11.  
*Interlocking twins.*

challenges. Once the presenting part of the second twin is engaged, amniotomy followed by assisted breech delivery is performed. After delivery, the placenta should be examined for completeness, and the chorionicity should be confirmed by histologic examination.

### NONVERTEX-ANY PRESENTATION

If the first twin is not in a vertex presentation, it is customary to deliver by a cesarean. When twin A is in breech presentation and twin B in cephalic presentation, there is a possibility of interlocking of twins (Figure 13.11). This uncommon but potentially disastrous situation results from the entrapment of the aftercoming head of twin A below the chin of twin B, making it impossible to deliver twin A. The frequency of interlocking twins is

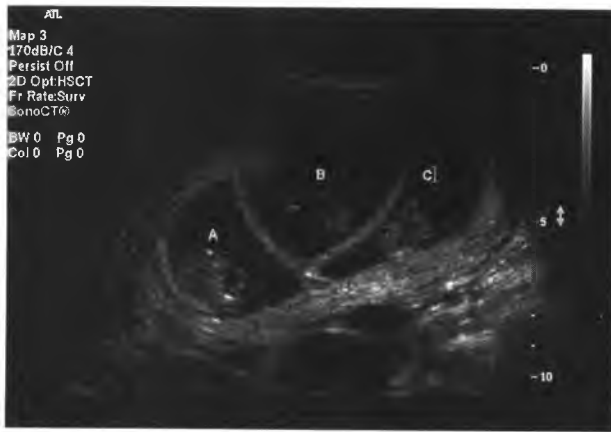


FIGURE 13.12.  
*Ultrasound scan showing early triplet gestation.*



FIGURE 13.13.  
*Ultrasound scan showing quadruplet gestation.*

approximately one per 1,000 twin deliveries and carries a fetal mortality rate of 31% [144].

### High-order Multiple Gestation

Triples or more constitute high-order multiple (HOM) gestations (Figures 13.12 and 13.13). As multiple births from ART continue, higher-order births are more frequently encountered. Multiple embryos are transferred in one cycle to improve the pregnancy rates, and the increase in HOMs is a natural outcome of such aggressive practice. In recent years, thanks to the guidelines issued by the Society for Assisted Reproductive Technology and American Society for Reproductive Medicine, there has been a declining trend in triplets or higher-order births [145,146]. Maternal complications are sim-

ilar to those in twin pregnancies but are increased manifold. HOMs also impose psychological stress, and parental counseling before fertility therapy is begun should be given. Preterm labor occurs in about 76% of triplets and in over 90% of quadruplet pregnancies [147].

### SELECTIVE REDUCTION

Although the survival rates of preterm infants have improved with the excellent neonatal care now available, the risk of delivering an extremely low-birthweight (ELBW;  $\leq 1,000$  g) infant is still very high, and this increases the long-term neurologic morbidity of these infants.

Multifetal pregnancy reduction (MFPR) decreases the fetal numbers with an aim to reduce the spontaneous losses and premature deliveries; this option is available to parents faced with HOM gestations. Typically performed in the first trimester, this procedure involves fetal intrathoracic injection of potassium chloride under ultrasound guidance.

Ideally, counseling should begin in the preconception period, and parents should be informed of the possible HOM births, the MFPR procedure, and its benefits and risks.

Prior to reduction, abnormal fetuses are identified and selectively reduced. Increased use of NT screening test helps to identify possible abnormal fetuses early in the first trimester. Alternatively, CVS can be performed, and karyotype of the fetus can be determined by fluorescent in situ hybridization (FISH) or by complete karyotype (results available in 1–2 weeks). Overall, experienced centers reported lower fetal loss rates with this procedure. Transvaginal and transcervical procedures are associated with greater fetal loss rates than are transabdominal procedures [148].

Although quadruplets are more usually reduced to twins, the reduction of triplets to twins or singletons is controversial. With improvements in the survival rates in triplet pregnancies, some practitioners are reluctant to perform reduction of triplets to twins. As the medical indications for reductions decrease, the burden of choice increasingly falls on the couple. The fetal loss rates (before 24 weeks) and the prematurity rates (delivery between 25 and 28 weeks) increased with higher starting and finishing numbers. In one multicenter study, 20%

fetal losses before 24 weeks were reported when sextuplets were reduced and a 6% loss rate when triplets were reduced to twins [148]. Spontaneous loss rates (before 24 weeks) were higher in unreduced triplets against those of triplets reduced to twins (25% vs. 6.3%,  $p = 0.07$ ) [149]. Boulot and coworkers reported comparable loss rates in both unreduced and reduced triplets (6% vs. 5.4%), but the rates of prematurity and low-birthweight infants in unreduced triplets were much higher. In HOM gestations, monochorionic twins are usually reduced to prevent complications.

In 2003, more than 50% of ART cycles using fresh nondonor embryos or eggs were performed on women over 35 years of age [151]. More women over 40 years of age are now seeking reduction to singletons from twins [152]. Reduction of natural twins to singletons poses ethical problems, and some have questioned its justifications [153]. Evans and others compared the outcome of reductions of 52 twins to singletons with the twin gestations from the national registries and reported 1.9% fetal loss rates from reductions and much higher rates of losses in the on-going twin gestations [153].

### **PSYCHOSOCIAL ASPECTS IN MULTIPLE GESTATIONS**

It is seldom that the prospective parents undergoing fertility treatment are fully aware of the problems of multiple gestations. After prolonged infertility, pregnancy transports them into a blissful state. Even after extensive preconception counseling, the couples are only marginally aware of the full implications of multiple births. Studies find that 20% to 40% of women undergoing IVF treatment actually consider multiple births as a preferred outcome [154,155]. A diagnosis of an HOM gestation poses fresh challenges when couples are confronted with the possibility of multifetal pregnancy reduction (MFPR).

Although mourning for the lost fetus was predominant in women undergoing MFPR, many were able to overcome their grief in 1 month. Frequent use of ultrasound monitoring was directly related to the emotional reactions to the procedure [156]. Detection of anomalies in one fetus, undergoing invasive procedures, and possible selective fetocide or pregnancy termination are all anxiety-provoking instances in women with multiple gestations. The

fetal death of one twin and its consequences on the surviving twin in a monochorionic pregnancy can be devastating. The grief is sometimes delayed by several days after birth. Preterm deliveries are much higher in multiple gestations, and the consequences of prematurity, particularly cerebral palsy, can be devastating. The emotional and financial burden in raising these children can strain the couple's relationship and has led to divorce in some cases. Couples undergoing fertility treatment should have adequate counseling by experienced providers so that they are better prepared to face any complications that might occur. Special problems of multiple pregnancies should be highlighted, and information should be provided about the support groups in the community.

### **MEDICOLEGAL ISSUES IN MULTIPLE GESTATIONS**

In the wake of ART and other fertility treatments, counseling begins in the preconception period. Even treatment with ovulation-inducing drugs like clomiphene is associated with multiple gestations, and failure to counsel the patient leads to liability. Physicians must be able to foresee these possible outcomes and counsel prospective parents accordingly.

Diagnosis of multiple gestations, establishing chorionicity, identifying anomalies, foreseeing possible maternal and fetal complications, prevention and treatment of preterm labor, and management of growth restriction are some of the areas of medicolegal concerns in multiple gestations. HOM gestations entail counseling and appropriate referral to experts for pregnancy reduction. Although many of these complications might not be prevented, explicit counseling helps couples to choose among the available options.

For example, if a discordant anomaly is detected, the couple should be informed of the available options, including selective fetocide. Appropriate prenatal screening tests should be offered, and if NT screening is available, its significance should be explained with reference to twin gestations. Appropriate invasive testing and sampling of both amniotic sacs without contamination are essential to prevent liability.

Monochorionic twins are more likely to have complications, and they should be monitored more

closely. They should be referred to maternal-fetal medicine specialists for frequent ultrasound monitoring and managed if any complications such as TTTS arises.

Another area of concern is preterm delivery. Although it cannot be prevented in most cases, screening tests like transvaginal ultrasonography for cervical length and fetal fibronectin are recommended in symptomatic patients. Although routine use of antenatal corticosteroids is not recommended in twin gestations, antenatal corticosteroids can be considered in triplet gestations since the available evidence supports its role in the prevention of respiratory distress syndrome and grade III/IV IVH in newborns.

Timing and mode of delivery of twins are other areas of concern. Most twin gestations deliver by 36 to 37 weeks. Recent reports indicate an increase in the cerebral palsy rates in twins born after 38 weeks (or for twins weighing over 2,500 g). Although induction is not routinely recommended at 38 weeks, the parents should be counseled appropriately about it. Vaginal delivery of twins in vertex-vertex presentations is recommended, but the couple should be aware of the possible surgical delivery of the second twin, with appropriate consent taken. In vertex-nonvertex presentations, counseling on assisted breech delivery or breech extraction should be done before delivery, and, if the provider is not experienced and if the couple wishes a vaginal delivery, the assistance of an experienced obstetrician should be obtained.

## REFERENCES

- Martin JA, Hamilton BE, Sutton PD, et al: Births: Final Data for 2003. National Vital Statistics Reports 2005;54(2).
- Reynolds MA, Schieve LA, Martin JA, et al: Trends in multiple births conceived using assisted reproductive technology, United States, 1997–2000. *Pediatrics* 2003;111:1159–1162.
- Centers for Disease Control and Prevention: Infant mortality and low birth weight among black and white infants: United States, 1980–2000. *MMWR Morb Mortal Wkly Rep* 2002;51:589–592.
- Alexander GR, Wingate MS, Salihu H, Kirby RS: Fetal and neonatal mortality risks of multiple births. *Obstet Gynecol Clin North Am* 2005;32:1–16.
- Rydhstroem H, Heraib F: Gestational duration, and fetal and infant mortality for twins vs. singletons. *Twin Res* 2001;4:227–231.
- Pharoah POD, Cooke T: Cerebral palsy and multiple births. *Arch Dis Child* 1996;75:F174–177.
- Scher AI, Petterson B, Blair E, et al: The risk of mortality or cerebral palsy in twins: A collaborative population-based study. *Pediatr Res* 2002;52:671–681.
- Blickstein I: Do multiple gestations raise the risk of cerebral palsy? *Clin Perinatol* 2004;31:395–408.
- Kohl SG, Casey G: Twin gestation. *Mt Sinai J Med* 1975;42:523–539.
- Imaizumi Y: Twinning rates in Japan, 1951–1990. *Act Genet Med Gemellol (Roma)* 1992;41:165–175.
- Fakeye O: Perinatal factors in twin mortality in Nigeria. *Int J Gynecol Obstet* 1986;24:309–314.
- Nylander PPS: Biosocial aspects of multiple births. *J Biosoc Sci* 1971;3:29–38.
- Heuser RL: Multiple Births: United States 1964. Washington, DC: National Center for Health Statistics, 1967;DHEW (series 21; No.14).
- MacGillivray I: Epidemiology of twin pregnancy. *Semin Perinatol* 1986;10:4–8.
- Schenker JG, Yarkone S, Granat M: Multiple pregnancies following induction of ovulation. *Fertil Steril* 1981;35:105–123.
- Schachter M, Raziel A, Friedler S, Strassburger D, Burn O, Ron-El R: Monozygotic twinning after assisted reproductive techniques: A phenomenon independent of micromanipulation. *Hum Reprod* 2001;16:1264–1269.
- Behr B, Fisch JD, Milki AA, et al: Blastocyst transfer is associated with an increased incidence of monozygotic twinning. The 15th Annual Meeting of the European Society of Human Reproduction and Embryology, Tours, France. *Hum Reprod* 14 1991 (Abstract Bk. 1), P-081, p. 181.
- Blickstein I, Verhoeven HC, Keith LG: Zygotic splitting after assisted reproduction. *N Engl J Med* 1999;340:738–739.
- Kaufman MH, O'Shea KS: Induction of monozygotic twinning in the mouse. *Nature* 1978;276:707–708.
- Bamforth F, Brown L, Senz J, Huntsman D: Mechanisms of monozygotic (MZ) twinning: A possible role for the cell adhesion molecule, E-cadherin. *Am J Med Genet* 2003;120A:59–62.

21. Benirschke K: Multiple gestation, incidence, etiology and inheritance. In Creasey RK, Resnik R (eds): *Maternal-Fetal Medicine: Principles and Practice*, 2nd ed. Philadelphia: W.B. Saunders, 1989; pp. 565–579.
22. Monteagudo A, Timor-Tritsch IE, Sharma S: Early and simple determination of chorionic and amniotic type in multiple gestation in the first 14 weeks by high frequency transvaginal ultrasonography. *Am J Obstet Gynecol* 1994;170:824–829.
23. Carroll SGM, Soothill PW, Sherif AA, Porter H, Montague I, Kyle PM: Prediction of chorionicity in twin pregnancies at 10–14 weeks of gestation. *Br J Obstet Gynecol* 2002;109:182–186.
24. Shetty A, Smith APM: The sonographic diagnosis of chorionicity. *Prenat Diagn* 2005;25:735–739.
25. Sebire NJ, Sniijders, RJ, Hughes, K, et al: The hidden mortality of monochorionic twin pregnancies. *Br J Obstet Gynaecol* 1997;104:1203.
26. Benirschke K, Kaufmann P: *Pathology of the Human Placenta*, 2nd ed. New York: Springer-Verlag, 1990; pp. 662–677.
27. Monga M: Maternal cardiovascular and renal adaptation to pregnancy. In Creasy RK, Resnik R (eds): *Maternal-Fetal Medicine: Principles and Practice*, 5th ed. Philadelphia: W.B. Saunders, 2004; pp. 111–120.
28. Pritchard JA: Changes in blood volume during pregnancy and delivery. *Anesthesiology* 1965;23:393–399.
29. Veille JC, Morton MJ, Burry KJ: Maternal cardiovascular adaptations to twin pregnancy. *Am J Obstet Gynecol* 1985;153:261–263.
30. Leroy B, Lefort F, Jenny R: Uterine heights and umbilical perimeter curves in twin pregnancies. *Acta Genet Med Gemellol (Roma)* 1982;31:195–198.
31. Franchini M: Hemostasis and pregnancy. *Thromb Haemostat* 2006;95:401–413.
32. MacGillivray I: Twin pregnancies. *Obstet Gynecol Annu* 1978;7:135–151.
33. Davidson KM, Simpson LL, Knox TA, D'Alton ME: Acute fatty liver of pregnancy in triplet gestation. *Obstet Gynecol* 1998;91:806–808.
34. Casele HL, Dooley SL, Metzger BE: Metabolic response to meal eating and extended overnight fast in twin gestation. *Am J Obstet Gynecol* 1996; 175:917–921.
35. Schwartz DB, Daoud MA, Zazula P, et al: Gestational diabetes mellitus: Metabolic and blood glucose parameters in singleton versus twin pregnancies. *Am J Obstet Gynecol* 1999;181:912–914.
36. Redford DHA, Whitfield CR: Maternal serum alpha-fetoprotein in twin pregnancies uncomplicated by neural tube defects. *Am J Obstet Gynecol* 1985;152:550–553.
37. Spinillo A, Capuzzo E, Piazzzi G, et al: Risk of spontaneous preterm delivery by combined body mass index and gestational weight gain patterns. *Acta Obstet Gynecol Scand* 1998;77:32–36.
38. Luke B, Minogue J, Witter FR, et al: The ideal twin pregnancy: Patterns of weight gain, discordancy, and length of gestation. *Am J Obstet Gynecol* 1993; 169:588–597.
39. Institute of Medicine: *Nutrition during Pregnancy*. Washington, DC: National Academy Press, 1990.
40. Luke B: Nutrition in Multiple Gestations. *Clin Perinatol* 2005;32:403–429.
41. Luke B: Nutrition and multiple gestation. *Semin Perinatol* 2005;29:349–354.
42. Martin J, Hamilton B, Ventura S, et al: *Births: Final Data for 2001*. National Center for Health Statistics Reports, 2002; p. 51.
43. Sibai BM, Hauth J, Caritis S, et al: Hypertensive disorders in twin versus singleton gestations. *Am J Obstet Gynecol* 2000;182:938–942.
44. Heyborne KD, Porreco RP: Selective fetocide reverses preeclampsia in discordant twins. *Am J Obstet Gynecol* 2004;191:477–480.
45. Ananth C, Demissie K, Smulien J, Vintzeleos AM: Placenta previa in singleton and twin births in the United States, 1989 through 1998: A comparison of risk factor profiles and associated conditions. *Am J Obstet Gynecol* 2003;188:275–281.
46. Ananth C, Smulien J, Vintzeleos AM, Knuppel R: Placental abruption among singleton and twin births in the United States: Risk factor profiles. *Am J Epidemiol* 2001;153:771–778.
47. Guttmacher AF: An analysis of 573 cases of twin pregnancy. II. The hazards of pregnancy itself. *Am J Obstet Gynecol* 1939;38:277–288.
48. Chervenak FA, Youcha S, Johnson RE, Berkowitz RL, Hobbins JC: Twin gestation: Antenatal diagnosis and perinatal outcome in 385 consecutive pregnancies. *J Reprod Med* 1984;29:727–730.
49. National Center for Health Statistics: Final natality data. [www.marchofdimes.com/peristats](http://www.marchofdimes.com/peristats).
50. Jacquemyn Y, Martens G, Ruyssinck G, et al: A matched cohort comparison of the outcome of twin



- versus singleton pregnancies in Flanders, Belgium. *Twin Res* 2003;6:7–11.
51. Ballabh P, Kumari J, Al-Kouatly HB, et al: Neonatal outcome of triplet versus twin and singleton pregnancies: A matched case-control study. *Eur J Obstet Gynecol Reprod Biol* 2003;107:28–36.
  52. Dyson D, Danbe K, Bamber J, et al: A multicentre randomised trial of three levels of surveillance in patients at risk for preterm labour – twin gestation subgroup analysis. *Am J Obstet Gynecol* 1997;176: S118.
  53. Goldenberg R, Iams J, Miodovnik M, et al: The preterm prediction study: Risk factors in twin gestations. *Am J Obstet Gynecol* 1996;175:1047–1053.
  54. Terrone DA, Rinehart BK, Kraeden U, Morrison JC: Fetal fibronectin in symptomatic twin gestations. *Prim Care Update Ob Gyn* 1998;62:135–139.
  55. Souka AP, Heath V, Flint S, et al: Cervical length at 23 weeks in twins in predicting spontaneous preterm delivery. *Obstet Gynecol* 1999;94:450–454.
  56. Mueller-Heubach E: Complications of multiple gestation. *Clin Obstet Gynecol* 1984;27:1003–1013.
  57. Dickey RP, Taylor SN, Lu PY, et al: Spontaneous reduction of multiple pregnancy: Incidence and effect on outcome. *Am J Obstet Gynecol* 2002;186:77–83.
  58. Dickey RP, Olar TT, Curole DN, et al: The probability of multiple births when multiple gestational sacs or viable embryos are diagnosed at first-trimester ultrasound. *Hum Reprod* 1990;5: 880–882.
  59. Blickstein I: Growth aberration in multiple pregnancy. *Obstet Gynecol North Am* 2005;32:39–54.
  60. Blickstein I, Kalish RB: Birth weight discordance in multiple pregnancy. *Twin Res* 2003;6:526–531.
  61. Blickstein I, Keith LG: Neonatal mortality rates among growth discordant twins, classified according to the birth weight of the smaller twin. *Am J Obstet Gynecol* 2004;190:170–174.
  62. Reece EA, Yarkoni S, Abdalla M, et al: A prospective study of growth in twin gestations compared with growth in singleton pregnancies. I. The fetal head. *J Ultrasound Med* 1991;10:439.
  63. Graham D, Shah Y, Moodley S, et al: Biparietal diameter femur length growth in normal twin pregnancies [abstract 112]. Society of Perinatal Obstetricians, Annual meeting, February, 1984.
  64. Dudley DK, D'Alton ME: Single fetal death in twin gestation. *Semin Perinatol* 1986;10:65–72.
  65. Hanna JH, Hill JM: Single intrauterine fetal demise in multiple gestation. *Obstet Gynecol* 1984;63: 126–130.
  66. Carlson NJ, Towers CV: Multiple gestation complicated by the death of one fetus. *Obstet Gynecol* 1989;73:685–689.
  67. Johnson CD, Zhang J: Survival of other fetuses after a fetal death in twin or triplet pregnancies. *Obstet Gynecol* 2002;99:698–703.
  68. D'Alton ME, Simpson LL: Syndromes in twins. *Semin Perinatol* 1995;19:375–386.
  69. D'Alton ME, Newton ER, Cetrulo CL: Intrauterine fetal demise in multiple gestation. *Acta Genet Med Gemellol (Roma)* 1984;33:43–49.
  70. Dudley DK, D'Alton ME: Single fetal death in twin gestation. *Semin Perinatol* 1986;10:65–72.
  71. Pharaoh POD, Adi Y: Consequences of in-utero death in a twin pregnancy. *Lancet* 2000;355:1597–1602.
  72. Pomplar HJ, Madjar H, Klosa W, et al: Twin pregnancies with single fetal death. *Acta Obstet Gynecol Scand* 1994;73:205–208.
  73. Fusi L, Gordon H: Twin pregnancy complicated by single intrauterine death: Problems and outcome with conservative management. *Br J Obstet Gynaecol* 1990;97:511–516.
  74. Onyskowova A, Dolezal A, Jedlicka V: The frequency and the character of malformations in multiple births. *Acta Universitatis Carolinae* 1970;16: 333–376.
  75. Hall JG: Twinning. *Lancet* 2003;362:735–743.
  76. Mohammed SN, Swan MC, Wall SA, Wilkie AOM: Monozygotic twins discordant for frontonasal malformation. *Am J Med Genet* 2004;130:384–388.
  77. Rodis JF, Egan JF, Craffey A, Ciarleglio L, Greenstein RM, Scorza WE: Calculated risk of chromosomal abnormalities in twin gestations. *Obstet Gynecol* 1990;76:1037–1041.
  78. Gilbert B, Yardin C, Briault S, et al: Prenatal diagnosis of female monozygotic twins discordant for Turner syndrome: Implication for prenatal genetic counseling. *Prenat Diagn* 2002;22:697–702.
  79. Jenkins TM, Wapner RJ: The challenge of prenatal diagnosis in twin pregnancies. *Curr Opin Obstet Gynecol* 2000;12:87–92.
  80. Evans MI, MD, Goldberg JD, MD, Horenstein J, et al: Selective termination for structural, chromosomal, and Mendelian anomalies: International experience. *Am J Obstet Gynecol* 1999;181:893–897.

81. Nicolini U, Poblete A, Boschetto C, Bonati F, Roberts A: Complicated monochorionic twin pregnancies: Experience with bipolar cord coagulation. *Am J Obstet Gynecol* 2001;185:703–707.
82. Challis D, Gratacos E, Deprest JA: Cord occlusion techniques for selective termination in monochorionic twins. *J Perinat Med* 1999;27:327–338.
83. Deprest JA, Van Ballaer PP, Evrard VA, et al: Experience with fetoscopic cord ligation. *Eur J Obstet Gynecol Reprod Biol* 1998;81:157–164.
84. James WH: A note on the epidemiology of acardiac monsters. *Teratology* 1977;16:211–216.
85. Wong AE, Sepulveda W: Acardiac anomaly: Current issues in prenatal assessment and treatment. *Prenat Diagn* 2005;25:796–806.
86. Bajoria R, Wigglesworth J, Fisk NM: Angioarchitecture of monochorionic placentas in relation to the twin-twin transfusion syndrome. *Am J Obstet Gynecol* 1995;172:856–863.
87. Denboow M, Cox P, Taylor M et al: Placental angioarchitecture in monochorionic twin pregnancies: Relationship to fetal growth, fetofetal transfusion syndrome, and pregnancy outcome. *Am J Obstet Gynecol* 2000;182:417–426.
88. Mahieu-Caputo D, Dommergues M, Delezoide A, et al: Twin-to-twin transfusion syndrome: Role of the fetal renin-angiotensin system. *Am J Pathol* 2000;156:629–636.
89. Quintero RA, Morales WJ, Allen MH, et al: Staging of twin-twin transfusion syndrome. *J Perinatol* 1999;19:550–555.
90. Denbow M, Foglian R, Kyle P, et al: Hematological indices at fetal blood sampling in monochorionic pregnancies complicated by fetofetal transfusion syndrome. *Prenat Diagn* 1998;18:941–946.
91. Rychik J: Fetal cardiovascular physiology. *Pediatr Cardiol* 2004;25:201–209.
92. Barrea C, Alkazaleh F, Greg G, et al: Prenatal cardiovascular manifestations in the twin-to-twin transfusion syndrome recipients and the impact of therapeutic amnioreduction. *Am J Obstet Gynecol* 2005;192:892–902.
93. Saunders NJ, Sniijders RJM, Nicolaides KH: Therapeutic amniocentesis in twin-twin transfusion syndrome appearing in the second trimester of pregnancy. *Am J Obstet Gynecol* 1992;166:820–824.
94. Pinette MG, Yuqun P, Pinette SG: Treatment of twin-twin transfusion syndrome. *Obstet Gynecol* 1993;82:841–846.
95. Mari G, Roberts A, Detti L, et al: Perinatal morbidity and mortality rates in severe twin-twin transfusion syndrome: Results of the International Amnioreduction Registry. *Am J Obstet Gynecol* 2001;185:708–715.
96. Mari G, Detti L, Oz U, Abuhamad AZ: Long-term outcome in twin-twin transfusion syndrome treated with serial aggressive amnioreduction. *Am J Obstet Gynecol* 2000;183:211–217.
97. Dickinson JE, Evans SF: Obstetric and perinatal outcomes from The Australian and New Zealand Twin-Twin Transfusion Syndrome Registry. *Am J Obstet Gynecol* 2000;182:706–712.
98. Quintero R, Morales W, Mendoza G, et al: Selective photocoagulation of placental vessels I twin-twin transfusion syndrome: Evolution of a surgical technique. *Obstet Gynecol Surv* 1998;53:s97–103.
99. Senat MV, Deprest J, Boulvain M, et al: Endoscopic laser surgery versus serial amnioreduction for severe twin-to-twin transfusion syndrome. *N Engl J Med* 2004;351:136–144.
100. Fisk NM, Galea P: Twin-twin transfusion – as good as it gets? *N Engl J Med* 2004;351:182–184.
101. Quintero RA, Dickinson JE, Morales WJ, et al: Stage-based treatment of twin-twin transfusion syndrome. *Am J Obstet Gynecol* 2003;188:1333–1340.
102. Saade G, Moise K, Dorman K, et al: A randomized trial of septostomy versus amnioreduction in the treatment of twin oligohydramnios polyhydramnios sequence (TOPS). *Am J Obstet Gynecol* 2002;187:S54.
103. De Lia JE, Kuhlmann RS, Lopez KP: Treating previable twin-twin transfusion syndrome with fetoscopic laser surgery: Outcomes following the learning curve. *J Perinat Med* 1999;27:61–67.
104. Hecher K, Diehl W, Zikulnig L, Vetter M, Hackloer BJ: Endoscopic laser coagulation of placental anastomoses in 200 pregnancies with severe mid-trimester twin-to-twin transfusion syndrome. *Eur J Obstet Gynecol Reprod Biol* 2003;92:135–139.
105. Moise JM Jr, Dorman K, Lamvu G, et al: A randomized trial of amnioreduction versus septostomy in the treatment of twin-twin transfusion syndrome. *Am J Obstet Gynecol* 2005;193:701–707.
106. Hartung J, Chaoui R, Bollman R: Amniotic fluid pressure in both cavities of twin-to-twin transfusion syndrome: A vote against septostomy. *Fetal Diagn Ther* 2000;15:79–82.

107. Cook TL, Shaugnessy R: Iatrogenic creation of a monoamniotic twin gestation in severe twin-to-twin transfusion syndrome. *J Ultrasound Med* 1997;16:853–855.
108. Allen VM, Windrim R, Barrett J, Ohlsson A: Management of monoamniotic twin pregnancies: A case series and systematic review of the literature. *Br J Obstet Gynaecol* 2001;108:931–936.
109. Demaria F, Goffinet F, Kayem G et al: Monoamniotic twin pregnancies: Antenatal management and perinatal results of 19 consecutive cases. *Br J Obstet Gynaecol* 2004;111:22–26.
110. Roque H, Gillen-Goldstein, Funai E, et al: Perinatal outcomes in monoamniotic twin gestations. *J Matern Fetal Neonat Med* 2003;13:414–421.
111. Benirschke K, Kim CK: Multiple pregnancy: 1. *N Engl J Med* 1973;288:1276–1284.
112. Meyer B, Elimian A, Royek A: Comparison of clinical and financial outcomes of triplet gestations managed by maternal-fetal medicine versus community physicians. *Am J Obstet Gynecol* 2001;184:S102.
113. Giles W, Bisits A, O'Callaghan S, et al: The Doppler Assessment in multiple pregnancy randomized controlled trial of ultrasound biometry versus umbilical artery Doppler ultrasound and biometry in twin pregnancy. *Br J Obstet Gynaecol* 2003;110:593–597.
114. Megann E, Chauhan S, Whitworth N, et al: Determination of amniotic fluid volume in twin pregnancies: Ultrasonographic evaluation versus operator estimation. *Am J Obstet Gynecol* 2000;182:1606–1609.
115. Pandya PP, Snijders RJM, Johnson SJ, de Lourdes, Brizot MJ, Nicolaides KH: Nuchal translucency thickness, crown-rump length in twin pregnancies with chromosomally abnormal fetuses. *J Ultrasound Med* 1995;14:565–568.
116. Jeanty P, Shah D, Roussis P: Single-needle insertion in twin amniocentesis. *J Ultrasound Med* 1990;9:511–517.
117. Buscaglia M, Ghisoni L, Bellotti M, et al: Genetic amniocentesis in biamniotic twin pregnancies by a single transabdominal insertion of the needle. *Prenat Diagn* 1995;15:17–19.
118. Crowther CA: Hospitalization and bed rest for multiple pregnancy. *Cochrane Database of Systematic Reviews* 2001;1:CD000110.
119. Newman R, Krombach R, Myers M, et al: Effect of cerclage on obstetrical outcome in twin gestations with a shortened cervical length. *Am J Obstet Gynecol* 2002;186:634–640.
120. Grant A: Cervical cerclage to prolong pregnancy. In Chalmers I, Enkin M, Keirse M (eds.): *Effective Care in Pregnancy and Childbirth*. Oxford: Oxford University Press, 1989, pp. 633–645.
121. Bernasko J, Lee Robert, Pagano M, Kohn N: Is routine prophylactic cervical cerclage associated with significant prolongation of triplet gestation? *J Matern Fetal Neonatal Med* 2006;19:575–578.
122. Rouse DJ, Caritis SN, Peaceman AM, et al: A trial of 17 alpha-hydroxyprogesterone caproate to prevent prematurity in twins. *N Engl J Med* 2007;357:454–461.
123. Blickstein I, Shinwell ES, Lusk A, et al: Plurality-dependent risk of respiratory distress syndrome among very-low-birth-weight infants and antepartum corticosteroid treatment. *Am J Obstet Gynecol* 2005;192:360–364.
124. Blickstein I, Reichman B, Lusk A, et al: Plurality-dependent risk of severe intraventricular hemorrhage among very-low-birth-weight infants and antepartum corticosteroid treatment. *Am J Obstet Gynecol* 2006;194:1329–1333.
125. Caughey AB, Parer JT: Tocolysis with beta-adrenergic receptor agonists. *Semin Perinatol* 2001;25(4):248–255.
126. Elliott J, Istwan N, Rhea D, Stanziano G: The occurrence of adverse events in women receiving continuous subcutaneous terbutaline therapy. *Am J Obstet Gynecol* 2004;191:1277–1282.
127. Antenatal Corticosteroids Revisited: Repeat Courses – National Institutes of Health Consensus Development Conference Statement, August 17–18, 2000. *Obstet Gynecol* 2001;98:144–150.
128. Bianco AT, Lapinski R, Lockwood C, Lynch L, Berkowitz RL: The clinical outcome of preterm premature rupture of membranes in twin versus singleton pregnancies. *Am J Perinatol* 1996;13(3):135–138.
129. Hsieh YY, Chang CC, Tsai HD, Lee CC, Tsai CH: Twin vs. singleton pregnancy: Clinical characteristics and latency periods in preterm premature rupture of membranes. *J Reprod Med* 1999;44(7):616–620.
130. Cheung YB, Yip P, Karlberg J: Mortality of twins and singletons by gestational age: A varying-coefficient approach. *Am J Epidemiol* 2000;152:1107–1116.

131. Hartley RS, Emanuel I, Hitti J: Perinatal mortality and neonatal morbidity rates among twin pairs at different gestational ages: Optimal delivery timing at 37 to 38 weeks' gestation. *Am J Obstet Gynecol* 2001;184:451-458.
132. Sairam S, Costeloe K, Thilaganathan B: Prospective risk of stillbirth in multiple-gestation pregnancies: A population-based analysis. *Obstet Gynecol* 2002; 100:638-641.
133. Multiple gestation: Complicated twin, triplet, and high-order multifetal pregnancy. *ACOG Practice Bulletin* 2004;56:1-15.
134. Leveno KJ, Quirk JG, Whalley PJ, Herbert WM, Trubey R: Fetal lung maturation in twin gestation. *Am J Obstet Gynecol* 1984;148:405-411.
135. McElrath TF, Norwitz ER, Robinson JM, et al: Differences in TDx fetal lung maturity assay values between twin and singleton gestations. *Am J Obstet Gynecol* 2000;182:110-112.
136. Whitworth NS, Magann EF, Morrison JC: Evaluation of fetal lung maturity in diamniotic twins. *Am J Obstet Gynecol* 1999;180:1438-1441.
137. Chervenak FA, Johnson RE, Youcha S, Hobbins JC, Berkowitz RL: Intrapartum management of twin gestation. *Obstet Gynecol* 1985;65:119-124.
138. Rayburn WF, Lavin JP, Midovnik M Jr, Varner MW: Multiple gestation: Time interval between delivery of the first and second twins. *Obstet Gynecol* 1984;63(4):502-506.
139. Brown HL, Miller JM Jr, Neumann DE, et al: Umbilical cord blood gas assessment of twins. *Obstet Gynecol* 1990;75(5):826-829.
140. Rydhstrom H, Ingemarsson I: Interval between birth of the first and the second twin and its impact on second twin perinatal mortality. *J Perinat Med* 1990;18(6):449-453.
141. Leung TY, Lok IH, Tam WH, et al: Deterioration in cord blood gas status during the second stage of labour is more rapid in the second twin than the first twin. *Br J Obstet Gynaecol* 2002;109:63-67.
142. Wells SR, Thorp JM Jr, Bowes WA Jr: Management of the nonvertex second twin. *Surg Gynecol Obstet* 1991;172:383.
143. Chervenak FA, Johnson RE, Berkowitz RL, et al: Intrapartum external version of the second twin. *Obstet Gynecol* 1983;62:160-165.
144. Acker D, Leiberman M, Holbrook H, James O, Phillipe M, Edelin KC: Delivery of the second twin. *Obstet Gynecol* 1982;59:710-711.
145. American Society for Reproductive Medicine: Guidelines on Number of Embryos Transferred. Birmingham, AL, American Society for Reproductive Medicine, 1998.
146. The Practice Committee of the Society for Assisted Reproductive Technology and the American Society for Reproductive Medicine: Guidelines on the number of embryos transferred. *Fertile Steril* 2004; 82(Suppl 1):S1-S2.
147. Evans MI, Ciorica D, Britt DW, Fletcher JC: Update on selective reduction. *Prenat Diagn* 2005;25:807-813.
148. Evans MI, Berkowitz RL, Wapner RJ, et al: Improvement in outcomes of multifetal pregnancy reduction with increased experience. *Am J Obstet Gynecol* 2001;184:97-103.
149. Yaron Y, Bryant-Greenwood P, Dave N, et al: Multifetal pregnancy reductions of triplets to twins: Comparison with nonreduced triplets and twins. *Am J Obstet Gynecol* 1999;180:1268-1271.
150. Boulot P, Vignal J, Vergnes C, et al: Multifetal reduction of triplets to twins: A prospective comparison of pregnancy outcome. *Hum Reprod* 2000; 15:1619-1623.
151. Assisted Reproductive Technology Success Rates: National Summary of Fertility Clinic Reports. Atlanta, Centers for Disease Control and Prevention, December 2005.
152. Evans MI, Kaufman MI, Urban AJ, et al: Fetal reduction from twins to a singleton: A reasonable consideration? *Obstet Gynecol* 2004;104:102-109.
153. Rorty MV, Pinkerton JV: Elective fetal reduction: The ultimate elective surgery. *J Contemp Health Law Policy* 1996;13:53-77.
154. Ryan GL, Zhang SH, Dokras A, et al: The desire of infertile patients for multiple births. *Fertil Steril* 2004;81(3):500-504.
155. Child TJ, Henderson AM, Tan SL: The desire for multiple pregnancy in male and female infertility patients. *Hum Reprod* 2004;19(3):558-561.
156. Schreiner-Engel P, Walther VN, Mindes J, et al: First-trimester multifetal pregnancy reduction: Acute and persistent psychologic reactions. *Am J Obstet Gynecol* 1995;172:541-547.

James J. Nocon

*To find a fault is easy; to do better may be difficult.*

Plutarch (46–120 CE)  
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*Shoulder dystocia* is a well-known and much-feared obstetric emergency. It is not hard to imagine the thoughts in the doctor's mind when a baby's head delivers and the shoulder remains impacted. No matter what is done thereafter, at least some newborns have an observable injury, which can include a brachial plexus injury, fractures of the clavicle or humerus, neonatal asphyxia, and even death. Fortunately, most such neonatal injuries are transitory. Maternal consequences can involve vaginal or cervical lacerations, uterine atony, and postpartum hemorrhage. Thus, every clinician who provides obstetric care is expected to be able to manage a shoulder dystocia.

A brief review of the approach to shoulder dystocia reveals that the onus of responsibility for the outcome has been placed squarely on the shoulders of the doctor (pun intended). Researchers have identified several "risk factors" associated with shoulder dystocia. One theory asserts that if doctors were able to identify the patient "at risk," then they could take some action or intervention to prevent the risk from occurring. Clinicians have also devised maneuvers to "safely" dislodge the stuck shoulder, even asserting that some of these maneuvers are superior to others or that such maneuvers should be attempted in a specific sequence to be successful. Finally, the theory of the mechanism of the most common injury, a *brachial plexus injury*, postulates that excessive downward traction on the baby's head and neck stretches the brachial plexus and thereby causes injury.

The best evidence of the last 25 years regarding this traditional approach to shoulder dystocia, and especially brachial plexus injury, indicates that most if not all of the former presumptions are incorrect, inconsistent, and incomplete. In 1987, Gross and coworkers reported that even if a risk factor were of statistical significance, it had little to no predictive value [1]. Nocon and coworkers confirmed that the traditional risk factors for shoulder dystocia had no predictive value and also that no single maneuver or sequence of maneuvers was superior to any other in preventing brachial plexus injury [2]. Finally, researchers have demonstrated that when shoulder

dystocia occurred with two different 4,700-g babies and traction exerted on each baby's head was four times greater than in a normal delivery, there was no permanent injury [3]. This study and others indicate that excessive downward traction cannot be the only cause of brachial plexus injury.

Certain findings are clear. Shoulder dystocia remains an unpredictable event. Regardless of the approach used to dislodge the shoulder, up to 32% of all such babies will have some observable injury [4]. Fortunately, over 90% of those injured are transitory and thereby not permanent. This creates a problem in physician accountability; that is, if the occurrence is not predictable and the choice of management yields similar rates of poor outcome, then there is little basis to subject the physician to a fault-based system of liability. This chapter reviews and examines the best evidence available about the nature and scope of shoulder dystocia, including reasonable management options and the challenging ethical and legal aspects surrounding this common obstetric emergency.

The author performed an extensive analysis of the occurrence of shoulder dystocia and neonatal injury from the records of 14,297 parturients with 12,532 vaginal and 1,765 cesarean deliveries (12.4%) at the Wishard Memorial Hospital, from January 1986 through June 1990 [2]. Briefly, Wishard Memorial Hospital is the county hospital for Indianapolis and a major teaching center for the Indiana University Medical School. Resident physicians under direct faculty supervision render all care. Between 1986 and 1990, the hospital had the following patient characteristics: 55% African American, 45% Caucasian, and 95% on Medicaid. The author refers to the Wishard Memorial Hospital (Wishard) study in subsequent sections for comparison and contrast with other reported data.

## **CLINICAL ISSUES**

### **Prevalence of Shoulder Dystocia**

The definition of shoulder dystocia categorically affects its prevalence, but a functional definition includes any difficulty in extracting the shoulders after delivery of the head [5]. This view might be overly broad and might lead to a higher incidence of reported cases with a lower rate of complications. A more specific definition indicates that "true" dys-

tocia requires maneuvers to deliver the shoulders, combined with gentle downward traction and episiotomy [6]. This view might be too narrow in scope and skew the incidence downward, however.

Although the actual prevalence is unclear, shoulder dystocia does appear to be increasing, presumably because of increasing birthweight [7]. Other reasons for this rise include increasing maternal age, obesity, improved prenatal care, and fewer factors leading to preterm delivery. Most important, there is an increase in the reporting of shoulder dystocia as the need for greater documentation of obstetric care has been emphasized.

The reported incidence of shoulder dystocia varies from 0.6% to 1.4% of all vaginal deliveries [8]. Some authors report only the frequency among vaginal deliveries, whereas others include frequency for all births, including cesarean deliveries. Some include all birthweights, whereas others exclude those newborns less than 2,500 g. Some exclude deliveries that require only mild traction and no special maneuvers. Finally, the degree of documentation can vary from institution to institution, and even year to year within the same institution as importance of the diagnosis is emphasized.

### **Morbidity and Mortality**

It is well documented that perinatal morbidity and mortality rates are increased in shoulder dystocia. Boyd and coworkers noted severe asphyxia in 143 per 1,000 births associated with shoulder dystocia in contrast to 14 per 1,000 births in the general population [9]. Although some neonatal morbidity is readily apparent in about 20% of newborns with shoulder dystocia, most infants with shoulder dystocia experience no significant injury. Investigators at Parkland Hospital in Dallas, Texas, report brachial plexus injuries in 4 of 737 infants delivered vaginally weighing 4,000 g to 4,500 g and in 4 of 118 infants weighing more than 4,500 g [10]. Of note, the Parkland group reports that 99.5% of infants weighing 4,000 g to 4,500 g had a safe vaginal delivery.

### **Neonatal Injury**

The range of injuries to the newborn following a shoulder dystocia typically include trauma to the brachial plexus or phrenic nerve, fractures of the clavicle or humerus, neonatal asphyxia, and even

death. Clavicular fractures are commonly associated with shoulder dystocia but also occur frequently in infants weighing less than 4,000 g. They are transitory, unavoidable, and not considered an indicator for quality improvement [11].

The classic injury is a brachial plexus palsy (BPP). In 1872, Duchenne ascribed the injury to traumatic delivery, and in 1874, Erb described the most common form of trauma involving the fifth and sixth cervical nerves [12].

*The Anatomy of the Brachial Plexus:* The source of the brachial plexus is the anterior primary rami of spinal segments C5, C6, C7, C8, and T1. These rami form the three trunks of the plexus, which in turn form anterior and posterior divisions. The upper trunk contains fibers from C5 and C6, the middle trunk is derived from the undivided fibers from C7, and the lower trunk comes primarily from the fibers from C8 and T1. The divisions form three cords: the lateral, posterior, and medial. Figure 14.1 illustrates the anatomic relationships of the brachial plexus.

*Classification of Brachial Plexus Injuries:* The upper trunk injury (C5–C7), *Erb's palsy*, is the most common form of brachial plexus injury. The infant appears to have the humerus adducted and internally rotated, and the elbow is extended. Paralysis usually affects the muscles of the upper arm, and

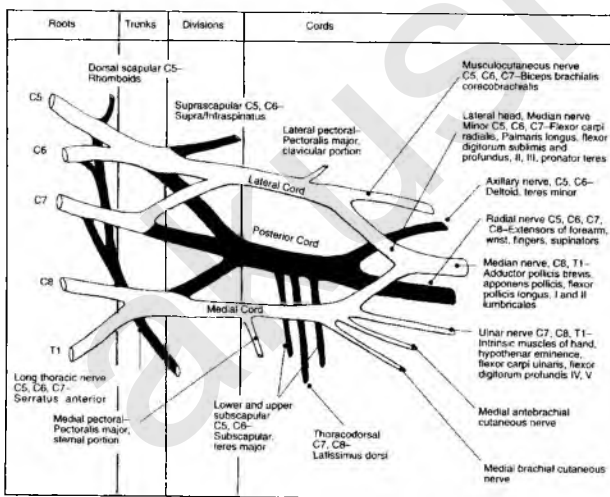


FIGURE 14.1. *The complex branching of the brachial plexus from its origin from cranial roots C5–T1 to the eventual peripheral nerves depicted.*

winging of the scapula is common. The supinator muscles and the extensors (C6) of the wrist can be affected. Sensory deficit is usually limited to the distribution of the musculocutaneous nerve.

The lower trunk lesion (C8 and T1), called *Klumpke palsy*, generally affects the forearm and wrist. The elbow is flexed with the forearm supinated, and a characteristic clawlike deformity of the hand is observed. Sensation in the palm can be depressed. Horner's syndrome is often present in the affected side owing to the involvement of the sympathetic fibers that traverse T1.

Rarely, a severe BPP involves the entire plexus and causes complete paralysis of the arm. The physician should be alerted to an associated spinal cord injury in such circumstances. There can be blood in the spinal cord because of avulsion of the roots of the plexus. Another rare injury, involving the fourth cervical root, might not be associated with a brachial plexus injury. This injury involves trauma to the phrenic nerve, and the infant presents with features of respiratory distress (*Weigert palsy*).

Most infants having an observable BBP at birth have transitory symptoms and recover with no permanent injury. Studies indicate the occurrence rate of BPP varies from 0.05% to 0.26% of all deliveries, and full return of function occurs in 70% to 95% [13]. An early study by Eng found that 30% of those with brachial plexus injury recovered by 6 months and 55% recovered by 1 year. Eventually, of those injured, approximately 15% demonstrated some residual handicap [14]. More recent studies from Johns Hopkins Hospital indicate that 116 of 127 (91.3%) brachial plexus injuries were temporary, and these resolved by 2 years [15].

In the Wishard study of 185 patient records coded for shoulder dystocia, there were 28 brachial plexus injuries (15.1%) and 14 fractured clavicles (7.5%; Table 14.1). All of the brachial plexus injuries were Erb's palsies, and those injured were followed for up to 5 years. Of interest, Brett found that brachial plexus injuries occur more often on the right, ostensibly because the predominant left occipitoanterior position leaves the right shoulder against the pubic arch for longer than other presentations [16].

At Wishard, six of ten such injuries involved the left shoulder. All but one brachial plexus injury resolved, and that child (3,864 g birthweight) had

TABLE 14.1 Wishard Study\*

Weight (g)	No.	Not Injured	Sustained Injuries	
			Clavicle Fracture	Brachial Plexus Injury
<b>Group A: Coded Shoulder Dystocia, Birthweight, and Injury<sup>†</sup></b>				
>4500	23	16	1	6 (26%)
4000–4449	83	65	7	11 (13.2%)
3500–3999	59	45	5	9 (15.2%)
3000–3499	19	16	1	2 (10.9%)
≤3000	1	1	0	0
Total	185	143	14	28 (15.1%)
<b>Group B: Not Coded for Shoulder Dystocia; Injury Occurred</b>				
2000–2999	3		2	1
3000–3999	12		9	3
>4000	4		3	1
Total	19		14	5

\*Group A compared with B for birthweight,  $p \leq 0.01$ ; Group A compared with B for injury,  $p \leq 0.01$ .

<sup>†</sup>Data from 12,532 vaginal and 1,765 cesarean deliveries, Wishard Memorial Hospital, Indianapolis, IN, January 1986 to June 1990. See text for additional details [3].

a residual mild arm weakness. All fractures of the clavicle resolved without incident. In the 5 years preceding the author's study, there were approximately 12,000 vaginal deliveries with four permanent brachial plexus injuries. In the author's experience, permanent brachial plexus injury is a rare event that occurs in about 1 in 4,000 vaginal deliveries and most likely varies from 1 in 2,000 to 1 in 4,000.

*Brachial Plexus Injury without Shoulder Dystocia:* There is substantial evidence that brachial plexus injury does occur without shoulder dystocia. Jennett found that 22 of 39 BBPs were not associated with shoulder dystocia [17]. In addition, Gilbert found only 53% of BPP associated with shoulder dystocia, and even in macrosomic infants, there was no shoulder dystocia associated with 26% of cases of BPP [13]. Likewise, Graham and coworkers also found only 53% of BPP associated with shoulder dystocia [18].

In the Wishard study, there was a group of 19 patients not coded for shoulder dystocia whose infants sustained an injury (see Table 14.1). There were 14 clavicular fractures and 5 brachial plexus injuries. When these infants were compared with the group coded for shoulder dystocia (14 fractured clavicles and 28 brachial plexus injuries) the nature

of injury was significantly different ( $p \leq 0.01$ ). The second group had a mean birthweight of 3,528 g compared with 4,112 g ( $p \leq 0.01$ ) for the recognized shoulder dystocia group. There were 12 spontaneous vaginal deliveries (3 brachial plexus injuries), 5 elective low forceps (1 brachial plexus injury, 2,892 g) deliveries, and 2 midforceps deliveries for fetal distress (1 brachial plexus injury, 3,205 g).

Statistically, this cohort represents a different population, particularly regarding the nature of the predominant injury (clavicular fractures) and the infants' smaller size. There was also no evidence of prolonged labor, diabetes, or other risk factors in this group. Gurewitsch and coworkers noted very similar findings in an extensive review of BPP with and without shoulder dystocia [15]. In this study, they found 49 cases of nonshoulder-dystocia-related BPP, and 30% lacked all risk factors for shoulder dystocia.

These studies indicate that various and diverse mechanisms result in a shoulder dystocia. Likewise, there are various, diverse, and most likely, multiple mechanisms involved in a BPP. The following sections discuss the multiplicity of risk factors, predictability, mechanisms of impaction, and theories of injury.



### Macrosomia and Related Risks

The definition of macrosomia varies with associated risks [19–21]. A consistent definition holds that in a nondiabetic patient, *macrosomia* occurs at 4,500 g and in a diabetic patient, 4,000 g. Some observers propose that a fetal weight above the 90th percentile for gestational age constitutes macrosomia [20]. More recently, taking into account the imprecision in diagnosing macrosomia, the ACOG Practice Bulletin No. 40 suggests that prophylactic cesarean delivery may be considered in nondiabetic infants over 5,000 g and diabetic infants over 4,500 g [8]. Although birthweight over 5,000 g is uncommon, about 5% to 7% of babies weigh more than 4,000 g, and about 1% will exceed 4,500 g [21]. It appears that the recent trend is toward the delivery of larger infants.

Multiple factors contribute to macrosomia, and many of these are interrelated. The most significant factors include a large maternal habitus, male fetus, multiparity, maternal diabetes or obesity, post-term pregnancy, and macrosomia in a prior infant [22]. It should be stressed, however, that most patients with these factors have normal-weight babies.

### Maternal Weight

Maternal height, weight, and prepregnancy weight are associated with increased infant weight [23]. In other words, large women have large babies. A corollary of this finding is that the mother's own birthweight is directly related to fetal macrosomia [24]. Although maternal obesity and weight gain during pregnancy are directly related to the infant's birthweight, the influence of these factors varies markedly with prepregnancy weight, age, parity, and level of education [25].

Unfortunately, most of these risk factors for macrosomia have limited clinical value. For example, male infants are larger than female infants and are twice as likely to weigh more than 4,000 g [26]. This fact does not lend itself to the development of a decision-making protocol, however. Spellacy and coworkers noted a high-risk group for macrosomia having a triad of obesity, diabetes, and postdates, and recommended liberal use of cesarean delivery if macrosomia were found [27]. The problem with this recommendation and all similar ones, however, is that it is virtually impossible to document macro-

somia with any kind of reliability sufficient to justify routine operative delivery.

### Reliability of Ultrasonography

In macrosomia, the trunk appears to grow larger relative to the head. Elliot and coworkers used ultrasound examination to document this growth pattern in diabetic patients and developed an index of macrosomia by subtracting the biparietal diameter from the chest diameter [28]. If the difference was more than 1.4 cm, then cesarean delivery was recommended, ostensibly to reduce the incidence of traumatic morbidity. Recent evaluations of the positive predictive value (PPV) of ultrasound examination indicate that accurate sonographic evaluation of the suspected large fetus is beyond the current capability, however.

Although the best estimates of macrosomia include abdominal circumference and femur length, the range of error in one study was 22% [29]. Delpapa and Mueller-Heurbach compared the outcomes in 242 women with sonographic estimates of macrosomia and concluded that cesarean delivery or elective induction to avoid continued fetal growth was inappropriate when based only on the sonogram [30]. Thus, protocols for determining the route of delivery based solely on estimates of fetal weight are too simplistic and merely result in unnecessary operative deliveries.

### Post-term Pregnancy

The effect of length of gestation on development of macrosomia is well recognized [31]. In the Wishard Study, the majority of shoulder dystocias (42.2%) occurred between 40 and 41 weeks of gestation. The incidence of shoulder dystocia decreased relative to the total number of deliveries thereafter, and only three episodes of shoulder dystocia were noted at 43 weeks, with no trauma in this group.

### Prior Macrosomic Infant

Patients who delivered a prior macrosomic infant have a higher relative risk for shoulder dystocia than is present with weight gain, height, and parity. Women who deliver an infant weighing more than 4,500 g are more likely to have had a prior macrosomic infant (4,000 g) [32]. Although Ouzounian

and coworkers indicate that the rate of recurrent shoulder dystocia is increased (3.8%) over the general population (0.6%), it is lower than previously estimated [33].

The Wishard study revealed that in 185 shoulder dystocia patients, 31 of 106 women who gave birth to an infant weighing more than 4,000 g had a previous infant weighing more than 4,000 g, whereas only 1 of 79 whose infants weighed less than 4,000 g had a prior macrosomic infant. Common sense would dictate that the prior delivery of a large infant might have clinical significance, especially if the delivery were difficult or associated with trauma.

### Maternal Diabetes

It is universally recognized that diabetes, pregestational and gestational, is associated with macrosomia. An extensive study of macrosomia by Boyd and coworkers, however, found that only 32% of diabetic mothers had macrosomic infants [9]. Acker found that the incidence of shoulder dystocia increased to 31% in diabetic patients whose infants weighed more than 4,000 g, and the incidence in nondiabetic patients increased to 22.6% when their infants weighed more than 4,500 g [34]. Although this classic study was often cited to justify the use of cesarean delivery for diabetic mothers with a fetus weighing more than 4,000 g and the liberal use of cesarean delivery for nondiabetic parturients with an estimated fetal weight exceeding 4,500 g, especially if labor is abnormal, it is not a standard of care. As previously noted, the ACOG Practice Bulletin No. 40 suggests that prophylactic cesarean delivery might be considered in nondiabetic infants weighing more than 5,000 g and diabetic infants over 4,500 g. This is because no birthweight category, even 2500 g, is entirely free of shoulder dystocia risk.

Other maternal factors associated with macrosomia noted in Boyd's study include

- Multiparas over age 35
- Prepregnant weight greater than 70 kilograms
- Ponderal index (weight/height<sup>3</sup>) in the upper tenth percentile
- Height exceeding 169 cm
- Greater than a 20-kg weight gain
- Delivery more than seven days post term.

It is clear that multiple factors contribute to macrosomia, and some of these are interrelated with diabetes; however, most patients with these risk factors have normal-weight babies.

Moreover, almost one half (47.6%) of all shoulder dystocias occur in infants weighing less than 4,000 g [35]. Furthermore, many diabetic mothers do not have macrosomic infants, the majority of macrosomic infants are not infants of diabetic mothers, and injury does not occur more often in this group. Moreover, macrosomia is as difficult to predict in the diabetic as in the nondiabetic population. Benson and coworkers found that the use of standard formulas for predicting macrosomia by ultrasonography was correct in only 47% of infants [36]. Nonetheless, the liberal use of selective cesarean delivery in diabetic mothers meets little clinical opposition.

### Intrapartum Factors

#### *Labor Abnormalities*

Benedetti and Gabbe reported that the incidence of shoulder dystocia in deliveries with prolonged second stage plus midpelvic delivery was statistically significant compared with those without these factors [37]. In this review, *prolonged* second stage is defined as more than 2 hours in the nulliparous patient and more than 1 hour in the parous patient, with arrest of descent at station +3 cm or higher. This observation remains as one of the strongest subsets of complications associated with shoulder dystocia. *The predictive value for shoulder dystocia in prolonged second stage and midpelvic delivery increases only when the fetus is actually macrosomic, however.*

In contrast, in the Wishard study, only nine episodes of prolonged second-stage labor were identified in the shoulder dystocia study group, and five of these patients had newborns weighing less than 4,000 g. Two were delivered spontaneously; the shoulder dystocia was resolved by suprapubic pressure in one (left Erb's palsy), and the other had no technique listed (left clavicular fracture). There were three low-forceps deliveries with one right brachial plexus injury and no injury in the other two. Of the remaining four patients with a midforceps rotation, there were no injuries. There were no permanent injuries in this group.

Other labor patterns associated with shoulder dystocia appear to have little or no significance independent of macrosomia. For example, prolonged latent phase is independently associated with increased maternal and fetal morbidity and should alert the physician to an increased risk for further problems in labor and delivery [38]. Protracted active phase disorders appear to carry no inherent threat to the fetus unless accompanied by operative (especially midforceps) delivery [39]. Because shoulder dystocia is a complication of macrosomia, an increased incidence of labor disorders would be expected.

### ***Oxytocin and Anesthesia***

It would be logical to expect an increased incidence of oxytocin augmentation and induction in patients with shoulder dystocia owing to the observed labor abnormalities associated with macrosomia. No studies have implicated any other significance, however. In the Wishard group, there was also no statistical significance found with the use of oxytocin, either by induction or augmentation, between the study and reference groups. Oxytocin was used in 78 of 185 patients with shoulder dystocia (42.1%), compared with 49.2% of all vaginal deliveries. There was also no statistical difference found in the use of anesthesia. Epidural anesthesia was used in 110 of 185 shoulder dystocia cases (57.4%) and in 67.9% of vaginal deliveries in a control population.

### ***Episiotomy***

An extensive episiotomy in the presence of shoulder dystocia was frequently recommended, ostensibly to relieve any resistance from the perineal floor that could prevent egress of the shoulders. There is no statistically significant relationship between the absence of episiotomy and subsequent neonatal injury, however. In the author's study, there were 17 shoulder dystocia patients without episiotomy and 5 neonatal injuries (29.4%): four fractured clavicles and one transitory brachial plexus injury. In comparison, in 168 patients with shoulder dystocia who had an episiotomy, there were 37 injuries (22%), including 10 fractured clavicles and 27 brachial plexus injuries.

### **Risk Factor Profile**

The occurrence of shoulder dystocia increases in direct relationship to the birthweight; this becomes statistically significant in infants weighing more than 4,000 g (see Table 14.1). What is striking, however, is the frequency with which shoulder dystocia occurs in newborns weighing less than 4,000 g. In this respect, over 90% of all vaginal deliveries account for slightly less than one half of all shoulder dystocias. What is most important is that this single observation refutes the general notion that shoulder dystocia is always predictable and therefore preventable.

Apparently, none of the frequently noted risk factors are reliable in predicting the occurrence of shoulder dystocia without macrosomia. Even the strong association of a prior macrosomic infant did not result in a shoulder dystocia in more than 70% of women. Conditions such as diabetes or midforceps delivery, after a prolonged second stage of labor, become significant only in the presence of a large fetus. Moreover, other traditional risk factors such as obesity, multiparity, and postdate pregnancy are not statistically significant or predictive of shoulder dystocia. Finally, there seems to be no association of shoulder dystocia with episiotomy, oxytocin, or anesthesia.

Gherman and coworkers found diabetes to be more common in transitory BPP. They found operative delivery equally common in transitory and permanent BPP [40]. In contrast, Gurewitsch found no difference in the rate of diabetes between shoulder dystocia and nonshoulder-dystocia-related BPP or between temporary and permanent BPP [15].

The limiting factor is the inability to predict macrosomia with the requisite degree of certainty on which a clinical decision should be based. Until the macrosomic infant can be accurately identified, no reasonable risk factor profile can be established.

### **Pathophysiology of Shoulder Impaction**

After delivery of the head, restitution or external rotation returns the head to its normal axis to the spine and its perpendicular relationship to the shoulders. The shoulders are usually in an oblique axis under the pubic rami. Maternal pushing drives the anterior shoulder under the pubis. If the shoulder fails to rotate into this oblique axis and remains in

the anteroposterior position, a large fetus can impact its anterior shoulder against the symphysis [41].

In 1926, J. Whitridge Williams noted that in most cases the anterior shoulder will deliver spontaneously just after external rotation [42]. Occasionally, a delay occurs, and the physician is advised to seize the occiput and chin with two hands and apply downward traction until the anterior shoulder is seen. In the case of prolonged delay, Williams states, "indeed, even when the former method of extraction is applied, traction should be exerted only in the direction of the long axis of the child, for if it be made obliquely, the neck will be bent upon the body, when excessive stretching of the brachial plexus on its convex side will occur, with subsequent paralysis" [42].

Although Williams postulates that a brachial plexus injury results from excessive stretching of the brachial plexus and not necessarily from excessive downward traction during delivery, this theory has never been substantiated. Moreover, Williams did not consider the role of maternal expulsion efforts, compression of the brachial plexus against the pubic symphysis, torque or twisting forces during rotation of the head against an impacted shoulder, or the failure of the shoulders to adduct during a rapid descent [41].

The primary difficulty with the shoulders arises from their relatively large size respective to the inlet. Although dystocia can occur in the presence of pelvic deformity, it can also occur when the shoulders fail to rotate into the anteroposterior diameter. Thus, some degree of fetopelvic disproportion is present in a shoulder dystocia. Similarly, fetopelvic disproportion is a relative condition and therefore varies in its presentation. This would account for the unpredictable occurrence of shoulder dystocia in the same patient who might have a subsequent large infant without dystocia as well as in newborns weighing less than 4,000 g.

### ***Forces Operating in a Shoulder Impaction***

As the head descends through the birth canal, the maternal expulsive forces impact the shoulder against the pubic symphysis, and to a much lesser degree, the sacral promontory. As early as 1936, Moir noted that the maternal expulsive forces, converted to pounds-weight, of the average uterine contraction is about 16 pounds per square inch [44].

With maternal pushing, the force doubles to 32 pounds per square inch. More recently, observers measured maternal expulsive forces as well as the forces applied to the head and found them to vary with the result of the load required for delivery, and that the largest amounts of brachial plexus stretching occurred with maternal pushing [3,45].

As the shoulder approaches the symphysis, it either rotates into the oblique axis or remains impacted. The shoulder either then stays impacted or overrides the symphysis. As the head continues its outward journey but the shoulder stays impacted, the soft tissues of the neck and cervical spine are stretched. After the head delivers, it retracts against the perineum; this is frequently observed as the so-called *turtle sign*. Thus, before any traction is placed on the head, two forces have stressed the brachial plexus, that is, stretching forces and compression forces. If the head rotates after delivery, a third torque or twisting force can also occur.

What also appears to be an important mechanism of shoulder dystocia is the ability of the shoulders to mold themselves in the pelvis. In the normal fetal position, the shoulders are forced anteriorly as they enter the inlet. This would reduce the usual bisacromial diameter (12 cm) and allow the shoulders to follow the contours of the birth canal, with the posterior shoulder preceding the anterior one.

As mentioned previously, before any traction is placed on the head, the brachial plexus has been stretched, compressed, and possibly twisted. Subsequently, downward traction is placed on the head, often without any evidence of a shoulder impaction. Most often, the patient is encouraged to push with this traction, and additional stretching and compression take place. At this point, shoulder dystocia is recognized, and some disimpaction maneuver is then performed. Most commonly, the application of suprapubic pressure and McRoberts' position is used. This combination is noted to relieve about 80% of shoulder dystocias. If this combination fails, other maneuvers must be used. Eventually, the baby must be delivered, with a wide spectrum of outcomes ranging from no injury to complete paralysis of the shoulder girdle and arm, phrenic nerve injury, and Horner's syndrome. Fortunately, over 90% of such injuries are not permanent.

Injury to the brachial plexus can occur from stretching, twisting, or compression of nerve trunks

resulting in partial to complete involvement of the nerves. Injury can also include avulsion of cervical nerve roots from the spinal cord. In addition, compression of the vascular supply and hypoxia, which often occur in such deliveries, compromise the neural tissue, making the nerve trunks more susceptible to injury. If the nerve is severely compressed, its functional ability appears as though it were torn, but the tear is usually not complete. The atrophy is not as intense, and the conduction loss is not as extensive [46]. Stretching of the brachial plexus appears to result in a similar range of injury. Most likely some combination of all the above forces contribute to a BPP.

The above pathophysiology of a shoulder impaction explains the entire range of observable outcomes, including

- A wide range of injuries from none to fractures of the clavicle and humerus and BPP
- Transitory and permanent BPP
- BPP in the absence of observable shoulder dystocia
- BPP in which no traction was applied
- BPP where appropriate maneuvers were used.

### ***Excessive Traction and Brachial Plexus Palsy***

*A review of the best obstetric literature does not reveal any consistent empirical evidence to support the conclusion that excessive traction causes BPP.* At best, the conclusion is a limited one that does not consider the various forces described previously that affect the head, neck, and shoulder during a normal delivery and an obstructed one. Thus this theory does not even qualify as Level III evidence.

In contrast, peer-reviewed and evidence-based studies do not support the opinion that extreme or excessive traction causes brachial plexus injuries. There are three articles in the obstetric literature that contain substantial data about the use of traction (described as greater than normal or excessive) in the delivery of infants during a shoulder dystocia.

On the surface, an article by Gross, Shime, and Farine in 1987 indicates that fundal pressure, in the absence of other maneuvers, resulted in a 77% complication rate and was associated with orthopedic and neurologic damage [46]. Within the next few

years, references to this article were cited as the reason to avoid fundal pressure in shoulder dystocias. A closer look at this article, however, clearly reveals that some of the conclusions are questionable, especially that the use of fundal pressure causes brachial plexus injuries.

Gross retrospectively reviewed 10,662 vaginal deliveries for which 91 shoulder dystocias were identified. The shoulder dystocia cases were divided into two groups: Group 1 (n = 24) included *true* shoulder dystocia, defined as deliveries requiring maneuvers in addition to downward traction and episiotomy, whereas Group 2 (n = 67) included deliveries that required *increased traction*. The authors noted that fundal pressure and traction were used in 13 patients in Group 1.

Morbidity in Group I consisted of six cases of Erb's palsy (6/24 or 25%), five fractured clavicles, and one respiratory arrest. Two infants sustained multiple injuries. Thus, 10 of 24 newborns in Group I had some morbidity (42%). In Group 2, however, when increased traction was used, there were no injuries. Moreover, Gross did not indicate whether any of the Erb's palsies were permanent. Thus, there is no information in this study to indicate that fundal pressure causes any type of permanent injury.

At best, only two valid conclusions can be drawn from the Gross study. First, there were six brachial plexus injuries in 24 true shoulder dystocia cases (incidence = 25%). The authors note that all orthopedic and neurologic injury was associated with a combination of increased traction and fundal pressure. There are no data to suggest that fundal pressure alone is associated with any damage. Second, and most important, there were no injuries associated with 67 cases of shoulder dystocia in which only increased traction was applied. This latter observation refutes the opinion that increased traction alone causes permanent neurologic injury.

In the second study, Baskett documented that when only "strong downward traction" was used in 48 shoulder dystocia cases, there were only 12 brachial plexus injuries (25%) [47]. In other words, 75% of babies delivered with strong downward traction were *not* injured.

The third study, previously cited, is most interesting [3]. In this study of 29 vaginal deliveries, there were only two shoulder dystocias, seven deliveries defined as *difficult*, and 20 classified as *routine*.

An obstetrician wore a specially designed glove that measured the forces applied in these deliveries. As expected, the peak force rates in the shoulder dystocia group were substantially higher than in the normal deliveries. The peak force rate used in the two shoulder dystocias was not significantly different from that used in the difficult deliveries, however. There were no injuries in the latter difficult group. Furthermore, in the two shoulder dystocia cases, in which each infant sustained only one injury (a transitory Erb's palsy and fractured clavicle) the peak forces were identical, but the *rate* of application and duration of the force in the "injured" infant differed somewhat. Both of these babies weighed 4,790 g and 4,775 g, respectively. In summary, there were big babies and excessive traction; one baby was uninjured, and the other was not permanently injured.

Allen's study showed that even when the baby is large for gestational age (LGA) and the force is greater than usually applied, there was no correlation to any level of injury. In this study, there were nine deliveries in which the force was higher than usual, and there were no injuries.

From these observations from respected physicians, based on clinical experience, one cannot support the concept that extreme or excessive traction causes brachial plexus injury. In fact, from a probability perspective, it is more likely than not that extreme or excessive traction does not cause brachial plexus injury.

### Fundal Pressure and Brachial Plexus Palsy

Virtually every study of the injuries associated with shoulder dystocia distributes the injuries, both brachial plexus and fractures, among the entire population of shoulder dystocia cases. In this way, selective bias tends to be diminished. For example, Gherman's study identified 285 cases of shoulder dystocia in 50,114 vaginal deliveries with 71 injuries (24.9%). In this study, there were 48 brachial plexus injuries (16.9%), 28 fractured clavicles (9.5%), and 12 humeral fractures (4.2%). No use of fundal pressure occurred in this study, but there were brachial plexus injuries and fractures. In addition, only four of the brachial plexus injuries were permanent.

Similarly, the Wishard study identified 185 shoulder dystocias among 12,552 vaginal deliveries with

44 injuries (22.7%) [2]. There were 28 brachial plexus injuries and 14 fractures. There was one case of permanent Erb's palsy in this study, and fundal pressure was not used in any of the shoulder dystocia cases.

Likewise, Baskett found 254 shoulder dystocias in 40,518 vaginal deliveries for which fundal pressure was not used [48]. There were 46 injuries, with 33 brachial plexus injuries and 13 fractures (18.1% total injuries). In this study, about 80% of infants with brachial plexus injuries improved by the time they were discharged from the nursery.

Gherman, Nocon, and Baskett reported on three extensive studies on shoulder dystocia and injury in the obstetric literature. When the Gross and Allen studies are included for comparison, only a few valid conclusions can be made:

- No method of delivery in a shoulder dystocia case is free of injury.
- Permanent brachial plexus injury is a rare event and is clearly not associated with the method of delivery.
- The evidence does not support the conclusion that fundal pressure causes permanent brachial plexus injury.
- Most important, the evidence does not support the conclusion that increased traction or strong downward traction is the only cause of brachial plexus injury.

### Disimpaction Maneuvers

Historical surveys of obstetric procedures used to resolve difficult births reveal very consistent patterns [50]. In most situations, the mother's legs are drawn back to the hips and the midwives or attendants support the fetal head while applying some force to the uterus, just over the shoulder. Beer conducted an extensive review of the history of maneuvers used to resolve a shoulder dystocia and found citations as early as 1753 involving the extraction of the posterior arm and what is now known as the *McRoberts maneuver* [51].

Protocols for the management of shoulder dystocia abound in the literature. Most interesting, the older texts describe techniques that are remarkably similar to more recent descriptions of the management of this emergency. In 1947, McCormick's



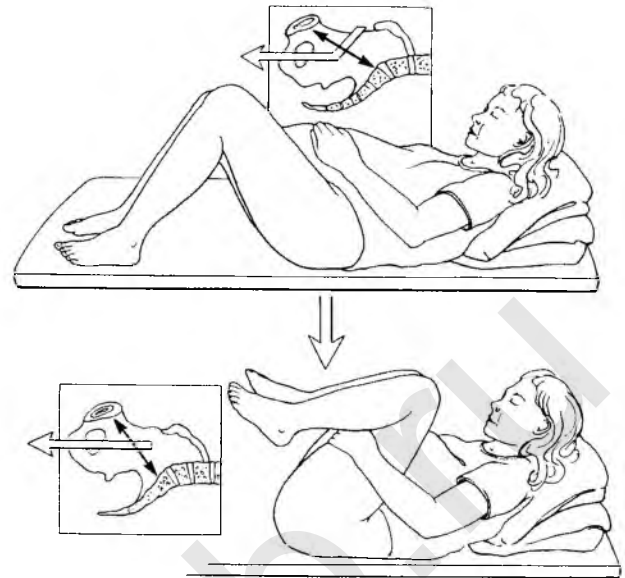
**FIGURE 14.2.**  
*Shoulder dystocia: Hibbard/Resnick maneuver. Oblique suprapubic pressure is applied by the surgeon while gentle traction is applied to the posterior shoulder.*

description of a disimpaction maneuver used at Indiana University was quite astute [52]. He first noted that shoulder dystocia frequently comes as a surprise and develops into an emergency. McCormick then described a technique used for “seven to eight years” of “screwing” the baby out of the pelvis after freeing the posterior arm. Castallo and Ullery’s timely advice is to place the patient in the Walcher position and have an assistant push from above the symphysis to facilitate the shoulders coming into the inlet [53]. The Walcher position involves hyperflexion of the thighs against the abdomen.

### **Simple Maneuvers**

Perhaps the easiest and quickest of the disimpaction maneuvers is the application of *suprapubic pressure* recommended by Hibbard in 1969 [54] and reiterated by Resnick in 1980 [6]. An assistant applies suprapubic pressure, and gentle downward traction is applied by the physician (Figure 14.2).

Gonik and collaborators named a maneuver after William A. McRoberts, Jr.; this maneuver involves hyperflexion of the thighs [55]. Figure 14.3 illustrates this technique, which is used on the patient to straighten the sacrum relative to the lumbar spine. This rotates the symphysis cephalad, with a resulting decrease in the angle of inclination from 25° to 10°. Although this maneuver does not actually increase



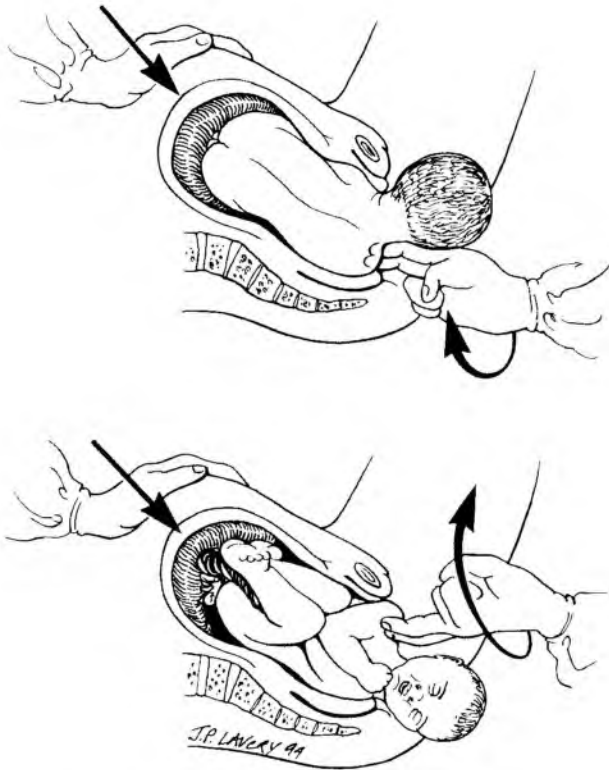
**FIGURE 14.3.**  
*The McRoberts maneuver. Hyperflexion of the patient’s thighs changes the relationship of the pelvis to the lumbar spine, facilitating delivery of the fetal shoulders. (From Beckmann CR, Ling FW, Barzensky BM, et al. [eds]: *Obstetrics and Gynecology for Medical Students*. Baltimore: Williams & Wilkins, 1992; with permission.*

the dimensions of the birth canal, it appears to allow easier disimpaction of the anterior shoulder.

In a laboratory model, Gonik tested the physical forces involved in this maneuver and noted that it did reduce fetal extraction forces, brachial plexus stretching, and the likelihood of clavicular fracture [56]. The McRoberts maneuver appears to be one of the most popular techniques, and many operators use it prophylactically when they suspect a large fetus or when the second stage is prolonged. Poggi and coworkers found that the use of this maneuver provides no reduction in the forces used in multiparas, however, and questions the use of this maneuver prophylactically [57]. In addition, Beall, Spong, and Ross found that prophylactic use of McRoberts’ maneuver and suprapubic pressure did not differ significantly from maneuvers used after delivery of the head with respect to delivery times, admissions to the special care nursery, or birth injuries [58].

### **Rotation Maneuvers**

The most classic and one of the earliest descriptions of the management of shoulder dystocia is by Woods, who likened the shoulders to the



**FIGURE 14.4.**  
*Shoulder dystocia: Woods corkscrew maneuver. The posterior fetal shoulder is rotated anteriorly, freeing the obstruction.*

longitudinal section of a screw, and determined that the fetus should be rotated through the birth canal, because traction on the neck is mechanically incorrect [59]. In the *Woods corkscrew maneuver*, the physician exerts downward thrust on the uterine fundus with one hand while inserting two fingers of the other hand on the anterior aspect of the posterior shoulder and gently rotating clockwise (Figure 14.4). This delivers the posterior shoulder. Then, with synchronized downward pressure, the two fingers make gentle counterclockwise pressure upward around the circumference of the arc to and beyond 12 o'clock. This "unscrews" and delivers the remaining shoulder.

Note that fundal pressure is appropriate in a disimpaction maneuver. Likewise, fundal pressure is appropriate once the anterior shoulder rotates into the oblique angle of the inlet; this minimizes the forces exerted to deliver the baby when applied with downward traction.

A variation on the theme of rotation is the *rocking maneuver* suggested by Rubin [60]. In this tech-

nique, the obstetrician rocks the shoulders from side to side by applying lateral suprapubic force. Thereafter, the most accessible shoulder is pushed toward the anterior surface of the fetal chest, resulting in abduction of the shoulders and a subsequently smaller bisacromial diameter. Gurewitsch and coworkers noted that Rubin's maneuver provides less tractional force than McRoberts' and thereby requires the least amount of brachial plexus tension [61].

#### *Delivery of the Posterior Arm*

Schwartz and Dixon concluded that *extraction of the posterior arm* was safe and simple [62]. Figure 14.5 illustrates the extraction of the posterior arm and delivery of the fetus. The hand is gently inserted along the curvature of the sacrum and the fingers follow along the curvature of the humerus to the antecubital fossa (see Figure 14.5, A and B). With pressure, the forefinger flexes the forearm across the chest (Figure 14.5C). As the arm flexes, the infant's forearm is grabbed with the index finger and swept across the chest and face of the fetus and out of the vagina (Figure 14.5D). Often, the anterior shoulder will slide under the symphysis after the posterior arm is removed.

Sometimes it is necessary to rotate the baby to complete the delivery. Carefully supporting the posterior arm with one hand, the operator places the other on the back of the head or up to the back of the anterior shoulder, and the baby is then rotated much as in the corkscrew maneuver (Figure 14.5, E and F). Fracture of the humerus is a recognized complication of this technique. This is one situation for which deep anesthesia is ideal, but extraction of the posterior arm can be safely performed without any anesthesia. Poggi, Spong, and Allen report that using extraction of the posterior arm reduces the obstruction by more than a factor of two, relative to the McRoberts' maneuver, and recommend its earlier use [63].

A wide episiotomy can be helpful in allowing the hand to reach the posterior shoulder when one performs a rotation maneuver or removes the posterior arm. There is no evidence to suggest that a lack of an episiotomy impedes such a technique, however. Moreover, Gurewitsch and coworkers found that if the delivery can be performed without an episiotomy, perineal trauma is minimized and an episiotomy offered no benefit in avoiding BPP [64].





FIGURE 14.5.  
Shoulder dystocia: Schwartz-Dixon maneuver. Delivery of the posterior arm is followed by fetal rotation.

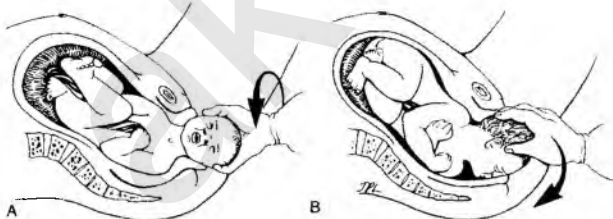


FIGURE 14.6.  
Shoulder dystocia: Zavanelli maneuver. Following tocolysis, the fetal head is rotated to the occipitoanterior or posterior position, flexed, and returned to the birth canal; cesarean delivery follows.

### Other Techniques

The *Zavanelli maneuver* has been described as replacing the head in the vagina so that a cesarean delivery can be performed [65]. In this procedure, the head is returned to the occipitoanterior or occipitoposterior position, and it is then flexed and slowly pushed back into the birth canal (see Figure 14.6). A cesarean delivery is then performed. Although the procedure appears straightforward, many observers have found it to be difficult [66].

Although virtually every text describes deliberate *fracture of the clavicle* as a method to reduce the shoulder width and thereby disimpact the anterior shoulder, this procedure might actually be much easier to describe than to accomplish. The author has found it to be extremely difficult. Other recognized but rarely performed procedures include *cleidotomy* (i.e., cutting of the clavicle with a scissors) and *symphysiotomy*. (Symphysiotomy is discussed in Chapter 18, Cesarean Delivery and Surgical Sterilization.)

### Prediction and Prevention

Each of these aforementioned procedures has its proponents. O'Leary and Leonetti suggest a protocol to grade shoulder dystocia from mild to severe and recommend a treatment plan consistent with the grade, described as mild, moderate, or severe [67]. In addition, they also hold that many cases are preventable by the proper identification of historical and antepartum risk factors.

In contrast, Gross and coworkers have emphasized that even if a factor is statistically significant, it might not be useful as a predictor of shoulder dystocia [1]. For example, the combination that would best predict shoulder dystocia is birthweight, prolonged deceleration phase, and length of the second stage. This combination predicts only 16% of patients with shoulder dystocia and trauma, however.

The Wishard study is in complete agreement with the observations of Gross and others. It indicates that most of the traditional risk factors for shoulder dystocia have limited or no predictive value, that shoulder dystocia itself is an unpredictable event, and that infants at risk for permanent injury are virtually impossible to predict.

TABLE 14.2 Wishard Study: Management of Shoulder Dystocia and Neonatal Injury\*

Maneuver Performed	No.	Not Injured	Injuries Sustained	
			Clavicle Fracture	Brachial Plexus Injury
McRoberts	74	63 (85.1%)	4	7
Rotations	42	36 (85.7%)	0	6
Posterior arm	29	18 (62.1%)	1	10 <sup>†</sup>
Suprapubic pressure	20	16 (80%)	1	3
Traction	3	2 (66.7%)	1	0
None listed	17	8 (47.5%)	7	2
Not coded <sup>‡</sup>	19	0	14	5
Totals	204	143 (70.1%)	28 (13.7%)	33 (16.2%)

\*Data from 12,532 vaginal and 1,765 cesarean deliveries, Wishard Memorial Hospital, Indianapolis, IN, January 1986 to June 1990. See text for additional details [3].

<sup>†</sup>One right brachial plexus injury and left humerus fracture in same patient.

<sup>‡</sup>Injuries noted in newborns where no shoulder dystocia was found in record.

### Management and Injury Profile

In the Wishard study, there were 17 different techniques identified in 168 patients in the study group; no maneuver was noted or described in the remaining 17 patients. The McRoberts maneuver was, by far, the most common initial approach taken in 94 patients (50.8%). The various techniques were grouped into six major treatment categories and related to the frequency and nature of the trauma that occurred (Table 14.2).

None of the major categories revealed a statistically significant difference when compared with each for incidence of brachial plexus injury. Within the McRoberts category, 74 primary attempts successfully disimpacted the anterior shoulder, and 20 attempts failed. One McRoberts' maneuver failed as a secondary procedure. Failed McRoberts' maneuvers obviously were followed by some other approach. Both the successful and the failed McRoberts groups had the same number of injuries: seven brachial plexus injuries and one fractured clavicle.

Although no disimpaction maneuver was significantly superior to any other with respect to injury, there was a tendency to less injury with rotation maneuvers. Likewise, other studies indicate that the anterior Rubin maneuver was associated with less tractional force than the McRoberts [61]. There appears to be no rationale for choosing one technique over another. No significant reason

was found to suggest that the subjective degree of shoulder dystocia (i.e., mild, moderate, or severe) should be managed by any particular approach, and thus, no protocol should substitute for clinical judgment.

Despite the fact that the removal of the posterior arm resulted in a slightly higher incidence of brachial plexus injury, the clinical importance of this approach should be emphasized. Namely, it was the only procedure that resolved the impaction when other maneuvers failed. For this reason, all physicians who deliver babies should be competent in its use.

### Routine Cesarean Delivery for Macrosomia

The Wishard study provides substantial follow-up information identifying the severity and persistence of injuries associated with shoulder dystocia. This allows physicians to balance maternal and fetal risks when they consider the routine use of cesarean delivery in cases of macrosomia. In this respect, two facts become clear. First, the *risk of permanent fetal injury is very small* (about 1 in 2,000 to 4,000 vaginal deliveries). Second, *protocols for determining the route of delivery based solely on estimates of fetal weight result in a substantial number of unnecessary operative deliveries*. For these reasons, the routine use of cesarean delivery in suspected macrosomia cannot be justified.

The Wishard study illustrates this point well. If the newborns weighing more than 4,000 g could have been accurately predicted, routine cesarean deliveries would have prevented 106 shoulder dystocias but not one permanent injury. The caveat here is that the obvious relationship between shoulder dystocia and progressive fetal birthweight cannot be denied. Thus, one cannot fault the logic of clinical judgment in the selective use of cesarean delivery when there is objective evidence that the fetus is macrosomic.

## SPECIAL ISSUES

### **Shoulder Dystocia: An Obstetric Emergency**

Shoulder dystocia is an obstetric emergency, and although one might suspect it, one cannot predict it with any degree of reliability. Any delay in its resolution therefore cannot be tolerated. It makes little difference which approach is used to resolve the impact of the anterior shoulder. The key to its resolution is to execute a reasonable plan of management. Although the author's approach is similar to others proposed in the literature, it is offered with the recognition that any reasonable approach is just as effective, and thus the failure to follow this management plan in no way constitutes a deviation from a standard of care.

### ***Anticipate a Shoulder Dystocia***

What distinguishes the professional from the amateur is an attention to detail that the amateur does not even consider. Is there a reason to suspect a large infant? Although no single risk factor or set of risk factors is predictive of macrosomia, important risk factors for macrosomia include diabetes, a previous large infant, and the patient's weight at her birth. As previously stated, large women tend to have large babies. Clinical suspicion of a large fetus should rise when

- The estimated fetal weight is greater than the 90th percentile on routine screening ultrasound scan.
- The fundal height is persistently greater than expected.
- The fundal height is greater than 41 cm at term.

- The estimated fetal weight by Leopold maneuvers exceeds 4,000 g.
- Maternal perception suggests a baby larger than a prior infant.
- A single ultrasound scan at term has the widest margin of error.

### ***Determine the Optimal Route for Delivery***

Consider early delivery of the suspected macrosomic infant; induction at term is reasonable when the cervix is favorable for a good outcome. The routine use of cesarean delivery in suspected macrosomia cannot be justified in the general population; however, liberal use of cesarean delivery is ostensibly more justifiable in the diabetic population with evidence suggestive of macrosomia. Abnormal labor has been well documented to portend a poor outcome. Studies indicate that labor abnormalities might not serve as clinical predictors of shoulder dystocia, however, and no characteristic of second-stage labor predicts BPP [68,69]. Nonetheless, one should avoid a vacuum or forceps on a fetus at a +2/5 station in a prolonged second stage. Shoulder dystocia confirms the adage that to be forewarned is to be forearmed.

### ***Call for Help, Take a Deep Breath, and Stop Pushing***

Virtually all disimpaction maneuvers require an assistant. Even anesthesiologists and pediatricians can apply suprapubic pressure and other lifesaving procedures. It is just as important to have the patient's confidence and cooperation as it is to have nurses assist in the delivery. *Most important, resist the urge to tell the patient to push.* Keeping the patient from pushing decreases the pressure of the shoulder against the pubic bone and can assist the shoulder in moving to the oblique angle of the inlet either by suprapubic pressure or a rotation maneuver. This also greatly assists in the removal of the posterior arm.

### ***Episiotomy***

If the perineum is "tight" or room is needed to insert the hand, make a large episiotomy. Although there is no evidence that it does anything other than allows

one to insert one's hand in the vagina, it indicates that the operator is functioning logically and systematically. Again, not performing an episiotomy has not been shown to contribute to any injury.

- *McRobert's position and suprapubic pressure will disimpact most tight shoulders.* These maneuvers are easy to perform, and the McRoberts position can also enhance the ability to perform a rotation maneuver or remove the posterior arm successfully.
- *Avoid excessive traction or even the appearance of excessive traction on the neck.* Observers in the birthing room have often been asked to testify in malpractice claims involving a brachial plexus injury. To the uninitiated, even gentle downward traction can appear excessive.
- *Perform a rotation maneuver.*
- *Extract the posterior arm.*
- *Know when and when not to use fundal pressure.*

When the anterior shoulder moves to the oblique angle of the inlet, either after suprapubic pressure or after a rotation maneuver, fundal pressure is indicated. Gentle but firm pressure decreases the amount of force applied to the head. Likewise, after the posterior arm is removed, fundal pressure will also enhance the delivery without requiring excessive traction to the head. Fundal pressure should not be used as the sole means to disimpact a shoulder unless all other maneuvers fail and time is of the essence to save the baby's life.

- *Replace the head and perform a cesarean delivery.*
- *Most important, write a clear and contemporaneous delivery note that describes the elements of the obstetric intervention.* In addition, it is wise to dictate the note.

### The Medical Record

The medical record should reflect what happened in such a way that no one would question the veracity of the note. Acker has developed a shoulder dystocia intervention form that encourages the physician to be clear and concise in the documentation of an incident that is highly probable to result in a legal action [70]. Included in this note is the delivery time, episiotomy, anesthesia, suction, initial traction, maneu-

vers, force, maneuvers and their duration, personnel present, estimated fetal weight, and actual birth-weight. The author offers a medicolegal caveat: the note should not appear blatantly self-serving. Many dictated delivery summaries appear to be read word for word from a textbook. Moreover, do not forget to include which shoulder was anterior. In an evaluation of resident's notes, most did include the correct order of maneuvers used, but most failed to document which shoulder was anterior [71].

The infant's chart should include a physical examination that documents the presence or absence of any injury and whether there was any improvement. Most injuries are not permanent. Especially important is documenting that adequate referral and follow-up were offered.

The medical record is the single most important instrument that can prove a doctor was not negligent in a malpractice claim. If the physician can articulate a reasonable basis for the clinical judgment, and that information is documented in the medical record, then it is extremely difficult for the plaintiff patient to prevail in the action. This is because the plaintiff patient cannot show, through the testimony of a physician expert witness, that the defendant doctor deviated from the standard of medical care in the first instance.

### MEDICOLEGAL ISSUES

The standard of care on which the physician is legally judged is based on reasonableness, not scientific certainty. *Reasonable conduct* is that degree of care expected of the average competent physician, in the same or similar area of expertise, under like or similar circumstances, based on the state of the art at the time. The standard is not based on optimal care. In this respect, the law is much more forgiving than medical peer review.

One practical application of this reasonable standard of care is that the courts are compelled to recognize areas in which even experts disagree [72]. For example, physicians agree that most episodes of shoulder dystocia are unpredictable and rarely result in permanent injury. Thus, the routine use of cesarean delivery for the prevention of dystocia and related injuries is difficult to justify; however, some physicians recommend liberal use of cesarean delivery for those fetuses that one can reasonably believe to weigh more than 4,500 g. In a case in

which there was a large infant, failure to perform a cesarean delivery does not automatically constitute negligence.

It is well recognized that a brachial plexus injury will trigger a claim of medical malpractice. Typically, the plaintiff alleges that some risk factor, sign, or screening procedure associated with a large baby was not recognized or performed by the doctor. The usual scenario involves the failure to perform a glucose screen. A physician expert testifies that this was a deviation from the standard of care and, if performed, it would have been abnormal, ostensibly because the baby was macrosomic. Hindsight can be very accurate. Knowing that the patient was at risk for macrosomia, it is argued, a “reasonable” physician would have either treated the patient for diabetes to prevent macrosomia or performed a cesarean delivery, thereby avoiding the trauma encountered by the vaginal route.

The problems with this scenario are obvious. There might be no association between a positive glucose screen and macrosomia, or between treatment of diabetes and macrosomia, especially in this specific case. Moreover, there is no reasonable way to predict macrosomia with any degree of accuracy to justify a cesarean delivery. Nonetheless, the defendant doctor is at great risk for self-incrimination during a deposition, because the associations in question are well documented in the obstetric literature. The problem lies in the extrapolation of general information to a specific case. The caveat here is this: lawyers are trained to make such inferences, whereas physicians are not.

Another common allegation made in a shoulder dystocia case is that the doctor applied excessive traction on the baby’s neck. The plaintiff will point to the medical record, which often lacks specifics about the method of delivery, and claim that no appropriate maneuver was performed to disimpact the shoulder. The only reasonable conclusion that can be drawn from the events, therefore, is that there was excessive traction on the neck, which caused the brachial plexus injury. With a sparse medical record, a wise lawyer can lead the defendant doctor down the path of self-incrimination based on the inference that a “good” physician documents the procedures performed, especially when there is a poor outcome.

Rarely does a legal case discuss a standard of care, but there is such a case applicable to shoulder dys-

tocia [73]. In this case, the court’s characterization of the defendant doctor’s testimony resulted in his acquittal. The doctor testified that on discovering that the baby had shoulder dystocia, he enlarged the episiotomy, placed his hands behind the baby’s armpits, and attempted to rotate the child. The court noted that this was an indisputably non-negligent act. Note that the doctor did not succeed in preventing an injury; the important fact was that he did what was expected of the average competent physician under the circumstances.

From a medicolegal perspective, *any* reasonable method to resolve the impacted anterior shoulder conforms to the level of care expected of the average competent physician. If the physician can articulate a reasonable basis for the clinical judgment, and that information is documented in the medical record, then the physician has the best defense against a medicolegal entanglement.

Both obstetricians and attorneys agree that shoulder dystocia and its complications are fertile ground for medicolegal arguments. The many reported appellate decisions concerning cases of shoulder dystocia emphasize the potential monetary risks of permanent injury. In one case, a jury awarded \$50,000 for a child’s pain and suffering from a total brachial plexus palsy encompassing both an Erb and Klumpke palsy; the appellate court held the award was too small and ordered the defendant to pay \$300,000 in pain and suffering or retry the case. The defendant chose to retry the case, with the unsurprising result of a \$700,000 verdict for the child’s pain and suffering [74]. The statistics concerning the outcome of shoulder dystocia litigation are deceiving. Whereas most reported appellate decisions involving shoulder dystocia cases resulted in jury verdicts and ultimate decisions for the defendant medical practitioners, numerous other cases settle every year. The number of cases tried with defense verdicts is skewed by the out-of-court settlement of other cases. Obstetricians therefore should not necessarily take solace in the fact that most reported decisions are favorable for the defendants.

### Standards of Care

A review of decisions involving shoulder dystocia indicates a series of factors that lead to successful

lawsuits against physicians. These fall into two general categories: 1) *failure to take appropriate steps, which could have led to diagnosis of probable dystocia prior to an attempt at vaginal delivery*; and 2) *failure to adhere to a proper and safe protocol in actually managing a shoulder dystocia delivery*.

Several risk factors for shoulder dystocia are discussed in the literature. As mentioned previously, among the best established and most problematic is fetal macrosomia, or a large infant. Although the author indicates that there are few absolute indicators to guide the obstetrician in determining when cesarean delivery should be performed (if ever) to prevent problems with fetopelvic disproportion, it is also fair to state that this position is highly controversial. The problem for the clinician is to determine the fetal weight in advance of delivery accurately, and to judge the fetopelvic relationship just as accurately.

Several reported appellate court cases involving shoulder dystocia recite expert witness testimony outlining stepwise plans for dealing with shoulder dystocia as the standard of care [5,75,76]. Much of the liability testimony in the reported appellate shoulder dystocia cases works backward from the injury; that is, it hypothesizes that the trauma would not have occurred in the absence of excessive traction or incorrect maneuvers. Invariably, cases with jury verdicts for the plaintiff include testimony that the defendants applied excessive or improperly directed traction to release the shoulders, thus injuring the baby [77,78]. Based on expert testimony that brachial plexus palsies do not occur in shoulder dystocia cases except for the negligence of the physician involved, several courts have considered whether the legal doctrine of *res ipsa loquitur* ("it speaks for itself") applies in shoulder dystocia cases. This is an important issue that gently calls into question the conclusion of the author of this chapter, based on 19 uncoded patients in the Wishard study, that a brachial plexus injury can and does occur spontaneously.

The doctrine of *res ipsa loquitur* permits a jury to infer negligence based on circumstantial evidence from the mere occurrence of an event in which the injury is of a character that would not ordinarily occur in the absence of negligence. At least two appellate courts in different states have held that the

doctrine of *res ipsa loquitur* is applicable to shoulder dystocia cases involving brachial plexus injuries. This is based on expert testimony that brachial plexus palsies do not occur without someone's negligence. A third appellate court in still another state held that, although the plaintiff's expert in the case testified that the infant's injury could not have occurred without the physician's negligence, the expert witness for the physician presented credible testimony that the injury resulted because the forces of labor placed a strain on the infant's shoulder. The conclusion was that *res ipsa loquitur* did not apply because the appraisal of the circumstances attendant upon the injury-causing event was within the confidence of the ordinary lay jury, as supplemented by the testimony of expert witnesses [79].

### Prevention Strategies

How does the prudent practitioner avert a potential medical negligence lawsuit for shoulder dystocia and a resulting nerve injury? The reported appellate decisions in the shoulder dystocia cases illustrate that the prudent physician should undertake fetal and pelvic evaluations in any case for which there is reason to believe that there is a reasonable possibility of a macrosomic infant. The best answer is thorough evaluation of pelvic size and fetal lie, presentation, position, and weight, using both clinical means and the best available modern technology. With the universal availability of ultrasonography, physicians who do not use ultrasonic imaging when there is suspicion of disproportion or macrosomia are probably inviting a medical negligence lawsuit. Such a lawsuit will probably end favorably for the plaintiff if after delivery the child is found to have sustained a permanent injury. The importance of the mother's obstetric and medical history needs emphasis. Prior difficult deliveries, shoulder dystocia, or macrosomic infants should alert the clinician to possible trouble. A detailed discussion with the mother/family *before* a trial of vaginal delivery in a suspect case, with careful notation of the specifics of the discussion in the medical record, is especially important.

Acute management of dystocia remains a major problem. Some practitioners, on encountering a shoulder dystocia, fail to approach the problem systematically and sometimes panic. Those who do

so often end up the losers in subsequent medical negligence cases. In the event that shoulder dystocia cannot be suspected in advance and avoided, the physician who encounters a dystocia must have an organized and practical plan of approach, involving a practical series of actions performed without panic that avoid excessive traction.

### **Injuries from Shoulder Dystocia: A No-fault System?**

In most shoulder dystocia cases, the prenatal care met the standards expected of the reasonably competent physician. The shoulder dystocia was unpredictable; there was no indication for a cesarean delivery; there was no fetal distress or an obstructed labor; the second stage was normal, and the head delivered easily without episiotomy; the shoulder impacted, and appropriate maneuvers were used without any evidence of excessive traction. If there was a BPP, the physician will be held at fault. Even though the burden of proof is on the plaintiff, for all practical purposes the doctor must defend his/her own innocence. Offering payment, either by settlement or judgment, means the physician is deemed negligent when in fact no such negligence occurred. This situation is ideal for the development of a no-fault compensation system, which spreads the risk among physicians, hospital insurers, and patients (taxpayers) [80]. Such a system entails enacting legislation for it to become viable.

The most practical application of such a system would be to use a panel process based on the Indiana system, whereby a panel of three physicians reviews all of the records in the case to determine whether malpractice occurred. If so, then the case proceeds according to traditional tort litigation. If no malpractice is found, then a patient's compensation panel is left to determine compensation based on an appropriate amount of funds to cover medical expenses to treat the problem, including ongoing physical therapy and surgery, if needed. In addition, a reasonable award for net economic loss should be offered.

This system would ensure that the child receives the proper care. The physician does not suffer the consequences of an adverse determination of negligence, and the cost of litigation would be substantially reduced. A patient's compensation fund would be set up, derived from mandatory contribu-

tions from physicians, hospitals, insurers, attorneys, and the taxpayers. In this system, the majority of the money necessary to compensate the injury would be directed to the child. This system also has an impact on liability premiums and encourages physicians to join and continue in the specialty of obstetrics and gynecology.

### **REFERENCES**

1. Gross TL, Sokol RJ, Williams T, Thompson T: Shoulder dystocia: A fetal-physician risk. *Am J Obstet Gynecol* 1987;156:1408-1418.
2. Nocon JJ, McKenzie DK, Thomas LJ, Hansell RS: Shoulder dystocia: An analysis of risks and obstetrical maneuvers. *Am J Obstet Gynecol* 1993;168:1732-1739.
3. Allen R, Sorab J, Gonik B: Risk factors for shoulder dystocia: An engineering study of clinician-applied forces. *Obstet Gynecol* 1991;77:352-355.
4. Christoffersson M, Kannisto P, Ryhdstroem H, Stale H, Walles B: Shoulder dystocia and brachial plexus injury: A case-controlled study. *Acta Obstet Gynecol Scand* 2003;82:147-151.
5. Dignam WJ: Difficulties in delivery, including shoulder dystocia and malpresentations of the fetus. *Clin Obstet Gynecol* 1976;19:577-585.
6. Resnik R: Management of shoulder girdle dystocia. *Clin Obstet Gynecol* 1980;23:559-564.
7. Hopwood HG Jr: Shoulder dystocia: Fifteen years' experience in a community hospital. *Am J Obstet Gynecol* 1982;144:162-166.
8. American College of Obstetricians and Gynecologists: Shoulder dystocia. Practice Bulletin No. 40, November 2002. Washington, DC: American College of Obstetricians and Gynecologists.
9. Boyd ME, Usher RH, McLean FH: Fetal macrosomia—prediction, risks and proposed management. *Obstet Gynecol* 1983;61:715-722.
10. Cunningham GF, MacDonald PC, Gant NF, Leveno KJ, Gillstrap LC III: *Williams Obstetrics*, 19th ed. Norwalk, CT: Appleton & Lange, 1993; pp. 509-514.
11. Chez R, Carlan S, Greenberg S, Spellacy W: Fractured clavicle is an unavoidable event. *Am J Obstet Gynecol* 1994; 171:797-798.
12. Swaiman KF, Wright FS: *The Practice of Pediatric Neurology*, 2nd ed. St. Louis: CV Mosby, 1982; pp. 1178-1179.

13. Gilbert WM, Nesbitt TS, Danielsen B: Associated factors in 1611 cases of brachial plexus injury. *Obstet Gynecol* 1999;93:536–540.
14. Eng GD: Brachial plexus palsy in newborn infants. *Pediatrics* 1971;41:713–719.
15. Gurewitsch ED, Johnson E, Hamzehzadeh S, Allen RH: Risk factors for brachial plexus injury with and without shoulder dystocia. *Am J Obstet Gynecol* 2006;194:486–92.
16. Brett EM: *Pediatric Neurology*. New York: Churchill Livingstone, 1983.
17. Jennett RJ, Tarby TJ, Kreinick CJ: Brachial plexus palsy: An old problem revisited. *Am J Obstet Gynecol* 1992;166:1673–1676.
18. Graham EM, Forouzan I, Morgan MA: A retrospective analysis of Erb's palsy cases and their relation to birth weight and trauma at delivery. *J Matern Fetal Med* 1997;6:1–5.
19. American College of Obstetricians and Gynecologists: Fetal macrosomia. Technical Bulletin No. 159. Washington, DC: American College of Obstetricians and Gynecologists, 1991.
20. Tamura RK, Sabbagha RE, Depp R, Dooley SL, Soel ML: Diabetic macrosomia: Accuracy of third-trimester ultrasound. *Obstet Gynecol* 1986;67:828–832.
21. Miller JM Jr: Identification and delivery of the macrosomic infant. In: Plauché WC, Morrison JC, O'Sullivan MJ (eds): *Surgical Obstetrics*. Philadelphia: WB Saunders, 1992; pp. 313–323.
22. Modanlou HD, Dorchester WL, Thorosian A, Freeman RK: Macrosomia: Maternal, fetal, and neonatal implications. *Obstet Gynecol* 1980;55:420–424.
23. Anderson GD, Blinder IN, MacClermont S, Sinclair JC: Determinant of size at birth in a Canadian population. *Am J Obstet Gynecol* 1980;150:236–244.
24. Klebanoff MA, Mills JL, Berendes HW: Mother's birth weight as a predictor of macrosomia. *Am J Obstet Gynecol* 1985;153:253–257.
25. Seidman DS, Ever-Hadani P, Gale R: The effect of maternal weight gain in pregnancy on birth weight. *Obstet Gynecol* 1989;74:240–246.
26. Klebanoff MA, Yip R: Influence of maternal birth weight on rate of growth and duration of gestation. *J Pediatr* 1987;111:287–293.
27. Spellacy WN, Miller S, Winegar A, Peterson PQ: Macrosomia – maternal characteristics and infant complications. *Obstet Gynecol* 1985;66:158–160.
28. Elliot JP, Garite TJ, Freeman RK, McQuown DD, Patel JM: Ultrasonic prediction of fetal macrosomia in diabetic patients. *Obstet Gynecol* 1982;60:159–162.
29. Hirata GI, Medearis AL, Horenstein J, Bear MB, Platt LD: Ultrasonographic estimation of fetal weight in the clinically macrosomic fetus. *Am J Obstet Gynecol* 1990;162:238–242.
30. Delpapa EH, Mueller-Heurbach E: Pregnancy outcome following ultrasound diagnosis of macrosomia. *Obstet Gynecol* 1991;78:340–343.
31. Eden RD, Seifert LS, Winegar A, Spellacy WN: Perinatal characteristics of uncomplicated postdate pregnancies. *Obstet Gynecol* 1987;69:296–299.
32. Lazer S, Biale Y, Mazor M, Lewenthal H, Insler V: Complications associated with the macrosomic fetus. *J Reprod Med* 1986;31:501–504.
33. Ouzounian J, Naylor CS, Gherman R, Kamath M, Johnson M, DeLeon J, Anguiano H: Recurrent shoulder dystocia: How high is the risk? *Am J Obstet Gynecol* 2001;185:6:S108.
34. Acker DB, Sachs BP, Friedman EA: Risk factors for shoulder dystocia. *Obstet Gynecol* 1985;66:762–768.
35. Acker DB, Sachs BP, Friedman EA: Risk factors for shoulder dystocia in the average-weight infant. *Obstet Gynecol* 1985;67:614–618.
36. Benson CB, Doubilet PM, Saltzman DH: Sonographic determination of fetal weights in diabetic pregnancies. *Am J Obstet Gynecol* 1987;156:441–444.
37. Benedetti TJ, Gabbe SG: A complication of fetal macrosomia and prolonged second stage of labor with mid-pelvic delivery. *Obstet Gynecol* 1978;52:526–529.
38. Chelmow D, Kilpatrick SJ, Laros RK: Maternal and neonatal outcomes after prolonged latent phase. *Obstet Gynecol* 1993;81:486–491.
39. Friedman EM: *Labor: Clinical Evaluation and Management*. New York: Appleton-Century-Crofts, 1978.
40. Gherman RB, Ouzounian JG, Satin AJ, Goodwin TM, Phelan JP: A comparison of shoulder dystocia-associated transient and permanent brachial plexus palsies. *Obstet Gynecol* 2003;102:544–548.
41. Swartz DP: Shoulder girdle dystocia in vertex delivery – clinical study and review. *Obstet Gynecol* 1960;15:194–206.
42. Williams JW: *Obstetrics*, 5th ed. New York: D. Appleton & Co., 1926.



43. Sandmire HF, DeMott RK: Erb's palsy: Concepts of causation. *Obstet Gynecol* 2000;95:941-942.
44. Moir JC, Myerscough PR: *Munro Kerr's Operative Obstetrics*, 8th ed. London: Baillière-Tindall and Cassell, 1971; p. 66.
45. Gonik B, Zhang N, Grimm MJ: Prediction of brachial plexus stretching during shoulder dystocia using a computer simulation model. *Am J Obstet Gynecol* 2003;189:1168-1172.
46. Wechsler IS: Ch. 24 Peripheral nerve injuries. In *Clinical Neurology*, 9th ed. Philadelphia: W.B. Saunders Co., 1972.
47. Gross SJ, Shime J, Farine D: Shoulder dystocia: Predictors and outcomes - A five-year review. *Am J Obstet Gynecol* 1987;156:334-336.
48. Baskett TF, Allen AC: Perinatal Implications of Shoulder Dystocia. *Obstet Gynecol* 1995;86:14-17.
49. Gherman RB, Ouzounian JG, Goodwin TM: Obstetrical maneuvers for shoulder dystocia and associated fetal morbidity. *Am J Obstet Gynecol* 1998;178:1126-1130.
50. Speert H: *Iconographia Gyniatrix: A Pictorial History of Gynecology and Obstetrics*. Philadelphia: FA Davis, 1973.
51. Beer E: History of extraction of the posterior arm to resolve shoulder dystocia. *Obstet Gynecol Survey* 2006;61(3):149-151.
52. McCormick CO: *Pathology of Labor, the Puerperium, and the Newborn*, 2nd ed. St. Louis: CV Mosby, 1947.
53. Castallo MA, Ullery JC: *Obstetric Mechanisms and Their Management*. Philadelphia: FA Davis, 1957.
54. Hibbard LT: Shoulder dystocia. *Obstet Gynecol* 1969;34:424-429.
55. Gonik B, Stringer CA, Held B: An alternate maneuver for management of shoulder dystocia. *Am J Obstet Gynecol* 1983;145:882-884.
56. Gonik B, Allen R, Sorab J: Objective evaluation of the shoulder dystocia phenomenon: Effect of maternal pelvic orientation on force reduction. *Obstet Gynecol* 1989;74:44-47.
57. Poggi SH, Allen RH, Patel CR, Ghidini A, Pezzullo JC, Spong CY: Randomized trial of McRoberts versus lithotomy positioning to decrease force that is applied to the fetus during delivery. *Am J Obstet Gynecol* 2006;195:1544-1549.
58. Beall MH, Spong CY, Ross MG: A randomized controlled trial of prophylactic maneuvers to reduce head-to-body delivery time in patients at risk for shoulder dystocia. *Obstet Gynecol* 2003;102:31-35.
59. Woods CE: A principle of physics as applicable to shoulder delivery. *Am J Obstet Gynecol* 1943;45:796-804.
60. Rubin A: Management of shoulder dystocia. *JAMA* 1964;189:835-837.
61. Gurewitsch ED, Kim EJ, Yang JH, Outland KE, McDonald MK, Allen RH: Comparing McRoberts' and Rubin's maneuvers for initial management of shoulder dystocia: An objective evaluation. *Am J Obstet Gynecol* 2005;192:153-60.
62. Schwartz BC, Dixon DM: Shoulder dystocia. *Obstet Gynecol* 1958;11:468-471.
63. Poggi SH, Spong CY, Allen RH: Prioritizing posterior arm delivery during severe shoulder dystocia. *Obstet Gynecol* 2003;101:1068-1072.
64. Gurewitsch ED, Donithan M, Stallings SP, Moore PL, Agarwal S, Allen LM, Allen RH: Episiotomy versus fetal manipulation in managing severe shoulder dystocia. *Am J Obstet Gynecol* 2004;191:911-916.
65. Sandberg EC: The Zavanelli maneuver extended: Progression of a revolutionary concept. *Am J Obstet Gynecol* 1988;158:1347-1353.
66. Graham JM, Blanco JD, Weu T, Magee KP: The Zavanelli maneuver: A different perspective. *Obstet Gynecol* 1992;79:883-884.
67. O'Leary JA, Leonetti HB: Shoulder dystocia: Prevention and treatment. *Am J Obstet Gynecol* 1990;162:5-9.
68. McFarland M, Hod M, Piper JM, Elly M-JX, Langer O: Are labor abnormalities more common in shoulder dystocia? *Am J Obstet Gynecol* 1995;173:1211-1214.
69. Poggi SH, Stallings SP, Ghidini A, Spong CY, Deering SH, Allen RH: Intrapartum risk factors for permanent brachial plexus injury. *Am J Obstet Gynecol* 2003;189:725-729.
70. Acker DB: A shoulder dystocia intervention form. *Obstet Gynecol* 1991;78:150-151.
71. Deering S, Poggi S, Hodor J, Macedonia C, Satin AJ: Evaluation of resident's delivery notes after a simulated shoulder dystocia. *Obstet Gynecol* 2004;104:667-670.
72. Keeton WP, Dobbs DD, Keeton RE, Owen DG: *Prosser and Keeton on the Law of Torts*, 5th ed. St. Paul, MN: West Publishing, 1984.

73. *Dunne v. Somoano*, 550 So. 2d 5 (Fla. App. 3 Dist. 1989).
74. *Sutherland v. County of Nassau*, 593 N.W. 2d 287 (1993).
75. *James v. Woolley*, 523 So. 2d 110 (Ala 1988).
76. *Smith v. Nguyen*, 855 S.W. 2d 263 (Tex App Houston [14th Dist.] 1993, writ denied).
77. O'Grady JP: *Modern Instrumental Delivery*. Baltimore: Williams & Wilkins, 1988.
78. *Reilly v. Straub*, 282 N.W. 2d 688 (Iowa 1979).
79. *Abbott v. New Rochelle Hospital Medical Center*, 529 NYS 2d 352 (1988).
80. Minkin MJ: A no-fault approach to shoulder dystocia. *Contemporary OB/GYN* 2004;49(12):48-50.

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## Chapter 15 INTRAPARTUM AND POSTPARTUM: LEGAL COMMENTARY II

### Kevin Giordano

*At a time when reasoning from real facts and accurate observations has taken the place of idle theory in almost every other science, and has with particular advantage been applied to many branches of medicine, no apology seems necessary for trying the same method of reasoning on this important subject, which has hitherto been too much governed by arbitrary custom, and ignorant prejudice.*

Charles White (1728–1813)

*A Treatise on the Management of Pregnant and Lying-in Women.*

London, Dilly, 1773, p. viii

Obstetricians practice in a highly litigious specialty. In fact, the most prevalent patient condition that gives rise to claims of malpractice cases against physicians is pregnancy [1]. In cases involving allegations of birth trauma resulting in a severely impaired infant, jury verdicts can be remarkable. Obviously, sympathy can be a very significant component in large jury verdicts. Furthermore, when the jury has determined liability, in certain cases a large verdict can be intended by the jury as a message about a physician's particular method of practice or or his/her uncaring demeanor as established by the evidence. The potential for substantial verdicts in obstetric cases are the significant driving force behind tort reform, particularly with respect to damage caps, which establish a ceiling to which the jury can award damages for noneconomic awards.

The impediment to developing tort reform, however, is the difficulty in developing a system that is fair to the litigants and to those responsible for paying the claims. Those who advocate against tort reform argue that large verdicts do not necessarily represent verdicts that are excessive, particularly in cases of the so-called brain-damaged baby. In determining an award of damages, a jury is instructed that there are two separate types of compensatory damages, economic and noneconomic. Both types of damages are intended to compensate victims of negligence for their injuries; however, they do so in different ways. Economic damages, often referred to as out-of-pocket losses, include the past and future cost of medical and rehabilitation care, educational care, and other related losses (i.e., future support needs and the child's loss of earning capacity). Typically in such cases, evidence of the cost of future care is established during the trial by economists and life care planners about the costs of future care and maintenance of the child. These costs can be astronomical. Noneconomic losses include claims for pain and suffering, mental anguish, injury, and disfigurement, and juries are instructed that consideration must be given to the extent of any

permanent neurologic injury suffered by the child and emotional and physical demands placed on the parents caring for a severely compromised child.

Although the intent of each type of damage is intended to be compensatory in nature, they are fundamentally different. The determination of noneconomic damages is subjective, and the jury is left to its collective wisdom to determine the amount of money to award, whereas economic damages can be mathematically calculated, and the jury relies upon the evidence to determine the amount to award. Because of the catastrophic nature of many obstetrical cases, the question ultimately becomes whether a jury's verdict on damages, albeit large, is truly excessive. Consider the following case. In *Gourley v. Nebraska Methodist Health System*, the plaintiff was carrying twin fetuses [2]. During the 36th week of her pregnancy, the plaintiff noted less movement, so she contacted her obstetrician. The plaintiff was advised that a decrease in fetal movement was common and that everything appeared to be normal. Two days later, the plaintiff again contacted her physician with the same concern. In response, she was told to come to the office for evaluation. Examination revealed a lack of amniotic fluid and that one of the fetuses suffered from bradycardia. The patient was referred to the hospital for further assessment. Following her clinical evaluation at the hospital by a specialist in maternal-fetal medicine, an immediate cesarean delivery was ordered. Shortly thereafter, both babies were delivered; one was born with brain damage. By the time of trial, the injured child had been diagnosed with cerebral palsy and significant physical, cognitive, and behavioral difficulties.

The mother filed suit, alleging failure to properly monitor her pregnancy. At trial, the plaintiff presented evidence of damages that included a specialist in physical medicine and rehabilitation was called to testify about the life care plan that had been developed for the child. A *life care plan* is a comprehensive document that is developed to establish the likely expenses and costs associated with the needs of caring for and supporting a disabled person. The included costs are for reasonable value of medical, hospital, nursing, therapy, rehabilitation, medical equipment, and similar care and supplies that a disabled person will need over the course of his/her life, as well as the cost of developmental education. In this case, the evidence showed that the child suffered severe brain damage and for the rest of his

life would be afflicted by cerebral palsy and extensive physical, cognitive, and behavioral deficiencies. The economic evidence presented was that the child would need a total of \$12,461,500.22 for all of the items identified in the life care plan. Discounting for present-day value, the amount was a minimum of \$5,943,111. Apparently because of the fact that the plaintiff's expert was unable to state with reasonable certainty that all costs identified in his plan would actually be necessary, the jury awarded \$5 million in damages. Thus, the jury essentially compensated the plaintiff for her economic damages. Unbeknownst to the jury, however, tort reform capping damages had been enacted, limiting damage awards to \$1,250,000. The plaintiff appealed the case in an effort to avoid application of the cap because it represented only 25% of the total economic damages awarded. The statute at issue conveyed a privilege to all healthcare providers whose negligence causes catastrophic damages, defined as damages in excess of \$1,250,000. For damages caused that exceed that amount, healthcare providers are no longer liable. The plaintiff's appeal was denied because the court determined the legislative intent at the time of enacting the legislation was clear. As a result of the legislation in effect at the time, the family would receive less than one fourth of child's economic expenses alone. In denying the plaintiff's appeal, the *Gourley* court noted that "... the facts of the instant case demonstrate the callous effect of denying recovery for economic damages."

There are those who would argue that even when legislation capping jury awards exclude economic damages, such limitations are unjust. They rely on the catastrophic damages incurred by a family beyond the economic damages. For instance, consider the case of *Wareing v. United States* [3]. The defendant obstetrician conceded that he departed from good practice by failing to perform a cesarean delivery in a timely manner. As in all tort cases involving the United States, the presiding Federal District Court judge and not a jury makes the findings and awards damages. In this case, after hearing the evidence, the judge awarded \$1.5 million in noneconomic damages. He based his award on expert testimony that established that the child was profoundly and permanently neurologically impaired and that such deficits would leave him intellectually, socially, and functionally limited. In fact, the judge determined that the weight of the

evidence suggested that, although the minor plaintiff was a happy child who is active and attempts to function within a normal range in his peer group, he would forever be limited by the brain injury he had suffered. Among other things, the evidence revealed that the child would ultimately function at a ten- to twelve-year-old age level and that as the child got older he would become increasingly aware of his limitations. This awareness created concerns regarding the plaintiff's ability in "dealing with sexual changes, sexual differences, and possible impulsivity and aggressiveness." Because the plaintiff was unable to think abstractly, it was determined that he would be locked into a child's level of concrete functioning. Indeed, at the age of 10 years, the plaintiff's cognitive functioning in some areas had plateaued and would not improve. The judge concluded that there was evidence that the child was "... already experiencing problems in Easter Seals with the friends he has made. They are progressing at a more rapid rate than he is, and so therefore they are leaving him by the wayside and going and playing with their other friends." This would essentially always be the case. In connection with toiletry, the plaintiff's mother testified that "[he] took a real long time to toilet train," and, although he was taught the use of the toilet, he remained "really bad" at taking care of himself in the bathroom. The mother stated that he would often urinate on the floor and that "his underwear ha[s] a residue of stool most days" and even after toileting it was soiled. The father testified that he and his wife continually had been working with him to improve his personal hygiene skills, but he has not significantly improved over the course of many years.

As to his adult years, his neurologic impairments would restrict his employment options to a position through a charitable organization such as Goodwill, which provides menial labor jobs with required supervision. The better weight of the evidence suggested that the plaintiff would require a "supported employment environment," where he can be placed with an outside employer with a job coach and real supervision. The *Wareing* case illustrates the burden that negligence causing severe brain damage can have on a permanent basis, as well as the impact imposed on parents and caregivers by such medical negligence. Determining a fair verdict is difficult under any circumstances. With caps on noneconomic damages, a child born with an affliction that is

the result of medical negligence is limited in his/her ability to recover damages. Many states impose a cap of \$250,000, whereas in other states it is \$500,000. In this case, or in cases in which the child is even more severely harmed, the amount of noneconomic damages is reduced to the cap amount. Consumer advocacy groups argue that capping damages in cases like the *Wareing* case does not provide adequate compensation to the injured parties given the emotional demands and challenges of raising a severely impaired child can have upon the individual parents and family.

Regardless of whether the award is justified, awards following jury trials represent only a fraction of medical malpractice payments. It has been reported that jury verdicts constitute approximately 3% of payments made by medical malpractice insurers. In a study of Florida malpractice cases closed between 1990 and 2004, investigators found that there were in excess of 800 cases involving payments made by an insurer of more than \$1 million. Of those 800 cases or more, only 54 cases involved jury trials; and as a subset of the 54 cases, only 6 were obstetric cases. Furthermore, of the 800 cases analyzed, there were 34 cases in which an indemnity payment of \$5 million or more was made by an insurer. Of these 34 cases, only two involved jury verdicts in an obstetric case, the other 32 payments of \$5 million or more came as a result of the insurer settling the case or other alternative dispute resolution.

Tort reform will remain an on-going debate, because clearly the impact of insurance premiums has placed a difficult burden on the healthcare system. This study appears to suggest that excessive jury verdicts by themselves are not in and of themselves the predominant factor having an impact on the increase in indemnity payouts, assuming that similar studies in other states would be somewhat consistent. The insurer's decision to take a case to trial or settle is largely risk dependent. That risk analysis must include the evaluation of an adverse jury verdict and potential sympathy factors that could create a concern about a runaway jury verdict. Support for tort reform seeks to address this aspect of the insurer's risk. Risk assessment also depends greatly on the clinical care that was provided, documentation, and the bedside manner or appearance of concern by the obstetrician – all issues that are within the control of the obstetrician.

Concomitant with efforts to obtain tort reform, obstetricians also must focus on improving health-care and outcomes and on better documentation of the clinical thought process and events surrounding care. Ultimately the best way to reduce liability claims is for obstetricians to avoid the avoidable injury. In a study analyzing 90 closed claim files involving obstetrics and gynecology, the investigators concluded that 78% of the cases that were evaluated appeared to have at least one potentially preventable cause [4]. The authors of this study do not suggest that in all the cases malpractice was present. Instead, the suggestion is that adverse outcomes could have been prevented and the potential for a lawsuit thereby averted. Events most commonly associated with an adverse obstetric outcome were inpatient monitoring and treatment of complications of pregnancy, including preeclampsia, preterm labor, premature rupture of membranes, vaginal birth after cesarean (VBAC), and abruption. Diagnosis and treatment-related errors were common in 49% of all cases. Notably, communication failures were identified in 31% of the cases; these failures occurred among caregivers, in patient education, or in communications that upset the patient or family. Documentation errors were found in 9% of the cases, including two cases in which failure to document had a direct impact on care.

A common misconception is that preventable adverse events are the product of human error, some individual deficiency in failing to meet performance requirements, or from sheer bad luck. Even in many cases in which the negligence can be attributed to one particular individual failure, systems errors contribute to or permit the error to occur [5]. Additionally, often other providers perceive or should perceive the significance of the events but fail to take any steps to “rescue” the situation.

Obviously, one additional step toward reducing medical malpractice claims is for obstetricians to undertake efforts to improve patient communication. These efforts should be aimed at educating the patient about the care plan and expectations, effectuating proper informed consent, and face-to-face conversations in the event of an unexpected outcome. One of the more commonly cited reasons for a malpractice lawsuit is the failure of the physician to explain the events surrounding a bad outcome. Plaintiffs have often indicated that they filed a lawsuit to “find out” what happened to their child.

## **OBSTETRIC ANESTHESIA**

Pregnancy, labor, and delivery are associated with major physiologic changes that can decrease maternal reserves. Consequently, various techniques of analgesia and anesthesia can have profound effects on maternal physiology. Furthermore, obstetric pain management and operative obstetric anesthesia are recognized secondary causes of neonatal respiratory depression. The practicing obstetrician therefore must have an understanding of the general principles and techniques for obstetric anesthesia.

This risk for serious adverse medical outcome, when coupled with the high risk that both obstetrics and anesthesiology carry, creates a significant concern for legal action in the event of a complication. It is true that maternal mortality from anesthetic causes has fallen in the United States in recent decades. Currently, the leading causes of mortality during pregnancy include hemorrhage, embolism, and hypertensive disorders [25]. This decline in anesthesia-related deaths in pregnancy is mostly a result of the marked reduction in deaths associated with regional anesthesia. Improvements in the types of anesthetic drugs administered and implementation of test-dose regimens are two significant factors that have led to this decline in mortality. Despite the dramatic reduction in maternal mortality, the number of deaths that occur remains a concern. Although there has been a significant improvement in maternal mortality rates from regional anesthesia, there has not been any significant improvement in the number of deaths attributable to general anesthesia. From a risk management perspective, however, whether related to regional or general anesthesia, many anesthesia-related maternal deaths are preventable [25].

Serious but nonfatal events remain a concern as well. Claims for maternal brain death and newborn brain damage are among the most common claims made. Difficulties with airway management, including intubation and pulmonary aspiration, represent a significant portion of malpractice claims involving obstetric anesthesia [25]. Thus, in addition to maternal death, maternal and fetal injury, including brain damage, permanent nerve injury, and aspiration-related illness such as pneumonitis, are among the complications that have resulted in a significant number of malpractice claims. Even injuries of a relatively minor degree, such as postdural

headache, pain occurring during anesthesia, and chronic back pain, have a greater likelihood of generating a malpractice claim in obstetric patients when compared with the nonobstetric population [25].

Why is anesthesia-related malpractice anything other than of interest to the obstetrician, given that a separate specialty of medicine is involved in its administration and management? It is true that the *captain of the ship doctrine*, which would hold an obstetrician-surgeon liable essentially for all malfeasance that occurs in the operating suite, has been abandoned [pun intended]. Similarly, the obstetrician does not have *per se* liability for all malfeasance that arises from an order to implement anesthetic analgesia. To the contrary, courts have recognized that, given the distinct areas of medicine involved, an obstetrician is not in control of all care provided to his/her patient. As the court recognized in *Lanzet v. Greenberg* [26], during a cesarean it is the anesthesiologist's responsibility to maintain the vital functions of the patient as near normal as possible. Thus, the *Greenberg* court determined that because it was the anesthesiologist's role to monitor the patient's vital signs, holding the obstetrician liable for the failure to resuscitate sooner would make physicians susceptible to malpractice even though the negligence was attributable to a provider not under his/her control or direction. This line of reasoning is almost universally accepted.

An obstetrician cannot merely wash his/her hands of the situation, however; liability can arise if, in the eyes of the court, there is evidence that the obstetrician was able to control the situation. In fact, a Kansas Court held in *Oberzan v. Smith* [27] that a surgeon usually is liable for the negligence of an anesthesia resident or nurse anesthetist under the captain of the ship doctrine. The court premised its determination on the "right of control" and a determination that an agency relationship existed. In the eyes of the court, in these circumstances the obstetrician-surgeon has control of both the care provided by the non-anesthesiologist and the manner in which it is performed. Ultimately, this is not a significant divergence from most jurisdiction. The Kansas Court would appear to agree that if that right of control did not exist, even though the obstetrician might be considered to be supervising, that role does not convey automatic liability for the actions of the nurse anesthetist or anesthesia resident. The

liability in each case rises and falls on its own specific facts.

There are certain acts or omissions that traditionally have given rise to medical negligence actions, including claims against the obstetric team. The most common allegations of malfeasance include 1) failure to properly train and supervise the staff and medical personnel attending patients, employees or agents of the hospital, including but not limited to the labor and delivery nursing staff; 2) negligently failing to provide an adequate and accurate record of the anesthetic drugs administered and the patient's responses to those anesthetic drugs; 3) failure to properly inform the patient and obtain her consent before administering obstetric anesthesia; 4) failure to follow established anesthesia procedures or protocols or the failure to have such procedures and protocols established.

Considerable disparity among hospitals remains about both the availability and implementation of obstetric anesthesia services. Efforts at standardization of management of obstetric analgesia and anesthesia through American Society of Anesthesiologists' (ASA) guidelines and hospital policies and protocols has been effective at reducing complications, as well as supporting that the provider's compliance has been consistent with the standard of care when an adverse outcome does occur. Practice often does not mirror the guidelines or policies, and rather than shielding against liability, they are used to create it. For instance, the ASA guidelines state that an anesthesiologist should initiate regional anesthetic, whereas a CRNA may monitor its effect. After the ASA promulgated this guideline, many hospitals' policies adopted very similar language. Meanwhile, in the clinical setting, as these policies were being created, CRNAs routinely provided all aspects of anesthesia services to obstetric patients [28]. When a complication in this setting became the substance of a malpractice case, plaintiffs could then cite this violation of the hospital's own anesthesia policy as the evidence for a deviation from the standard of care. This disparity became the issue in the case of *Denton v. LaCroix* [29]. The *Denton* case involved a woman who during a cesarean delivery suffered a hypoxic brain injury after the onset of a seizure that prevented intubation by the CRNA. When the patient arrived at the hospital in labor, the hospital required her to sign an anesthesia consent form. The consent form authorized that any physician in the

anesthesia group could provide care and specifically identified each anesthesiologist by name. None of the anesthesiology attending physicians knew that the hospital was obtaining the anesthesia consent for patients in this manner, nor were they aware of the specific form that was being used. Prior to signing the consent form, the patient had never received a preanesthetic evaluation by an anesthesiologist, nor had any anesthesiologist ever explained the anesthesia consent to her. Ultimately, the patient needed a cesarean delivery because of a nonreassuring fetal heart rate tracing and slow progress in her labor. The cesarean was considered by the obstetrician to be an emergency in that it was unscheduled; however, it was not an urgent situation nor considered to be a true emergency in the obstetric sense of needing to be performed immediately. While the patient was being prepped for delivery, she developed a seizure. The CRNA could not establish an airway because the patient's teeth were clenched shut. The obstetrician had already commenced the abdominal incision when the seizure occurred, and he continued to deliver the baby. By the time of delivery, the mother became apneic and had to be resuscitated. The CRNA was finally able to establish an airway. To do so, however, she had to paralyze the patient with succinylcholine and administer sodium pentothal. It was only then that the CRNA was able to intubate.

Unfortunately, it was determined that an esophageal and not a tracheal intubation had occurred. The obstetrician, who was still working inside the mother's abdomen, pointed this out to the CRNA. The CRNA removed the tube, and a successful intubation was subsequently accomplished. Unfortunately, marked hypoxia had developed, and, as a consequence of these events, the parturient suffered an irreversible brain injury. She was comatose for 3 days and hospitalized for a total of 13 days, after which she was then transferred to a rehabilitation hospital. Her subsequent intellectual function was seriously impaired, and she was totally and permanently disabled. At trial, the jury found for the plaintiff.

It is possible to criticize the outcome in *Denton*, in that there was considerable evidence that the outcome would not have been different had an anesthesiologist been present from the outset. The verdict favoring the plaintiff is presumed largely to be based on the uncontested violation of hospital pol-

icy, which stated that regional anesthetics were to be administered by an anesthesiologist. Also of significance was that the patient's consent, perhaps without her even being cognizant of the fact, authorized only the identified anesthesiologists to administer regional anesthesia. These factors were both under the control of the hospital and/or the anesthesia department. Although the practice at the institution was to have CRNAs administer regional anesthesia and monitor the patient afterward, they had instituted a policy contradictory to that approach. In the high-stakes setting of obstetrics and obstetric anesthesia, policies and protocols are important. These policies and protocols play an important role in legal actions should there be a departure from the substance that is contained within.

Medication errors in any setting are a constant theme in malpractice lawsuits. Extreme care must be taken to monitor for the effects of analgesics or sedatives that are administered to a laboring patient. Failure to position the patient properly during the administration of such medications can lead to supine hypotension, the obstruction of blood flow from the legs and pelvis of the patient back to the patient's heart. Failure to recognize an adverse drug reaction, prescribing or administering too much drug at one time, or administering an excessive amount over a period of time, and the choice of route (i.e., intravenous instead of intramuscular) have all been identified as leading to malpractice litigation, not just against the anesthesia or nursing staff, but against the obstetrician as well. Vigilance by all members of the obstetrical team will help reduce the avoidable bad outcomes, thereby decreasing the potential of a lawsuit. It is always valuable to develop a good working relationship with the anesthesiology staff, who might be called upon to provide care in an emergency situation. Obtaining adequate informed consent is also an important feature in reducing bad outcomes. As with any treatment option, the material risks and benefits of anesthesia or analgesia must be conveyed to the patient as well as the alternatives to the proposed treatment and consequences that may occur of not proceeding with the discussed treatment option.

The obstetrician should consider the following:

- Obstetric analgesia and anesthesia modify or suspend a variety of normal functions, can affect



labor, and involve the risk of complications to the parturient as well as the baby. Therapeutic strategies must be developed to circumvent these effects. These strategies must contemplate interaction with the compounding influences of obstetric agents as well as possible illicit drugs.

- In all areas of anesthesia, patients must have realistic expectations and a full understanding of the potential major and minor complications associated with their procedure. The consequences of inappropriate expectations, even about pain management, can lead to patient dissatisfaction and a greater potential for a malpractice case in the event of an adverse outcome, even if that outcome is a relatively minor injury.
- A team approach from obstetricians, anesthesiologists, and nurses, with good communication overall, improves the patient's confidence and can make a claim less likely for an unexpected outcome.
- Obstetric anesthesia for purposes of operative intervention requires attention to the health of both the mother and the baby. Appropriate anesthetic selection and administration, with proper monitoring, can reduce the inherent maternal and fetal risks. Administration and management must comply with the guidelines and standards of anesthesia care, however.
- Although general anesthesia is still recommended in certain circumstances (e.g., prolapsed cord or massive hemorrhage), ACOG advocates greater use of regional anesthesia for emergency cesarean delivery. Antepartum risk assessment minimizes potential complications if and when emergency anesthesia and intervention are necessary.
- Hospital policies and protocols governing obstetric analgesia and anesthesia must be reviewed regularly to ensure that they comply with the current standards of care. In addition, clinical practice should be consistent with these policies and protocols.
- Obstetric and anesthesia teams should continually work together to improve procedures and communication. Drills rehearsing emergency situations that might be encountered should be imple-

mented to ensure that competent care is delivered when time is of the essence.

## LABOR

Improper management of labor is the common claim in obstetrical malpractice cases. Malpresentation and/or dystocia are some of the most fertile areas for medical negligence lawsuits. The delivery of an infant requires balancing risks to the mother against those to the infant. In this respect the advent of modern technology has given the physician the tools to assist in this balancing act; however, successful lawsuits abound in which the practitioner fails to use or delays usage of available diagnostic techniques, such as real-time ultrasonography, clinical pelvimetry, or electronic fetal monitoring. The physician might also be held liable when there is an unjustified delay in performing a cesarean delivery. In addition, the practitioner can be liable for the negligent administration of or failure to monitor oxytocin during induction or augmentation of labor. In this brief critique, these and several related issues are considered.

Unfortunately, there is no available diagnostic technique except labor that can establish which fetus will or will not successfully negotiate the maternal pelvis. All experienced clinicians have had the experience of confidently predicting dystocia only to witness a rapid, uncomplicated labor. Despite the inability to predict an abnormal labor short of a trial of labor (TOL), juries have held physicians liable for failure to use pelvimetry, ultrasound scanning, and/or other types of fetal evaluation such as biophysical profile monitoring in assessing maternal prenatal status or abnormal labor [30]. In addition, a jury might also hold a physician liable where electronic fetal monitoring is readily available but is not used. The legal peril facing the experienced accoucheur who fails to use ultrasound, pelvimetry, or electronic fetal monitoring is demonstrated by this typical instruction given, in this instance, to a jury by the court in a Rhode Island obstetric negligence case:

**Now, further, members of the jury, in considering this question [medical negligence], I instruct you: If a physician, as an aid to treatment or diagnosis, does not avail himself of all of the scientific means and facilities available**

**to him so that he can obtain the best factual data upon which he can make a diagnosis and treatment of the patient, such an omission can be considered as evidence of negligence [31].**

Even when a jury fails to find the physician liable for not using available equipment, some appellate courts have reversed the jury's finding and held the physician liable as matter of law. An older case that serves as an example is in the instance in which an Illinois Court of Appeals found that the failure of the defendant doctor to have available or use Piper forceps during delivery of a baby in breech presentation required an entry of judgment against the doctor, notwithstanding the jury verdict [32].

Real-time ultrasound scanning is a new and important tool in assessing fetal positioning and station. Transperineal or transvaginal ultrasound scans can identify specific landmarks, including the maternal symphysis and the fetal calvarium, the fetal orbits, and early signs of edema. An experienced sonographer can rapidly determine the position of the fetal head and if it is engaged. Although radiographic pelvimetry in evaluating dystocia in cephalic presentations is of limited value, the situation is more complex for breech presentations. Based upon expert testimony given at trial, at least one appellate court has found the failure to have x-ray pelvimetry or ultrasound a basis for sustaining a jury verdict in favor of the plaintiffs in a breech presentation/head dystocia case [33]. Similarly, the fact finder often considers expert testimony as to whether the physician ordered or performed either x-ray pelvimetry or ultrasound studies to determine whether either cephalopelvic disproportion or a macrosomic fetus is likely to be present [34].

In addition, there are numerous cases every year involving the failure to use x-ray pelvimetry or ultrasound in fetopelvic evaluation that are settled and do not reach the appellate system, particularly in light of the legal community's increasing reliance on alternative dispute resolution. For example, in one case a \$900,000 settlement resulted from a defendant's failure to order either a sonogram or an x-ray pelvimetry to determine fetal size and/or estimate the fetopelvic relationship [35].

Pelvimetry, both clinical and radiographic, is alive and well in the expert testimony of negligence cases when the fact finder is evaluating the procedures and equipment used by the defendant/doctor to judge

the fetopelvic relationship. This is especially true in cases of failure to progress. In breech presentation, delay in ordering and procuring x-ray pelvimetry to determine fetopelvic relationship, fetal position, and/or fetal presentation can lead to a finding of negligence. Further, plaintiff's experts and attorneys can cite literature in which similar recommendations have been made. For instance, one source states:

**In cases of breech presentation, x-ray pelvimetry or a combination of pelvimetry and ultrasound measurements of the fetus, when combined within standard management protocols, significantly reduce rates of cesarean delivery. A combination of pelvimetry and ultrasound also appears to be useful in the management of macrosomia and failure to progress [36].**

There is no available technique that provides sufficient accuracy to *absolutely* establish which fetus will or will not successfully navigate the maternal pelvis except a TOL. To rely on a TOL alone as the sole measure of possible disproportion is unwise. *In uncertain cases, the medical record must reflect reasonable efforts to evaluate both fetal size and maternal pelvic capacity.* Juries have found that the practice of simply allowing a patient to proceed in labor to determine whether there was true cephalopelvic disproportion was negligent when there were other data indicating that this course was inappropriate [37]. In one reported case, the physician had obtained an x-ray pelvimetry that suggested disproportion; however, despite being faced with a mother at high risk, the court noted that the physician did not use sonographic data or information from a glucose tolerance test or electronic fetal monitoring. In fact, the doctor stated that it was his practice to let patients labor, even in cases of possible cephalopelvic disproportion, in order to check progress. In this case, the jury found the death of the fetus was due to the physician's negligence, in that he did not make use of available methods of evaluation [37].

Juries consistently evaluate the actions taken by doctors to determine the well-being of both the fetus and the mother. Fetal heart rate should be monitored before and during labor either by electronic means or intermittent auscultation following the protocol of the institution. Although ACOG

states that all laboring women need some form of fetal monitoring, it does not recommend one type of monitoring over another in normal cases [38]. Despite the fact that the ACOG guidelines are the minimal recommendations for the specialty, as mentioned previously, the guidelines do not necessarily form the standard of care to which a practitioner will ultimately be judged. It should also be noted that the ACOG guidelines indicate that if auscultation is the means for evaluation, a one-to-one nurse-to-patient ratio should exist. Thus, the physician must be acutely aware of staff availability and capability when relying on this method of evaluation. Furthermore, historically, juries have not looked favorably on obstetric practitioners when continuous electronic fetal monitoring is available but is used inappropriately.

Oxytocin increases labor contractions and has the potential to overstimulate the uterus and result in distress to the baby or maternal injury. Experts on both sides of the courtroom agree that excessive uterine activity can cause compression of the umbilical cord and thus has the potential to impede blood and oxygen flow. Not long ago, the risks of oxytocin were considered to be significantly graver than at present. One treatise described the risk as follows:

**Oxytocin is a powerful drug, and it has killed or maimed mothers through uterine rupture and even more babies through hypoxia from markedly hypertonic uterine contractions . . . Failure to treat uterine dysfunction exposes the mother to increased hazards from maternal exhaustion, intrapartum infection, and traumatic operative delivery. At the same time, failure to treat uterine dysfunction may expose the fetus to an appreciably higher risk of death, whereas the risk from intravenous oxytocin should be negligible when used appropriately . . . It should be used for no longer than a few hours; if, by then, the cervix has not changed appreciably and if a predictably easy vaginal delivery is not imminent, cesarean delivery should be performed [39].**

With the advancement of intravenous administration of oxytocin, previously described disasters are uncommon today [40]. Administration of oxytocin where true cephalopelvic disproportion is suspected has led to physician culpability [41]. Further, liability

has been found where the plaintiffs have claimed that augmentation was not indicated since labor was progressing adequately. Physician liability has also resulted from the administration of oxytocin when fetal distress is present or when labor induction is attempted prior to engagement of the fetal head.

More commonly today than in the past, claims of improper monitoring and failure to intercede are coupled with claims alleging improper administration and/or management of oxytocin. Clinicians must recognize that *fetal monitoring and constant medical supervision are mandatory during administration of uterine stimulants*. Failure to closely monitor both mother and baby are frequent charges and may well result in a successful negligence claim. The setting for the use of oxytocin is another important issue. Both the medical literature and the legal case law indicate that a physician capable of performing a cesarean delivery must be readily available when labor is induced or augmented. Rarely, maternal death from uterine rupture has resulted from the negligent administration and monitoring of oxytocin. However, more routinely plaintiffs are alleging that a baby's neurologic impairment is the result of the cumulative effect of hyperstimulation and draining the fetus's reserves resulting in hypoxic ischemic encephalopathy.

The obstetrician should consider the following:

- The management of labor dystocia depends on the type of specific abnormality, the maternal-fetal condition, and the results of the evaluation of the fetopelvic relationship. Abnormalities of the latent phase should be treated with either therapeutic rest (with or without sedation) or amniorrhexis and oxytocin infusion.
- The most useful tool for immediate evaluation of fetal anatomy is real-time ultrasonography. Although it cannot evaluate the anatomy of the maternal pelvis, real-time ultrasound scan does have the ability to easily document the lie, presentation, and position of the fetus, to estimate gestational age, to evaluate fetal anatomy, and, with a limited degree of reliability, to estimate fetal weight.
- For active-phase labor abnormalities when progress is poor, the presentation is cephalic, and absolute disproportion and malpresentation have been excluded by the suggested examinations, the

best measure of pelvic adequacy is a trial of oxytocin labor stimulation under close maternal-fetal observation.

- In second-stage arrests in patients with epidural anesthesia, augmentation with oxytocin should be considered. When second-stage progress is tardy, patient repositioning, use of epidural analgesia as opposed to anesthesia, simply prolonging the second stage, and patient encouragement are often successful in achieving vaginal delivery or, minimally, in advancing the fetal head to a lower station to avoid a complex or rotational instrumental delivery.
- Trials of labor augmentation require especially close attention to possible maternal and fetal stress. The pattern of uterine activity is commonly documented by continuous monitoring using an intrauterine pressure catheter or transducer (IUPC), while the FHR is recorded electronically. Such invasive monitoring is not required in all cases, but at least in nulliparous patients.
- Induction of labor is now second only to cesarean delivery as the most common obstetric procedure. Induction of labor is indicated when the maternal or fetal benefits of induction outweigh the risks of continuing the pregnancy
- Physicians should discuss with their patients the indications, methods, and the increased possibility of cesarean delivery prior to proceeding with a trial of induction. The gestational age, an estimate of fetal size, notation of presentation, a clinical statement concerning pelvic adequacy, and a cervical examination should be included in the hospital admission documents. ACOG has specific guidelines to assist in choosing a date for induction.

### THE THIRD STAGE OF LABOR

After delivery, close and critical review of obstetric practice is never more intense than when a neurologically damaged or "bad baby" results from a delivery. Such cases are often complex, difficult to defend legally, and can prove remarkably expensive. Evidence supports that a complete histologic examination of the placenta can provide important data concerning the etiology of an infant's injury [42,43]. Placental findings of nucleated red blood cells, chronic ischemia, intimal cushions, intervillous

fibrin, and acute and chronic meconium staining, among others, can help to determine whether acute or chronic neonatal asphyxia was a factor in the etiology of a child's observed deficits. At present, many institutions follow the recommendations of the College of American Pathologists' consensus committee in determining which placentas to study [43]. Some institutions have implemented various programs for routine gross placental examination, with preparation and permanent storage of microscopic blocks should subsequent histologic examination be required, even years later. All chiefs of service should carefully review the handling of placentas within their institutions. Arguably, the successful avoidance of even just one legal judgment on a "bad baby" could justify a program of placental block storage/gross examinations.

As to maternal care at this stage, many of the themes of safe practice in the third stage of delivery are no different from those in other stages of pregnancy. The third stage is more likely to trap the unwary, however, because of the relaxation that occurs after the stressful delivery of the baby has been completed. The obstetrician should be fully alert and cognizant of the substantial risks involved in the third stage of labor, while not overreacting to those possibilities. The clinician must be fully aware of the general predisposing factors to complications in the third stage of labor. Complete history and current evaluation of the patient, as well as anticipatory monitoring and evaluation, are necessary to be prepared to handle possible complications. The obstetrician should always take postoperative complaints seriously. The case of *Gabaldoni v. Bd. of Physicians* [44] is an example of what can go wrong when a physician does not sufficiently consider the clinical situation or does not fully comprehend the seriousness of a patient's condition, despite the findings. The case also highlights difficulties that are created from poor documentation. In *Gabaldoni*, the complaint was brought by the physician's state licensing board and was not a medical malpractice case. The underlying facts are as follows: On July 8, 1995, at 5:11 p.m., the patient delivered a healthy baby boy. After delivery, she began to hemorrhage because of uterine atony and retained placental fragments; estimated blood loss exceeded 600 ml. At 7:35 p.m., the patient expelled a large blood clot. Her blood pressure then fell to 67/42. At 8:30 p.m., the obstetrician was called at home and ordered a CBC

to be performed the following morning. The standard preprinted orders, which were already in the chart, also called for a CBC in the morning.

On July 9th, at 8:30 a.m., the nurse caring for the patient phoned the physician at home and told him that the hospital laboratory had reported the patient's CBC results showed that she had hemoglobin levels of 5.4 and a hematocrit of 14.8. The obstetrician ordered that a repeat CBC be performed at noon that day. He also ordered the blood specimen be typed and cross-matched, and that the patient's blood pressure be checked regularly. The patient's repeat hematocrit was 14.0.

The physician made rounds on the afternoon of July 9th and again on the afternoon of July 10th. What he told his patient during these two visits and whether there were any other visits are issues that the parties vigorously disputed. The physician testified during the hearing that he discussed the possibility of a transfusion and the patient vehemently refused. The patient denied that any discussion about a transfusion transpired. At 4:30 p.m. on July 10th, the patient experienced slight nausea, shortness of breath, and blurred vision. Less than three hours later, at 7:10 p.m., her condition worsened; it was noted that her blood pressure was very high (162/104), as was her pulse rate (124 beats/min), and she needed to lean forward to breathe. The nursing staff also observed that she was "shaky" and short of breath. There were crackles in her lungs, indicating a buildup of moisture in the lungs.

The RN who was caring for the patient spoke with the obstetrician at 7:30 p.m., advising him of the patient's condition. A repeat CBC was ordered and arterial blood gases (ABGs) were done immediately. The test results showed that the hematocrit was 13.5 and the hemoglobin was 4.7 g. The patient's arterial oxygen content of blood was 56. When these results were reported to the obstetrician at 8:20 p.m., the doctor instructed nurse to tell the patient that she should "strongly reconsider" accepting blood. At 8:30 p.m., the RN offered the patient a blood transfusion, explaining to her and her husband the risks and benefits of the procedure. The plaintiff did not refuse the procedure; however, she did not authorize consent until 9:20 p.m. At the time the blood transfusion started, the patient was in severe respiratory distress. At 3:55 a.m. on July 11th, the RN again called the physician at home to

report to him that there had been no improvement in the patient's condition. At 4:05 a.m., the nurse once more called the obstetrician, reporting that the patient's condition was worsening, that she now had crackles in both lungs, front and back, all the way up.

The patient's condition continued to deteriorate. At approximately 4:45 a.m., the nurse advised the physician that his patient was ashen in color, unresponsive, and sweating. She also told the physician that it was urgent that he come to the hospital. He arrived at the hospital at 4:55 a.m., at which point his patient went into respiratory arrest. She died on July 13th. The cause of death was determined to be a cardiac arrhythmia complicating postpartum hemorrhage and severe anemia.

The Board based its decision to discipline the obstetrician on his failure to respond appropriately to the clinical situation. The Board determined that at the very least, the obstetrician should have acted at once, when the second hematocrit reading of 14.0 was recorded at 12:00 noon on July 9th. His patient was at this point not oxygenating her organs, and any competent physician should have recognized the crucial need for a blood transfusion. The physician did not order a blood transfusion and did not even order further hemoglobin and hematocrit (H&H) testing until 7:30 p.m. on the following day. During this period, she frequently displayed many of the symptoms of severe anemia, including tachycardia, shortness of breath, vomiting, and dizziness.

The Board also determined that the obstetrician did not request any nurse to offer the patient a blood transfusion until after 8:20 p.m. on July 10th, after cardiac decompensation had begun and she was in respiratory distress. Although the physician had conversations indicating that she would need a blood transfusion before being discharged, the obstetrician had no conversations with the patient or her family in which he informed her of the potential adverse consequences that could occur if she failed to have a blood transfusion. Another criticism from the board was the doctor's failure to be present in the hospital at any point between early evening on July 10th and when the patient went into cardiac arrest.

The Board also found that the physician breached the obligation to create an accurate medical record. Two days after the patient died, he added notations to the records in such a way that it would not be clear to a reader of the progress note that additions had been made. The entries were

inaccurate in that, among other things, they recorded an incorrect hematocrit level; the time ("a.m.") was inaccurately recorded for a July 10th entry; he added the words "feels much better" between the phrases "no dizziness now" and "refuses transfusion," and also added the words "consider transfusion at later date" at the end of the entry. The record for both dates incorrectly reported that continued H&H testing had been ordered, and the record of July 9th incorrectly stated that the patient refused a transfusion. Not only did the physician fail to note that the additions were added later, but he also used two different pens, a blue pen that matched the blue ink on the original note concerning July 9th and a black pen that matched the black ink used on the original note concerning July 10th. In addition, for July 10th, the additions were interspersed throughout the note from beginning to end, in such a way that it would be natural to mistake the record as one that had been written all at one time. This type of record keeping violates both the letter and the spirit of the standard of care enunciated previously. What was very important in the Board's determination on this issue was that the changes were of critical significance.

In cases when there is a poor outcome owing to omissions of treatment, a retrospective evaluation can make the omissions seem very obvious. Perhaps many obstetricians, even without the benefit of hindsight, would have reacted differently to the clinical situation in *Gabaltoni*; certainly most would not have amended the records in similar fashion. As is well recognized from the standpoint of being involved in a clinical setting without the benefit of hindsight, however, those obvious clinical features are not always so evident. The *Gabaltoni* case establishes the importance of contemporaneous documentation, especially in the context of a patient who refuses recommended care. Of note in *Gabaltoni* is that the Administrative Law Judge who heard the evidence during the hearing concluded that the obstetrician had appropriately advised the patient of the need for a blood transfusion on the morning of July 9, 1995. In fact, based on the evidence and evaluation of the demeanor of the witnesses, the judge agreed with the physician and determined that the patient had refused to have a transfusion until 9:20 p.m. on July 10th. The judge also concluded that, after the July 9th morning visit, the physician repeatedly advised his patient to have a

transfusion, but the advice was consistently rejected up until 9:20 p.m. on July 10th.

Regarding the amendments that were made to the records, the Administrative Law Judge concluded that although the Board did establish the physician's failure to make additions to his progress notes properly, and that the notes did not accurately reflect the severity of the patient's condition, she did not believe the nature of the amendments amounted to falsification. In such settings, the findings of the Administrative Judge are considered to be recommendations. The Board of Physicians had the right to adopt or refuse them, and in this case failed to accept the findings, thus determining that the obstetrician's conduct amounted to misconduct. The physician appealed, however, and the Board's decision was upheld on appeal. It is clear from the ruling that many of the details that ultimately became determinant were interwoven in the documentation. If, in fact, the physician had advised the patient on repeated occasions to have a blood transfusion as he stated, then the difficulty in defending the case arose primarily from his poor documentation and subsequent modification of the records.

Although most women experience a healthy postpartum course, serious complications can and do occur. The obstetrician should be prepared for catastrophic emergencies, specifically hemorrhage/hypovolemia. Postpartum hemorrhage (PPH) is a common complication of pregnancy and is the single most important cause of maternal death. The incidence of PPH is estimated to range from >5% to 10% of all deliveries, depending on definition. Only 5% of vaginal births are associated with a 1,000 ml or greater blood loss, however [45].

In addition to hemorrhage, eclampsia and preeclampsia are also serious causes of maternal mortality worldwide. Complications of hypertension are the third leading cause of pregnancy-related deaths, superseded only by hemorrhage and embolism [46]. Preeclampsia/eclampsia can develop before, during, or after delivery. Up to 40% of eclamptic seizures occur before delivery; however, approximately 16% occur more than 48 hours after delivery [47].

Other common postpartum complications include urinary tract problems, such as infections, urine retention, or incontinence. Many women also experience pain in the perineum and vulva for several weeks, especially if tissue damage occurred

or an episiotomy was performed during the second stage of labor. The perineum should be regularly inspected to make sure that it is not infected. Psychological problems in the postpartum period are also not uncommon. These problems can be lessened by adequate social support and support from trained caregivers during pregnancy, labor, and the postpartum period.

Although data are not collected nationally, the percentage of women readmitted to the hospital in the postpartum period is estimated at 1.2% to 3% [48]. After cesarean birth or assisted vaginal birth, women have an increased risk for rehospitalization from PPH, uterine infection, obstetric surgical wound complications, cardiopulmonary and thromboembolic conditions, gallbladder disease, genitourinary tract conditions, pelvic injury, and appendicitis, compared with patients who have had spontaneous vaginal birth [49]. Although readmission to the hospital occurs relatively infrequently, the women who are admitted are very ill. The sequelae of their illness affects not only their postpartum recovery but also the physical and mental health of their infants and families.

When hemorrhage occurs, the goals of management are directed toward rapid control of blood loss, prevention of maternal cardiovascular collapse, and close patient monitoring. Active management of the third stage with routine administration of parenteral uterotonics can avoid many but not all cases of PPH.

Early PPH, defined as events of hemorrhage occurring within the first 24 hours after delivery, are mostly due to uterine atony or retained products of conception [50]. Nearly nine out of ten of these deaths take place within 4 hours of delivery, because a woman who is suffering the physiological effects of labor and delivery is usually less able to cope with blood loss than a woman who is well nourished. Late PPHs occur more than 24 hours after delivery but usually prior to 6 weeks after parturition. Delayed bleeding results largely from placental site subinvolution, a condition that is usually combined with chronic infection and retained products or placental polyps. Because of the difficulties in the clinical estimation of the volume of hemorrhage and the wide range of values for normal, clinical suspicion must rest on the observations of maternal signs and symptoms and estimated blood lost.

Although every postpartum patient has some potential for puerperal hemorrhage, high-risk cases are identified based on events of labor and deliv-

ery, prior history, or preexisting medical condition. Among women undergoing cesarean delivery, general anesthesia, amnionitis, preeclampsia, and protracted active phase or second-stage arrest disorders increase the risk for bleeding. In vaginal deliveries, multiparity, amnionitis, and overdistension of the uterus from multiple gestation, hydramnios, or placental abnormalities can also increase the risk.

There are other, rarer causes of PPH. Fortunately, most of these medical conditions are recognized prior to parturition and are managed prospectively. Beyond such special cases, the most common obstetric cause for an acquired postpartum coagulopathy is simply severe bleeding, with severe loss of clotting factors (i.e., loss coagulopathy).

- No matter how experienced and qualified the obstetrician is, he/she should insist on qualified personnel to be supplied by the hospital. Misfortune can occur in the third stage of labor because the "eyes and ears" of the obstetrician, the nursing staff, are not sufficiently attentive to the patient or did not know what to look for in anticipating complications before they became serious. However in some circumstances the obstetrician's "eyes and ears" may function very appropriately, but the physician's delay in the clinical recognition of a complication can be due to his failure to process the information he receives or she appropriately.
- The obstetrician should be fully aware of the activities of the anesthesiologist at all times. In particular, the obstetrician must know all of the medications administered. The claim that the obstetrician left all of these matters entirely up to the anesthesiologist is not persuasive in a courtroom. There is no question that the obstetrician should be fully aware of the consequences, indications, and risks of the major anesthetic techniques, including epidural and spinal anesthesia and a wide variety of medications that might modify the normal third stage of labor and either increase or decrease the likelihood of a PPH or have an adverse effect on its subsequent therapy. An example is the administration of an inhalation agent that increases the risk for uterine atony during a cesarean delivery for failure to progress involving a macrosomic infant.
- Because many things done in the third stage of labor involve judgment, an important factor is

candor with the patient. Rather than making a decision without discussing it with the patient, the physician should advise the patient fully about what is occurring, and the discussion should be documented. Many bad results do not offend or upset a patient if a reasonable discussion preceded the event.

- Informed consent is as important in the third stage of labor as it is elsewhere in the practice of medicine. Numerous lawsuits have been filed about episiotomies, alleging improper performance or follow-up. These are difficult cases for the patient to pursue; nonetheless, it is imperative to have discussed the choice to perform (or not perform) an episiotomy and the risks and complications of the alternatives.
- With the proper anticipation, most of these situations can be managed preemptively without serious complications. Waiting until the catastrophe has occurred to take action means losing vital minutes or even seconds, which can turn a correctable temporary problem into a serious disaster with permanent consequences.
- The initial maternal response to hemorrhage varies and can be confusing to the clinician. Common indicators of circulatory function, including arterial pressure and pulse rate, are often normal in pregnant women despite substantial blood loss. Unfortunately, the usual orthostatic measurements and tests for orthostatic hypotension are inconsistent signs.
- Most deaths from maternal hemorrhage occur within 4 hours of delivery. A woman who is suffering the physiological effects of labor and delivery in the immediate postpartum period typically has less reserve to combat blood loss than a woman who is well nourished. During the first hours after the birth, the obstetrician must establish that the uterus remains well contracted and that there is not significant blood loss.
- Vaginal bleeding is the most common sign of hemorrhage. In cases of active hemorrhage, blood loss is almost always underestimated. If the bleeding is particularly severe, blood transfusion might be the only way of saving a woman's life.
- In selected high-risk patients with strong histories of prior atony, or those in whom heavy blood loss is anticipated owing to coagulation or

placental abnormalities, discussion of autogenous blood donation for potential delayed transfusion is appropriate.

- Current obstetric treatment in the United States has resulted in a shift of eclampsia toward the postpartum period, with most cases being seen late postpartum. To reduce the rate of late postpartum eclampsia, efforts should be directed to the education of the healthcare providers and patients about the importance of prompt reporting and evaluation of symptoms of preeclampsia during the postpartum period.

### BREECH PRESENTATION

Until 1959, vaginal delivery for breech presentation was the norm; it was then that cesarean section began to be considered the method of choice for delivery. With the liberalization of indications for cesarean section, the proportion of breech presentations delivered by cesarean rose from approximately 14% in 1970 to 60.1% in 1978. Thus, by the late 1970s, the standard of care strongly suggested, if not mandated, that breech babies be delivered by a cesarean unless there was a strong contraindication. As scientific advancements were made that reduced the risks associated with abdominal delivery and anesthesiology, the cesarean section rate rose to 86% by 1986. In 2001, ACOG issued an opinion that "patients with persistent breech presentation at term in a singleton gestation should undergo a planned cesarean section" [51].

Obstetricians have long recognized the excessive perinatal morbidity and mortality associated with the breech-presenting fetus [52,53]. Even when a cesarean is the presumed mode of delivery, a breech infant can be a difficult challenge to the obstetrician. Breech presentation complicates 3% to 4% of all pregnancies and is associated with an increase in both morbidity and mortality compared with cephalic presentation, regardless of whether the method for delivery is vaginal or cesarean [54]. Multiple factors are responsible for the increase in poor outcomes, including congenital malformations, prematurity, and traumatic birth injury [55,56].

Despite the potential advantage of cesarean delivery to ensure an acceptably low complication rate for the newborn infant in certain breech presentations, a cesarean is not always necessary and a role still remains for the vaginal delivery. Because



cesarean delivery carries with it a four- to fivefold increased risk of significant maternal mortality and a substantially greater risk of significant morbidity and prolonged recovery in comparison to vaginal delivery, there has been a careful reassessment of routine cesarean delivery for breech presentations. The potential risks of the cesarean operation and its limited maternal/fetal benefits from surgery in certain breech presentations are the issue. Furthermore, vaginal breech delivery is still a method to be employed in certain circumstances: patients presenting in advanced labor, patients who have had multiple prior cesareans, or patients who anticipate a larger family. Thus, clinicians will still be called on to conduct vaginal breech procedures. The necessity is to establish how these deliveries can be conducted safely.

The most important elements for the safe conduct of breech delivery are 1) preselection of candidates for vaginal delivery, 2) continuous fetal monitoring, and 3) a policy of noninterference until spontaneous delivery of the breech to the umbilicus has occurred. Preselection limits vaginal breech delivery to average-sized fetuses (under 3,500 g) in frank breech presentation with an adequate maternal pelvis. Early in the course of labor, appropriate preparation should be made for immediate cesarean section should that prove necessary. Anesthesia should be available, the operating suite should be ready, and appropriate informed consent must be obtained. Two obstetricians should be in attendance, as well as a pediatric team. Premature or overaggressive assistance can adversely affect the breech birth, and cervical dilation must be maximized and complete dilatation sustained for sufficient duration to ensure retraction of the cervix and avert entrapment.

As a result of the increased risk of morbidity and mortality in a breech presentation, regardless of the method of delivery, informed consent is an important aspect of care. In cases in which there is a serious impairment, it is very significant to the jury that the outcome was an understood and recognized complication of the clinical presentation. If there were any opportunity to have altered the outcome by a different approach, the issue then becomes was the injury avoidable, and if so, was the mother given the opportunity to do so?

Open and frank discussion with the patient of the risks and options that are presented her and appropriate documentation are of utmost impor-

tance. This approach can avoid a bad medical event, but in the event of an adverse outcome, it provides strong evidence in the defense of a malpractice claim. Failure to provide informed consent is a claim that an injured child can pursue, even though there is no argument that he/she as the injured person ever had the opportunity of making an informed choice.

Consider *Draper v. Jasionowski*, in which, at the time of delivery, the injured plaintiff, the fetus at the time of the events, presented in frank breech position with a large cranial vault while in his mother's womb [57]. The delivering physician was aware of his presentation prior to the onset of labor. The patient signed consent forms for both vaginal and cesarean deliveries. She gave birth to the plaintiff by vaginal breech delivery, which was complicated by a torn umbilical cord. The plaintiff's board-certified obstetric expert opined that the plaintiff suffered from anemia, hypoxia, and neurologic damage, indicating a tremendous loss of blood secondary to the torn umbilical cord. The plaintiff was also born with bilateral Erb's palsy. The plaintiff's claim was that the defendant neither informed his mother of the option to do a cesarean rather than a vaginal delivery nor left the decision of his manner of delivery to the mother's choice.

The plaintiff initiated suit in 2002, 20 years after his birth. The defendant's contention was that the obligation to disclose the risks of and alternatives to obstetric care is solely to the mother and not the child. In setting forth the argument, the defendant contended that plaintiff's informed consent claim is strictly derivative of the mother, and consequently the case was barred because of the expiration of the statute of limitations, which gave a 2-year period to commence a lawsuit. It was determined that a doctor who fails in the duty of securing informed consent violates a duty owed to both the mother and the child. Furthermore, the plaintiff's injuries are independent of any injury to the mother, and thus, even though any claim that the mother might have had was barred by the statute, the child's claim was proper because the statute provided for a minor who was allegedly injured as a result of negligence to bring suit at any time prior to 2 years after his/her eighteenth birthday.

Although academic interest continues in the efficacy of breech vaginal delivery to reduce the number of cesareans, vaginal delivery of the breech infant has been modestly used in the clinical setting. To the extent that there is variance, there is

evidence to support that hospital factors are associated with vaginal breech delivery. Public hospitals had the most vaginal breech deliveries, and private non-teaching hospitals were least likely to use this procedure [58]. The study concludes that regardless of the reason, there is considerable variation in the practice of vaginal breech delivery. Given this variance, the *Draper* case underscores the need for educational counseling to the patient, particularly in the event that vaginal breech delivery is attempted. Ultimately, the consequences of the decision are borne by the child, the mother, and the family. In the event of bad outcomes, patients often reconsider their decisions. The aftermath is not the time for them to learn of the particular risks associated with the decision or that there were alternative approaches. Certainly, frank discussions should always transpire when there is an unfortunate result; however, the ideal time to offer the patient the opportunity to clear up any misconceptions on which the decision was based has passed.

The other issue that becomes underscored in any debate about the efficacy of breech vaginal delivery to reduce the number of cesareans and maternal risk is the decline of physician operative skill. Unfortunately, the level of physician skill, training, and experience in performing many traditional obstetric procedures, including assisted breech delivery and/or extraction, has steadily declined over the past three decades [59]. The relative infrequency of vaginal breech presentation and the difficulty acquiring experience will result in poorly trained and inexperienced obstetricians who will be called on to perform a vaginal breech presentation, yet the standard of care will require the obstetrician to perform the procedure using reasonable skill and care and the obstetrician will be subject to valid criticism for injuries caused by his/her lack of competence.

The obstetrician should consider the following:

- Breech presentation during labor is a high-risk situation and requires liberal use of cesarean delivery. By using a selective approach, however a TOL and vaginal delivery may in certain circumstances be reasonable. Implementing a selective approach, a physician can balance maternal surgical risk and fetal delivery risk.
- External cephalic version (ECV) should be offered to most women who are of at least 36 weeks' gestation. There are contraindications for this maneuver, however. Multiple gestations with a breech-presenting fetus, nonreassuring fetal heart rate tracing, and mothers in whom vaginal delivery is contraindicated are not candidates for ECV.
- Before attempting an ECV, the obstetrician should evaluate for any fetal anomalies or other conditions that are associated with malpresentation.
- The risks associated with an ECV procedure must be described to the mother, as well as the possibility that the attempt could fail. Risks of ECV include rupture of membranes, onset of labor, placental abruption, and creating problems with the baby's heart rate. Before proceeding, therefore, the obstetrician should discuss the benefits, the potential for failure, and the accompanying risks.
- For most patients with breech presentation, cesarean delivery is the best option; however, this might not be possible for patients who present in advanced labor or who have had multiple gestations. Furthermore, a cesarean delivery does not avoid all difficulties associated with breech presentation. Thus, the risks attendant to vaginal delivery and a cesarean for a breech infant must be discussed with the mother during the informed consent process. The maternal risk associated with cesarean delivery must be included in the counseling for the decision to be truly informed.
- Although a cesarean is performed for most women with a breech fetus, selective TOL in women with known breech presentation in labor can be a reasonable approach to delivery. If ECV is contraindicated or simply refused by the patient, then a next step to consider is a TOL.
- Because all breech births have inherent risks that are often uncertain and unpredictable, this information should be shared with the family whenever possible as part of the decision-making process.
- If one is going to attempt a vaginal breech delivery, a qualified anesthesiologist or nurse anesthetist must be in attendance, one who can give agents to relax the uterus when and if use of such is indicated.
- Although documenting the counseling session is very important, the counseling session itself is most important.

- It is incumbent on the obstetrician who knows or suspects that due to limited training and experience he or she might not be qualified to undertake a vaginal breech delivery to obtain the assistance or intervention of a practitioner who is capable of managing the delivery properly.

### MULTIPLE GESTATIONS

There are several main areas of medicolegal concern in multiple gestations: diagnosis, management of preterm labor, anomaly surveillance, counseling, evaluation of gestational age, and delivery management. The most obvious and probably the most crucial area of liability is the failure to diagnose the presence of a multifetal gestation. Although many of the complications surrounding these pregnancies (i.e., preterm labor or growth, or congenital anomalies) cannot be totally avoided in any gestation, the knowledge that they are much more common in these pregnancies can enable an earlier diagnosis and promote more efficient management.

In terms of general obstetric management, once the diagnosis has been made, it is the physician's responsibility to perform suitable and timely assessment of fetal and maternal status. Complications of multiple gestations are common and well documented in the literature. Failure to recognize or appropriately treat evolving problems is fertile ground for legal entanglements, particularly related to issues of disordered growth, malpresentation, and preterm labor. Weekly office visits after 20 weeks of gestation and liberal use of sonography and non-stress testing to evaluate fetal well-being are strongly encouraged. Early detection of growth problems can give the physician sufficient time to develop a well-reasoned plan of care, obtain consultations, and arrange for management of labor and delivery at a tertiary care center if necessary. When fetuses are at 24 to 26 weeks of gestation, errors of gestational age calculation are critical. If a physician has determined that the fetal age is less than 24 weeks, he/she might believe that the fetuses are potentially not viable and treat accordingly. If in fact the gestational age is 26 weeks and the fetuses *are* potentially viable, catastrophic consequences can result from a treatment plan that assumes otherwise.

With many obstetric practices today, the delivering physician might have examined the patient only once, twice, or not at all during the antenatal period.

Ultrasound examinations done early in pregnancy for dating purposes are particularly useful, as are follow-up scans to evaluate fetal growth and well-being. Critical mistakes in the calculation of gestational ages are possible, and care is needed. Even in cases of pregnancies induced by fertility drugs, when the date of conception is known, physicians have incorrectly calculated the gestational ages, sometimes with catastrophic results. Before finalizing a plan of intrapartum management, sonography is mandatory to ascertain fetal position and obtain estimated fetal weights.

Given the high-risk nature of the labor and delivery, the physician must take the necessary steps to ensure that all relevant data about fetal well-being are obtained. The failure to do so in the face of a poor outcome will create issues in defending any medical negligence case. This failure was the issue in the case of *Mundell v. La Pata* [60]. During the patient's 28th week of a twin pregnancy, she sought medical attention because of decreased fetal movements and contractions. Over the course of 2 days, the defendant doctors attended to the patient and her twin fetuses, primarily by monitoring the twin fetuses' heart rates, conducting ultrasound examinations, and reducing maternal contractions. On the second day, an ultrasound examination revealed that one of the twins had died in utero. The ultrasound test and a Doppler study indicated that the other twin had "no major anomalies." Later that day, however, the other twin died in utero, as confirmed by a second ultrasound examination. The patient then underwent a cesarean for delivery of the dead fetuses. The preliminary postoperative diagnosis of the cause of death was twin-to-twin transfusion syndrome. The alleged negligence forming the basis of this action arose out of the direct patient care provided to the patient during her pregnancy, and the management, treatment, and delivery decisions that were made when she sought medical attention because of decreased fetal movements and contractions. She alleged that the defendant doctors were negligent by failing to provide proper medical treatment, primarily testing, and by failing to intervene surgically to save the life of the remaining twin after one had died in utero. Her expert witnesses testified that the defendant doctors breached the standard of care by failing to perform certain tests to determine not just whether the twins were alive but also whether they were in distress. The expert witnesses

further opined that the standard of care required that a recommendation be made to the parents to proceed with delivery, especially after one of the twins had died in utero. The burden was then placed on the physicians to establish that the requisite testing had been performed, and despite evidence of the opportunity to rescue, that there was no clinical indication to do so.

Multiple gestations often pose intrapartum management problems. This is especially true if the gestation is complicated by preterm labor or disordered or discrepant growth.

The debate continues to rage over whether particular presentations should be delivered vaginally or operatively. From the medicolegal standpoint, the simplest course is to perform cesarean delivery when either twin A or twin B is in a nonvertex presentation. Some studies have concluded that vaginal delivery of nonvertex-presenting twin B does not increase perinatal mortality. *No study, however, has concluded that twin B suffers a greater morbidity or mortality when undergoing cesarean delivery.* It is inevitable that the physician who delivers the vertex-presenting twin A and nonvertex-presenting twin B vaginally will always be second guessed if unavoidable crises develop during labor and delivery and one of the twins is born impaired. The physician who has delivered the twins by timely cesarean delivery eliminates that medicolegal risk but might not have made the best obstetric decision.

When faced with a vertex-nonvertex-presenting delivery, a vaginal TOL is prudent only if 1) the patient meets all of the criteria for vaginal deliveries; 2) there is a double set-up present in the delivery suite; 3) anesthesia is present; 4) the physician is experienced in delivering nonvertex fetuses; and 5) continuous electronic fetal monitoring and real-time ultrasound scanning are available. If all of the above criteria cannot be met, cesarean delivery is best.

Multiple gestations place the patient at increased risk for preterm labor. Problems resulting from prematurity are thought to be responsible for the increased incidence of morbidity and mortality in multiple gestations. The physician must consider, in cases of preterm labor, the possible use of tocolytic drugs and other modalities to prolong the pregnancy. Routine antepartum use of oral tocolytics and home uterine monitoring have not been shown to prevent preterm delivery. Thus, these treatments should be used sparingly if at all in otherwise uncomplicated

cases, with their limited goals clearly understood by the clinician and family.

The use of tocolytic agents after documented preterm contractions or labor is another matter. Prolonging some pregnancies by as few as 2 or more weeks can significantly improve the chances of fetal survival and reduce morbidity. Before using tocolytic agents, however, the physician must, to the extent reasonable, confirm the absence of chorioamnionitis or other contraindications to such therapy. The standard of care requires that the doctor managing a multiple gestation make reasonable efforts to prolong pregnancy to at least 32 weeks of gestation or beyond, with due regard to maternal well-being.

Some states recognize a patient's right to recover damages if a patient is not advised of the possibility of congenital abnormalities or deformities in the fetus and of the ability of modern fetal surveillance techniques to identify these problems in time to terminate the pregnancy safely. Multiple gestations produce a higher incidence of congenital anomalies than do single pregnancies. The risks and benefits of maternal serum  $\alpha$ -fetoprotein (MSAFP) values, chorionic villous sampling, amniocentesis, and early ultrasound examination must be thoroughly discussed with each patient, in addition to the risk of congenital anomalies.

The obstetrician should consider the following:

- Multiple gestations are high-risk situations for all concerned: the patient, the fetuses, and the physician. The physician managing the patient with multiple gestations must be ever vigilant for any sign or symptom suggesting a complication. Knowledge of the full range of complications involving multiple gestations and the appropriate procedures for handling each potential complication is mandatory.
- The failure to perform ultrasound evaluation in the presence of clinical evidence suggesting a multiple gestation (e.g., increased fundal height, elevated MSAFP levels, early or unanticipated maternal carbohydrate intolerance, or exaggerated gestational hypertension), can be considered below the standard of care.
- The physician must carefully review the patient's chart and be certain that the estimated date of confinement (EDC) has been properly calculated

from ultrasonic and menstrual data, and that an accurate gestational age is known. If the precise gestational age is not known because it has been calculated by reference to an uncertain last menstrual period (LMP) or sonogram done late in pregnancy, the physician should err on the side of caution and assume the more advanced gestational age in cases of borderline viability. Any other approach means taking unnecessary and unwise risks.

- Frequent cervical examinations in patients presenting with unusual signs or symptoms are useful in detecting early changes in the cervix that could alert the physician to the possibility of preterm labor and other potential problems.
- The appropriate method of delivery depends on close attention to clinical detail and full evaluation of maternal and fetal data. Diligent monitoring of maternal and fetal status and prompt intervention in instances of presumed fetal jeopardy can aid in achieving optimal maternal and fetal outcomes. As always, detailed notation in the medical record about the choices made and the consent process is prudent.
- Patients must be instructed on the signs and symptoms of potential complications of multiple gestations, particularly those of preterm labor. Preterm labor can be painless, and its symptoms often confused with minor abdominal discomforts. The physician must be particularly alert for symptoms that the patient describes as “cramping” and “pressure,” especially in those patients with abnormalities of the cervix.
- Giving patients a short instructional sheet or pamphlet explaining the warning signs and symptoms of preterm labor and other complications of multiple gestations helps to reduce confusion and misunderstanding in the event that problems are encountered later in the pregnancy. If such sheets or pamphlets are used, documentation in the patient’s medical record of her receipt of this information costs nothing and is strongly recommended.

### SHOULDER DYSTOCIA

Shoulder dystocia has been a controversial and contentious subject medically and legally. A classic description of shoulder dystocia made by Mor-

ris almost 50 years ago illustrates the physician’s dilemma in management of an unanticipated shoulder dystocia [61]:

**After delivery of the head, fat cheeks, and double chin, perhaps with a little difficulty, time passes. The child’s face becomes suffused. It endeavors unsuccessfully to breathe. Abdominal efforts by the mother or by her attendants produced no advance. Gentle head traction is equally unavailing. Usually equanimity forsakes the attendants. They push, they pull; alarm increases. Eventually by greater strength of muscle or some infernal juggle, the shoulders of a goodly child are delivered. The pallor of its body contrasts with the plum-colored cyanosis of the face and the small quantity of freshly expelled meconium about the buttocks. It dawns on the attendants that their anxiety was not ill-founded. The baby lies limp and voiceless and only too often remains so despite all efforts at resuscitation.**

Until more recently, there was little consensus about the ability to anticipate shoulder dystocia and the role of cesarean delivery in avoiding the problems of shoulder dystocia. Different maneuvers were advocated to release the shoulder when dystocia was diagnosed; however, there was debate over whether any particular maneuver or combination of maneuvers was superior. Given the low incidence of the presentation, the individual provider typically had less than ample opportunity to become an expert in these delivery techniques. Meanwhile, the mechanism for injury remained poorly understood; the only consensus was that excessive lateral traction during delivery was thought to damage the nerves structures of the brachial plexus. The ability to argue that the only identifiable cause of the injury is the force used by the obstetrician, together with the controversy surrounding management, is the reason that lawsuits began to focus on shoulder dystocia, and in essence this injury became the “flavor of the day” for plaintiffs’ attorneys.

Because of the body of literature suggesting the existence of risk factors that were predictors for shoulder dystocia, and the poorly understood mechanism for a brachial plexus injury, it was common for plaintiff’s expert witnesses to work backward from

the injury, hypothesizing how it could have been avoided, and the cause of the injury itself. Typically allegations of medical malpractice in shoulder dystocia cases involved either or both of the following: 1) the failure to perform a cesarean delivery section in the presence of maternal risk factors, or 2) failure to adhere to a proper and safe protocol in managing a shoulder dystocia delivery. Relying on risk factors that permeated the literature, plaintiffs' experts would suggest that a prophylactic cesarean should have been undertaken to avoid the risk entirely. Frequently the risk factors relied on were macrosomia, gestational diabetes, maternal obesity, postdatism, and prior history of deliveries being complicated with shoulder dystocia. A prolonged second stage would provide further fodder, because the plaintiff's expert could testify that any misguided decision to attempt a TOL should have been aborted.

Invariably, the plaintiff's argument would question the manner in which the delivery was performed, alleging that the defendant's use of excessive or improperly directed traction to release the shoulders caused the resulting harm. Expert testimony based its premise on the belief that that brachial plexus palsies do not occur in shoulder dystocia cases except when there is excessive downward traction. The plaintiff's attorneys and experts painted a portrait of an unanticipated presentation resulting in chaotic response to the emergent situation. They argued that the only recognized indisputable cause of brachial plexus injury in this setting is "the hands of the obstetrician." Given the inability to provide a contrary explanation for the injury, plaintiffs were emboldened, with some success, to argue that courts should recognize the *res ipsa* doctrine and instruct juries that they could draw an inference that a defendant acted negligently in cases of brachial plexus injury, thus putting the burden on the defendant to prove otherwise. In upholding a jury's verdict in favor of the plaintiff who argued *res ipsa* to establish liability, the court in *Stennis v. Rekkas* gave this explanation: The record shows that evidence was admitted from which a jury could conclude that [the child] suffers from Klumpke's palsy; the injury was received while the delivery of the shoulder dystocia was under the defendant's control and management, and in the normal course of events, the injury would not have occurred if the defendant had used ordinary care during the delivery of the shoulder dystocia [62]. Given this record,

the jury could have based its verdict for plaintiff on the *res ipsa loquitur* theory.

In courts that were persuaded to allow a plaintiff to use the *res ipsa* argument, the expert would merely be called on to testify in some manner similar to the following [62]:

**Q:** Do you have an opinion, based on a reasonable degree of medical certainty, whether a brachial plexus injury ordinarily occurs in a vertex or headfirst delivery in the absence of negligence or in the absence of a departure from the standard of care by the delivering physician?

**A:** That would be extremely rare.

**Q:** In your opinion, does that injury then not ordinarily occur in the absence of negligence?

**A:** Right.

In this context, the jury then could make a presumption that the injury was the result of negligence without having to establish the particular act of the obstetrician that departed from the standard of care. If the jury determined to make this presumption, then it becomes incumbent on the defense to establish proof of the converse. *In essence then, the burden of proof in such a case rests on the defendant obstetrician to prove lack of negligence, and not the plaintiff's obligation to establish departure from the standard of care.* (See Appendix 1 for a more detailed discussion of burden of proof.)

In response to the explosion of litigation, the obstetric community has been able to amass a strong rebuttal and consequently has significantly increased the defensibility of these cases. Evidence-based medicine, improved training techniques, and better documentation techniques have provided a strong rebuttal in defense of the care provided. Evidence-based medicine has established that shoulder dystocia is an unpredictable event and that identifying pregnancies in which the fetus is at risk for permanent injury is impossible. Research supported the proposition that even risk factors that are statistically significant for shoulder dystocia have no usefulness as predictors and that fetal size estimations are routinely inaccurate. This body of literature provided considerable support to the proposition that routine use of cesarean delivery for the prevention of dystocia and related injuries is difficult to

justify. Of utmost importance is the body of literature that has developed to refute the contention that brachial plexus injuries occur only when excessive force is used during the maneuvers employed to disengage the shoulder.

In addition to attacking the medical propositions offered by plaintiffs, however, the obstetric field placed education and training aimed at training physicians how to effectively respond when confronted with shoulder dystocia in the forefront, along with proper documentation techniques. Emphasizing the shoulder dystocia was a true obstetric emergency, and greater emphasis was placed on team approach, including neonatal resuscitation. Shoulder dystocia drills were successfully implemented so that providers would have preplanned the individual manner in which they would respond if confronted with an impacted shoulder. Training models were developed to allow the maneuvers to be practiced, including the use of traction, to give additional hands-on experience for a condition that occurs infrequently. Last, significant emphasis has been placed on the importance of documentation, which has provided direct evidence at trial of the prenatal course, informed consent counseling, and labor and delivery issues, including the implementation of a well-thought-out plan when shoulder dystocia was first identified.

The totality of the response mounted by physicians practicing in obstetrics has more than leveled the playing field. The ability to respond to allegations of malpractice by producing evidence that neurologic shoulder injuries occur even in the best of hands, when due care has been used, has contributed to a significant decline in the number of adverse verdicts in cases involving shoulder dystocia. More often, insurers are making the decision to defend through trial shoulder dystocia cases with good results.

The obstetrician should consider the following:

- Obstetricians should undertake fetal and pelvic evaluations in any case in which there is a reasonable possibility of a macrosomic infant. The best answer is thorough evaluation of pelvic size and fetal lie, presentation, position, and weight, using both clinical means and the best available modern technology.
- With the universal availability of ultrasonography, physicians who do not use ultrasonic imaging when there is suspicion of disproportion or macrosomia are likely inviting a medical negligence lawsuit. Such a suit will probably end favorably for the plaintiff if it is discovered after delivery that the child sustained a permanent injury.
- The importance of the mother's obstetric and medical history needs emphasis. Prior difficult deliveries, shoulder dystocia, or macrosomic infants should alert the clinician to possible trouble. A detailed discussion with the mother and family prior to a trial of vaginal delivery in a suspect case, with careful notation of the specifics of the discussion in the medical record, is especially important. When the events of a previous delivery are unclear, consider obtaining the records from that delivery.
- Acute management of dystocia remains a major problem. The physician who encounters a dystocia must have an organized and practical plan of approach, involving a practical series of actions performed without panic and avoiding excessive cranial traction.
- Arguably, it has become a standard of practice, as reflected in the literature, to perform cesarean delivery when there is an estimated fetal weight of 4,500 g or more. When the mother has diabetes, the weight limit for a vaginal trial is commonly 4,000 g. The problem for the clinician is to determine the fetal weight accurately in advance of delivery and to judge the fetopelvic relationship just as accurately. When these issues are in play, informed consent is very significant should there be a bad outcome.
- Virtually all disimpaction maneuvers require assistance. Even anesthesiologists and pediatricians are capable of applying suprapubic pressure, in addition to other life-saving procedures.
- It is as important to have the patient's confidence and cooperation as it is for nurses to assist in the delivery. Once shoulder dystocia has been identified, the provider must ensure that there is adequate support. The maneuvers should be implemented deliberately, without haste, reflecting a consistent and logical plan of management.
- Make a large episiotomy. Although there is no evidence that it does anything other than enhances

the ability to insert one's hand in the vagina, the performance of an episiotomy indicates that the operator is functioning logically and systematically. The failure to perform an episiotomy has not been shown to contribute to any injury, however.

- Use McRobert's position and suprapubic pressure to disimpact most tight shoulders. These maneuvers are easy to perform, and the McRoberts position can also enhance the ability to successfully perform a rotation maneuver or remove the posterior arm. *In all cases, avoid excessive traction.*

The medical record can play an important role in establishing that the doctor was not negligent in a malpractice claim. If the physician can articulate a reasonable basis for the clinical judgment and that information is documented in the medical record, then it is extremely difficult for the plaintiff patient to prevail in the action. Effective documentation regarding prenatal workup, informed consent, and events during labor and delivery are important aspects of this response. Through the totality of these efforts, the likelihood of defending shoulder dystocia cases successfully has increased significantly.

## REFERENCES

1. Physician's Insurers Association of America (PIAA): Closed Claims Study, 2005.
2. *Gourley v. Nebraska Methodist Health System*, 663 N.W.2d 43 (2003).
3. *Wareing v. United States*, 943 F. Supp 1504 (1996).
4. White AA, Pichert JW, Bledsoe SH, Irwin C, Entman SS: Cause and effect analysis of closed claims in obstetrics and gynecology. *Obstet Gynecol* 2005; 105:1031–1038.
5. Institute of Medicine: *To Err is Human: Building a Safer Health System*. Washington DC: The National Academics Press, 2000.
6. American College of Obstetricians and Gynecologists: *Surgery and patient choice*. Committee Opinion No. 289. Washington, DC: American College of Obstetricians and Gynecologists, 2003.
7. Glazener CMA, Abdalla M, Stroud P, Naji S, Templeton A, Russell IT: Postnatal maternal morbidity: Extent, causes, prevention and treatment. *Br J Obstet Gynaecol* 1995;102:282–287.
8. Minkoff H, Chervenak FA: Elective primary cesarean delivery. *N Engl J Med* 2003;348:946–950.
9. Pritchard JA, Baldwin RM, Dickey JC, Wiggins KM: Red blood cell loss and changes in apparent blood volume during and following vaginal delivery, cesarean section, and cesarean section plus total abdominal hysterectomy. *Am J Obstet Gynecol* 1962;84:1271.
10. Sultan AH, Stanton SL: Preserving the pelvic floor and perineum during childbirth – elective caesarean section? *Br J Obstet Gynaecol* 1996;103:731–734.
11. Hemminki E, Merilainen J: Long-term effects of cesarean sections: Ectopic pregnancies and placental problems. *Am J Obstet Gynecol* 1996;174:1569–1574.
12. Greene R, Gardeil F, Turner MJ: Long-term effects of cesarean sections. *Am J Obstet Gynecol* 1996; 176:254–255.
13. Scott JR: Putting elective cesarean into perspective. *Obstet Gynecol* 2002;99:967–968.
14. Clark SL, Koonings PP, Phelan JP: Placenta previa/accreta and prior cesarean section. *Obstet Gynecol* 1985;66:89–92.
15. *Schreiber v. Physicians Ins.*, 579 N.W.2d 730 (1998).
16. The Boston Globe, *Meador v. Stahler and Gheridian*, Wednesday, June 16, 1993.
17. Varner MW: Neuropsychiatric sequelae of midforceps deliveries. *Clin Perinatol* 1983;10:455–460.
18. Dennen PC: *Dennen's Forceps Deliveries*, 3rd ed. Philadelphia: FA Davis, 1989.
19. O'Grady JP: *Modern Instrumental Delivery*. Baltimore: Williams & Wilkins, 1988.
20. Laufe LE, Berkus MD: *Assisted Vaginal Delivery: Obstetrical Forceps and Vacuum Extraction Techniques*. New York: McGraw-Hill, 1992.
21. American College of Obstetricians and Gynecologists: *Obstetric forceps*. Committee Opinion No. 71. Washington, DC: American College of Obstetricians and Gynecologists, 1989.
22. American College of Obstetricians and Gynecologists: *Operative vaginal delivery*. Technical Bulletin No. 152. Washington, DC: American College of Obstetricians and Gynecologists, 1991.
23. American College of Obstetricians and Gynecologists: *Operative vaginal delivery*. Technical Bulletin No. 196. Washington, DC: American College of Obstetricians and Gynecologists, 1994.
24. Healy DL, Laufe LE: Survey of obstetrical forceps training in North America in 1981. *Am J Obstet Gynecol* 1985;1:54–58.
25. Ross BK: ASA closed claims in obstetrics: Lessons learned. *Anesthesiol Clin North Am* 2003;21:183–197.



26. *Lanzet v. Greenberg*, 594 A. 2d 309 (1991).
27. *Oberzan v. Smith*, 869 P. 2d 682 (1994).
28. Hawkins JL, Gibbs CP, Orleans M, Martin-Salvaj G, Beaty B: Obstetric anesthesia work force survey, 1981 versus 1992. *Anesthesiology* 1997;87:135-143.
29. *Denton v. LaCroix*, 947 S.W. 2d 941 (1997).
30. *Forrestal v. Magendantz* 848 F.2d 303 (1988); *Kavanaugh v. Nussbaum* 523 N.E. 2d 284 (1988).
31. *Forrestal v. Magendantz* 848 F.2d 303 (1988).
32. *Carmen v. Dippold* 379 N.E. 2d 1365 (1978).
33. *Williams v. Lallie Kemp Charity Hospital.*, 428 So.2d 1000 (1994).
34. *Mundaell v. La Pata No.* 1-92-1 245, 1994 WL 202583 (Ill. Ct. App 1994).
35. *Simpson v. Wilkinson No.* 21265188 (Kings Cty Sup. Ct. Brooklyn, NY (1992).
36. Comstock C, Mathies H: X-ray pelvimetry. In Sciarra JJ (ed): *Gynecology and Obstetrics*. Philadelphia: JB Lippincott, 1994:1.
37. *Jones v. Karraker* 440 N.E. 2d (1982).
38. American College of Obstetricians and Gynecologists: Intrapartum fetal heart rate monitoring. Technical Bulletin No. 132. Washington DC: American College of Obstetricians and Gynecologists, 1989.
39. Cunningham FG, MacDonald PC, Leveno KJ, et al. (eds): *Williams Obstetrics*, 19th ed. Norwalk, CT: Appelton and Lange, 1993:487.
40. Cunningham FG, MacDonald PC, Gant NF, et al. (eds): *Williams Obstetrics*, 20th ed. Stamford, CT: Appelton and Lange, 1997:427.
41. *Mertsaris v. 73rd Corp* 482 N.Y.S. 2d 792 (1984).
42. Atshuler G: Placenta within the medicolegal imperative. *Arch Pathol Lab Med* 115:688-695, 1991.
43. Atshuler G, Deppisch LM: College of America Pathologists Conference XIX on the examination of placenta: Report of the working group on indications for placental examinations. *Arch Pathol Lab Med* 115:701-703, 1991.
44. *Gabaldoni v. Bd. of Physicians* 785 A.2d 771 (2001).
45. Pritchard JA, Baldwin RM, Dickney JC, et al: Blood volume changes in pregnancy and the puerperium. *Am J Obstet Gynecol* 84:1271-1273, 1962.
46. Mackay AP, Berg CJ, Atrash HK: Pregnancy-related mortality from preeclampsia and eclampsia. *Obstet Gynecol* 97:533-538, 2001.
47. Witlin AG, Sibai BM: Magnesium sulfate therapy in preeclampsia and eclampsia. *Obstet Gynecol* 92: 883-889, 1998.
48. Borders N: After the afterbirth: A critical review of postpartum health relative to method of delivery. *J Midwifery Women's Health* 514:242-248, 2006.
49. Lydon-Rochelle M, Holt VL, Martin DP, Easterling TR: Association between method of delivery and maternal rehospitalization. *JAMA* 283:2411-2416, 2000.
50. American College of Obstetricians and Gynecologists: Diagnosis and management of postpartum hemorrhage, Technical Bulletin No. 143. Washington, DC: The American College of Obstetricians and Gynecologists, 1990.
51. American College of Obstetricians and Gynecologists: Committee Opinion No. 265. Mode of term singleton breech delivery. Washington, DC: American College of Obstetricians and Gynecologists, 2001.
52. DeCrespinsky LJC, Pepperell RJ: Perinatal mortality and morbidity in breech presentation. *Obstet Gynecol* 1979;53:141-145.
53. Hall J, Kohl S: Breech presentation: A study of 1456 cases. *Am J Obstet Gynecol* 1956;72:977-990.
54. Gimovsky ML, Petrie RH: Strategy for choosing the best delivery route for the breech baby. *Contemporary OB/GYN* 1983;21:201-215.
55. Brenner WE, Bruce RS, Hendricks CH: The characteristics and perils of breech presentation. *Am J Obstet Gynecol* 1974;118:700-712.
56. Cruishank DP, Pitkin RM: Delivery of the premature breech. *Obstet Gynecol* 1977;50:367-369.
57. *Draper v. Jasionowski*, 858 A. 2d 1141 (2004).
58. Gregory KD, Korst LM, Krychman M, Cane P, Platt LD: Variation in vaginal breech delivery rates by hospital type. *Obstet Gynecol* 2001;97:385-390.
59. Healy DL, Laufé LE: Survey of obstetrical forceps training in North America in 1981. *Am J Obstet Gynecol* 1985;151:54-58.
60. *Mundell v. La Pata*, 635 N.E. 2nd 933 (1994).
61. Morris WIC: Shoulder dystocia. *Br J Obstet Gynecol* 1955;62:302-306.
62. *Stennis v. Rekkas*, 599 N.E. 2d 1059 (1992).

# SURGICAL PROCEDURES

## Chapter 16 SURGERY IN PREGNANCY

Reinaldo Figueroa

J. Gerald Quirk

*The Chirurgeon must have a goode eyes and a stedfast hande (for chirurgy taketh its name of this). He must have goode witte and memory and goode judgement.*

*Chirurgeons ought to be wyse and gentil, sober and circumspect.*

*They muste be learned and not drunken.*

*Nor must they promise more than they can perform with God's helpe.*

Andrew Boorde (1490–1549)  
*The Brevyary of Health (1547?)*

Most conditions requiring surgery during pregnancy are due to complications unique to gestation, such as obstetric hemorrhage from abnormal placentation, or result from problems encountered during vaginal or cesarean delivery. Pregnant women also can suffer from acute abdominal conditions such as acute cholecystitis, appendicitis, trauma, and various neoplastic diseases of the genital tract, however. To treat these patients, the obstetric surgeon must know the unique physiologic changes associated with pregnancy, the limitations imposed by uterine size, and the peculiarities of the clinical presentation modified by the pregnancy changes. This chapter considers selected aspects of surgical technique, complications, and the management of some surgical problems that develop in association with pregnancy.

### ESTABLISHING THE DIAGNOSIS

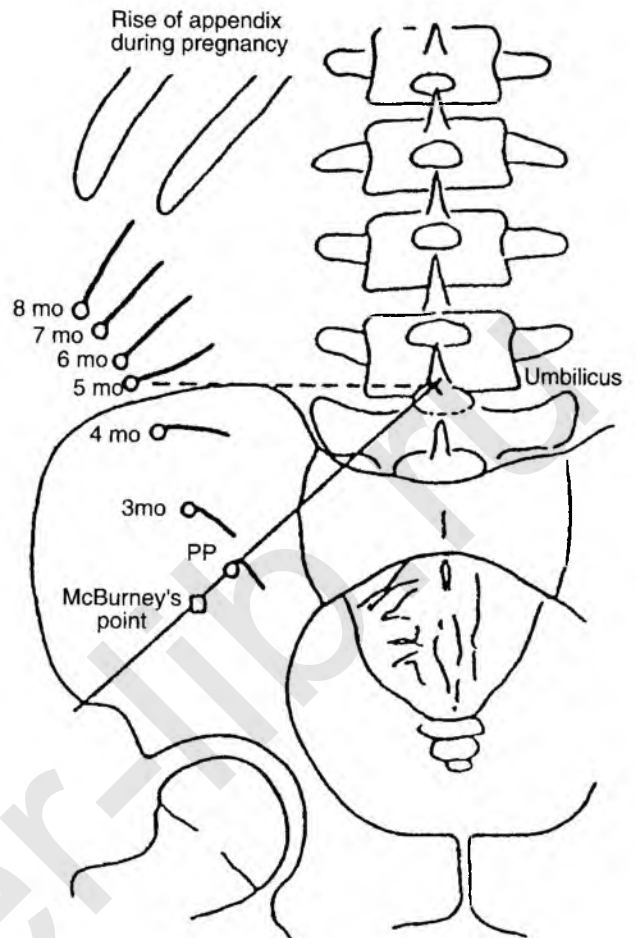
#### History and Physical Examination

Most surgical conditions that occur outside of pregnancy also occur in pregnant women. Prompt diagnosis and judicious timing of procedures are imperative during pregnancy because unnecessary delays result in increased morbidity and mortality to both mother and fetus. It is potentially dangerous to attribute all reports of abdominal pain in pregnant women to obstetric conditions such as labor, placental abruption, degenerating uterine leiomyoma, or the round ligament syndrome. The diagnostic evaluation should include a carefully taken history, a complete physical examination, and the appropriate use of laboratory studies. The signs and symptoms of various surgical conditions are modified by the anatomic and physiologic changes that accompany pregnancy, paradoxically often resulting in their exacerbation, an apparent reduction in intensity, or a change in the location of the expected physical signs. For example, failure to consider normal gestational

changes in the digestive tract can delay the diagnosis of cholecystitis. Nausea and vomiting during the first trimester might be attributed to hyperemesis gravidarum and not be recognized as symptoms of cholecystitis, appendicitis, or bowel obstruction [1,2]. Gastroesophageal reflux and pyrosis, from physiologic reduction of lower esophageal tone and increased gastric pressure, can incorrectly suggest peptic ulcer disease. Intestinal obstruction might not be promptly recognized because constipation is deemed physiologic owing to elevated progesterone levels or from mechanical compression by the gravid uterus. Conversely, constipation during pregnancy can be severe enough to cause a pseudo-obstruction, inciting clinical concern but requiring conservative treatment rather than surgery [3].

During pregnancy, the location and progression of pain from various surgical conditions change over time, mostly from anatomic displacement by the enlarging uterus. Best known is the progressive upward and counterclockwise displacement of the appendix as the uterus grows out of the true pelvis during the second trimester (Figure 16.1). In appendicitis, the point of maximal tenderness in the third trimester rises into the right upper quadrant, and an erroneous diagnosis of cholecystitis or pyelonephritis is sometimes considered [4]. The pain and tenderness can be less well localized and more diffuse as the omentum is displaced by the uterus and is less effective at walling off the inflammatory process.

Women of childbearing age are increasingly victims of trauma, a trend that does not spare pregnant women [5]. Trauma is the leading cause of nonobstetric maternal death in the United States [6,7]. Injuries range in severity from a minor fall, which the pregnant woman might not remember but that could result in a ruptured spleen, to motor vehicle accidents, stab and gunshot wounds, criminal assaults, or battering [8]. In cases of obvious trauma, the physical examination must focus first on airway patency, ensuring adequate breathing, and maintaining vital signs, keeping in mind the physiologic tachycardia, mild second-trimester reduction in arterial pressure, and expanded blood volume normal in pregnancy. Shock can be aggravated in the third trimester by uterine compression of the inferior vena cava [9]. Ultrasonography or computed tomography (CT) can confirm free intraperitoneal fluid, suggesting a hemoperitoneum, or can otherwise indicate the site of injury. If these stud-



**FIGURE 16.1.** Location of the appendix at varying stages of pregnancy. (From Baer JL, Reis RA, Arens RA: *Appendicitis in pregnancy with changes in positions in areas of normal appendix in pregnancy.* JAMA 1932;98:1359-1364; with permission.)

ies are inconclusive and hemoperitoneum is suspected, paracentesis with peritoneal lavage can be performed safely at any gestational age, with care taken to avoid direct uterine puncture. Some investigators have recommended that pregnant women with suspected abdominal trauma should undergo electronic fetal monitoring (EFM) for a minimum of 4 hours of monitoring [10,11]. If uterine activity is not present, the risk of placental separation is low, and the patient can be safely discharged. If there is uterine irritability or tenderness, vaginal bleeding, or a nonreassuring fetal heart rate pattern, the patient should receive at least 24 hours of continuing fetal heart rate monitoring because of the risk of delayed placental separation [11].

## Laboratory and Other Tests

Laboratory data add to the information gathered from the clinical signs and symptoms in establishing a diagnosis. The interpretation of laboratory results must take into account the physiologic changes of pregnancy, which alter normal values. For example, a moderate leukocytosis is normal in pregnancy, but a white blood count of 20,000 or more, or a marked decrease, should not be ignored. The hypervolemia of pregnancy accounts for a mild decrease in hemoglobin concentration, but hemoglobin levels below 10.0 g/dl in a patient with tachycardia or hypotension suggests blood loss. Apart from labor, red blood cells in the urine imply urinary tract pathology such as a calculus, infection, or tumor.

Radiographs should be kept to the minimal number necessary. Clinically indicated imaging studies should be neither avoided nor delayed because of pregnancy, regardless of the period of gestation, however. Plain abdominal radiographs suggest a diagnosis of bowel obstruction if intestinal distension and air fluid levels are present. Free air under the diaphragm indicates a perforated viscus, unless the film closely follows abdominal surgery. Depending on its location, a radiopaque calculus documents cholelithiasis or nephrolithiasis in the presence of suggestive clinical findings. Contrast studies might be indicated to identify the level of occlusion when there is a strong suspicion of bowel or ureteral obstruction. Avoidance of multiple exposures and prolonged fluoroscopy, or a limited intravenous pyelogram and modified techniques to minimize exposure to the fetus, usually provide sufficient information to arrive at a diagnosis. The risk of inducing a congenital anomaly or later development of a malignancy or leukemia is miniscule. Most diagnostic radiographic procedures result in an exposure to the fetus of between 0.02 centigray (cGy) and 5 cGy, well below the estimated minimal dose of 20 cGy to produce growth restriction, possible mental retardation, or gross anatomic malformation. Based on controversial data, the risk for a childhood carcinogenic effect for a calculated 1 cGy fetal exposure is very low, with estimates varying from 3.4 in 10,000 to 5 in 1,000,000 [12]. In contrast, ultrasonography can be used liberally for a wide number of applications because no fetal ill effects are documented. Recently, magnetic resonance imaging (MRI) has been used more frequently in the diagno-

sis of various surgical conditions during pregnancy [13,14].

## SURGICAL TECHNIQUE

### Operative Incisions

The choice of the surgical incision in a gravid woman depends on the disease process for which surgery is indicated, gestational age and fetal presentation, experience of the surgeon, and the urgency of intervention required. The advantage of a vertical incision is rapid easy access in a relatively bloodless plane, with potential for extension, if required. A vertical incision is also useful when the diagnosis is uncertain. The transverse incision has the advantages of superior cosmetic results and decreased pain, resulting in less pulmonary depression; its disadvantages include increased operative time, more bleeding, and creation of multiple potential spaces. In the first trimester of pregnancy, operations on the pelvic organs are performed through a Pfannenstiel, Cherney, or Maylard incision, unless a neoplasm such as carcinoma of the ovary is suspected, for which a midline incision is recommended. The uterus grows outside of the pelvis by the twelfth to fourteenth week of gestation. If a laparotomy is needed after this time, a midline incision, extended cephalad as necessitated by the size of the gravid uterus, provides better exposure. Properly closed midline incisions, as compared to transverse incisions, are not associated with an increased rate of dehiscence [15–17]. For suspected appendicitis, a muscle-splitting incision is made over the point of maximal tenderness. The appendix is usually displaced upward and outward toward the right upper quadrant as pregnancy advances, and the progressively enlarging fundus elevates the cecum [4]. In positioning the patient for surgery during the third trimester, a left lateral decubitus position is preferable to avoid supine hypotension from uterine compression of the inferior vena cava [9]. During abdominal procedures, manipulation of the pregnant uterus is minimized to avoid uterine irritability.

An incision perpendicular to the skin, which avoids tangential cutting or a jagged, erratic line, yields the best cosmetic results. In incising the tissues, countertraction, applied to the skin and underlying tissues by the assistant or the surgeon's non-dominant hand, fixes the area to be entered and

guides the incision through natural anatomic planes, minimizing tissue disruption and bleeding. The skin and subcutaneous tissues are incised with one sweep of the scalpel to the desired depth, restricting tissue damage [18]. The common practice of using a second knife for the subcutaneous tissues after incision of the skin does not reduce the risk of infection and is unnecessary because the knife blade is not a vehicle for bacterial contamination [19–21]. Skin incisions made with an electrosurgical unit result in less blood loss and are accompanied by a rate of wound infection similar to that of incisions made by a scalpel [22–24].

### **Skin Preparation, Hemostasis, and Wound Closure**

Hemostasis by obliteration of bleeding vessels reduces the incidence of wound hematoma and infection and restricts blood loss. Pinpoint electrocoagulation below the dermis, using the lowest effective energy delivered to isolated bleeding vessels, minimizes thermal tissue injury. Ligature with fine polyglycolic acid suture material is also acceptable. For oozing and minor bleeding, gentle continuous pressure applied to the bleeding surface with a saline-moistened sponge is often effective and has the advantage of not devitalizing surrounding tissues or leaving foreign material in the wound [25].

Hair removal as a preparation for surgery has been a traditional practice based on two rationales: hair harbors bacteria, which can be a source of contamination, and hair can interfere with skin closure. Shaving hair from the operative site the evening before surgery, however, increases the rate of wound infection by creating microcuts and microabscesses in the skin. No shaving at all is associated with the lowest risk of infection. If hair removal is required, it is best done immediately before the surgery by clipping the hair instead of shaving it [26,27].

A strong and dependable closure of the wound is most important during pregnancy because of the increased intraabdominal pressure. Clinical observations and experimental data provide valuable information concerning wound closure. The parietal peritoneum need not be routinely closed. Its closure does not strengthen the wound, and peritoneal closure leads to focal ischemic areas, favoring the formation of adhesions [28–30]. The peritoneal defect rapidly fills with an inflammatory exudate, which is replaced within 72 hours by fibroblasts and the

development of new mesothelium. The edges of the fascia should be closely, but not tightly, approximated. Excessively tight sutures cause strangulation of tissue and ischemic necrosis, increasing the potential for dehiscence. The tensile strength of closely approximated wounds is far stronger than those in which a “tight” closure has been attempted [31,32]. A continuous suture incorporating a “1.5 cm wide, 1 cm apart” bite of fascia has the advantage of a better distribution of tension along the entire length of the incision, compared with interrupted sutures [33]. Such running closures have greater wound-bursting pressure than figure-of-eight or Smead-Jones sutures. They save significant operative and anesthesia time, with a significant decrease in the rate of incisional hernias and no difference in wound infection or dehiscence rate [34–36]. In a continuous suture, however, the integrity of the entire closure rests on a single suture and knot. Thus, close attention to detail to avoid damage to the suture and to the technique of knot tying is important. In particular, the suture should not be crushed with the needle holder, weakening the material.

Dead space within the wound favors the collection of blood and serum, providing a good culture medium, and interferes with the local immune response. Attempts to obliterate dead space by suturing the subcutaneous tissues are inappropriate, however. Suture material in the subcutaneous plane acts as a foreign body, resulting in local areas of ischemia and tissue necrosis. Closure of the subcutaneous tissues does not add tensile strength to the wound and is best avoided [37]. In a particularly wet case, a closed suction drain (Jackson-Pratt type) removes fluid and obliterates the dead space but can also increase the risk for wound infection. (Drains are discussed in greater detail later in this chapter.) A pressure dressing can reduce dead space formation in the wound, but as usually applied, such dressings are largely ineffective.

### **Suture Materials**

The role of the suture material is to restore normal anatomic relationships while awaiting the patient's own repair mechanisms to restore tissue integrity. If a perfect suture material were available, it would be easy to handle, have low tissue drag, maintain good knot security, and have lasting tensile strength. It would also be nonallergenic, provoke minimal

inflammation, retain holding power in the presence of infection, and eventually resorb in a predictable fashion. Although this theoretically perfect material does not exist, several new materials do approach the ideal. Suture materials are divided based on their origin (natural or synthetic), absorbability within tissues, and whether they have a monofilament or braided structure. Each suture material has specific handling characteristics, advantages, and disadvantages. Silk is a natural, nonabsorbable suture material, which has poor knot security in body fluids and loses its tensile strength within 14 days as it is degraded by hydrolysis, proteolysis, and phagocytosis [38]. Classic surgical gut has a tendency to fray easily with knot tying and causes an intense tissue reaction. In modern surgery, silk and gut have been replaced by synthetic materials. Both polyglycolic acid (Dexon) and polyglactin (Vicryl) are absorbable, multifilament, braided sutures that maintain tensile strength for 14 to 21 days and are completely resorbed in 28 to 70 days. Polydioxanone (PDS) and polyglyconate (Maxon) are synthetic, delayed, absorbable, monofilament sutures; they maintain 50% of their tensile strength at 4 weeks and are completely absorbed at 180 days. They are therefore excellent choices for fascial closure in abdominal incisions. Polypropylene (Prolene) is a nonabsorbable, or permanent, monofilament suture, which can be an advantage in infected wounds. In some thin patients the knots from polypropylene are palpable through the skin and are a source of discomfort or chronic sinus formation [39]. Burying the knot below the fascia largely prevents this problem. As a group, monofilament sutures are theoretically less likely to be colonized with bacteria than the multifilament sutures, because they contain no interstices in which bacteria can hide.

The tying characteristics and knot security of surgical sutures depend on the configuration of the knot and number of throws. Two throws on a square knot or surgeon's knot or three throws on a sliding knot have a high rate of knot failure, and these techniques are therefore not recommended. The loop-holding capacity of surgeon's knots and square knots are comparable; the only benefit of a surgeon's knot is that the double-loop first throw does not slip easily. With thicker-gauge sutures, such as 0, square knots are clearly superior to sliding knots; with smaller diameter sutures, such as 3-0, the strength of the square knots and sliding knots with an extra

throw are identical. When the loop-holding capacities of five-throw and three-throw sliding knots are compared, the additional two throws result in significantly less knot failure for monofilament as opposed to multifilament synthetic sutures [40,41].

### Skin Closure

Skin edges are approximated with stainless steel staples, fine nylon sutures, adhesive strips (Steri-Strips) or tissue adhesive (cyanoacrylate). For an incision with minimal tension, closure with an adhesive strip or tissue adhesive provides the best cosmetic result and the lowest rate of infection [42,43]. In terms of infection risks, skin staples have been considered superior to the least reactive nonabsorbable suture, monofilament nylon [44]. Recent reports suggest that a running subcuticular closure with polydioxanone or polyglactin, even though it takes longer to place, results in less postoperative discomfort and better appearance than staples [45,46]. Silk is not a good choice for skin closure because it is among the most reactive suture materials, and its braided multifilaments allow organisms to gain access to the wound more easily.

### Drains

Drains are sometimes indicated in pregnant women undergoing surgery. The principal role of a drain is to prevent the accumulation of blood or other body fluids within the wound or other body cavity. Drains are also used to remove a purulent collection, as with an appendiceal or pericolic abscess. A sump or vented drain that uses constant suction is less prone to blockage by fibrin or tissue and is more effective for evacuating abscesses than is any passive drain. Drains are left in place long enough for a sinus tract to form and prevent premature healing of the skin over an abscess cavity. Drains can also be used prophylactically to prevent the formation of a hematoma or a seroma in the pelvis or in the subcutaneous tissues in obese or other high-risk patients. Even a sterile collection within the wound impairs healing, but the combination of a hematoma with bacterial contamination is an excellent nidus for wound infection. Based on these observations, the authors recommend a closed suction drainage system that emerges through a separate stab wound at cesarean hysterectomy or other major surgery,

evacuates serum and blood, and collapses the potential dead space within the wound. The drain is removed when the volume of drainage is minimal, usually within 24 to 48 hours. Drainage has potential complications. The risk of a prophylactic drain is that skin contaminants can gain retrograde access to the wound through the drain puncture site, especially if an open passive drain such as a Penrose drain is used. Such nonsuctioning open drainage is best avoided. A drain in the wound also acts as a foreign body, potentially further impairing the host's defense mechanisms. When the pros and the cons are weighed, it is best to drain for specific indication only, using closed, constant-suction drains exiting through a stab wound separate from the original incision [47–51].

### **WOUND COMPLICATIONS**

Impaired healing and wound infections are among the most common complications of surgery, in pregnant as in nonpregnant patients. Wound complications are often prevented by careful technique. Gentle handling of tissues, adequate hemostasis, débridement of devitalized tissue, secure but not excessively tight fascial approximation, appropriate suture material, and avoidance of dead space usually lead to uneventful wound healing by primary intention. Most pregnant women are young, in good general health, and do not suffer from additional risk factors for delayed healing such as malnutrition, cancer, diabetes mellitus, or immunosuppressive states. Nonetheless, pregnancy provides unique risks to wound integrity. When abdominal surgery is performed during pregnancy, the incision is subjected to increasing intraabdominal pressure as the pregnancy advances. This additional stress tends to pull the edges of the wound apart, especially with vertical incisions. Poor wound healing can result in an asymptomatic incisional hernia but also can result in a complete disruption of all wound layers, leading to evisceration of abdominal contents with potentially high mortality rates (10%–35%) [52].

#### **Incisional Hernia**

Pressure symptoms and discomfort around the incision, accompanied by palpation of a protruding mass through a defect in the fascia, are diagnostic of an *incisional hernia*. The defect occurs when the edges

of the fascia either fail to heal initially or separate after inadequate healing. The defect can be small or involve the entire length of the incision. At the site of the hernia, the attenuated tissues are limited to the peritoneum, the subcutaneous tissues, and the skin. As long as the hernial sac contents are reducible into the abdominal cavity, there is no urgency. When the herniated tissue cannot be reduced, however, incarceration is possible, potentially leading to bowel or omental strangulation. If an abdominal incisional hernia develops during pregnancy but before delivery, it is best managed conservatively, with definitive repair performed electively some weeks after parturition. As pregnancy progresses, the enlarging uterus usually displaces the bowel and omentum away from the abdominal incision site, reducing the likelihood of visceral herniation or strangulation. An attempted repair during pregnancy increases the risk of premature delivery and has a high failure rate. Indications for immediate operation include signs or symptoms suggestive of incarceration or strangulation. During correction of the hernia, the principles of repair include 1) reconstitution of the normal anatomic planes, 2) freeing up, opening, and excising the hernial sac, 3) restitution of the herniated viscera into the abdomen, and 4) approximation of the separated aponeurotic and fascial structures with a nonabsorbable, continuous suture such as polypropylene [53].

#### **Wound Dehiscence and Evisceration**

*Wound dehiscence* or complete disruption of the fascia usually occurs between the fifth and fifteenth postoperative day. A copious serosanguinous discharge from the wound early in the postoperative period is an ominous sign of impending evisceration. If the skin is intact, absence of a palpable healing ridge at the level of the fascia confirms its separation. Such a wound must be explored in the operating suite and not at the bedside. Extrusion of abdominal contents with potential bacterial contamination of the peritoneal cavity carries a high rate of morbidity and potential mortality [52]. If evisceration occurs outside of the surgical suite, the defect and viscera should be immediately covered with moist sterile saline packs, and the patient must be promptly returned to the operating room. Broad-spectrum parenteral antibiotics to cover skin flora, as well as genital or gastrointestinal tract organisms,

are administered. At surgery, the edges of the fascia are débrided, and a mass closure is performed using a continuous, large, monofilament nonabsorbable suture such as polypropylene. This closure incorporates all tissue layers at a good distance from the fascial edges. Some surgeons reinforce this repair with retention sutures of similar material placed through the entire abdominal wall several centimeters from the wound edge. These retentions are placed under direct vision to avoid puncture or entrapment of intestinal loops. Specially designed bridges are used below these retention sutures to distribute the pressure of the suture over a wider skin surface and prevent ulceration or cutting at the cutaneous puncture sites.

### Wound Infection

Wound infections usually manifest between the fifth and the seventh postoperative day. The definition of *wound infection* includes 1) *wound pain*, combined with fever, and marginal cellulitis with or without minimal purulent exudate, 2) *wound cellulitis* with a significant amount of purulent material, or 3) a *positive culture* from a separated wound. Not every stitch abscess or minimal wound erythema qualifies as a wound infection. An infected wound can lead to additional complications, including bacteremia, fascial dehiscence, or rarely, necrotizing fasciitis or septic shock. The risk of developing a wound infection is related to the size of the bacterial inoculum, the surgical technique, and the immunocompetence of the host [54]. Most bacteria responsible for wound infection are endogenous and originate from the gastrointestinal or genitourinary tract; therefore, despite the risk of uterine irritability, a preoperative mechanical bowel preparation with fluid diet and cathartics is indicated in the unusual case of elective colonic surgery during pregnancy. Exogenous skin contaminants are less important in the etiology of wound infection since their numbers are significantly reduced by the usual chlorhexidine or povidone-iodine surgical skin scrub. Surgical technique plays an important role in prevention, with adequate hemostasis, preservation of blood supply, débridement of necrotic tissue, gentle handling of tissues, and wound irrigation as prominent features of good technique. Prophylactic antibiotics are indicated when the intestinal or genitourinary tract are likely to be entered, the host's

immunocompetence is compromised, or contamination of the operative site is likely due to known or suspected chorioamnionitis. Adequate coverage for bowel surgery includes a single-dose cephalosporin such as cefazolin, gentamicin plus clindamycin, or metronidazole [54]. For cesarean delivery in high-risk patients, the recommended prophylactic treatment is 1 g or 2 g of intravenous cefazolin administered after the umbilical cord is clamped.

Despite such preventive measures, wound infections still occur. Responsible pathogenic organisms vary considerably with the site of surgery, the operation performed, and the prevalent flora in the hospital environment. Common pathogens include *Staphylococcus aureus*, Group A *streptococcus*, *enterococci*, *Escherichia coli*, *Pseudomonas aeruginosa*, *Proteus mirabilis*, *Bacteroides fragilis*, and other anaerobic bacteria [54]. The clinical signs and symptoms of an infected wound include localized pain, erythema, edema, seropurulent discharge, and fever. Staphylococcal infections lead more to abscess formation and thick odorless pus; streptococcal infections are less localized, with diffuse cellulitis, lymphangitis, vesicular formation, and less tendency for abscess formation. With a gram-negative aerobic infection, the local signs are often less impressive, but the patient has more systemic manifestations such as fever, tachycardia, hypotension, or shock [55].

The treatment of an infected wound consists of opening the wound for drainage, débridement of necrotic tissue, evacuation of pus, and irrigation. Systemic antibiotic therapy is indicated in more severe infections or in immunocompromised hosts; the choice being guided by the suspected organism, the appearance of the wound, the presence of systemic signs, and the local hospital antibiotic resistance pattern. The wound edges can be reapproximated after a few days when clean granulation tissue is present, using adhesive strips or interrupted monofilament sutures placed with the area under local anesthesia. Bacterial counts within the wound can be estimated by culture from a biopsy, but the clinical appearance of the wound in most cases is as reliable an indicator as laboratory studies that secondary closure can be attempted [56].

### *Necrotizing Fasciitis*

*Necrotizing fasciitis*, also known as streptococcal gangrene of mixed bacterial infection, is a severe type



of wound infection that can involve an abdominal incision or, uncommonly, a perineal wound such as an episiotomy site. Tissue necrosis develops from the synergistic action of gram-positive cocci and both aerobic and anaerobic gram-negative bacilli. Certain debilitating systemic diseases such as cancer or advanced diabetes, uncommon in pregnant women, increase the risk of necrotizing fasciitis. In addition to cellulitis, multiple skin ulcers are sometimes observed, draining a thin reddish-brown foul-smelling exudate. On palpation, there can be crepitus and altered sensation, ranging from severe tenderness to anesthesia. The clinical course of this unusual disorder is variable, either indolent or fulminant. Gangrene of the skin sometimes occurs owing to thrombosis of cutaneous vessels. Clinical manifestations range from dusky areas to areas of frank sloughing; pus formation is scant. Systemic signs can include fever and chills, or even septic shock. Blood cultures are frequently but not invariably positive. The diagnosis is suggested when probing a wound demonstrates easy separation between the subcutaneous tissues and the fascia, and interconnection between skin ulcers. A common finding is a thin gray "dishwater" wound discharge and the observation that wound probing fails to elicit bleeding. A biopsy reveals a characteristic pattern of tissue necrosis and bacterial invasion. Gram's stains most often reveal a mixture of gram-positive and gram-negative organisms, unless the patient suffers from streptococcal or clostridial gangrene [57].

Aggressive therapy is required for necrotizing fasciitis because the risk of mortality is high [58]. The most important treatment is urgent, radical débridement, resecting all necrotic tissue and exposing normal fascia. When a full-thickness abdominal wall defect is involved, a synthetic mesh graft can be necessary to allow closure of the defect. Postoperative irrigation of the wound removes residual purulent and necrotic material. Ancillary therapy with broad-spectrum antibiotic coverage is appropriate, including an aminoglycoside, metronidazole or clindamycin, combined with high-dose ampicillin. Other widespread antibiotic regimens can be appropriate, based on local pathogens and sensitivities. Hyperbaric oxygen can reduce morbidity and mortality by improving tissue oxygenation but is only ancillary to surgical débridement [59]. The prognosis in necrotizing fasciitis is always guarded and depends largely on the underlying disease pro-

cess, overall immunocompetence of the patient, and especially the delay until the initiation of definitive, surgical therapy.

## SYSTEMIC COMPLICATIONS

### **Febrile Morbidity**

*Fever* is a common occurrence after any surgical procedure. By itself, a temperature elevation is neither a serious complication nor necessarily an indication for antibiotic therapy. Postoperative fevers are so common that there is no consensus as to what represents a normal postoperative temperature or what is considered febrile morbidity. In one review of the literature, 32 definitions of postoperative febrile morbidity were referenced from 92 publications [54]! Diagnostic criteria were more or less stringent, ranging from 38.6°C in the first 24 hours after surgery, or more than 38.3°C thereafter, to 37.5°C on two or more consecutive days more than 24 hours after surgery.

Practically, the magnitude of a fever and the presence or absence of other clinical signs, including tachycardia, tachypnea, hypotension, oliguria, jaundice, abdominal distension, wound pain, confusion, or drowsiness, can help to discriminate a minor complication from a more serious one. The differential diagnosis includes atelectasis, dehydration, superficial phlebitis at the intravenous infusion site, drug fever, a mild transfusion reaction, pneumonia, urinary tract infection, wound infection, intraabdominal abscess, anastomotic leak, or central venous line sepsis. Potential noninfectious etiologies of fever include thromboembolism, myocardial infarction, pancreatitis, or tumor, among others [60]. In the pregnant patient, any serious systemic infection can result in fetal tachycardia or potentially be associated with premature labor.

A systematic approach to the differential diagnosis is based on the site and type of surgery, and the time since the initial operation. In the initial 24 to 48 hours, atelectasis is the most common source of fever. This is particularly true with surgery in the upper abdomen because the patient's deep inspiratory efforts are impaired by pain. The fever usually associated with atelectasis is low grade, less than 38.5°C, but occasionally can be as high as 40°C. Most patients with atelectasis appear well, without other morbid clinical signs, unless excessive

sedation is the primary cause leading to poor ventilation. Auscultation of the lungs usually reveals poor inspiratory efforts and fine inspiratory crackles heard at the bases. Because most pregnant women are neither debilitated nor immunocompromised, the risk of pulmonary consolidation is low. In most cases, a chest radiograph is not indicated. The surgical site should be examined even though wound infection is uncommon in the first 2 days after surgery. The patient's hydration status and any intravenous sites should also be checked for signs and symptoms of inflammation. Urinalysis and urine culture should also be performed, because urinary tract infection is a common and serious cause of morbidity, especially in the pregnant woman. Treatment of atelectasis is conservative and includes encouraging the patient to take deep breaths and cough, use incentive spirometry, and ambulate. Occasionally, chest physiotherapy is indicated. Analgesics can be increased if incisional pain is perceived to be the problem or reduced if the patient is oversedated. Ambulation and vigorous pulmonary toilet speed up the recovery process. If the diagnosis is correct, the fever will promptly disappear without additional treatment.

Fever that persists or begins four or more days after surgery is suggestive of infection, especially if the patient complains of unusual pain, the anticipated postoperative leukocytosis does not resolve, or there is a shift to the left in the differential count. Pain, erythema, edema, or increased warmth over the wound suggests a wound infection. Chest examination or radiography could indicate pneumonia. Microscopic examination of the urine for white blood cells and bacteria, and urine culture and sensitivity, is mandatory. Empirical antibiotic therapy for the pregnant woman with presumed pneumonia, urinary tract infection, or uterine infection pending culture results is appropriate. The choice of agent is based on the severity of the patient's clinical condition, any known hypersensitivity to medications, the hospital flora and local resistance pattern for nosocomial infection, and the stage of pregnancy. If the patient has either an arterial or venous indwelling catheter or central line and no other apparent source of infection is identified, it is best to remove or replace the catheter, perform cultures, and initiate broad-spectrum antibiotic coverage.

An intraabdominal abscess is suspected when abdominal surgery for an infectious process or a

bowel resection was performed and then an otherwise unexplained fever developed after 4 to 7 days. Persistent abdominal pain or localized tenderness, abdominal distension, and ileus suggest the same diagnosis. An abdominal abscess is a major complication in any patient but even more so during pregnancy. The inflammatory process can prove irritating to the gravid uterus, or the infection could spread to the uterus and the membranes, risking amnionitis and preterm labor. Whenever an abscess is suspected in a pregnant woman, it must be promptly evaluated to prevent further morbidity and potential mortality. Several studies are potentially useful. Ultrasound examination of the abdomen could identify an abnormal collection. A CT or MRI is sometimes indicated in a sick patient if there is a high index of suspicion. Localization of an abscess can allow safe percutaneous catheter drainage of the collection, obviating the need for another laparotomy. If the gravid uterus prevents safe access to the collection, and the patient remains ill despite adequate antibiotic therapy, a second laparotomy is indicated, because the morbidity associated with an untreated abscess is unacceptable.

Most pregnant women with postoperative febrile morbidity or infection respond to supportive therapy, antibiotics, or surgical drainage. Nonetheless, the surgeon must remain alert for other potential complications if the original infection does not resolve. The patient's condition can change rapidly if the organism responsible produces an endotoxin. Such infections can progress to septic shock, with multisystem effects. A cascade of events in the microcirculation and at the cellular level results in hypotension, initially increased cardiac output then changing to myocardial depression, arteriovenous shunting in the lung, alveolar-capillary leakage or pulmonary edema, oliguria, thrombocytopenia, disseminated intravascular coagulation, and central nervous system changes. Such severely ill patients need intensive care and often require invasive monitoring to direct therapy properly. This management is better provided in an intensive care unit setting, with consultation and concurrent care by an intensive care specialist.

### Deep Vein Thrombosis and Pulmonary Embolism

The incidence of *deep vein thrombosis* (DVT) in pregnancy is approximately five times that of

nonpregnant women of comparable age [61]. Deep vein thrombosis has been reported to complicate 0.13 per 1,000 women in the antepartum period and 0.61 per 1,000 women in the postpartum period [62]. Pregnancy is a hypercoagulable state. Maternal physiologic adaptations to gestation include elevations of several coagulation factors and fibrinolytic proteins that are believed to contribute to the prevention of hemorrhage at the time of delivery. Plasma levels of fibrinogen increase approximately 50%; coagulation factors VII, VIII, X, and XII increase significantly, and prothrombin (factor II) to a lesser degree; there is some reduction in factors XI and XIII. Coagulation factors V and IX do not change during pregnancy. The net result is a shorter prothrombin time (PT) and partial thromboplastin time (PTT). Despite elevated maternal plasminogen, there is decreased fibrinolytic activity. Levels of Protein S are significantly decreased during pregnancy, whereas concentrations of the antifibrinolytic type I plasminogen activator inhibitor are increased by up to threefold [63]. Antithrombin III (AT-III) and Protein C levels are unchanged in normal gestation. With all of this, the risk of hemorrhage is reduced, but more important, the risk of thromboembolism is increased.

Surgery independently increases the risk of DVT. The pregnant woman who undergoes surgery is believed to be particularly at risk, even though the literature contains minimal data on perioperative thromboembolic complications during pregnancy. A previous history of DVT significantly increases the risk of thromboembolism. Interference with the congested venous system during deep pelvic or retroperitoneal surgery can lead to subclinical endothelial injury, which can initiate the coagulation process. Venous stasis in the lower extremities increases as pregnancy advances, mostly because of extrinsic compression of the iliac veins by the enlarged uterus at the pelvic brim [64]. Reduced ambulation after surgery further gives rise to stasis. Other clinical risk factors for DVT include varicosities with venous vascular insufficiency, increased parity, trauma, nephrotic syndrome, obesity, and increased bedrest [65]. DVT occurs more often after cesarean than after vaginal delivery [66].

Pregnant women with the *antiphospholipid syndrome* or the *inherited thrombophilias* have a further increased risk of venous thrombosis [67,68]. The antiphospholipid antibody syndrome (APS)

is the most common acquired thrombophilia and accounts for 14% of venous thrombosis in pregnancy [67,68]. These antibodies are directed against proteins bound to negatively charged (anionic) phospholipids. The most commonly encountered antiphospholipid antibodies are isolated lupus anticoagulant antibodies, anticardiolipin antibodies, and anti- $\beta$ 2-glycoprotein-I antibodies [62]. The inherited forms of thrombophilia include a wide variety of relatively common genetic conditions that predispose women to DVT. They include gene mutations of Factor V Leiden and prothrombin G20210A, antithrombin III deficiency, deficiency of Protein S or Protein C, and hyperhomocysteinemia because of abnormality of the methyl-tetrahydrofolate reductase gene. Inherited thrombophilias increase the risk of thromboembolism in pregnancy eightfold [62].

The major impetus for the prevention and treatment of DVT in pregnant women is the avoidance of pulmonary embolism (PE). The occurrence of PE depends on whether treatment for DVT has been initiated in a timely fashion. If untreated, up to 25% of patients will develop PE, with a mortality rate of 15%. In contrast, fewer than 5% of treated patients will develop PE, with a less than 1% mortality rate. Unfortunately, the medical literature is of limited assistance in reaching treatment decisions in many cases. Most clinical trials evaluating various preventive measures for perioperative thromboembolism were conducted outside of pregnancy; therefore, their findings and conclusions cannot be automatically applied to the pregnant patient. Conversely, clinical trials of low-dose heparin in pregnancy generally recruited patients with high-risk factors, mostly previous thrombosis, and have not focused on otherwise uncomplicated surgical patients. Some extrapolations from published data and clinical experience are reasonable, however. In nonpregnant patients undergoing major surgery, the incidence of fatal thromboembolic events is reduced by low-dose heparin therapy, 5,000 units SC twice a day, started before the procedure and continued until the patient is ambulatory [69]. These results were not reproducible in a trial of low-dose heparin in patients undergoing major gynecologic surgery, however [70]. Given the uncertainty and lack of data in the literature, the authors recommend the use of low-dose heparin to reduce the risk of thromboembolic complications if major elective surgery is performed on a pregnant

woman when prolonged anesthesia or extensive dissection, especially around major vessels, is expected, or who might have malignant disease. The dose and frequency of administration of heparin might need to be adjusted in pregnancy because of the increased distribution volume and the hypercoagulable state. In one study aimed at prophylaxis in women with a previous history of thrombophlebitis, the dose of heparin was titrated according to the plasma heparin concentration, as determined by the anti-factor Xa activity; the total dose required per 24 hours ranged from 13,000 units to 23,000 units, with a mean of 16,000 units [71]. The risk-benefit ratio and the effectiveness of prophylactic heparin administration in pregnant women undergoing elective surgery have not been adequately evaluated and therefore remain controversial. Heparin is the preferred systemic drug for anticoagulation during pregnancy because this agent does not cross the placental barrier, leaving the fetal coagulation system unaffected.

Besides pharmacologic prophylaxis, mechanical interventions, such as intermittent pneumatic compression stockings, reduce the risk of thrombosis by promoting venous return in the lower extremity. Their efficacy in nonpregnant surgical patients has been demonstrated, but no published series addresses their use in pregnant women undergoing surgery [72]. As there is no risk associated with the use of these stockings, and some benefits have been documented, their routine use in high-risk patients undergoing surgery is recommended.

Postoperative DVT in the lower extremity can be suspected when the classic signs and symptoms of pain, calf or thigh tenderness, edema, erythema, and flow redistribution to the superficial venous system are noted. The clinical manifestations of venous thrombosis are often subtle or absent, making the clinical diagnosis of DVT in pregnancy inaccurate and unreliable. A high index of suspicion is necessary, because more than one half of the patients exhibiting the classic features of DVT do not have the condition. The clinical impression of thrombosis must be confirmed by objective investigations because of the unacceptably high false-positive and false-negative diagnostic rate for unaided clinical evaluation. Compression color Doppler ultrasonography of the lower extremities should be performed to confirm the diagnosis, because it has been found to be >98% sensitive and >96% specific in detect-

ing thromboses of the deep femoral and popliteal veins [73]. If ultrasound findings are abnormal, DVT is diagnosed and treatment initiated with a follow-up scan within 3 days to confirm the results [62]. If ultrasound findings are normal but there is a high index of suspicion (i.e., positive history, clinical progression), contrast venography is indicated [62]. Venography with an abdominal lead shield exposes the fetus to low levels of radiation. Contrast venography is the most accurate method of diagnosing DVT in pregnancy. Recently, MRI or CT has been used to make the diagnosis.

A patient with or without peripheral venous thrombosis can present with chest pain, tachypnea, hemoptysis, air hunger, or anxiety, strongly suggesting the diagnosis of PE. Often, however, the initial symptoms are not this dramatic. There are often no physical signs of right ventricular strain. Hypoxemia and respiratory alkalosis, although suggestive, are not diagnostic. In establishing the correct diagnosis, a plain chest radiograph excludes major consolidation, atelectasis, pneumothorax, or pulmonary edema. Echocardiographic findings in severe cases can include right ventricular dilation and hypokinesis, tricuspid regurgitation, and pulmonary artery dilation [62]. An electrocardiogram can reveal right bundle branch block, right axis shift, Q wave in leads III and a ventricular fibrillation (VF), S wave in leads I and a VL >1.5 mm, T wave inversions in leads III and a VF, or new-onset atrial fibrillation (AF) [62]. As for DVT, anticoagulation can be initiated before a definitive diagnosis of PE if there is a strong clinical suspicion. Before committing the patient to long-term therapy, the diagnosis must be confirmed.

The traditional initial evaluation of a woman suspected of having a PE has been a ventilation and perfusion scan (V/Q scan). The V/Q scan can be normal or show a low, moderate, high, or indeterminate probability of PE, based on the presence or absence of a mismatch, where a portion of the lung is ventilated but not perfused. No further investigation is required if the V/Q scan is normal, and heparin, if started, can be discontinued. The scan has a high specificity, and patients whose studies are interpreted as indicating a high probability of embolism are best treated with anticoagulants. Because of the relatively low sensitivity of this study and the serious potential risk if a case of embolism is not treated, however, pulmonary angiography is advised in patients with a low-probability scan, if the clinical

signs and symptoms are suggestive [74]. Pulmonary angiography is also indicated in selected cases for which known pulmonary infiltrates or pneumonia renders the V/Q study noninterpretable. Spiral CT can be used instead of angiography when a V/Q study is nondiagnostic [75]. At the present time, spiral chest CT is being used in many centers as the noninvasive alternative to the V/Q scan for initial evaluation of PE. Radiation exposure to the fetus is lower than that of a V/Q scan [76]. Spiral CT is highly sensitive and specific for diagnosing central pulmonary artery thrombi, but it is insensitive for diagnosing subsegmental clots. A high-risk patient with a positive spiral CT requires therapy, but if the spiral CT is negative, she should undergo pulmonary angiography.

The goals of therapy for DVT are to prevent clot extension and embolization. The primary therapy includes anticoagulation, analgesia, and elevation of the leg until the clot becomes organized. For PE, heparin is given parenterally, and supplemental oxygen and standard resuscitative measures are prescribed to maintain adequate oxygenation and cardiac output. Specific therapy for thromboembolism consists of anticoagulation until the end of the pregnancy and through the early postpartum period, until the hypercoagulable state and the risk of venous stasis abate.

Heparin, a large and highly polar molecule, is the anticoagulant of choice during pregnancy because it does not cross the placenta, appears to be safe for the fetus, and does not enter breast milk. Earlier concerns about heparin's possible detrimental effects in pregnancy have been dispelled [77,78]. A comprehensive review of the literature yields a rate of 13.3% for adverse fetal outcomes, including abortion, stillbirth, neonatal death, or congenital anomalies for patients receiving heparin alone. After excluding pregnancies with maternal comorbid conditions independently associated with poor fetal outcome, and cases of prematurity with a normal outcome, the rate of adverse fetal outcomes fell to 3.0% compared with 16.9% for patients receiving oral anticoagulants. Based on these data, heparin remains the drug of choice in pregnancy if anticoagulation is indicated for the treatment or the prevention of thromboembolic disease [77]. The major drawback of heparin is its parenteral route of administration. Most pregnant women are capable of self-administering subcutaneous heparin twice

or three times a day with the dose adjusted to maintain the activated partial thromboplastin time (aPTT) at 1.5 to 2 times the control. The platelet count should be monitored periodically because of the risk of thrombocytopenia. Early-onset heparin-induced thrombocytopenia is mild, and patients are usually asymptomatic; delayed-onset heparin-induced thrombocytopenia is a more severe complication, which can result in thrombotic episodes, limb amputation, or even death [79]. Osteoporotic vertebral fractures are also possible and can occur in up to 2.2% of pregnant women on prolonged heparin therapy [80].

Low-molecular-weight (LMW) heparin is a safe and effective alternative to unfractionated heparin because it does not cross the placenta and has no teratogenic effects [81]. LMW heparin has a longer half-life and bioavailability, a more predictable dose-response relationship, and decreased risk of thrombocytopenia and hemorrhage than unfractionated heparin. In pregnancy it can be used to treat patients with DVT, PE, or thrombophilic disorders. The goal of therapy is to maintain the anti-factor Xa activity between 0.5 units/ml and 1.2 units/ml and the heparin level between 0.2 units/ml and 0.4 units/ml [82]. To avoid epidural hematoma formation after regional anesthesia while receiving LMW heparin, patients can be switched to unfractionated heparin at or near term (36–37 weeks). Regional anesthesia should not be used within 24 hours of the last dose of LMW heparin. Consultation with an experienced anesthesiologist is indicated [83]. (see Chapter 9, Obstetric Anesthesia)

During a completely uneventful, spontaneous vaginal delivery, intrapartum bleeding is not usually increased in the patient who is receiving heparin, because most obstetric hemostasis results from obliteration of the vascular bed at the implantation site by contraction of the myometrium. The risk of excessive bleeding is substantial with any operative delivery, however, especially if there are episiotomy extensions or birth canal lacerations. Heparin therefore should be discontinued at the onset of labor or before an elective cesarean delivery, or the dose reduced until normal laboratory results are reported. The half-life of heparin is short, and its effects remit within 2 to 4 hours. In an emergency, protamine sulfate rapidly reverses the anticoagulation of heparin, although overdosing must be

carefully avoided. Heparin can be safely resumed 6 to 8 hours after delivery. Some clinicians prefer to allow the PTT to fall to normal or near normal with the onset of labor and then initiate mini-dose (1 IU/kg/hr–2 IU/kg/hr) or low-dose heparin (200 IU/hr–600 IU/hr titrated to not extend the PTT), maintaining this dose throughout labor. If either of these regimens is chosen, full heparin doses should be resumed 6 to 8 hours postpartum.

When DVT has been diagnosed during pregnancy, the woman should receive therapeutic anticoagulation for at least 3 months during the pregnancy followed by prophylactic therapy [82]. Kearon and colleagues have found that 6 months of anticoagulation treatment after a first episode of idiopathic DVT reduced the recurrence rate to 1.3% per patient year compared with 23% for patients on a 3-month regimen [84]. Postpartum anticoagulation should be continued for 6 to 12 weeks after DVT and for 4 to 6 months after PE or complex iliofemoral DVT. Oral anticoagulant therapy can be initiated postpartum by titrating the warfarin dose to maintain the patient's international normalized ratio (INR) at approximately 2.0. Heparin should be maintained during the initial 4 days of warfarin therapy or until a therapeutic INR is reached to avoid warfarin-induced skin necrosis and paradoxical thromboembolism [62].

Warfarin is a widely used anticoagulant outside of pregnancy because it offers ease of administration by the oral route and documented efficacy. The anticoagulant activity of warfarin is a result of its inhibition of vitamin K, which is a cofactor in the synthesis of factors VII, IX, X, and prothrombin (factor II). The risks of warfarin in pregnancy include a 33% risk of embryopathy when exposure is between gestational weeks 7 and 12, because it is loosely bound to albumin and crosses the placenta. The currently held pathophysiology for the embryopathy is that focal hemorrhages occur from anticoagulation in the embryo as the drug crosses the placenta. The anomalies most commonly reported are nasal hypoplasia and stippling of the vertebral and femoral epiphyses. Also observed are optic atrophy, cataracts, microcephaly, microphthalmia, blindness, mental retardation, and skeletal malformations. The risk of ingestion of warfarin in the third trimester is increased bleeding: specifically, intrapartum and postpartum hemorrhage in the mother, and internal bleeding in the fetus at delivery, including intracranial hemor-

rhage [77]. Vitamin K or fresh-frozen plasma can be used to reverse the effect of warfarin. If warfarin is used postpartum, the dose should be titrated to maintain the PT approximately 1.5 times the control. Because of significant variability between laboratories, the INR is currently used to monitor the anticoagulant effect of warfarin. For patients with DVT or PE, the aim is to keep the INR between 2.0 and 3.0. Pregnant women with mechanical heart valves might require warfarin use during the second trimester of pregnancy because current studies suggest an increase in thrombotic complications using unfractionated heparin [85,86]. Both warfarin and dicumarol (bishydroxycoumarin) are classified by the American Academy of Pediatrics as compatible with breastfeeding.

The placement of a Greenfield filter in the inferior vena cava is sometimes required in patients with recurrent PE despite adequate anticoagulant therapy, patients with PE or iliofemoral DVT who have a contraindication to anticoagulation therapy, and patients who develop serious hemorrhagic sequelae with anticoagulant therapy [62].

Women with inherited thrombophilias are at increased risk of thromboembolism in pregnancy. They can exhibit unusual thrombotic episodes of the sagittal, mesenteric, and portal veins. Eight percent to fourteen percent of Caucasians meet laboratory criteria for a thrombophilic disorder, excluding hyperhomocysteinemia; however, they account for 70% of venous thromboses diagnosed in pregnancy [87,88]. Pregnant women with low-risk thrombophilias (e.g., heterozygotes for the factor V Leiden and prothrombin G20210A mutations, Protein C or Protein S deficiencies, or hyperhomocysteinemia unresponsive to folate and vitamin B<sub>12</sub> therapy), and no personal history of DVT do not appear to require antepartum anticoagulation because they have a low incidence of DVT in pregnancy (0.2%–4%) (63). They should receive postpartum prophylaxis if they require a cesarean delivery or if they have other major risk factors for thrombosis (e.g., obesity, prior prolonged bedrest, strong family history) [89,90]. A pregnant patient with an inherited thrombophilia of low thrombotic potential and a history of thromboembolism should be treated with prophylactic unfractionated or LMW heparin during pregnancy. Patients with highly thrombotic thrombophilias (i.e., antithrombin deficiency or homozygotes for the Factor V Leiden or prothrombin G20210A

mutations), regardless of personal history of venous thrombosis, should be treated with full therapeutic anticoagulation during pregnancy. They should be maintained on anticoagulation for at least 6 weeks postpartum, and, if they have had a previous venous thromboembolism, anticoagulation should be continued for up to 3 months. Patients without an identifiable thrombophilia whose previous thrombosis occurred during pregnancy should probably be given low-dose heparin as antenatal prophylaxis.

## IATROGENIC INJURIES

### Avoidance

Whenever surgery is performed, there is a potential for injury to viscera or other structures. In pregnancy, this risk is increased owing to limited exposure, because the uterus occupies a good deal of intraabdominal space in the second and third trimesters. Contributing risk factors for injury are obesity, distortion of the anatomy by the pathologic process or adhesions from previous surgery, inadequate anesthesia, haste during surgical emergencies, inexperience of the surgeon, and failure to follow sound surgical technique. As with any type of medical injury, prevention or recognition and prompt treatment of the problem at the time of the original surgery are best. Prevention by careful case selection and close attention to detail avoids most surgical misadventures, thereby reducing immediate intraoperative and postoperative morbidity, long-term sequelae, and eventual medicolegal consequences. If an injury occurs despite careful attention to detail, the risk of complications is significantly lessened by its immediate recognition and repair. During abdominal surgery, iatrogenic injuries commonly involve the gastrointestinal and urinary tracts. Vascular injuries are uncommon; neurologic injuries occur even less frequently. Most iatrogenic lesions result from the inadvertent laceration, crushing, ligation, or transection of various structures, or by thermal injury from electrosurgical units.

If an inadvertent injury occurs, the surgeon must ensure a satisfactory repair. If the damaged viscus or structure is outside the area of expertise of the obstetrician, it is advisable to immediately consult a surgeon with more experience, or one from another specialty, for advice and help on how best to manage the problem. In all cases, the accident must be

fully reported in the operative note, including the mechanism and extent of injury, and a description of the repair. Soon after the surgery, the patient and, when appropriate, her family, must be informed of the injury. Attempts to conceal an injury are unethical and could lead to more questions, anger, and potential litigation. The surgeon can, when appropriate, stress complicating factors that led to the injury and the expected good outcome from the repair. It is always best to begin with an open and frank approach to the patient and seek appropriate consultation while fully documenting all events.

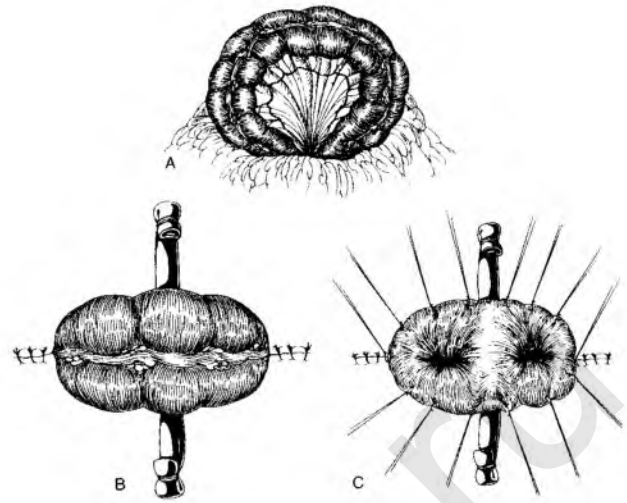
### Gastrointestinal Injuries

*Lacerations of the intestinal tract* occur during intraabdominal surgery with a reported incidence of approximately 0.8% [91]. Most bowel lacerations occur on opening the peritoneal cavity or during dissection of dense adhesions in patients with previous surgery. Opening through a previous incision is always best performed meticulously. When operating on patients who have had previous abdominal surgery, the surgeon should enter the peritoneum *away* from the site of the previous scar. Grasping and tenting the peritoneum with fine instruments and palpating for intestinal loops before incision of the peritoneum reduce the risk of laceration. Sharp dissection is preferable to blunt technique in separating dense inflammatory, neoplastic, or endometriotic adhesions. Although uncommon in obstetric patients, if elective bowel surgery is planned or in patients expected to be at high risk for bowel injury, a mechanical bowel preparation and preoperative intravenous antibiotics are indicated. A clear fluid diet followed by magnesium citrate, 300 ml, or iso-osmotic polyethylene glycol (GoLYTELY) 4 liters PO, and enemas to clear return, are an effective cathartic regimen. Intravenous gentamicin and metronidazole or clindamycin should be administered before the surgical incision, preferably at the induction of anesthesia. If such bowel treatments are given to pregnant women, close attention to maintenance of a normal vascular volume and normal electrolyte balance is essential.

Three of four iatrogenic bowel injuries involve the small intestine [91]. The characteristic greenish small bowel content can be visible at the time of the injury even if the enterotomy is not readily apparent. Such lacerations must be located and repaired immediately. Seromuscular tears not involving the

mucosa should be approximated with interrupted fine silk, polyglycolic acid (Dexon), or polyglactin (Vicryl) suture on a tapered intestinal needle. Full-thickness defects, even if in the longitudinal axis of the bowel, should be repaired transversely to avoid narrowing the intestinal lumen. The traditional closure includes continuous mucosal and submucosal absorbable suture such as polyglycolic acid or polyglactin, with interrupted silk sutures on the seromuscularis. A single-layer repair incorporating the full thickness of the bowel wall with interrupted nonabsorbable suture such as silk or continuous polypropylene (Prolene) is also adequate. A laceration that is very large, involves most of the circumference of the bowel, or has irregular edges is better treated by local resection and anastomosis; the continuity of the bowel can be restored by hand-sewn sutures as above or with automatic stapling instruments, which might result in a lower rate of leakage [92]. Whichever technique is used, the principles of the surgery are to 1) avoid tension on the anastomotic line, 2) maintain adequate perfusion to the proximal and distal segments, 3) create an adequate lumen at the anastomosis, 4) prevent hematomas in the suture line, and 5) avoid strangulation of bowel wall by excessively tight sutures. In the postoperative period, functional restoration of bowel continuity is monitored by routine clinical and radiologic techniques.

The pathophysiology and repair of lacerations to the large bowel differ inasmuch as the lumen is larger and the bacterial counts are several-fold higher than in the small bowel. The obstetrician-gynecologist should request the assistance of a general surgeon or a gynecologic oncologist, if available, in the unusual case that bowel repair, resection, or colostomy is required. The technique of repair is similar to that for the small intestine, except that compromise of the lumen is less likely by a suture line. Complex lacerations require resection and anastomosis, at the discretion of the senior surgeon. A low anterior anastomosis deep in the pelvis can be greatly facilitated by the use of the end-to-end automatic stapler. A temporary colostomy might be necessary when an optimal repair is not possible or with unprepared bowel. A major spill of bowel contents through a complex tear without previous bowel preparation and with suboptimal repair involves a high risk of postoperative peritonitis and abscess. In addition to profuse irrigation



**FIGURE 16.2.**  
*A, Preparation of colon, B, securing the colostomy, C, mucocutaneous suture.*

and postoperative antibiotics, the surgeon should consider a protective proximal colostomy. When no other surgeon is available, the easiest colostomy to perform for the gynecologist who infrequently performs bowel surgery is a transverse loop colostomy in the right upper quadrant. This is also the easiest one to close at a later date without the need for a formal laparotomy. In this procedure, the skin, subcutaneous tissues, and anterior rectus fascia are incised transversely to the right of the umbilicus, at the level of the transverse colon. The rectus muscle is transected by electrocautery, and the posterior rectus fascia, fascia transversalis, and peritoneum are incised. The transverse colon is mobilized into the wound after dissecting the infracolic omentum over that segment and creating a window through an avascular space in the mesocolon (Figure 16.2A). A glass rod or ostomy bridge is passed below the colon through this defect and secured to the skin (Figure 16.2B). The antimesenteric border of the colon is then incised transversely or longitudinally, and sutured to the dermis with 3-0 polyglycolic acid or polyglactin sutures, incorporating the full thickness of the bowel wall (Figure 16.2C). The bridge can be safely removed after 7 to 10 days [93].

Thermal injuries to the bowel wall are potentially serious. Thermal trauma sometimes goes unrecognized at the time of surgery, only to become apparent 2 to 4 days later when the patient presents with bowel perforation complicated by peritonitis or sepsis. The small and large bowels are at risk during



any abdominal surgery when the electrosurgical unit is used for hemostasis or to divide tissue planes. Thermal energy is conducted to the bowel more often by inadvertent direct contact with the structure than when the electrocautery is deliberately applied. Thermal energy coagulates the microcirculation, leading to ischemic necrosis and subsequent perforation. The operator who always uses the electrocautery under direct vision and takes care to retract the bowel away from the site of electrocoagulation prevents most thermal injuries. With the monopolar cautery, the current exits through a groundplate attached to the patient's skin; therefore, structures such as the bowel inadvertently included in that loop can be injured. The risk of thermal injury is reduced with bipolar cautery because the electrical current loop is between the two plates of the instrument and is less likely to exit through an alternative pathway. If damage occurs despite standard precautions, recognition of the injury at the time can save the patient significant morbidity. A small coagulation site limited to the serosa can be treated conservatively with observation. A deeper injury (i.e., extending more than 0.5 cm in diameter) is best treated by resection and anastomosis with a 3-cm to 5-cm margin of grossly normal-appearing bowel on either side. This wide resection border is prudent because the electrical current dissipates in the bowel, and microscopically the thermal injury extends well beyond the area of immediate or visually obvious burn [94].

Failure to recognize a thermal bowel injury when it occurs allows the inflammatory reaction to progress and coagulation necrosis of the intestinal wall to occur. The patient becomes symptomatic after 2 to 3 days with nonspecific abdominal pain, nausea, or vomiting. Objective signs of peritonitis progressively develop. A high index of suspicion is necessary not to miss this diagnosis, further delaying laparotomy and resection of the involved bowel [94]. It is advisable to consult with an experienced bowel surgeon as soon as a bowel injury is suspected.

### Urinary Tract Injuries

*Urinary tract injuries* during abdominal surgery primarily involve the ureter and the bladder. (See Urologic Complications, Chapter 19.) During pregnancy, variable hydronephrosis occurs. In addition, the left ureter is laterally and anteriorly displaced

by dextrorotation of the uterus [95]. In the second and third trimesters, the enlarging uterus impairs exposure to the lateral pelvic walls and increases the risk of injury. Common sites for iatrogenic ureteral injury include the pelvic brim during resection of an adnexal mass or, rarely, during a sigmoid resection, and immediately adjacent to the cervix. For example, the paracervical portion of the ureter can be injured at cesarean delivery if an extension of the uterine incision involves the broad ligament. During cesarean hysterectomy, especially after labor and cervical dilatation, injuries are possible because the cervix is difficult to delineate by palpation. Ureteral injuries include transection, ligation, or crushing, alone or in combination. Damage to the vascular supply of the ureter and bladder might not result in a visible injury at the time of the procedure, but subsequent postoperative ischemia can result in stricture or fistula formation.

Blind clamping, cutting, or suturing proximal to either ureter is imprudent. To expose the ureter properly, the uterus is mobilized medially, the sidewall peritoneum is incised posterior to the broad ligament, and the loose areolar retroperitoneal tissue is dissected bluntly, exposing the iliac vessels. The ureter crosses the bifurcation of the common iliac artery, is loosely attached to the medial leaf of the peritoneum, and courses lateral to the cervix below the uterine artery to enter the bladder trigone after turning medially.

De-ligation is sufficient therapy for inadvertent ureteric ligation as long as the ureter appears viable and the injury is recognized during the surgery. Ligation with visible damage to the ureter or crushing within a surgical clamp is treated by insertion of a 6 or 8 French double-J ureteral stent or a pediatric feeding tube by means of a suprapubic cystostomy. If there is an obvious injury to the ureter, or if a clamp is left on the ureter for longer than an arbitrary 30 minutes, the likelihood of more extensive trauma and devascularization mandates a local resection and repair. In most cases, the obstetrician-gynecologist should promptly consult with a urologist or a gynecologic oncologist if a ureteral complication occurs requiring resection or repair [96]. (See Urologic Complications, Chapter 19.)

During any lower abdominal laparotomy, the bladder dome can be inadvertently lacerated or punctured as the peritoneal cavity is entered. The risk for this complication increases as pregnancy

advances. The bladder progressively becomes an abdominal rather than pelvic organ because of its loose areolar attachment to the lower uterine segment. The most commonly performed surgical procedure during pregnancy, aside from an episiotomy, is cesarean delivery. The bladder is at risk for injury during its dissection off the lower uterine segment, especially during repeat cesarean operations. Previous pelvic surgery also can cause the bladder to adhere to the anterior abdominal wall, increasing the risk of injury at the time of peritoneal entry. A simple and mandatory measure is to empty or preferably drain the bladder continuously during surgery with an indwelling urethral (Foley) catheter. Prevention of injury is usually easy. It is safer to open the peritoneal cavity at the upper end of a midline incision or the lateral portion of a lower transverse incision. At cesarean delivery, bladder lacerations can be avoided by sharp dissection, rather than blunt avulsion, of the vesicouterine space, especially in a repeat operative delivery. If there is difficulty with exposure, the procedure should probably best be abandoned and the myometrium simply entered vertically or transversely at a different level, above the bladder reflection. If a bladder laceration is suspected but not readily apparent, the bladder can be filled with methylene blue or indigo carmine dye diluted in normal saline; sterile milk has also been used for the same purpose but is not always available in the operating suite.

If there is a bladder laceration, the most important step is to establish whether it involves the trigone. A superficial injury not involving the mucosa is simply repaired with a single continuous layer of 2-0 or 3-0 polyglycolic acid, polyglactin, or chromic catgut suture. A complete tear of the bladder away from the trigone should be repaired in two layers using the same suture materials. Continuous sutures that incorporate the full thickness of the bladder wall on the first layer can be imbricated by a superficial second layer that involves the muscular tissue. If the laceration is close to the trigone, it is best to open the bladder dome after dissecting the space of Retzius and place the sutures under direct vision to avoid the ureteral orifices. If the laceration involves the trigone, it is prudent for the obstetrician-gynecologist to consult a urologist or a gynecologist experienced with bladder injuries, because the risk of further injury to the ureter is significant. For simple bladder injuries

not involving the trigone or ureters, consultation is not required. After bladder repair, an indwelling urethral or suprapubic catheter should be kept in place for 7 to 10 days, depending on the extent of injury. When recognized and adequately managed, almost all bladder injuries heal uneventfully. Eventual follow-up by intravenous pyelogram or cystogram confirms satisfactory healing without leakage or stricture [97]. Such studies are unnecessary in asymptomatic patients.

### Neurologic Injuries

Peripheral nerves are at risk for injury during abdominal surgery, but the frequency of this complication does not appear to be increased during pregnancy. Mechanisms of injury include direct trauma such as undue pressure by a self-retaining retractor, ligation, electrosurgical thermal damage with attempts to control excessive bleeding, or accidental transection. Another mechanism of injury involves excessive traction on a nerve with local compression by instruments, packs, or ligatures. The *femoral*, *sciatic*, and *obturator nerves* are primarily at risk. Although nerve injuries are uncommon, they can result in a variably severe loss of motor or sensory function. The patient can suffer from paresthesias, pain, or weakness affecting her gait in the distribution of the injured nerve. Most cases improve to complete recovery, but residual deficits are possible.

Injury to the sciatic nerve can occur with dissection or suturing deep in the posterior pelvis where the nerve passes through the sacrosiatic notch. An exaggerated lithotomy position during surgery can overstretch the nerve over the sacrospinous ligament at the level of the greater sciatic notch, especially if the patient's leg is externally rotated and unsupported at the knee. The deficit varies with the specific fibers of this large nerve that are damaged. The lesion can result in weak leg flexion from denervation of the hamstring muscle with injury to the main trunk, a foot drop and inversion of the foot from weak extensor muscles of the ankle and abductor muscles of the foot after perineal nerve damage, or poor flexion of the foot from denervated calf muscles with tibial nerve deficit [98]. Treatment is symptomatic: physiotherapy, reassurance, analgesia, and passage of time. Electrophysiology studies are occasionally indicated if the return of function is tardy or incomplete.

Femoral nerve injuries can occur by pressure from the deep blade of a Balfour or other retractor during any pelvic surgery, including cesarean delivery, especially if the transverse abdominal incision extends close to the pelvic sidewall. The most notable clinical deficit with a femoral nerve injury is weakness of the quadriceps muscle with poor extension of the leg, which affects weightbearing and walking. Paresthesia and numbness over the anterior and medial portions of the thigh are also present. Femoral nerve complications can be mostly prevented by inserting the retractor blade superficial to the psoas muscle, and by protecting the nerve by placing a laparotomy pack behind the retractor. Periodic repositioning of the retractor during long procedures is also helpful in avoiding this complication. Treatment is symptomatic [99].

The obturator nerve is unlikely to be traumatized in an obstetric patient unless a radical hysterectomy is performed for cervical cancer. The most common misadventure is transection of the nerve with denervation of the adductor muscles of the leg and sensory loss over the medial thigh.

Although an uncommon procedure in pregnancy, radical hysterectomy interrupts several parasympathetic fibers with the radical excision of the cardinal and uterosacral ligaments. This leaves the patient with some degree of bladder atony and obstipation. This potential complication must be discussed preoperatively as part of the informed consent process [100].

### Reproductive Tract and Fetal Injuries

The uterus and adnexa are occasionally injured in the process of operating on extragenital disease during gestation or cesarean delivery. The increased perfusion to the gravid uterus causes marked venous congestion. Efforts to keep the laparotomy incision small reduce postoperative discomfort, but exposure can be limited. Simple retraction on the enlarged uterus to expose other viscera could lacerate the thin venous walls in the uteroovarian, infundibulopelvic, or broad ligaments and result in brisk bleeding. Rapid intervention is required to first tamponade the bleeding vessel, expose the lacerated segment, and then apply one or more suture-ligatures or hemostatic metal clips for control.

Accidental *lacerations of the fetus* can occur during cesarean delivery. Dessole and coworkers [101]

reported an overall rate of 3.1%, with a higher risk for fetal accidental lacerations when the cesarean delivery was emergent rather than elective. Risk factors associated with an increased risk of lacerations in the group undergoing emergent cesarean delivery were "fetal distress" during labor with premature rupture of the membranes (PROM) and PROM without labor [101]. Others have reported an incidence of fetal injury of 1.5% to 1.9% [102,103]. Strong pressure hastily applied to the scalpel on entering the abdominal cavity for an urgent cesarean delivery occasionally results in an inadvertent deep longitudinal cut through the myometrium; rarely, this can lacerate the baby. To minimize the risk of fetal accidental laceration at the time of cesarean delivery, the uterine incision should be suctioned meticulously, the uterus scored along the entire length of the incision with the scalpel, and the uterine cavity then entered bluntly with a finger into the central portion of the incision. Alternatively, a Kelly clamp can be used to expand the uterotomy at the time the uterus is opened, or the uterine incision can be elevated from the presenting part of the fetus with an Allis clamp or ring forceps [101,103].

Most accidental fetal lacerations are of cosmetic importance only but occasionally can have serious clinical consequences. They must be repaired immediately, and the situation subsequently discussed with the family. Superficial nicks, if bleeding is minimal, are best closed with adhesive strips. More extensive lacerations are reapproximated with fine, interrupted monofilament nylon or polyglycolic suture. If the injury involves the face, it is prudent to control bleeding by simple compression, apply adhesive strips, and consult a plastic surgeon for definite repair.

## SURGICAL COMPLICATIONS

### Gallbladder Disease

#### *Epidemiology*

The incidence and prevalence of cholelithiasis vary greatly among geographic regions and ethnic groups [104,105]. The incidence of symptomatic gallstones in women is approximately twice that in men [105]. It is estimated that acute cholecystitis occurs at a frequency of 1 to 6 per 10,000 pregnancies [106]. The

incidence of symptomatic cholelithiasis has been estimated to be 0.05% during pregnancy; 40% of these patients require surgery [107]. Asymptomatic cholelithiasis has been reported to occur in 3.5% of pregnancies [108]. After appendectomy, cholecystectomy and surgery for bowel obstruction are the most common nonobstetric surgeries performed in pregnant women [108]. Gallstone formation occurs frequently in pregnant women because of their altered metabolic state. Bile stasis, increased concentration of cholesterol, changes in the physiochemical nature of bile salts, and infection contribute to the formation and deposition of biliary calculi. Gallstones can remain silent or give symptoms at any time during pregnancy or the puerperium. Gallstones formed during pregnancy later can partially or, rarely, entirely resolve when the bile becomes less lithogenic postpartum.

### *Diagnosis*

The symptoms and signs of biliary colic are the same as in the nonpregnant patient. Characteristically, there is pain in the epigastrium or in the right subcostal region with radiation around the rib cage to the scapular region. The onset of pain can be abrupt. Tenderness and varying degrees of muscle guarding are noted on palpation, and, depending on the nature and extent of the disease, a mass can be felt. Nausea with or without vomiting occurs frequently. Fleeting icterus occurs without concomitant abnormalities in the common duct in approximately 20% of patients with isolated gallbladder disease; nonetheless, if icterus is noted, the possibility of stones within the common duct must be considered. Fever and a moderate leukocytosis are common. Differentiation from acute appendicitis is sometimes difficult because the appendix, elevated by the enlarged uterus, often resides in the right upper quadrant (see Fig. 16.1). The differential diagnosis includes gastric or duodenal ulcer, esophageal hiatal hernia, pneumonia, hepatitis, myocardial infarct, herpes zoster infection, pyelonephritis or renal lithiasis, appendicitis, adnexal torsion, or severe preeclampsia.

The documentation of cholelithiasis by ultrasonography confirms the diagnosis. Ultrasonography is the diagnostic test of choice, with a diagnostic accuracy of 97% for cholelithiasis [109,110]. If nondiagnostic, an oral or intravenous cholecystogram

can be necessary. Endoscopic retrograde cholangiopancreatography (ERCP) is sometimes necessary to detect choledocholithiasis [111]. With modification of the technique, shielding of the lower abdomen, and limiting fluoroscopic time, ERCP with endoscopic sphincterotomy, stone extraction, or stent insertion can be performed safely [112].

### *Management*

The initial treatment of acute cholecystitis has been conservative: hospitalize for observation, no oral intake, intravenous hydration, and adequate pain relief. If vomiting is repetitive, the stomach is kept empty with nasogastric suction. In more than 90% of cases, acute symptoms subside within 48 hours. Surgical intervention is necessary in about 10% to 20% of cases. Indications for surgical intervention generally include repeat attacks of biliary colic, failure to respond to medical therapy, suspected perforation with peritonitis, severe toxicity, obstructive jaundice, and cases in which a surgical condition such as appendicitis cannot be excluded. Recently, some authors favor a more aggressive surgical approach because pregnancy outcomes have been better when compared with those patients receiving conservative management [113–116].

When surgery is indicated, the type of operation depends on the findings and the condition of the patient. Jaundice with proved or suspected common duct stones mandates an exploration of the common duct unless technical difficulties or the condition of the patient are such that the simplest operation, cholecystotomy alone, is indicated. Mortality from surgery for uncomplicated cholecystitis is apparently not increased in pregnancy. The fetal loss rate is reported to be less than 5% with open cholecystectomy [117]. Both maternal mortality and fetal loss are increased when pancreatitis is present, however. Pancreatitis has been associated with maternal and fetal morbidity and mortality rates of 20% [118]. The traditional approach has been cholecystectomy by laparotomy, a technique with excellent results and mortality rate of less than 1% [119]. The potential morbidity associated with a large laparotomy incision remains, however, resulting in significant postoperative pain and immobility, respiratory splinting, delay in return to normal gastrointestinal function, and lengthy hospital stay. The pregnant patient can be at increased risk for postoperative

complications, especially thrombophlebitis and embolic events related to the hypercoagulable state.

Laparoscopic cholecystectomy has become the procedure of choice for most patients who require cholecystectomy [106,120]. Morbidity and mortality compare favorably with open cholecystectomy in the hands of skilled laparoscopists [120,121]. The benefits include a reduced hospital stay, significantly less postoperative pain, decreased length of disability, and improved cosmesis [119,122–125]. The risk of thromboembolic disease can be diminished by a reduction in postoperative atelectasis and immobility [106]. The laparoscopic approach has been found to be successful in all three trimesters [120,126]. These procedures are not for the inexperienced surgeon, however.

The following precautions are recommended when laparoscopic cholecystectomy is performed on a pregnant patient. The use of pneumatic compression stockings is recommended, because they improve venous return and reduce the risk of DVT. They should be on the patient's legs at the induction of anesthesia, and their use should be continued until normal ambulation is resumed. Furthermore, the operating table should be placed in lateral tilt to displace the uterus from the inferior vena cava so that venous return is unimpaired. Because stasis of blood in the lower extremities is common during pregnancy, the risk of thromboembolic disease during pregnancy is notably increased. Additionally, a lead shield should be placed over the uterus to maximize fetal protection from radiation, or ultrasound should be used instead of x-ray whenever possible. To avoid fetal respiratory acidosis, good maternal oxygenation and perfusion should be maintained, and end-tidal CO<sub>2</sub> or arterial blood gases should be monitored. Perioperative monitoring of the fetal heart rate and uterine contraction monitoring can be of benefit especially in the postoperative period.

### Appendicitis

Removal of the appendix was recorded for the first time in 1735. In 1880, Lawson Trait performed the first known successful appendectomy for a diagnosis of appendicitis [127]. Appendicitis complicating pregnancy was first reported by Hancock in 1848 [128]. Ten days after a preterm delivery, an appendiceal abscess was drained and the patient recovered.

### Epidemiology

Acute appendicitis is the most common nonobstetric indication for abdominal surgery during pregnancy. According to Black's 1960 literature review, appendicitis complicates 1 in 355 to 1 in 11,479 pregnancies, averaging one in 1,500 deliveries [129]. In 1977, Babaknia and coworkers found an incidence of one in 1,500 deliveries in their cumulative review of 503,496 deliveries [130]. In a 1990 study, Tamir and coworkers reported an incidence of one case of acute appendicitis in every 1,400 births [131]. Mazze and coworkers reported an incidence range from 1 per 1,500 to 1 per 6,600 pregnancies [132]. Appendicitis during pregnancy occurs at any age; however, 90% of patients are below the age of 30 years, and 75% are between the ages of 20 and 30 years. This is similar to the age groups affected in the nonpregnant population. Pregnant women are not more or less likely to suffer from appendicitis than the nongravid, but in pregnancy, rupture of the appendix occurs two to three times as frequently. Babaknia and coworkers reported the distribution of cases of appendicitis according to trimester of pregnancy as follows: 30% occurred in the first trimester, 40% in the second trimester, and 30% in the third trimester, labor, and puerperium [130]. In Black's review of 373 cases, he found 35% to be in the first trimester, 34% in the second, 27% in the third, 1.7% occurred during labor, and 2.3% in the puerperium [129]. Appendicitis is a potentially serious disorder, increasing the likelihood of abortion or preterm labor, especially if peritonitis is present. Delay in diagnosis is consistently the reason for a gangrenous and perforated appendix, with an associated increased risk of maternal morbidity and mortality and perinatal mortality [133]. In a 1977 review of 333 cases reported since 1963, Babaknia and coworkers found only three maternal deaths (1.0%), all three of which occurred among 70 cases with a ruptured appendix; this was accompanied by an 8.7% rate of fetal loss [130]. In a 1991 review, Mazze and Kallen reported 14 perinatal deaths among 778 cases of appendicitis [132]. The risk of preterm delivery was most significant during the first week after surgery; 16% of women delivered the day of the surgery [132]. In 1992, Mahmoodian reviewed 27 series reported between 1960 and 1992 and noted that perinatal mortality was 4.8% among patients with acute inflammation only, but 19.4%

TABLE 16.1 Comparison of Findings in Pregnant and Nonpregnant Patients with Appendicitis

	Pregnant	Nonpregnant
Diagnostic accuracy	72%	75%
Symptoms	Nausea, vomiting, increased frequency of urination, abdominal pain, anorexia	Nausea, vomiting, increased frequency of urination, abdominal pain, anorexia
Physical findings	Abdominal pain (100%) First trimester: right lower quadrant (100%) Second trimester: right lower quadrant (80%) Third trimester: right upper quadrant (20%) Rebound tenderness (75%) Guarding (60%) Fever >100.2 F (18%)	Abdominal pain (100%) Right lower quadrant (65%) Pelvis (30%) Flank (5%) Present
Laboratory findings	Normal pregnancy WBC =	Present Usually >100.4 F
White blood cell count (WBC)	12,500–16,000 per mm <sup>3</sup> with 80% bands	Normal WBC = 3,000–10,000 per mm <sup>3</sup> Most patients demonstrate a shift to the left. Not all demonstrate leukocytosis. Fewer than 4% have a normal WBC and no shift to the left.
Urinalysis	Pyuria can be present if the ureter or renal pelvis is in contact with the inflamed appendix.	Pyuria: rare

From DeVore GR: Acute abdominal pain in the pregnant patient due to pancreatitis, acute appendicitis, cholecystitis, or peptic ulcer disease. Clin Perinatol 1980;7:349–369; with permission.

in those with a perforated appendix. In a review of pregnancy outcome following nonobstetric surgical intervention, Cohen-Kerem and colleagues analyzed the appendectomy cases during pregnancy and found that 8% delivered prematurely; 2.6% had a fetal loss associated with appendicitis, but the fetal loss was 10.9% when peritonitis was present [135]. These data emphasize the risk(s) associated with appendiceal rupture and resulting peritonitis.

### Diagnosis

Acute appendicitis complicating pregnancy is basically the same disease process as in the nonpregnant patient; however, there are several confounders (Table 16.1). Nausea, vomiting, frequency of urination, constipation, and abdominal discomfort are common symptoms of pregnancy. The tenderness commonly associated with uterine contractions before and after delivery can easily obscure appendicitis. The surgeon, not recognizing that a mild leukocytosis and increased sedimentation rate are normal in pregnancy, could place more diagnostic value on these findings than they deserve. Conversely, a simple belief that these findings are only

normal physiologic variations in pregnancy could delay surgical intervention, with a consequently higher incidence of morbidity and mortality. Further diagnostic difficulty is related to the normal upward displacement of the appendix during pregnancy [4]. As gestation proceeds, the appendix is rotated counterclockwise. As term approaches, the appendiceal tip overlies the right kidney (see Fig. 16.1). If the appendix is fixed by adhesions, however, this migration does not occur. It is prudent to suspect acute appendicitis in any pregnant woman with right-sided pain, regardless of location. If, after several hours of observation, the clinical picture is still suggestive, exploratory laparotomy or laparoscopy should be performed without delay. *Suspicion, and not the constellation of classic clinical signs, is the indication for surgical intervention in pregnancy.* The removal of a normal appendix is justified to avoid the tragedies following expectant treatment. In 1972, Mahmoodian reported on 27 series of appendicitis during pregnancy between 1960 and 1992. Of 720 cases of appendicitis, 175 patients had a perforated appendix. Among the perforated cases there were five maternal deaths and a 22.3% incidence of premature labor. The mortality rate of appendicitis

today in the obstetric patient is essentially that associated with surgical delay.

Localization of tenderness to the right lower quadrant is a universal finding in the first trimester. The most important single sign, and often the only one, is persistent point tenderness at or near McBurney's point. As the uterus enlarges and the appendix is displaced upward and laterally, however, the point of maximal tenderness could be at the level of the umbilicus and often lateral to the anterior superior iliac spine, or even in the right upper quadrant. Involuntary guarding and rebound tenderness can be seen, caused by periappendiceal inflammation or peritonitis. Involuntary guarding and rebound tenderness are less reliable signs of peritonitis in late pregnancy because of the laxity of the abdominal wall. Other commonly occurring conditions can be responsible for similar history and physical findings. The differential diagnosis includes localized tenderness of the round ligament syndrome, a degenerating leiomyoma, and placental abruption. Pyelonephritis is the most common condition confused with appendicitis, however, especially during labor. Other conditions that are misdiagnosed as appendicitis include salpingitis, ectopic pregnancy, ovarian torsion and infarction, ruptured corpus luteum, ovarian vein thrombosis, premature labor, renal colic, pneumonia, pancreatitis, peptic ulcer, cholecystitis, mesenteric arterial occlusion or venous thrombosis with bowel gangrene, mesenteric adenitis, inflammation of a Meckel's diverticulum, and regional ileitis (Crohn's disease) [130,136–138]. A comparison of the findings in pregnant and nonpregnant patients with appendicitis is presented in Table 16.1.

In the first trimester, ultrasonography can help to differentiate among ovarian cysts, ectopic pregnancy, and appendicitis. Sonography can demonstrate appendiceal mucosal thickening and periappendiceal fluid, but the findings are usually nonspecific. Twenty-five percent of pregnant women with appendicitis develop appendiceal perforation. Appendiceal displacement predisposes to rapid development of generalized peritonitis after perforation, because the omentum is not nearby to contain the infection. Graded compression ultrasonography has been shown to be accurate in the first and second trimesters [139]. Helical CT is a newer technology and can be accomplished in 15 minutes with an exposure of 300 mrad to the fetus [140]. MRI

shows promise in the examination and diagnosis of acute appendicitis in pregnant women [13,14,141].

### *Management*

At all stages of pregnancy, the treatment is immediate appendectomy. The risk to the mother and fetus is small in cases treated early. Significant fetal loss occurs only with delay. Preoperative management includes intravenous hydration and correction of electrolyte abnormalities. During surgery, the best safeguards against subsequent abortion or premature labor are gentleness, avoidance of the uterus, unhurried maneuvers, and prevention of spread of infection. The type of incision is an individual choice, but because the appendix almost invariably lies beneath the point of maximal tenderness, a right midtransverse incision or a McBurney muscle-splitting incision over the point of maximal tenderness can be performed. Tilting the patient approximately 30° to the left side can assist in exposure.

If frank pus is present in the abdominal cavity, a culture is taken and appendectomy and copious peritoneal lavage follow. Inversion of the stump can be omitted if the cecal wall is edematous. A drain down to the appendiceal stump is recommended if perforation has occurred; otherwise, the peritoneal cavity should be closed and the wound drained. If there is no free pus, drainage is unnecessary. The question of routine postoperative antibiotic therapy is not settled. The authors recommend administration of intravenous antibiotics only if there has been perforation, peritonitis, abscess formation, or a periappendiceal collection. Tocolytic agents should be considered in women in preterm labor.

Seldom if ever is cesarean delivery indicated at the time of appendectomy. Aside from local tenderness, a recent abdominal incision presents no problem during labor and vaginal delivery. If the patient is in labor at the time of the diagnosis and vaginal delivery can be achieved without much delay, labor should be allowed to continue or proceed, and the appendix removed immediately postpartum. Conversely, if the patient is in early labor without an obstetric indication for abdominal delivery, awaiting vaginal delivery before performing a laparotomy is unwise. The decision to perform cesarean delivery at the time of appendectomy is made on the merits of the individual case and for obstetric

indications. This procedure should not be performed routinely. Theoretically, the best procedure is an extraperitoneal section followed by an appendectomy through an incision over the appendix. In the presence of a nonperforated appendix, however, cesarean delivery carries no additional morbidity. If perforation with peritonitis or abscess formation is apparent and abdominal delivery is indicated, a cesarean hysterectomy deserves consideration. (See Chapter 11.)

Laparoscopy should be considered during the first two trimesters of pregnancy for nonperforated appendicitis or when the diagnosis is uncertain. To reduce uterine injury, the open technique for establishment of pneumoperitoneum and careful introduction of additional trocars under direct visualization is recommended. Laparoscopic appendectomy is more widely accepted as safe and effective and has become the standard of care at some institutions [126,142,143]. Some authors propose the 28th week as the upper limit of gestational age for successful laparoscopic surgical intervention, although there are reports of it being done after that [144]. The major advantages of laparoscopic appendectomy are better visualization, limited uterine manipulation, and minimal morbidity for a negative exploration. In addition, there is earlier return of gastrointestinal function, earlier ambulation, and lower incidence of DVT, decreased hospital stay, and quicker return to routine activities. Furthermore, there are lower rates of wound dehiscence, infection, and hernia. Less pain and decreased narcotic use leads to a decrease in maternal hypoventilation and fetal narcotic depression.

If possible, the appendix should be routinely inspected at the time of cesarean delivery or at tubal ligation in the postpartum period. The complication of acute appendicitis in the first several weeks after cesarean delivery or pelvic laparotomy is rare but could have serious consequences because of the difficulty of interpreting physical signs and laboratory data in the postoperative patient. Larsson was one of the first to advocate incidental appendectomy at the time of cesarean delivery [145]. Since then there have been other proponents of this procedure. Sweeney compared 230 cesarean patients on whom appendectomy was performed with a control group of 230 cesarean patients without appendectomy [146]. Except for a 16-minute increase in

operative time for those with appendectomy, there were no significant differences between the groups. There was no increase in operative risk, no difference in postoperative febrile morbidity, and no increase in the duration of hospitalization among the patients in the appendectomy group. Douglas and Stromme reported no significant complications in more than 500 selected cases of cesarean delivery when incidental appendectomy was performed [147]. Wilson and associates found no increase in morbidity when appendectomy was combined with cesarean delivery, cesarean tubal ligation, cesarean hysterectomy, postpartum tubal ligation, or postpartum hysterectomy [148]. It has been suggested that incidental appendectomy should be performed with caution when cesarean delivery is done for a prolonged labor, prolonged rupture of the membranes, or amnionitis, and is most acceptable in patients who are having an elective cesarean operation. In 1986, Parsons and coworkers reported good results from performance of an incidental appendectomy at the time of elective cesarean delivery [149]. These authors also pointed out that a significant decrease in appendiceal disease in women could result from removal of the appendix during this procedure, since 20% to 25% of all deliveries were done by cesarean. Performing an incidental appendectomy at the time of cesarean delivery has been questioned by some and currently is not routinely done [150], even though there is ample evidence that, provided good judgment is used, no increase in morbidity or mortality occurs when incidental appendectomy is performed at the time of routine abdominal hysterectomy, salpingectomy, tubal ligation, or cesarean delivery. Appendectomy should be performed only when significant pathology of the appendix is found (e.g., inflammation, tumor, mass, or nonreducible stone).

### **NEOPLASTIC DISEASES UNIQUE TO PREGNANCY**

This section discusses a series of neoplastic disorders encountered at varying degrees of frequency during pregnancy, and outlines their clinical management. These diseases frequently require surgical treatment either during pregnancy or in the early postpartum period. Some are potentially life threatening to the mother, raising serious management



questions because appropriate therapy could well threaten the survival of the pregnancy.

### Gestational Trophoblastic Disease

Gestational trophoblastic disease was recognized by Hippocrates, who described the *hydatidiform mole* as dropsy of the uterus and attributed it to unhealthy water [151,152]. In 1700, the terms *hydatid* and *mole* were first used by William Smellie. In 1895, Marchand demonstrated that choriocarcinoma was preceded by the hydatidiform mole, and less commonly by a normal pregnancy or abortion [152]. By the early 1900s, several investigators had demonstrated that women with hydatidiform mole had an excess of chorionic gonadotropic hormone in the urine [152].

*Gestational trophoblastic disease* (also called *gestational trophoblastic neoplasia* or *gestational trophoblastic tumor*) is the term commonly applied to the spectrum of diseases that show abnormal proliferation of trophoblastic tissue. This general term encompasses the following histologically distinct conditions: complete and hydatidiform mole, invasive mole, choriocarcinoma, and placental site trophoblastic tumor. During the first half of the twentieth century, the morbidity and mortality from gestational trophoblastic disease, particularly choriocarcinoma, was substantial. In the late 1940s, Hertz demonstrated that fetal tissues required a large amount of folic acid and would be inhibited by the antifolic compound methotrexate [152]. In 1956, Li and coinvestigators reported the successful treatment of metastatic choriocarcinoma by using methotrexate [152,153]. Gestational trophoblastic disease is recognized as the most curable gynecologic malignancy as the knowledge and experience in its management has accumulated.

### Epidemiology

In the United States, hydatidiform moles are found in approximately 1 in 600 therapeutic abortions and in 1 of 1,500 pregnancies [152,154]. Earlier reports suggest a higher incidence of hydatidiform mole in Asia, possibly related to socioeconomic status, nutritional factors, and genetic predisposition [152]. Hydatidiform moles have been found to occur more often in older women (age >40 years) and women 15 years or younger, and are seen infrequently in women aged 20 to 29 years [152]. Parity does not

seem to be a risk factor. Age and parity do not appear to affect the clinical outcome of a woman with a hydatidiform mole [152]. Spontaneous remission occurs in 80% to 85% of all patients with a hydatidiform mole. Twenty percent of women develop malignancy that requires chemotherapy after evacuation of a mole [155,156]. Gestational age at the time of diagnosis does not appear to influence subsequent sequelae [152]. It is possible that nutritional factors, such as a deficiency of animal fat and fat-soluble vitamin carotene, have an effect on the incidence of hydatidiform mole [157].

Women with hydatidiform moles have an increased risk of trophoblastic disease in future pregnancies. In the United States, the reported incidence is 1% to 2% [158]. Sand and coworkers reported that after two episodes of gestational trophoblastic disease, the risk of molar disease in a later conception is 28% [159]. Although women with consecutive molar pregnancies can have subsequent normal pregnancies, they have an increased risk of persistent disease [158].

Gestational choriocarcinoma occurs in approximately 1 in 40,000 pregnancies and can follow any type of pregnancy [152]. Of these, 25% of choriocarcinomas follow hydatidiform moles, 25% follow an abortion or a tubal pregnancy, and 50% are associated with a term gestation [160].

### Pathology

An *invasive mole* is a trophoblastic tumor characterized by myometrial invasion by direct extension or through venous channels. Metastasis to distant sites, most commonly to the lungs and vagina, occurs in approximately 14% of cases. Histologically, it is characterized by swollen avascular placental villi and dysplastic and hyperplastic trophoblasts. Invasion or persistence is seen in approximately 10% to 17% of hydatidiform moles. The diagnosis is usually based on the detection of rising or persistently elevated human chorionic gonadotropin (hCG) levels after the evacuation of a hydatidiform mole.

Gestational choriocarcinoma is characterized by trophoblastic hyperplasia and anaplasia, absence of chorionic villi, hemorrhage and necrosis, direct uterine invasion, and vascular spread to the myometrium and distant sites. The most common sites for metastases are the vagina, lung, liver, and brain. It can also affect the pelvis, spleen, intestines, and kidneys.

The *placental-site trophoblastic tumor*, the rarest form of gestational trophoblastic disease, arises from the placental implantation site and resembles syncytial endomyometritis. Pathologically, tumor cells infiltrate the myometrium, growing between smooth muscle cells, but unlike syncytial endomyometritis, there is vascular invasion. A placental-site tumor differs from choriocarcinoma primarily by the absence of an alternating pattern of cytotrophoblast and syncytiotrophoblast; rather, there is a decrease in syncytiotrophoblasts. Hemorrhage and necrosis are less prominent. Human placental lactogen is present in tumor cells, whereas immunoperoxidase staining for hCG is positive only in scattered cells [152]. Serum hCG levels are relatively low in this disease, compared with choriocarcinoma. Although most of these tumors have a benign course, there is a 15% to 20% mortality rate from metastatic disease.

### Diagnosis

Gestational trophoblastic tumors are most often diagnosed following evacuation of a molar pregnancy or following passage of tissue resembling vesicles. The diagnosis of hydatidiform mole is suggested by an abnormally high hCG level, an enlarged uterus greater than expected for gestational age, and vaginal bleeding. Ultrasonography has replaced all other means (e.g., amniography, uterine arteriography) of diagnosing a hydatidiform mole. Usually, sonography will reveal an enlarged uterus with a diffuse mixed echogenic pattern replacing the placenta ("snowflake" pattern), formed by the interface between the molar villi and the surrounding tissue. Normally, a gestational sac or fetus is absent. In rare instances, a fetus coexists with a mole. In 15% to 25% of women with a complete mole, the ovaries are enlarged, with multiple cystic spaces identified (theca lutein cysts).

Techniques used in the past to evacuate a molar pregnancy have included dilatation and curettage (suction and sharp), hysterotomy, hysterectomy, and various induction techniques. Suction curettage is now the method of choice for evacuation of a mole regardless of the size of the uterus. The role of hysterotomy is extremely limited. It is recommended that all patients with molar pregnancy have evacuation by suction dilatation and curettage. The gynecologic surgeon should be prepared to perform a laparotomy, if necessary, in cases in which there is

major hemorrhage. After a moderate amount of tissue has been removed, administration of high-dose intravenous oxytocin is begun. When the suction curettage has been completed and involution has begun, a sharp curettage is usually performed, and this tissue should be submitted separately for subsequent histologic examination.

A primary hysterectomy with preservation of the adnexa can be selected as the method for evacuation if the patient does not wish to preserve child-bearing. If theca lutein cysts are encountered at the time of hysterectomy, the ovaries should be left in place, because these will regress to normal as the hCG diminishes to normal. Even if a hysterectomy is performed, the risk of postmolar gestational trophoblastic disease is approximately 3% to 5% [156,160]; therefore patients must be followed in the same manner as when other evacuation techniques are used.

The patient who has had a mole evacuated must be followed closely by serial determinations of the hCG titer, because hCG is produced by molar pregnancies and is a sensitive marker of trophoblastic cells present in the body. A sensitive quantitative  $\beta$ -hCG bioassay or radioimmunoassay capable of detecting  $\beta$ -hCG to values less than 5 mIU/ml should be used.

After evacuation, the patient should have a quantitative serum  $\beta$ -hCG level within 48 hours of evacuation, followed by serial  $\beta$ -hCG determinations at 1- to 2-week intervals while elevated and until there are three normal determinations. This would indicate a spontaneous remission and should occur in approximately 80% of patients with complete moles. Clinical residual disease following a partial mole is rare and unpredictable [161,162]. The hCG titer should be repeated monthly for 6 months, then every other month to complete 1 year. The patient must use some type of contraception during the monitoring period, because a subsequent normal pregnancy cannot be differentiated from gestational trophoblastic disease by the hCG determination. Unless otherwise contraindicated, oral contraceptives should be used because they do not increase the incidence of postmolar gestational trophoblastic disease or alter the pattern of regression of hCG values [156,163]. Regular pelvic examinations should be done at 2-week intervals until the hCG titers return to normal levels to monitor the involution of pelvic structures and to aid in identification of vaginal metastasis. During the first year,

pelvic examination should be done at 3-month intervals. Some investigators now think that a normal titer for 6 months is sufficient and permit subsequent pregnancies to occur after that time. Subsequent pregnancy outcomes following a molar pregnancy are very similar to those of women with normal pregnancies. No significant differences have been found between the two groups when compared for term live births, first- and second-trimester abortions, congenital anomalies, stillbirths, prematurity, and primary cesarean delivery rate. Subsequent pregnancy outcomes appear similar regardless of whether the mole is complete or partial [158]. Curry and coworkers [156] and Lurain and coworkers [164] suggest that the indications for treatment following evacuation of a hydatidiform mole include

- Plateauing hCG levels for 3 consecutive weeks
- Rising hCG levels for 2 consecutive weeks
- Persistently elevated hCG levels 6 months after evacuation
- Detection of metastases
- Histopathologic diagnosis of choriocarcinoma

Recently, the International Federation of Gynecologists and Obstetricians (FIGO) standardized hCG criteria for the diagnosis of postmolar gestational trophoblastic disease [165]. The following criteria were proposed:

- An hCG level plateau of 4 values plus or minus 10% recorded over a 3-week duration (days 1, 7, 14, and 21)
- An hCG level increase of more than 10% of 3 values recorded over a 2-week duration (days 1, 7, and 14)
- Persistence of detectable hCG for more than 6 months after molar evacuation

Of women with molar pregnancies, 85% have nonmetastatic disease, and only 20% of patients need treatment for trophoblastic tumor after evacuation [164]. Usually, a diagnosis of choriocarcinoma is made by a persistent hCG elevation, frequently in conjunction with demonstration of metastases. Tissue for pathologic examination is obtained by uterine curettage, from biopsy of a metastatic lesion, or through examination of a hysterectomy specimen or placenta.

*The symptom most suggestive of trophoblastic disease is continued uterine bleeding after hydatidiform mole evacuation or after any pregnancy.* Bleeding from uterine perforation or from a metastatic lesion can present as abdominal pain, hemoptysis, melena; or as headaches, seizures, or hemiplegia, as evidence of intracerebral hemorrhage. Patients also report respiratory symptoms, such as dyspnea, cough, and chest pain secondary to extensive lung metastases.

Signs suggestive of postmolar trophoblastic disease are an enlarged, irregular uterus and persistent bilateral ovarian enlargement from theca lutein cysts. Occasionally a metastatic lesion is noted on clinical examination, most frequently in the vagina.

### **Classification and Staging**

Once the diagnosis of gestational trophoblastic disease is made, its extent is evaluated by a thorough history and physical examination, and by laboratory and radiologic studies. Laboratory studies include complete blood and platelet counts, renal and liver function tests, clotting function tests, blood type and antibody screen, and a quantitative serum hCG. Radiographic studies include a chest radiograph or CT scan, pelvic ultrasonography, MRI or CT scan of the brain (if the patient has lung metastases), and abdominal and pelvic CT scans with contrast or MRI [165]. After these initial studies, staging is based on the extent of anatomic disease and the likelihood of response to various chemotherapeutic protocols.

Several classification and scoring systems are in use. Most major trophoblastic disease centers in the United States use a clinical classification system based on prognostic factors originally described by Hammond and coworkers in 1973 (Table 16.2) [166]. In this system, patients are divided into three disease groups: 1) *nonmetastatic*, 2) *low-risk metastatic*, and 3) *high-risk metastatic*. *High-risk metastatic* refers to patients whose disease is not likely to be cured by single-agent chemotherapy and therefore are at the highest risk of treatment failure. In 1983, the World Health Organization (WHO) adopted a modified prognostic scoring system proposed by Bagshawe based on nine factors (Table 16.3) [167,168]. An anatomic staging system conforming to other gynecologic cancers and based on data presented by Song and coworkers [169] was

**TABLE 16.2 Clinical Classification of Gestational Trophoblastic Tumors**

Nonmetastatic disease: No evidence of disease outside the uterus
Metastatic disease: Any disease outside the uterus
Low risk: Good prognosis metastatic disease
Low pretreatment, hCG titer (hCG $\leq$ 100,000 IU/24 hr urine or $\leq$ 40,000 mIU/ml serum)
Short duration (symptoms present for $\leq$ 4 months)
No brain or liver metastases
No prior chemotherapy
Pregnancy event is a hydatidiform mole, ectopic pregnancy, or a spontaneous abortion
High risk: hCG titer $>$ 100,000 IU/24-hr urine or $>$ 40,000 mIU/ml serum
Symptoms present for $>$ 4 months
Brain or liver metastases
Prior chemotherapeutic failure
Antecedent term pregnancy

From Hammond CR, Borchest LC, Tyrey L, et al: Treatment of metastatic trophoblastic disease: good and poor prognosis. *Am J Obstet Gynecol* 1973;115:451–457; with permission.

adopted by the FIGO Cancer Committee in 1982 (Table 16.4). The FIGO staging system was revised in 2000 because the original did not include hCG level, duration of disease, or type of previous pregnancy. The revised FIGO staging system includes a

**TABLE 16.4 FIGO Clinical Staging of Gestational Trophoblastic Tumors**

Stage	Tumor Site
I	Strictly contained to uterine corpus
II	Extending to adnexae, outside uterus, but limited to genital structures
III	Extending to lungs with or without genital tract involvement
IV	Metastatic to any other site(s)

From FIGO: Annual report on the results of treatment in gynecological cancer. *J Epidemiol Biostat* 2001;6:i–xiii, 1–184; with permission.

modification of the WHO prognostic index score for risk assessment [165].

#### **Management: Nonmetastatic Gestational Trophoblastic Disease and Low-risk Metastatic Disease**

Single-agent chemotherapy with methotrexate or dactinomycin is the treatment of choice for patients with nonmetastatic or low-risk metastatic disease who wish to preserve fertility [170–173]. Several chemotherapy protocols have yielded excellent and comparable results (Table 16.5). The treatment of choice in terms of efficiency and cost-effectiveness traditionally has been methotrexate

**TABLE 16.3 Prognostic Factor-based Scoring for Gestational Trophoblastic Tumors**

Prognostic Factors	Score*			
	0	1	2	4
Age (yr)	$\leq$ 39	$>$ 39	–	–
Antecedent pregnancy	HM <sup>†</sup>	Abortion	Term	
Interval (mo) <sup>‡</sup>	$\leq$ 4	4–6	7–12	$>$ 12
hCG (mIU/ml)	$\leq$ 10 <sup>3</sup>	10 <sup>3</sup> –10 <sup>4</sup>	10 <sup>4</sup> –10 <sup>5</sup>	$>$ 10 <sup>5</sup>
Largest tumor, including uterine tumor	3–4 cm	5 cm		
Metastatic sites	Lung, vagina	Spleen, kidney	GI tract	Brain, liver
No. of metastases identified	0	1–4	4–8	$>$ 8
Prior chemotherapy	–	–	1 drug	$\geq$ 2 drugs

\*Total patient score is obtained by adding individual scores for each prognostic factor.

Total score: 0–6 = low risk;  $\geq$ 7 = high risk.

<sup>†</sup>HM = hydatidiform mole.

<sup>‡</sup>Interval between end of antecedent pregnancy and start of chemotherapy.

From Kohorn EI: The new FIGO 2000 staging and risk factor scoring system for gestational trophoblastic disease: Description and clinical assessment. *Int J Gynecol Cancer* 2001;11:73–77; with permission.

**TABLE 16.5 Chemotherapy for Nonmetastatic and Low-risk Metastatic Gestational Trophoblastic Tumors**

Methotrexate	0.4 mg/kg PO, IV or IM qd × 5 days, repeated every 12–14 days (7- to 9-day window) 1 mg/kg IM days 1, 3, 5, 7 plus 0.1 mg/kg IM folic acid days 2, 4, 6, 8. Repeated every 15–18 days (7- to 10-day window)
Dactinomycin	10–13 µg/kg IV qd × 5 days, repeated every 12–14 days (7- to 9-day window) 1.25 mg/m <sup>2</sup> IV every 2 weeks

From Osathanondh R, Goldstein DP, Pastoride GB: Actinomycin D as the primary agent for gestational trophoblastic disease. *Cancer* 1975;36:863–866; Berkowitz RS, Goldstein DP, Bernstein MR: Ten years' experience with methotrexate and folic acid as primary therapy for gestational trophoblastic disease. *Gynecol Oncol* 1985;23:111–118; Smith EB, Weed JC Jr, Tyrey L, et al: Treatment of nonmetastatic gestational trophoblastic disease: Results of methotrexate alone versus methotrexate-folic acid. *Am J Obstet Gynecol* 1982;144:88–92; Baxter JF, Soong SJ, Hatch KD, et al: Treatment of nonmetastatic gestational trophoblastic disease with oral methotrexate. *Am J Obstet Gynecol* 1987;157:1166–1168; Goldstein DP: Prevention of gestational trophoblastic disease by the use of actinomycin D in molar pregnancies. *J Obstet Gynecol* 1974;43:475–479; Petrilli ES, Twigg LB, Blessing JA, et al: Single-dose actinomycin treatment for nonmetastatic gestational trophoblastic disease. *Cancer* 1987;60:2173–2176; Schlaerth JB, Morros CP, Nalick RH, et al: Single-dose actinomycin D in the treatment of postmolar trophoblastic disease. *Gynecol Oncol* 1984;19:53–56.

0.4 mg/kg (maximum 30 mg IV or IM) daily for 5 days per treatment course. An alternative regimen uses higher doses of methotrexate: 1.0 mg/kg to 1.5 mg/kg IM every other day for four doses, plus 0.1 mg/kg to 0.15 mg/kg IM folic acid 24 hours after each dose of methotrexate. The advantage of this protocol – decreased toxicity, especially stomatitis – is offset by disadvantages of increased cost, patient inconvenience, and increased need for a change in chemotherapy to achieve remission [174]. In both of these protocols, methotrexate courses are repeated as often as toxicity permits, usually every 2 weeks. Alternatively, in nonmetastatic postmolar trophoblastic disease, methotrexate produced remission rates of almost 75% in single weekly intramuscular doses of 30 mg/m<sup>2</sup> to 50 mg/m<sup>2</sup> and more than 85% when administered orally at standard doses for 5 days every 2 weeks [175,176]. Homesley and colleagues have shown a 70% to 80% primary remission rate for patients with nonmetastatic gestational trophoblastic disease treated with weekly intramuscular methotrexate at a dose of 30 mg/m<sup>2</sup> to 50 mg/m<sup>2</sup> with no apparent benefit of increas-

ing the dose to 50 mg/m<sup>2</sup> [176,177]. When efficacy, toxicity, and cost were taken into consideration, this weekly methotrexate schedule was preferred over the others [176,177]. Dactinomycin (10 µg/kg–13 µg/kg IV daily for 5 days every 2 weeks) is appropriate for patients with liver or renal disease, or effusions contraindicating methotrexate [178–180]. Dactinomycin can be given as a single dose of 1.25 mg/m<sup>2</sup> IV every 2 weeks [180].

An alternative agent should be chosen if hCG levels plateau or toxicity precludes adequate dosing or frequency of treatment. Multiagent chemotherapy is initiated if patients demonstrate resistance to single-agent chemotherapy, or if they develop metastases involving organs such as the brain or liver. Treatment is continued until three consecutive weekly normal hCG levels, and one or two courses of chemotherapy should be continued after the first normal hCG titer. After initial chemotherapy, 85% of patients are cured. With additional chemotherapy, most refractory patients are in permanent remission. With this protocol, fewer than 5% of patients require hysterectomy for resistant disease, thus preserving reproductive function in most patients [181].

Women with nonmetastatic trophoblastic tumors who no longer wish to preserve fertility should be offered a hysterectomy, which is also the treatment of choice for placental-site trophoblastic tumors, which are usually chemotherapy resistant [182–185]. Several series demonstrate the benefits of hysterectomy to treat nonmetastatic disease, followed by a reduced number of courses and duration of chemotherapy [181,186]. Hysterectomy is most often performed for 1) control of hemorrhage, 2) infection unresponsive to antibiotic therapy, and 3) localized disease in the uterus resistant to chemotherapy. Adjuvant single-agent chemotherapy at the time of operation can eradicate occult metastases and reduce the likelihood of tumor dissemination [187]. Hysterectomy is generally performed during the first cycle of chemotherapy. Chemotherapy is continued and administered for two cycles after a negative hCG value has been obtained [188]. No increase in postoperative morbidity has been reported with this sequence.

It is important to monitor patients carefully for evidence of drug resistance (i.e., plateau or rising hCG level or development of new metastases) so that chemotherapy can be changed promptly. To

TABLE 16.6 EMA-CO Regimen for High-risk Gestational Trophoblastic Tumors

Day 1	Etoposide	100 mg/m <sup>2</sup> IV infusion in 25 ml normal saline over 30 min
	Dactinomycin	0.5 mg IV push
	Methotrexate	100 mg/m <sup>2</sup> IV push stat 200 mg/m <sup>2</sup> IV infusion in 1000 ml D <sub>5</sub> W over 12 hr
Day 2	Etoposide	100 mg/m <sup>2</sup> IV infusion in 250 ml normal saline over 30 min
	Dactinomycin	0.5 mg IV push
	Folinic acid	15 mg PO or IM every 12 hr for 4 doses, beginning 24 hr after methotrexate start
Day 8	Vincristine	1 mg/m <sup>2</sup> IV push
	Cyclophosphamide	600 mg/m <sup>2</sup> IV infusion in 250 ml normal saline over 30 min Repeat cycle on days 15, 16, and 22 (every 2 weeks)

From Bagshawe KD: Treatment of high-risk choriocarcinoma. *J Reprod Med* 1984;29:813–820; with permission.

achieve complete remission, 10% to 15% of patients treated with sequential single-agent chemotherapy require combination chemotherapy with or without surgery [156,171,189].

#### **Management: High-risk Metastatic Tumors**

Aggressive multimodality therapy with appropriate combination chemotherapy, adjuvant radiation therapy, and surgery, as performed at trophoblastic disease centers, has resulted in cure rates of 80% to 90% in patients with metastatic high-risk gestational trophoblastic tumors [190–196]. The most important prognostic factors are a clinicopathologic diagnosis of choriocarcinoma, metastases to sites other than the lung or vagina, the number of metastases, and failure of previous chemotherapy. Other traditional high-risk factors, such as hCG level, disease duration, and antecedent pregnancy, have additional but more moderate impact on response.

Metastatic sites have a profound effect on survival. Lurain and coworkers noted that when metastatic disease was confined to the lung or vagina, survival was 91%, compared with 52% when other metastatic disease was present at initiation of treatment [197]. In their series, approximately one half the patients with brain metastases and two thirds with liver or intraperitoneal metastases died. Furthermore, survival decreased from 96% for patients with one to four metastases to 84% with five to eight, and to 47% with nine or more metastases.

Patients with high-risk metastatic gestational trophoblastic tumors are treated more aggressively with initial combination chemotherapy, with or without adjuvant radiotherapy or surgery. The regimen of etoposide, high-dose methotrexate infusion

with folinic acid rescue, dactinomycin, cyclophosphamide, vincristine (EMA-CO) formulated by Bagshawe (Table 16.6) or some variation of it is the treatment of choice for patients with high-risk disease [190]. Newlands and coworkers reported an 83% success rate with this regimen [190]. Since then it has generally replaced the methotrexate, dactinomycin, and cyclophosphamide or chlorambucil (MAC) and cyclophosphamide, hydroxyurea, dactinomycin, methotrexate, vincristine, and doxorubicin (CHAMOCA) regimens for high-risk disease because of greater efficacy and a lower risk of toxicity [198,199]. Schink and coworkers in 1992 reported 10 of 12 patients with high-risk disease (83%) had complete responses to EMA-CO [200]. The previously used MAC chemotherapy regimen [156,192,200,201] and the modified Bagshawe protocol [202], CHAMOCA, are no longer recommended as first-line therapy.

Other agents of proven activity in trophoblastic tumors, cisplatin and bleomycin, have been used in combination with etoposide or vinblastine to produce cures in some patients who fail initial therapy [203–206]. When either is used in primary therapy, however, significant cumulative toxicity before complete response often compromises the ability to deliver adequate salvage chemotherapy. Successful treatment for refractory trophoblastic disease with high-dose etoposide also has been reported, but side effects include nausea, bone marrow suppression, alopecia, amenorrhea, and ovarian dysfunction [207]. Colony-stimulating factors possibly play an important role in the future management of these patients.

When central nervous system (CNS) metastases are present, whole-brain irradiation is prescribed

simultaneously with initiation of combination chemotherapy. Whole-brain radiation therapy in combination with systemic chemotherapy has resulted in a 50% survival rate of patients presenting with brain involvement in the series of 75 cases collected by Jones [208]. This is virtually identical to the results of intrathecal methotrexate plus systemic chemotherapy reported from the United Kingdom [195]. Rustin and coworkers reported that 72% of 25 patients presenting with CNS metastases were cured with EMA-CO plus intrathecal methotrexate [209]. Evans and coworkers have shown that brain irradiation in combination with systemic chemotherapy in patients with brain metastases has a cure rate up to 75% [210]; therefore, the latest therapy for GTN with CNS metastasis remains controversial, but either intrathecal methotrexate or whole-brain radiation therapy is needed in addition to systemic chemotherapy. Brain metastases that appear during therapy are curable in only 5% to 25% of cases.

Adjuvant surgical procedures, especially hysterectomy and thoracotomy, can be used for isolated foci of chemotherapy-resistant disease [211]. Surgery also plays a significant role in controlling tumor hemorrhage, relieving bowel or urinary obstruction, treating infection, or dealing with other life-threatening complications [181]. Craniotomy with resection of metastases is performed for acute decompression in the presence of CNS hemorrhage [212].

### **Management: Follow-up**

After completion of chemotherapy, serial quantitative hCG levels are obtained at 2-week intervals for the first 3 months of remission and then at 1-month intervals until monitoring has shown 1 year of normal hCG levels [188]. The risk of recurrence after 1 year of remission is  $\leq 1\%$  [213]. Physical examinations should be done at 6- to 12-month intervals and other examinations (chest radiographs and CT scans) performed as indicated.

Contraception should be practiced during treatment and for 1 year after completion of chemotherapy. Pregnancies occurring after 6 months are probably safe. Barrier methods and oral contraceptives are acceptable; the latter are preferable because one half as many intercurrent pregnancies occur using

oral contraceptives as with the use of barrier methods [158].

Successful treatment of gestational trophoblastic disease with chemotherapy has resulted in the retention of reproductive capacity in an increasing number of women, despite exposure to drugs potentially toxic to the oocytes. A large number of successful pregnancies following treatment for gestational trophoblastic disease have been reported. There is no increased incidence of abortions, stillbirths, congenital anomalies, prematurity, or obstetric complications in these pregnancies [158]. They are, however, at greater risk for the development of a second gestational trophoblastic tumor in a subsequent pregnancy (1%–2%), although there is no evidence that gestation reactivates the original disease [159].

An early ultrasound examination is therefore recommended to confirm normal gestation in subsequent pregnancies. The products of conception from spontaneous abortion or termination of pregnancies require careful histopathologic examination, but the routine evaluation of term placentas is no longer recommended [214]. An hCG level should be obtained 6 weeks after any pregnancy.

## **OTHER NEOPLASMS**

### **Vulvar Malignancy**

#### ***Epidemiology***

The incidence of vulvar malignancy associated with pregnancy is unknown, but the disease is certainly rare since vulvar cancer constitutes 3% to 5% of all gynecologic malignancies [215–219]. Vulvar cancer is predominantly a disease of postmenopausal women, although 15% of vulvar cancer occurs in women younger than 40 years of age [219]. Fewer than 50 cases of invasive vulvar cancer associated with pregnancy have been reported [215,217–220], and recurrences are rare [221].

#### ***Pathology***

The most common vulvar tumors reported in association with pregnancy are invasive squamous cell carcinomas, followed by melanomas, sarcomas, and adenoid cystic adenocarcinomas [219]. Preinvasive

vulvar disease coexisting with pregnancy is occasionally encountered, because it has become more common in young women. Therapy for simple vulvar intraepithelial neoplasia is best delayed until postpartum.

### **Diagnosis**

Adequate biopsies of pruritic, pigmented, bleeding, eroded, or confluent hyperkeratotic vulvar lesions are essential to rule out invasive disease. The presence of intraepithelial neoplasia does not prohibit vaginal delivery. Histologic criteria for diagnosis are the same for pregnant as for nonpregnant women [222].

### **Classification and Staging**

Vulvar disease during pregnancy is staged and graded with the same criteria used in nonpregnant women. Staging of vulvar disease is surgical rather than clinical. Readers should refer to standard reference works [223].

### **Management**

Surgery is the treatment of choice and should be individualized. Invasive vulvar malignancy diagnosed during the first and second trimesters is treated by radical vulvectomy with bilateral groin dissection, preferably during the second trimester [218,219]. When the diagnosis is made in the third trimester, a wide local excision is recommended, with definitive surgery postponed until the postpartum period. Definitive therapy should be started within 1 week after delivery. Monaghan and colleagues have suggested definitive treatment until 36 weeks [224]. Many of the cases are diagnosed at the time of delivery or later because vulvar cancer occurs more frequently in poor women with no prenatal care. Pregnancy has not been found to alter the course of vulvar cancer. Women having carcinoma of the vulva who were treated with radical vulvectomy and bilateral inguinofemoral lymphadenectomy have subsequently become pregnant and delivered normally [219,225]. The decision whether the patient should deliver vaginally or undergo a cesarean delivery rests with the obstetrician but is heavily influenced by how well the vulva

has healed. A cesarean delivery should be the preferred route of delivery if there is fibrosis or vaginal stenosis [226].

### **Vaginal Malignancy**

#### **Epidemiology**

Cancer of the vagina accounts for less than 1% of all gynecologic malignancies. It is a disease that occurs primarily in women older than 50 years of age [225,227–229]. Despite the cluster of clear cell adenocarcinomas of the vagina associated with diethylstilbestrol (DES)-exposed offspring [230], vaginal cancer is uncommon during pregnancy [231]. The cancer risk to DES-exposed women is reported to be of the order of 1 in 1,000 to 1,400 [230]. Fortunately, the incidence in pregnancy is low since very few women have been pregnant at the time of diagnosis [230].

#### **Pathology**

Primary tumors occurring in the vagina are rare; most have been squamous cell cancers [217,232]. A few reports of sarcoma botryoides of the cervix and vagina in pregnancy have been recorded [233]. Secondary cancers comprise approximately 80% to 90% of all vaginal tumors. Most cases of vaginal cancer during pregnancy have been clear cell adenocarcinomas. Pregnancy is not known to adversely affect the outcome of clear cell adenocarcinoma of the vagina and cervix.

Primary squamous cell carcinoma of the vagina during pregnancy is exceptionally uncommon [217, 225,231,232]. Fujiat and colleagues have reviewed the reported cases of squamous cell carcinoma of the vagina complicating pregnancy and have noted the poor prognosis [231].

#### **Diagnosis**

Vaginal bleeding or foul watery discharge is the presenting symptom at diagnosis in about 50% of patients. These tumors are diagnosed usually by direct observation, occasionally by cytology, and by palpation of a nodule by bimanual examination. A biopsy is needed for histologic diagnosis.



### **Classification and Staging**

Vaginal cancer during pregnancy is staged and graded using the same criteria as in nonpregnant women. Staging for vaginal cancer is by clinical examination. Readers are referred to standard reference works [223].

### **Management**

Standard therapy for sarcoma occurring in the upper half of the vagina, with or without cervical involvement, includes radical hysterectomy, upper vaginectomy, and bilateral pelvic lymphadenectomy or exenteration, followed by postoperative adjuvant chemotherapy. Ortega has shown that survival with preliminary or complete treatment with chemotherapy is at least equal to radical surgical treatment, without the accompanying morbidity and mutilation [234]. Ortega and others used vincristine, actinomycin D, and cyclophosphamide (VAC) initially and later added doxorubicin to the regimen [234].

Surgical treatment of an early upper vaginal lesion and clear cell adenocarcinoma of the cervix and upper vagina is similar. Radical surgery alone is appropriate only for the Stage I lesions involving the upper vagina and/or cervix. In both instances, the pregnancy is disregarded if the patient is in the first or early second trimester. Should the pregnancy be further advanced, the appropriate timing for intervention depends on weighing the risks of prematurity against those of delayed therapy. The decision is based on the preferences of the parents and the judgment of the physician.

Superficial lesions can be treated with a wide local excision, with adjunctive radiation therapy after delivery [235]. Early-stage disease can be treated with surgery or radiation therapy [235,236]. When there is extensive involvement of the vagina by any malignant lesion, evacuation of the uterus by hysterotomy or cesarean delivery and initiation of appropriate radiation therapy should be seriously considered.

### **Prognosis**

Prognosis for vaginal neoplasia is unaffected by pregnancy. Survival statistics depend on tumor histology and the extent of spread at the time of initial diagnosis. For example, the actuarial 5- and 10-year sur-

vival rates for the 24 pregnant patients with clear cell carcinoma of the cervix and vagina (86% and 68%) were comparable to those of the 293 patients who had never been pregnant (87% and 79%) [230].

## **Cervical Disorders**

### **Cervical Intraepithelial Neoplasia**

A Papanicolaou smear of the cervix is an essential component of the initial prenatal visit. *Cervical intraepithelial neoplasia* (CIN) has a peak incidence during the reproductive years; the frequency of dysplasia in pregnancy varies with the patient population, with a reported prevalence ranging from 0.1% to 3% [237]. Risk factors for CIN include young age at first coitus, multiple sexual partners, cigarette smoking, a history of sexually transmitted diseases especially human papillomavirus (HPV), young age at first delivery, low socioeconomic status, and a high-risk sexual partner. The natural history of CIN is not affected by pregnancy, and conversely, in most cases CIN does not have an impact on the obstetric management.

The cytopathology laboratory should report cervical smears based on the Bethesda classification. Pregnant women with epithelial cell abnormalities including low-grade and high-grade squamous intraepithelial lesions (LGSIL, HGSIL), malignant cells, atypical glandular cells, and atypical squamous cells of undetermined significance (ASCUS) favoring dysplasia are referred for colposcopic evaluation of the cervix. Only an experienced colposcopist, who can appreciate the physiologic gestational changes of the cervix and recognize early signs of invasion, and who is also familiar with the decision-making process for the management of CIN during pregnancy, should perform this procedure.

Colposcopy of the cervix is a safe and noninvasive technique in pregnancy. In early pregnancy, the physiologic eversion of the external os facilitates the visualization of the squamocolumnar junction and the entire transformation zone where dysplasia originates, reducing the rate of unsatisfactory colposcopy. Late in the third trimester, the redundant vaginal walls can prevent complete visualization of the cervix, especially in obese women; abundant mucus can also impair visualization. The markedly increased uterine perfusion accentuates the vascular

findings, mimicking a mosaic pattern and suggesting a higher grade of dysplasia.

When the colposcopic findings suggest CIN, except for the most expert colposcopist, biopsies should be obtained from aceto-white lesions with or without abnormal vascular pattern, to correlate the results of the Pap smear, colposcopy, and histopathology. A lesion suspicious for invasive cancer must *always* be biopsied, regardless of the cytology. The number and depth of the biopsies should be controlled to avoid excessive bleeding; silver nitrate and Monsel's solution are used liberally for hemostasis. An endocervical curettage (ECC) is not done because of the risk of bleeding and rupture of the membranes.

When there is adequate correlation between the most severe abnormality on cytology, biopsy, and a satisfactory colposcopy for low-grade or high-grade CIN (including carcinoma in situ), treatment can be safely delayed until 6 to 8 weeks postpartum. The median interval for the progression from dysplasia to invasive cancer is 10 years, and the risk of developing an invasive cancer before delivery is minimal, as long as the patient is followed with periodic repeat colposcopy. Laser vaporization of the transformation zone would result in excessive bleeding, and its morbidity is not justified; the same applies to the loop electrosurgical excision procedure (LEEP). Cryotherapy might be less morbid given the superficial depth of freezing; however, given the natural history of CIN in pregnancy and the absence of data supporting the safety of cryotherapy in pregnancy, observation is appropriate.

Cervical conization in pregnancy is fraught with a high complication rate, including hemorrhage, spontaneous abortion, or preterm labor. The only indications for conization in pregnancy are when the Pap smear or colposcopy suggest invasive cancer that is not confirmed on biopsy; if the punch biopsy is interpreted as microinvasive carcinoma (i.e., invasion to less than 3 mm below the basement membrane), occult adjacent deeper invasion must be ruled out. A wedge resection limited to the suspicious area of the cervix is diagnostic and less morbid than a formal conization. Patients with microinvasive or Stage IA disease are allowed to continue their pregnancy to term, and the route of delivery should be guided by obstetric indications, regardless of the cervical neoplasia; definitive therapy after delivery ranges from simple hysterectomy to observation if the sur-

gical margins of the cone biopsy were negative and the patient wishes to maintain her fertility.

## Cervical Malignancy

### *Epidemiology*

*Carcinoma of the cervix* coexisting with pregnancy is relatively uncommon, but it is the most common gynecologic cancer encountered in pregnancy. Hacker and coworkers report an average incidence of carcinoma in situ in 1 of 770 pregnancies [238]. For invasive carcinoma, the average incidence is 1 in 2,200 pregnancies. They also noted that the average age of patients with carcinoma in situ of the cervix during pregnancy is 29.9 years and the average parity is 4.0. For invasive carcinoma, the average age is 33.8 years, and the average parity is 4.5.

Various studies have identified several associations between cervical cancer and the woman's history or laboratory findings. As an example, Orr and colleagues reported that abnormal vitamin levels were more common in patients with cervical cancer [239]. When compared with controls, levels of plasma folate, betacarotene, and vitamin C were significantly lower in patients with cervical cancer. There is a strong association between cigarette smoking or exposure to passive cigarette smoke and an increased risk of squamous cell carcinoma of the cervix [240]. Some studies have suggested that cancer of the cervix is more common among oral contraceptive users; however, these studies could have been influenced by confounding factors known to affect cancer risk, such as early onset of sexual activity, multiple sexual partners, and a previous history of sexually transmitted diseases. Compelling evidence now associates specific HPVs with cervical cancer. Although 70 different types of HPV have been described, HPV types 16, 18, 31, 33, 35, 39, 45, 51, 52, and 56 are found in cases of moderate and severe dysplasia, carcinoma in situ, and invasive cervical carcinoma and therefore constitute so-called high-risk types [241].

### *Diagnosis*

Although carcinoma in situ is generally asymptomatic, invasive cervical carcinoma often presents with abnormal vaginal bleeding, vaginal discharge, postcoital bleeding, and pelvic pain, similar to the

symptoms noted in nonpregnant women. Painless vaginal bleeding, the most common symptom, can readily be attributed to conditions such as threatened abortion or a low-lying placenta. Not surprisingly, the diagnosis is often delayed even though the patients are under regular medical surveillance.

Methods of diagnosis are generally the same as in nonpregnant patients. In most instances, screening cervical cytology and punch biopsy of a gross cervical lesion lead to the correct diagnosis. The pregnant cervix lends itself to colposcopic evaluation because the columnar eversion that occurs during pregnancy facilitates adequate visualization of the transformation zone. Pregnancy tends to exaggerate the colposcopic features of CIN, so that overdiagnosis is more likely than the reverse [242]. Cervical biopsies can be safely performed under colposcopic direction, but endocervical curettage should not be performed during pregnancy because of the risk of membrane rupture. When a visible lesion is present on the cervix, biopsy is indicated regardless of the cytology. Even if the biopsy is small, the increased vascularity of pregnancy can lead to excessive bleeding [242–244]. As a result, vaginal packing or placement of a suture is sometimes necessary. Some measures can be taken to prevent, reduce, or control bleeding: pressure by a cotton-tip applicator, application of ferric subsulfate (Monsel's solution), use of silver nitrate, or localized injection of vasopressin (Pitressin). Conization of the cervix, although it provides optimal diagnostic accuracy, is a relatively morbid procedure during pregnancy and should be avoided unless necessary for complete patient evaluation [242–244]. Averette and coworkers reported abortion as a complication in 11 of 33 patients who had conization during the first trimester [243]. Wedge resection or conization in pregnancy is indicated only if there is a real possibility of invasive cancer. Preterm labor is an additional serious complication. If required, conization is best performed during the second trimester to reduce risk of abortion and severe hemorrhage. When invasive cancer is suggested by cytology or colposcopy but has not been confirmed by directed biopsy, or if the transformation zone is not fully visualized, it is possible to do a wedge resection of the cervix, removing only the suspicious lesion or those areas incompletely visualized on colposcopy, rather than performing a complete conization.

TABLE 16.7 Malignant Tumors of the Cervix

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**Tumors of Epithelium**

Squamous cell carcinoma  
 Large cell nonkeratinizing  
 Large cell keratinizing  
 Small cell  
 Verrucous carcinoma  
 Adenocarcinoma  
 Common pattern  
 Adenoma malignum (minimal deviation adenocarcinoma)  
 Mucinous  
 Papillary  
 Endometrioid  
 Clear cell  
 Adenoid cystic  
 Adenosquamous carcinoma  
 Stem cell carcinoma (glassy cell carcinoma)

**Tumors of Mesenchymal Tissue**

Endocervical stromal sarcoma  
 Carcinosarcoma  
 Adenocarcinoma  
 Embryonal rhabdomyosarcoma  
 Tumor of Gartner duct (true mesonephroma)

**Others**

Metastatic tumors  
 Lymphoma  
 Melanoma  
 Carcinoid

---

**Pathology**

In most large series, 85% to 90% of malignant lesions of the cervix are squamous cell carcinomas, but other lesions are possible (Table 16.7). Most information about etiology and epidemiology is pertinent only to the more common squamous cell lesions.

**Classification and Staging**

The staging of cervical cancer is a clinical appraisal, often confirmed by an examination under anesthesia; the stage cannot be changed later if surgical findings or subsequent therapies reveal more advanced disease. Today, cervical cancers are staged almost exclusively according to the FIGO classification (Table 16.8) [245]. Palpation, biopsy, conization, colposcopy, hysteroscopy, cystoscopy, proctosigmoidoscopy, intravenous pyelography, barium

TABLE 16.8 FIGO Classification of Cancer of the Cervix

Stage 0	Carcinoma in situ, intraepithelial carcinoma
Stage I	The carcinoma is strictly confined to the cervix (extension to the corpus should be disregarded).
Stage IA	Preclinical carcinoma of the cervix (i.e., those only diagnosed by microscopy)
Stage IA1	Measured stromal invasion of no more than 3 mm in depth and extension of no more than 7 mm
Stage IA2	Measured stromal invasion of more than 3 mm and no more than 5 mm in depth, with an extension of no more than 7 mm. Larger lesions should be staged as IB.
Stage IB	Lesions of greater dimensions than Stage IA2, whether seen clinically or not
Stage IB1	Clinically visible lesion no larger than 4 cm
Stage IB2	Clinically visible lesion larger than 4 cm
Stage II	The carcinoma extends beyond the cervix but has not extended onto the pelvic wall. The carcinoma involves the vagina but not as far as the lower third.
Stage IIA	Involvement of the vagina but no evidence of parametrial involvement
Stage IIB	Infiltration of the parametria but not out to the sidewall
Stage III	Involvement of the lower third of the vagina or extension to the pelvic sidewall
Stage IIIA	Involvement of the lower third of the vagina but not out to the pelvic sidewall
Stage IIIB	Extension onto the pelvic sidewall or hydronephrosis or nonfunctioning kidney
Stage IV	The carcinoma has extended outside the reproductive tract.
Stage IVA	Involvement of the mucosa of the bladder or rectum
Stage IVB	Distant metastasis or disease outside the true pelvis

enema, and radiologic studies of the lung and skeleton can be used to stage a patient's disease. Specialized techniques such as lymphangiography, arteriography, venography, CT scan, MRI examination, and laparoscopy are not recommended for staging, because they are not uniformly available from institution to institution. Staging is a means of communication between institutions. More important, staging is a means of planning treatment. For these reasons, the method of staging should remain fairly constant. Staging does not limit the treatment plan,

and therapy can be tailored to the architecture of the malignancy in each patient. Findings uncovered by CT scan or MRI examination can be used in the planning of therapy but should not influence the initial clinical staging of the lesion. Unfortunately, clinical staging is only of rough value in prognosis, because widely variable lesions are often included under one subheading.

### Management

Carcinoma in situ diagnosed during pregnancy should be managed conservatively, with the pregnancy allowed to proceed to term. Vaginal delivery is anticipated, and appropriate therapy is carried out 6 to 8 weeks postpartum. *Microinvasive carcinoma* of the cervix diagnosed by conization during pregnancy can also be managed conservatively, with the pregnancy being allowed to continue to term with colposcopic surveillance every 6 weeks. At term, either cesarean hysterectomy or vaginal delivery, followed by postpartum extrafascial hysterectomy, is appropriate. Conization can be sufficient therapy if the surgical margins are negative and the patient wishes to maintain her fertility.

The decision to treat or delay treatment of cervical carcinoma during pregnancy is not difficult if the pregnancy is unwanted and the gestation less than 22 weeks. Similarly, if the cancer is diagnosed when fetal maturity has been attained, or if the cancer is far advanced and a delay will not change the maternal prognosis, there is little problem. Difficult decisions arise when the pregnancy is desired and the immature fetus approaches the period of viability [246–249].

In deciding on therapy, the physician must consider both the stage of disease and the gestational age. For Stage IB and IIA lesions, radical hysterectomy with bilateral pelvic lymphadenectomy with the pregnancy in situ, or radical hysterectomy with cesarean delivery, depending on the stage of pregnancy, is acceptable. The complications of radical surgery for cervical carcinoma in pregnant patients do not exceed those for nonpregnant patients when normal surgical principles are scrupulously followed. Patients in the first and second trimester are usually advised to undergo definitive therapy immediately; thus, interruption of the pregnancy usually is advised. Normal-appearing ovaries at the time of primary radical surgery in patients with

invasive carcinoma of the cervix can be preserved. Conservation of ovarian function does not adversely affect the cure rate [250].

At 24 or more weeks of gestation, one must balance potential fetal viability and maternal interests. Any decision to delay therapy should be made only after thorough discussion with the patient and her family. Highly complex ethical issues, religious beliefs, and emotional considerations for the patient and family are superimposed on this medical problem; the parents need to participate in the decision-making process. Optimal management includes consultation with a gynecologic oncologist, a radiation oncologist, a perinatologist, and a neonatologist. Delays longer than 6 to 8 weeks are difficult to justify, but with the expert neonatal care available in large centers, excellent fetal salvage should be expected after 28 weeks of gestation. Five-year survival rates reported by physicians advocating immediate treatment are not significantly different from those of physicians advocating delays in therapy of up to 8 weeks [246]. If it is decided to await fetal viability, it is important to evaluate the fetus by ultrasonography to rule out major congenital anomalies, and to administer at least 48 hours of steroid therapy before delivery.

Preoperative radiologic investigation should be performed only if radical surgery is contemplated and should be limited to chest radiographs with abdominal shielding and a limited intravenous pyelogram. Because of the increased risk of hemorrhage and infection with delivery through a cervix containing gross cancer, classic cesarean delivery is preferred to avoid the lower uterine segment. For patients in whom inadvertent vaginal delivery has occurred, however, there is no evidence to suggest that prognosis is worsened [248,251,252].

Radiation therapy is the treatment of choice for poor surgical candidates, bulky early-stage lesions, and advanced tumors (Stage IIB and beyond) [251,252]. During the first trimester, external whole-pelvis radiation therapy is usually a reliable abortifacient [253]. Spontaneous abortion usually occurs after 4,000 cGY, on average 35 days and 45 days following the onset of radiation therapy in the first and second trimesters, respectively. Abortion can be delayed 60 to 70 days in some patients treated during the second trimester [252,253]. Where radiation fails to induce abortion, dilatation and curettage (or evacuation) is performed before intra-

cavitary radium or cesium insertion. In a second-trimester pregnancy complicated by advanced cervical carcinoma, delivery of the products of conception is more complicated: at midtrimester, fetal tissues are more resistant to radiation therapy, and the abortifacient effects of treatment are less predictable. The resulting failure of abortion, despite radiation therapy, has led to reports of severely damaged yet viable neonates. The variable fetal effects following radiation therapy are accompanied by an adverse psychologic impact on the pregnant woman [254,255].

Hysterotomy is a useful alternative to radiation therapy-induced abortion, but this procedure carries its own risks (e.g., hematopyometria as a result of obstruction of the cervical canal by infected malignant tissue). Additional risks of laparotomy, including adhesions of bowel to the uterine scar, can place such tissues at risk of radiation therapy-induced damage. Scandinavian authors have suggested the *Porro operation*, a supracervical hysterectomy after hysterotomy [256]. This removes the nidus for hematopyometria formation, but resultant raw areas again could promote bowel adhesions and radiation therapy-induced damage. This procedure has not been widely accepted.

### **Prognosis**

The overall prognosis for all stages of cervical cancer in pregnancy is similar to that of nonpregnant women. Hacker and coworkers [238] noted an overall 5-year survival rate for pregnant women with invasive cervical cancer of 49.2%, similar to the 51% rate quoted for nonpregnant patients. Clinical stage is the most important determinant of prognosis (Tables 16.9 and 16.10). Age, parity, and gestational age at diagnosis have no effect on survival within a given stage. Similarly, the mode of delivery has no effect on maternal or fetal survival. Although patients diagnosed in the third trimester and postpartum do significantly worse than those diagnosed early in pregnancy, this is the result of more advanced disease. Bleeding in the late stages of pregnancy is often assumed to be of obstetric origin, and careful assessment of the cervix can be forgotten. The popular misconception that cervical cancer during pregnancy spreads more rapidly and aggressively than in the nongravid state is unfounded. Small cell carcinomas and glassy cell carcinomas

**TABLE 16.9 Carcinoma of the Cervix: Five-year Survival by Clinical Stage**

Stage	Treated	Survival	%
IB	474	348	74.5
II	449	214	47.8
III/IV	326	53	16.2
Total	1249	615	49.2

From Hacker NF, et al: Carcinoma of the cervix associated with pregnancy. *Obstet Gynecol* 1982;59:735-746; with permission.

**TABLE 16.10 Carcinoma of the Cervix: Five-year Survival by Period of Gestation**

Trimester	Treated	Survived	%
First	137	94	68.6
Second	51	32	62.7
Third	87	45	51.7
Postpartum	621	289	46.3
Total	896	460	51.3

From Hacker NF, et al: Carcinoma of the cervix associated with pregnancy. *Obstet Gynecol* 1982;59:735-746; with permission.

are fortunately uncommon and are associated with a poor outcome regardless of the treatment modality.

## Uterine Disorders

### *Leiomyomata*

#### *Epidemiology*

*Uterine leiomyomas* are the most common pelvic tumors in women [257-260]. Traditionally described as present in 20% of women over age 35; their appearance in 50% of postmortem examinations performed on women suggests a higher prevalence. Their reported incidence ranges from 0.3 to 2.6 per 100 births, depending on the age and race of the population studied. They usually have a minor impact on conception but can exhibit a profound effect on pregnancy maintenance. Leiomyomas are more common among African American than among Caucasian women. The growth of uterine leiomyomas is clearly related to circulating estrogens. These tumors are most prominent and demonstrate their maximal growth

during a woman's reproductive life, when ovarian estrogen secretion is maximal. With the onset of menopause, leiomyomas characteristically regress. Classic teaching has been that leiomyomas grow during pregnancy, reflecting their dependence on estrogen. Recent data suggest that this might not be true, however (see later discussion) [258,259].

#### *Pathology*

These neoplasms are variously referred to as leiomyomas, fibromyomas, myomas, leiomyofibromas, fibroleiomyomas, and fibroids. The most accurate term is *leiomyoma*, which best describes their origin and predominant cellular composition. Despite a general belief to the contrary, the best evidence is that pregnancy does not necessarily accelerate the growth of these tumors [259]. If growth occurs during pregnancy, the tumor can exceed its blood supply, leading to necrosis. Compromised tumors become dark and hemorrhagic, characteristic of the red or carneous degeneration classically described in pregnancy. Other secondary changes within leiomyomas include hyaline degeneration, cyst formation, calcification, fatty degeneration, and infection. Suppurative changes most commonly occur when a submucous myoma protrudes through the cervix into the vagina, ulcerates, becomes edematous, and is secondarily infected. Infection of a submucous leiomyoma can accompany puerperal endometritis and advance to endomyometritis with or without abscess formation. Necrosis and cystic changes are also common with torsion of a pedunculated leiomyoma. Malignant transformation of benign leiomyomas is extremely rare, occurring in less than 0.13% of uterine myomas [261]. Very few data exist regarding the incidence of malignant transformation of leiomyomas during gestation.

Unless strict criteria for determination of malignancy are used, cellular leiomyomas can be erroneously classified as leiomyosarcomas [261,262]. The microscopic diagnosis relies on the mitotic activity and degree of cellular atypia (i.e., nuclear hyperchromatism and pleomorphism). Tumors with fewer than five mitotic figures per ten high-power fields and little if any cytologic atypia are classified as cellular leiomyomas. Tumors with more than ten mitotic figures per ten high-power fields are considered malignant. Those tumors with five to ten mitotic figures per ten high-power fields and *no*

cellular atypia are termed smooth-muscle tumors of uncertain malignant potential (STUMP). Tumors with this level of mitotic activity and cellular atypia are usually classified as leiomyosarcomas. The prognosis is poor when tumors have a high mitotic count combined with cytologic atypia. Mitotically active smooth-muscle tumors of the uterus, with five to nine mitoses per ten high-power fields and no cellular atypia have a metastatic rate too low to be regarded as sarcomas. Hysterectomy need not automatically follow myomectomy in STUMP cases, because close clinical observation is a viable alternative if the patient is young and wishes to maintain fertility.

### *Diagnosis*

Most patients with uterine leiomyomas are symptom free. When symptoms occur, they are often related to the location of the leiomyomas, their size, or concomitant degenerative changes. Patients with a pedunculated leiomyoma often experience pain as the pedicle undergoes torsion, but pain can also occur with carneous degeneration. The discomfort is often acute and requires immediate attention. More commonly, pressure and increased abdominal girth develop insidiously and usually elicit vague complaints. Pressure on the bladder can provoke urinary frequency, especially when the leiomyoma is located in the subvesical region or when a large myomatous uterus fills the entire pelvis. Ureteral obstruction, usually silent, is one of the most serious complications resulting from pressure of the myomatous uterus on the ureter at the pelvic brim. Unless the kidney has suffered parenchymal damage, these anatomic changes are reversible once the pressure is alleviated. Rectal pressure is rare unless the myomatous uterus is incarcerated in the cul-de-sac or contains a solitary, large posterior wall leiomyoma. Constipation or tenesmus also is sometimes associated with a posterior leiomyoma, owing to pressure of the tumor on the rectosigmoid.

Uterine evaluation by ultrasound scan has increased understanding of the behavior of leiomyomas during gestation [258]. Before the use of sonography, only large leiomyomas were detectable in pregnancy, primarily by clinical examination. Aharoni and coworkers [259] serially followed 29 patients with 32 leiomyomas, using ultrasonography; an increase in size was apparent in only 7

(22%), whereas 6 (19%) myomas actually decreased in volume, and 19 (59%) changed in size by less than 10% of their initial volume. Thus, 78% of the uterine leiomyomas followed sonographically failed to increase in size during pregnancy. Although these data are reassuring, certain disturbances in reproductive performance, including infertility, spontaneous abortion, and premature delivery, increase with uterine leiomyomas. Associated complications include preterm PROM, malpresentation, dysfunctional labor, increased rate of abdominal delivery, retained placenta, postpartum hemorrhage, and puerperal uterine infections. In the series reported by Katz and coworkers, 2% of pregnant women had uterine leiomyomas diagnosed during gestation, and 10% of these women had complications attributable to the tumors [257].

### *Management*

Careful observation by serial follow-up examinations is suitable management for most leiomyomas. Most leiomyomas produce no symptoms and should not be confused with other pathologic conditions. These tumors are seldom malignant, especially in the absence of rapid enlargement.

### *Prognosis*

The use of progestational compounds or gonadotropin-releasing hormone agonists in an attempt to shrink leiomyomas is contraindicated during pregnancy. The best management scheme is early diagnosis and observation. If the tumors are large (>4 cm–5 cm) or symptomatic, serial ultrasonic studies are indicated to evaluate growth. Pain or other abdominal distress is best managed by bedrest, analgesia, and reassurance. Close observation during labor is prudent. Cesarean delivery is restricted to the usual obstetric indications. At cesarean delivery, the leiomyoma is avoided, if possible, as the uterus is entered. Myomectomy is generally to be avoided unless a pedunculated or otherwise easily removed tumor is encountered. In selected women and in experienced hands, myomectomy at the time of cesarean delivery can be a safe and effective procedure [264].

In the first trimester, the main problem is spontaneous abortion; in the second trimester the principal difficulty is red or carneous degeneration,

characterized by localized tenderness, leukocytosis, moderate fever, and signs of local peritoneal irritation. In most cases, bedrest, analgesics, sedation, and close observation are sufficient. After 4 to 7 days, the symptoms gradually subside and the pregnancy usually proceeds normally. More than one episode can occur, but as long as they are controlled by conservative treatment, the prognosis remains good. Myomectomy during pregnancy greatly increases the risk of abortion or premature delivery and is rarely indicated [257]. There are other opinions. Burton and coworkers in 1989 reported six women with leiomyomas ranging in size from 2 cm to 5 cm in diameter who underwent myomectomy without fetal loss [263]. They claimed that this operation can be performed safely in carefully selected patients. Although elective myomectomy at cesarean delivery might prove both safe and feasible in *selected* patients (e.g., when the mass is pedunculated), because of the potential complications this should not be a routine procedure.

During the third trimester, most leiomyomas are asymptomatic. The main problems occur during labor [257]. Although most patients with even large leiomyomas deliver normally, a cesarean rate of 25% to 30% is usual when there is significant uterine involvement. Problems arise from 1) obstruction from low cervical leiomyomas or prolapse of a large pedunculated tumor into the cul-de-sac; 2) uterine inertia with incomplete dilation requiring cesarean delivery; 3) abnormal placental separation, primarily from implantation over a leiomyoma interfering with development of a plane of cleavage necessary for placental delivery (the placenta sometimes adheres to a leiomyoma [partial or complete placenta accreta] and a hysterectomy is necessary); 4) postpartum uterine atony and hemorrhage owing to the inability of the myometrium to contract properly and compress the distended vessels; and 5) inadvertent or ill-advised extraction of a pedunculated submucous tumor on postpartum uterine exploration, because infection and hemorrhage almost always occur. Traction on a submucous tumor can lead to acute uterine inversion. In the puerperium, leiomyomas are usually asymptomatic, and if enlarged after 6 to 8 weeks, recede to their prepregnant size. Subsequent hysteroscopic resection of a submucous leiomyoma several months postpartum is much less risky than peripartum surgery. Postpartum use of leuprolide

acetate (Lupron), a luteinizing hormone-releasing hormone (LH-RH) agonist, results in an initial gonadotropin stimulation, but chronic administration results in decreased levels of LH and follicle-stimulating hormone (FSH), suppression of ovarian steroidogenesis, decreased estrogens, and shrinkage of leiomyomas and of the tumors of leiomyomatosis peritonealis disseminata [265]. Pregnancy must be rigorously excluded before this treatment is initiated, because spontaneous abortion might occur if this drug is administered during gestation.

## Uterine Malignancy

### *Epidemiology*

*Endometrial carcinoma* associated with pregnancy is extremely rare, with fewer than 30 cases reported [265–273]. More than one half of these cases were diagnosed at the time of dilatation and curettage for a spontaneous abortion or termination of pregnancy. Endometrial cancer after childbirth and term pregnancy is rare; Itoh and colleagues reviewed two cases at cesarean and ten cases after delivery [270].

### *Pathology*

When endometrial carcinoma does occur with pregnancy, it is usually focal, well differentiated, and minimally invasive or noninvasive. Leiomyosarcoma and carcinosarcoma have also been reported during pregnancy [274–276]. These tumors are usually found incidentally in surgical pathology specimens. Decidual transformation of the cervical stroma can resemble sarcoma and should not be confused with the more serious condition [277–279]. Benign metastasizing leiomyomas of the uterus has also been reported to complicate pregnancy [280]. The metastasis is usually solitary and composed of bland cells, but can be multiple; usually there is a history of prior myomectomy.

### *Management*

The recommended treatment for older patients with endometrial cancer is total hysterectomy with bilateral salpingo-oophorectomy and adjuvant radiation therapy for deeply invasive or high-grade tumors. In younger patients with noninvasive



adenocarcinoma, it is not necessary to remove normal-appearing adnexae [266–269]. Because these cases are so rare, the management must be individualized. In the face of a confirmed diagnosis of invasive, high-grade tumor, full operative extirpation of the genital organs and adjuvant radiation therapy is recommended, regardless of the patient's age. In less aggressive tumors in young women, adnexal sparing is a consideration after careful patient counseling. Conservative management with uterine preservation has been reported [281–282]. Young women desiring pregnancy and the need to preserve their reproductive organs can undergo progesterone treatment. Because the tumor can progress rapidly after a successful pregnancy, careful selection of patients and follow-up care are mandatory [283,284].

### Prognosis

The prognosis of endometrial cancer in pregnant women is presumed to be the same as that occurring for the same stage and grade among nonpregnant women. Readers are referred to standard reference works for these data [285,286].

## Ovarian Tumors

### Epidemiology

*Ovarian tumors* are a relative uncommon occurrence during pregnancy, with a reported incidence of 1 in 10,000 to 1 in 50,000 [287]. The diagnosis and management of ovarian tumors can be problematic because of the risk of malignancy and prematurity. Malignancy is very uncommon in ovarian tumors diagnosed during pregnancy, with an incidence of 2% to 5%. This disease is most common after age 50. Eastman and Hellman [288] quoted an incidence of ovarian cysts in pregnancy of 1 in 81 patients, and Grimes and coworkers [289] stated that in 1 of 328 pregnancies a cyst large enough to be potentially hazardous is present. The incidence of ovarian cysts in pregnancy by ultrasound scan is between 1 in 50 and 1 in 200 [290]. Beral has suggested that pregnancy could actually protect against the development of ovarian cancer, since this cancer is rare in populations that do not practice birth control [291]. Several studies have documented that the use of oral contraceptives diminishes the inci-

TABLE 16.11 Histologic Classification of Ovarian Tumors during Pregnancy

Histologic Classification	Number	%
Benign cystic teratoma (dermoid cyst)	25	36.0
Serous cystadenoma	17	25.0
Paraovarian cyst	9	13.0
Mucinous cystadenoma	8	12.0
Corpus luteum cyst	4	5.5
Malignant tumor*	3	4.0
Endometriotic cyst	1	1.5
Follicular cyst	1	1.5
Other	1	1.5

\*See text for details. Not otherwise specified.

From Struyk AP, Treffers PE: Ovarian tumors in pregnancy. *Acta Obstet Gynecol Scand* 1984;63:421–424; with permission.

dence of ovarian cancer, presumably by suppressing ovulation [292–296].

### Pathology

Beischer reported a series of 164 ovarian lesions diagnosed during pregnancy at the Royal Women's Hospital in Melbourne [297]. More than 50% were either mature cystic teratomas or mucinous cystadenomas, and only four (2.4%) were malignant.

Struyk described 90 pregnancies complicated by ovarian tumors (Table 16.11) [298]. Of the 69 tumors that progressed to surgery, no functional cyst was found in patients undergoing surgery after the 18th week of gestation. In eight patients, ovarian tumor enlargement was noted during a period of observation; two were malignant, one was a serous cystadenoma, and five were mature teratomas.

Thornton reviewed 131 ovarian enlargements in pregnancy; 81 (including one carcinoma and six borderline lesions) were removed [299]. Thirty-nine were larger than 5 cm in diameter and had simple internal echo patterns and smooth walls; three of these were borderline malignancies.

More recent studies of ovarian tumors during pregnancy diagnosed by ultrasonography have reported dermoid cysts as the most common diagnosis (36%), followed by serous cystoadenoma (15.5%), functional cysts (7.2%), low malignant tumors (2.4%), and adenocarcinoma (1.4%) [300–303].

## Diagnosis

The initial detection and the appropriate diagnosis are the most pressing problems with ovarian tumors in pregnancy [298]. The differential diagnosis includes a retroverted pregnant uterus, a normal corpus luteum of pregnancy, a pedunculated uterine myoma, carcinoma of the rectosigmoid, a pelvic kidney, or a congenital uterine anomaly. In the series by Struyk, 54% of the tumors were diagnosed in the first trimester [298]. Pain occurred in 26%, torsion in 12%, obstruction of labor in 17%, and rupture in 9% of the patients. Thirty-seven percent of the patients had no complications. Perinatal mortality was high (3 fetal demises, 7 neonatal deaths).

The diagnosis is difficult during the first trimester, because an asymptomatic adnexal mass is frequently detected on pelvic examination at the initial prenatal visit. Most of these ovarian masses are functional, follicular, or corpus luteum cysts, usually less than 6 cm and rarely up to 10 cm in diameter. More than 90% of functional cysts resolve spontaneously and are undetectable by the 14th week of gestation. The most common presentation for an ovarian tumor in pregnancy is as an incidental finding on an ultrasound examination. Tumors not diagnosed by the second trimester are likely to escape detection until labor and delivery, when they can result in obstructed labor or are detected as incidental findings at cesarean delivery.

In the second half of pregnancy, ovarian tumors are particularly difficult to diagnose; they ascend into the abdominal cavity beyond the reach of vaginal examination, and abdominal palpation becomes the chief method of clinical diagnosis. As in the nonpregnant patient, the most common symptoms of ovarian neoplasms in pregnancy are abdominal pain and swelling [304–308]. The pain is usually intermittent, vague, and can be associated with disturbances in gastrointestinal function, a common complaint during normal pregnancy. Whether these symptoms develop sooner because of uterine growth is not known. The pain can be acute as a result of tumor rupture, torsion, or hemorrhage. Torsion occurs more frequently during pregnancy owing to rapid growth of the uterus after the mid-trimester, elevating the tumor out of the pelvis and presumably permitting greater latitude for twisting on its narrow pedicle. With advanced malignancy,

adhesions tend to fix the mass in the pelvis despite uterine growth and prevent torsion. Rupture occurs in 2% to 3% of ovarian tumors in general, but 14% rupture during pregnancy [306,307]. Rupture and hemorrhage are most common during labor and delivery probably as a result of uterine contraction and descent of the presenting part.

Ultrasonography is particularly helpful in diagnosis. Initially, an asymptomatic mass detected in early pregnancy is assessed clinically for features suggestive of neoplasia. A cystic, unilateral, mobile ovarian enlargement less than 6 cm in diameter represents either a functional ovarian cyst or a benign cystic teratoma (dermoid) in more than 88% of cases. If there is doubt as to the nature of mass, an MRI, careful real-time review of anatomy as noted on ultrasonography, and CT scanning are helpful in establishing the correct diagnosis. Should the mass persist beyond 14 weeks of gestation or if at initial detection the mass is solid, bilateral, greater than 6 cm to 8 cm in diameter, or contains septal or surface irregularities, surgical intervention should be undertaken without delay. Metastatic disease to the ovaries must be considered in the differential diagnosis, as well as theca lutein cysts if there is a bilateral symmetric enlargement of the ovaries on pelvic examination. Carcinoma of the breast and colon are the most common malignancies to metastasize to the ovaries.

In the nonpregnant woman, an intravenous pyelogram and a barium enema are commonly ordered after ultrasound scan, if an ovarian tumor is suspected. Although not absolutely contraindicated in pregnancy, these studies must be used only after critical assessment of the perceived benefits versus the potential risks. Because laparotomy is ultimately required for a definitive diagnosis, the results of such radiologic investigations rarely alter the initial management.

Sonography and, in selected cases, serum tumor markers and color flow Doppler studies, are helpful in the evaluation. During the past decade, several investigators have confirmed that in patients with advanced ovarian cancer, cancer antigen-125 (CA-125) is useful in monitoring response to therapy and in detecting occult tumor recurrence [309,310]. Using the upper limit of normal for CA-125, only 1% of normal, healthy blood donors have an elevation of CA-125. Approximately 6% of women in the first trimester of pregnancy or with nonmalignant

disease, including active cirrhosis, pericarditis, uterine fibroids, and endometriosis, have an elevated CA-125 level, however. Elevated levels of CA-125 are also present in a variety of gynecologic and non-gynecologic malignancies – including pancreatic, colorectal, lung, endometrial, and breast carcinomas. Elevated CA-125 levels occur in most serous epithelial ovarian carcinomas but in only a few mucinous tumors.

Carcinoembryonic antigen (CEA) is a useful tumor marker in mucinous epithelial malignancies of the ovary. CEA is primarily used in the evaluation and management of patients with colon cancer, although it can be elevated in patients with carcinomas arising in the breast, endocervix, or pancreas. Choriocarcinoma and some dysgerminomas and embryonal cell tumors produce hCG, whereas endodermal sinus tumors produce  $\alpha$ -fetoprotein (AFP). Both of these markers are normally present during pregnancy, potentially confusing the diagnosis. Preoperatively drawing a tube of blood to be held for possible tumor marker assays following histopathologic identification of the ovarian tumor is likely the best approach, except when gestational trophoblastic disease is suspected. In this case, levels of serum hCG are essential to establishing the correct diagnosis.

### Classification and Staging

The simplest and most clinically oriented classification merely divides ovarian neoplasms into benign or malignant. This classification is clearly unsatisfactory because of the wide variation in the malignant behavior of ovarian neoplasms, even among tumors of the same general histologic type. In addition, there are critical differences in the spread pattern and response to treatment among the malignant tumors. Even the benign tumors have important clinical differences such as the variable frequency of bilateralism, the differences in endocrine activity, and the association of certain tumors with genetic disorders. The shortcomings of the benign-malignant division of ovarian tumors are even more apparent if the neoplastic-like ovarian enlargements are to be incorporated into the classification of ovarian tumors. Although the designation of *benign* or *malignant* is useful and important, it is not enough. A listing of benign ovarian tumors is seen in Table 16.12.

TABLE 16.12 Benign Ovarian Tumors

<b>Non-neoplastic tumors</b>
Germinal inclusion cyst
Follicle cyst
Corpus luteum cyst
Pregnancy luteoma
Theca lutein cysts
Sclerocystic ovaries
<b>Neoplastic tumors derived from celomic epithelium</b>
Cystic tumors
Serous cystoma
Endometrioma
Mucinous cystoma
Mixed forms
<b>Tumors with stromal overgrowth</b>
Fibroma, adenofibroma
Brenner tumor
Tumors derived from germ cells
Dermoid (mature cystic teratoma)

TABLE 16.13 FIGO Histologic Classification of the Common Primary Epithelial Tumors of the Ovary

1. Benign cystadenomas
2. Cystadenomas with proliferative activity of the epithelial cells and nuclear abnormalities but with no infiltrative destructive growth (borderline cases, low potential malignancy)
3. Cystadenocarcinomas

For many years, the existence of a group of epithelial ovarian tumors that have histologic and biologic features occupying a position between those of the clearly benign and frankly malignant ovarian epithelial neoplasms has been recognized (Table 16.13). Clinically, these tumors are characterized by a predominantly early stage at diagnosis, infrequent and late recurrence, and long survival rate with residual or recurrent malignancy. FIGO accorded these tumors official status in its 1971 classification of epithelial ovarian tumors, and designated them as *cystadenomas of low potential malignancy*. These borderline malignancies, which account for approximately 15% of all epithelial ovarian cancers, are also referred to as *proliferative cystadenomas*. Nearly three-fourths of borderline tumors are Stage I at the time of diagnosis. The average age

TABLE 16.14 World Health Organization Classification of Ovarian Tumors

I.	Common Epithelial Tumors	Teratomas
	Serous	Immature
	Mucinous	Mature (dermoid cyst or solid)
	Endometrioid (benign, borderline, or malignant)	Cystic with malignant transformation
	Clear cell	Monodermal (struma ovarii, carcinoid, strumal carcinoid)
	Brenner	Gonadoblastoma
	Mixed epithelial	IV. Tumor from nonspecific mesenchyme
	Undifferentiated	Hemangioma
	Carcinosarcoma and mixed mesodermal	Leiomyoma
	Unclassified	Lipoma
II.	Sex cord–stromal tumors	Lymphoma
	Granulosa cell tumor	Sarcoma
	Adult type	V. Tumors metastatic to the ovary
	Juvenile type	Gastrointestinal tract (Krukenberg)
	Thecoma	Breast
	Fibroma	Endometrium
	Cellular fibroma	Lymphoma
	Sclerosing stromal tumors	VI. Tumor-like conditions
	Arrhenoblastoma	Pregnancy luteoma
	Sertoli tumor	Hyperplasia of ovarian stroma
	Hilus or Leydig cell tumor	Stromal hyperthecosis
	Gynandroblastoma	Massive edema
	Sex cord tumor with annular tubules	Solitary follicle cyst
	Lipid cell tumor	Corpus luteum cyst
	Unclassified	Multiple follicle cysts (polycystic ovaries)
III.	Germ cell tumors	Multiple luteinized follicle cysts and/or corporalutea (hyperreactio luteinalis)
	Dysgerminoma	Surface epithelial inclusion cysts (germinal inclusion cysts)
	Endodermal sinus tumor	Simple cysts
	Embryonal carcinoma	Inflammatory lesions
	Polyembryoma	Paraovarian cysts
	Choriocarcinoma	
	Mixed germ cell tumor	

of women with the borderline tumors is between that of women with frankly malignant ovarian carcinoma and benign cystomas. The histologic criteria characterizing the borderline tumors can be summarized as follows: stratification of the epithelial lining of the papillae, formation of microscopic papillary projections or tufts arising from the epithelial lining of the papillae, epithelial pleomorphism, atypicality, mitotic activity, and no stromal invasion present.

There is now general agreement that the most useful classification of ovarian tumors is based on the presumed cell of origin; therefore, the best contemporary classifications are histogenetic. In this schema there are three major categories: 1) *tumors arising from the coelomic epithelium or mesothelium covering the ovary* (also termed germinal, surface, paramesonephric, and müllerian epithelium); 2)

*tumors arising from the specialized gonadal stroma* (sex cord–stromal tumors); and 3) *tumors arising from the germ cells*. The classification is completed by tumors that are derived from nonspecific mesenchyme and by tumors that involve the ovary but arise from other organs (Table 16.14).

The extent of the tumor at the time of diagnosis is the most important variable influencing the prognosis in ovarian carcinoma. For purposes of comparing treatment results among different institutions, the extent of the disease is usually expressed in terms of stages. The surgicopathologic staging system adopted by FIGO in 1985 is seen in Table 16.15.

### Management

In the presence of a fixed and solid pelvic mass or cul-de-sac deposits on clinical examination, or a complex multicystic mass with thick septae

TABLE 16.15 FIGO Staging for Carcinoma of the Ovary

Stage I	Growth limited to the ovaries.
Stage IA	Growth limited to one ovary; no ascites present containing malignant cells. No tumor on the external surface; capsule intact.
Stage IB	Growth limited to both ovaries; no ascites present containing malignant cells. No tumor on the external surfaces, capsules intact
Stage IC*	Tumor classified as either Stage IA or IB but with tumor on the surface of one or both ovaries, or with ruptured capsule(s), or with ascites containing malignant cells present or with positive peritoneal washings
Stage II	Growth involving one or both ovaries, with pelvic extension
Stage IIA	Extension and/or metastases to the uterus or tubes
Stage IIB	Extension to other pelvic tissues
Stage IIC*	Tumor either Stage IIA or IIB but with tumor on the surface of one or both ovaries, or with capsule(s) ruptured, or with ascites containing malignant cells present or with positive peritoneal washings
Stage III	Tumor involving one or both ovaries with peritoneal implants outside the pelvis or positive retroperitoneal or inguinal nodes. Superficial liver metastasis equals Stage III. Tumor is limited to the true pelvis but with histologically proven malignant extension to small bowel or omentum.
Stage IIIA	Tumors grossly limited to the true pelvis with negative nodes but with histologically confirmed microscopic seeding; nodes are negative.
Stage IIIB	Tumor of one or both ovaries with histologically confirmed implants on abdominal peritoneal surfaces, none exceeding 2 cm in diameter; nodes are negative.
Stage IIIC	Abdominal implants greater than 2 cm in diameter or positive retroperitoneal or inguinal nodes
Stage IV	Growth involving one or both ovaries, with distant metastases. If pleural effusion is present, there must be positive cytologic findings to classify a case as Stage IV. Parenchymal liver metastasis equals Stage IV.

\*Notes about the staging: To evaluate the impact on prognosis of the different criteria for classifying cases as Stage IC or IIC, it would be of value to know whether the rupture of the capsule was spontaneous or caused by the surgeon and if the source of malignant cells detected was peritoneal washing or ascites.

From the International Federation of Gynecology and Obstetrics: Annual report on the results of treatment in gynecological cancer. *Int J Gynecol Obstet* 1989;28:189–190; with permission.

and solid components on ultrasound scan, surgical exploration is mandatory. With suspected ovarian cancer, there are few treatment alternatives in early pregnancy. During the latter half of pregnancy, delay of surgery until fetal maturity must be considered, accepting the potential compromise in the maternal prognosis. It is recommended that surgery not be delayed, however. The possibility of preterm delivery should be anticipated; appropriate management involves consultation with a perinatologist and a neonatologist, and preoperative referral to a center where a neonatal intensive care unit and a gynecologic oncologist are available. The management of the pregnancy should be discussed preoperatively based on gestational age. This is particularly difficult when the diagnosis and the extent of surgery can be determined only intraoperatively. Pregnancy loss following a surgical procedure is lowest during the second trimester.

The use of laparoscopy in the management of adnexal masses during pregnancy has increased in

the past few years. Diagnostic laparoscopy has been used in the first trimester of pregnancy for the diagnosis and management of ectopic pregnancies and the evaluation of adnexal masses. Laparoscopy in the management of adnexal masses in pregnancy has been found to be efficacious and safe [311–313]. If laparoscopy is considered in a pregnant woman with an adnexal mass, the surgeon must be experienced and the surgery should be performed preferably in the second trimester. In addition, the mass should be mobile, accessible, and have the characteristics of a benign lesion.

Approximately 16 to 18 weeks of gestation is a judicious period for laparotomy both in terms of safety and elimination of functional ovarian cysts. This timing also permits sonographic evaluation to exclude major fetal anomalies. The outcome is influenced by the events leading up to the laparotomy; for example, acute ovarian accidents such as torsion or hemorrhage can affect the pregnancy adversely. Laparotomy for ovarian carcinoma is associated with

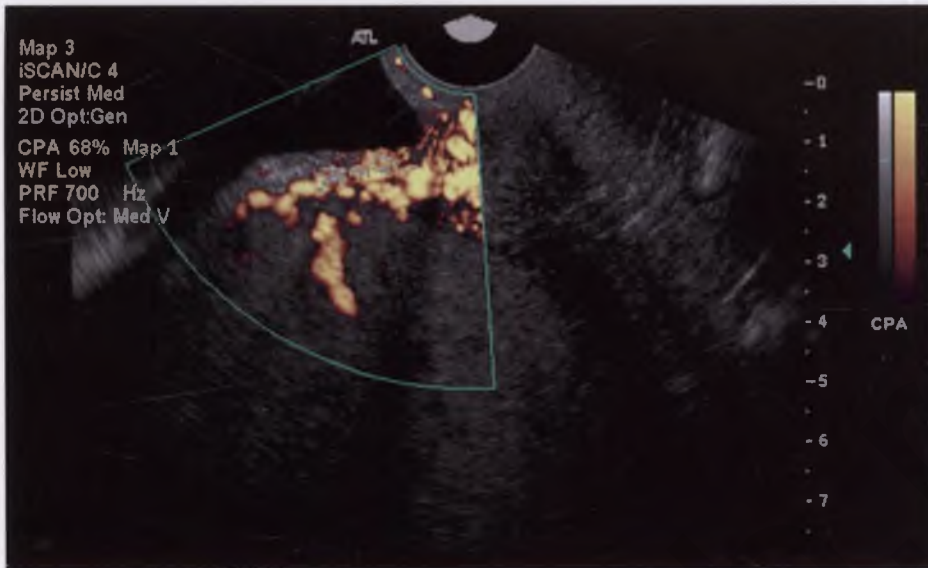


PLATE 1.  
*Ultrasound images of placenta accreta (transvaginal ultrasound, color Doppler).*

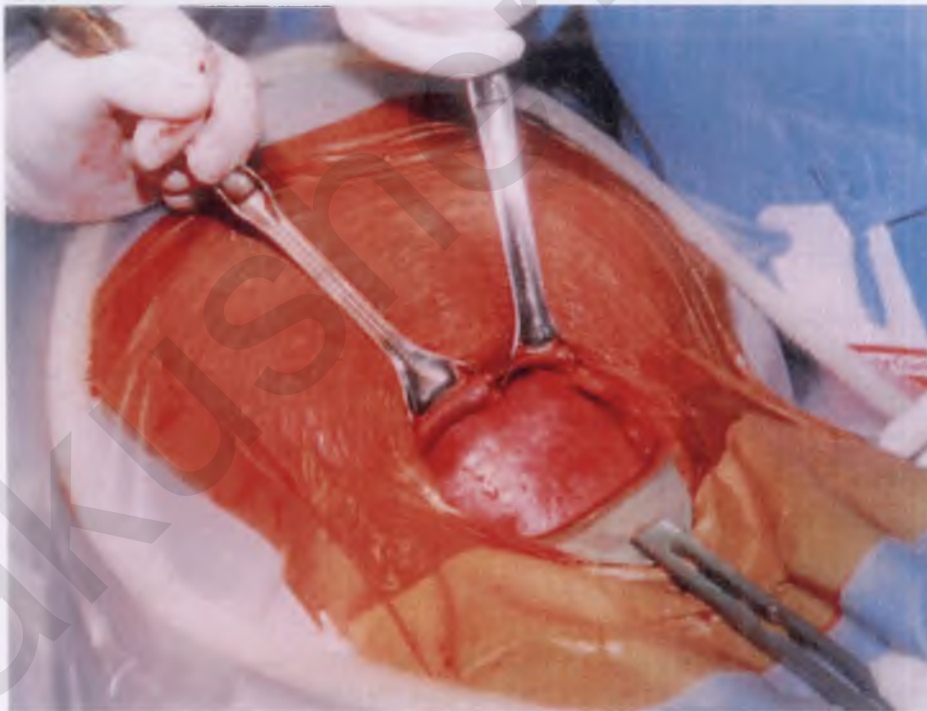
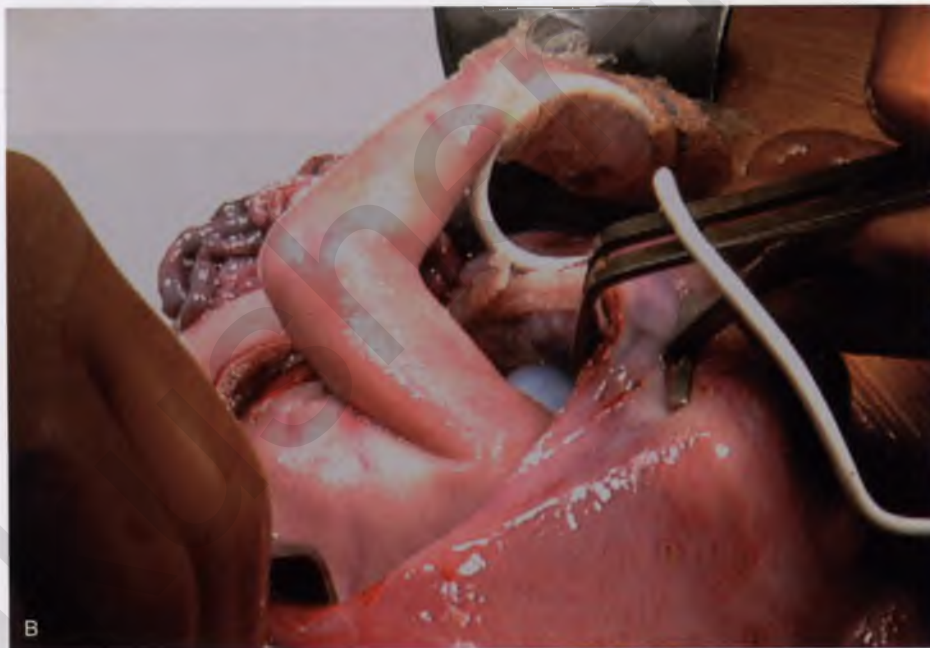
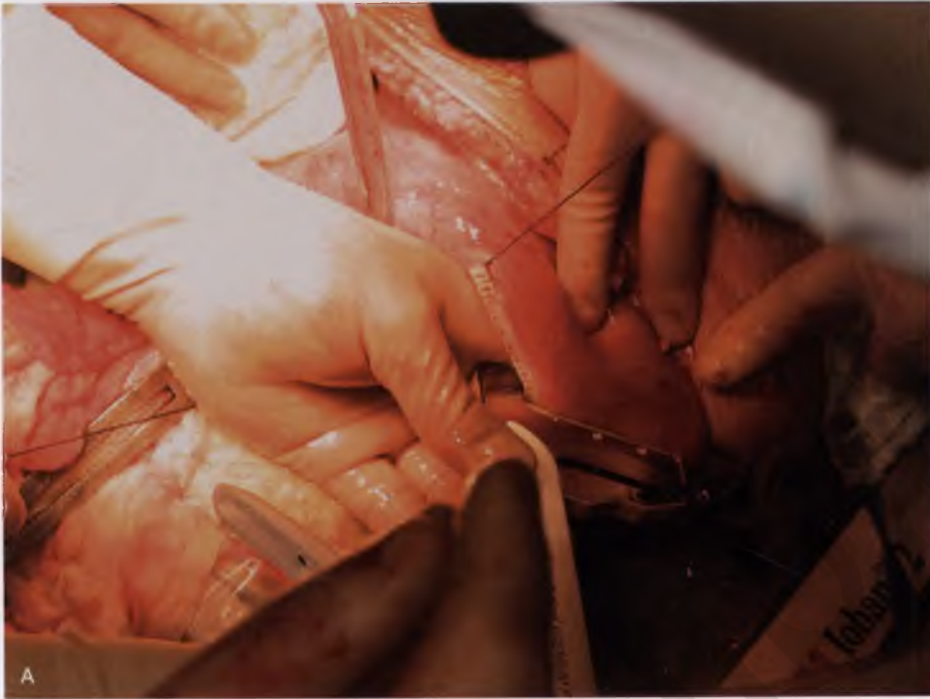


PLATE 2.  
*Without a Mobius retractor, adequate exposure requires two or more retractors and an assistant.*



**PLATE 3.**

*A, Mobius retractor results in good exposure without use of additional retractors. B, Subsequent fetal extraction is depicted.*



**PLATE 4.**

*A, In all open fetal surgery cases, a maternal hysterotomy is performed, using a specially designed uterine stapler device containing lactomer staples. B, Proper exposure of the fetal thorax in preparation for a left fetal thoractomy and lobectomy. A transcutaneous pulse oximeter is a useful adjunct for intraoperative fetal monitoring.*

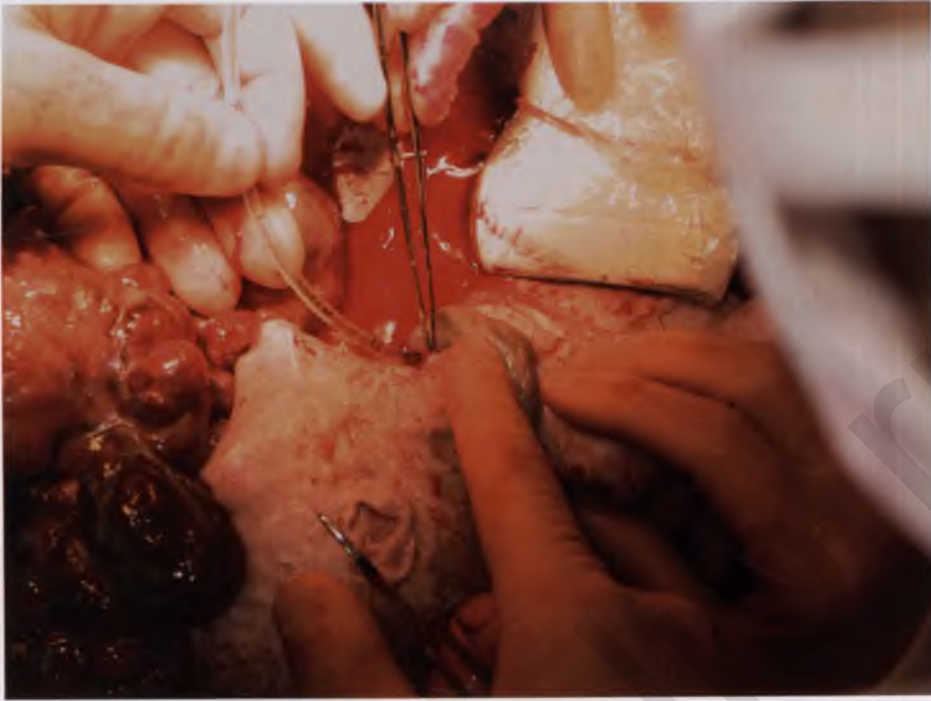




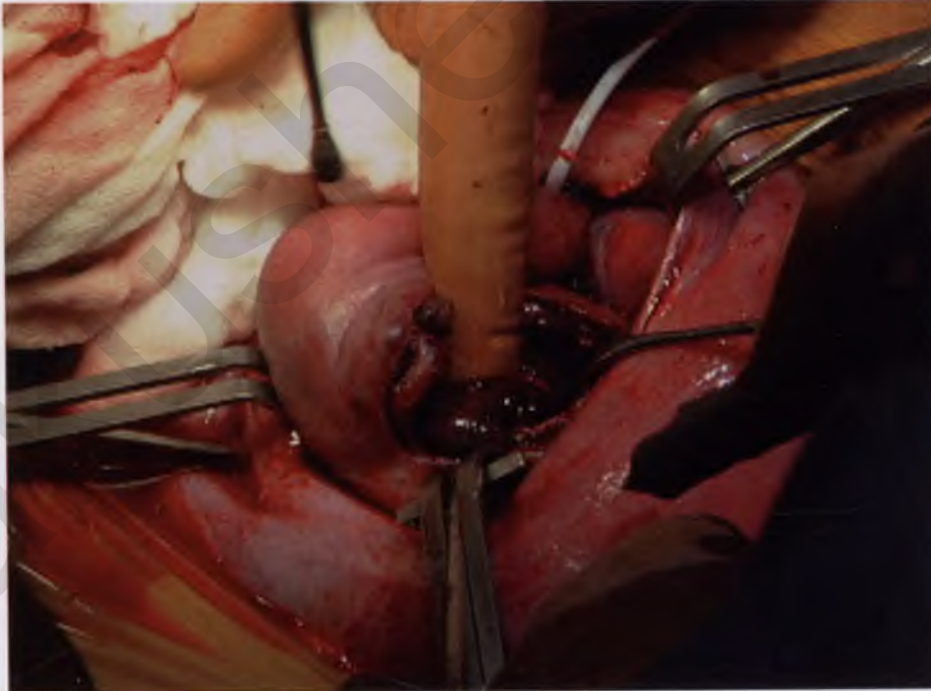
PLATE 5.  
*Fetoscopy after a maternal laparotomy.*



PLATE 6.  
*Percutaneous procedure under ultrasound guidance.*



**PLATE 7.**  
*A tracheostomy during the EXIT procedure, performed for massive airway obstruction.*



**PLATE 8.**  
*Fetal lobectomy for a cystic adenomatoid malformation.*

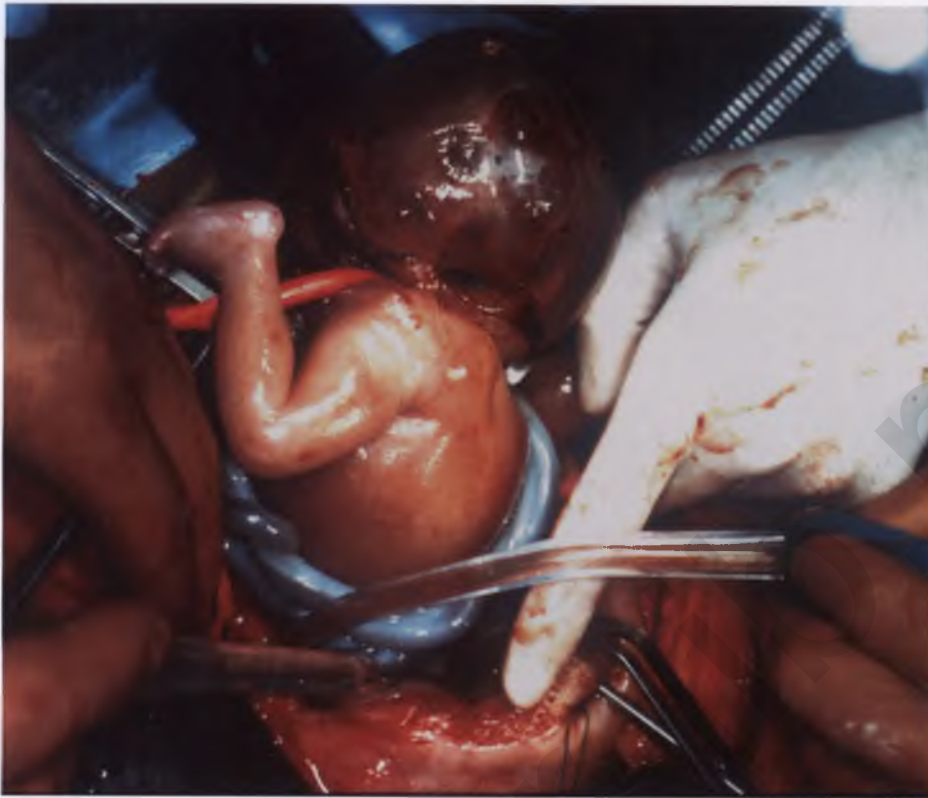


PLATE 9.  
*Sacrococcygeal teratoma prior to fetal resection.*



PLATE 10.  
*A simulated delivery used for medical student education.*



PLATE 11.

*A simulated perianal laceration is created with a beef tongue and turkey leg tendons. The tendons are inverted into the tongue to simulate the anal sphincter.*



PLATE 12.

*The goal of this shoulder dystocia simulator is to teach residents to deliver the posterior arm.*



PLATE 13.

*An inflatable uterus used to simulate uterine atony.*



PLATE 14.  
*Forceps or vacuum delivery can be simulated.*



PLATE 15.  
*A simulated vaginal breech delivery.*

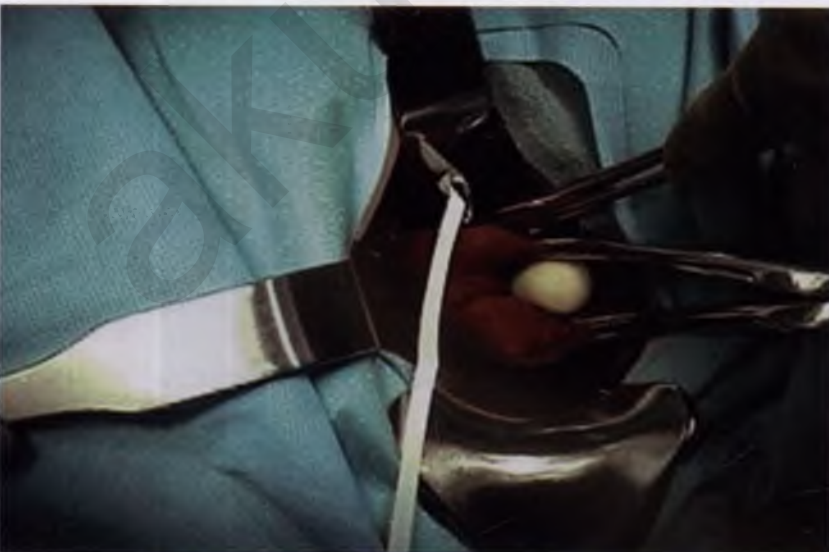


PLATE 16.  
*A simulation for cerclage placement.*

a significant risk of abortion in the first trimester, or premature labor during the third trimester.

Operative technique is similar to that used in the nonpregnant patient. Once a diagnosis of ovarian cancer is made, a full staging laparotomy is mandatory. A generous longitudinal incision is performed. All peritoneal surfaces are carefully inspected, and any rough areas or adhesions are excised. Ascitic fluid is collected for cytology; if no ascites is present, washings should be obtained for cytology. Multiple biopsies should be taken from the pelvic peritoneum and lateral gutters; the entire large and small bowel and their mesenteries are inspected. The stomach, pancreas, liver, and surface of the liver are palpated. Biopsies should be taken from the undersurface of the diaphragm. The appendix and the omentum are removed. These last three areas are of particular importance. The incidence of diaphragmatic metastases in apparent Stage I and Stage II disease has been reported at 11.4% and 23%, respectively, and the incidence of omental disease is 7% in combined Stage I and Stage II epithelial cancer of the ovary [314]. Studies by Sonnendecker [315], Malfetano [316], and Powell and coworkers [317] showed that 83%, 70%, and 63%, respectively, of Stage III and Stage IV patients with epithelial ovarian cancer had evidence of appendiceal metastasis.

Retroperitoneal lymphadenectomy should be included in the staging procedure to eliminate the possibility of more advanced disease. The incidence of positive pelvic and periaortic lymph nodes is approximately 8% and 10%, respectively, for Stage I and Stage II epithelial disease [318]. Evidence of disease in any of these areas represents Stage III disease, and treatment should be adjusted accordingly. Such complete assessment is imperative during the reproductive years, because conservation of a pregnancy and fertility can usually be considered only in Stage IA disease.

The traditional requirements for conservative management of Stage IA ovarian carcinoma are listed in Table 16.16. Unilateral salpingo-oophorectomy is the definitive treatment for young women of low parity found to have a well-differentiated serous, mucinous, endometrioid, or clear cell carcinoma of the ovary. The tumor must be unilateral, well encapsulated, free of adhesions, and not associated with ascites or extragonadal spread. Peritoneal washings for cytology should be taken from the pelvis and upper abdomen, and the oppo-

**TABLE 16.16 Requirements for Conservative Management of Epithelial Ovarian Cancer**

1. Stage IA
2. Well-differentiated tumor
3. A young woman of low parity
4. Otherwise normal pelvis
5. Encapsulated tumor, free of adhesions
6. No invasion of capsule, lymphatics or mesovarium
7. Peritoneal washings negative
8. Opposite ovary normal and omental biopsy negative
9. Close follow-up possible
10. Excision of residual ovary after completion of childbearing

*Modified from DiSaia PJ, Townsend PE, Morris CP: The rationale for less than radical treatment for gynecologic malignancy in early reproductive years. Obstet Gynecol Surv 1974;29:581-593.*

site ovary should be inspected and biopsied if suspicious in appearance. Munnell and others have calculated the incidence of microscopic metastasis in the opposite ovary to be approximately 12% [319]. The periaortic and pelvic wall lymph nodes must be carefully palpated and an adequate sample of the omentum taken for a biopsy. With the finding of carcinoma in any of these areas, conservative surgery is usually abandoned.

In the past, most authors recommended aggressive surgical treatment with sacrifice of the pregnancy; however, the definite surgical treatment for epithelial ovarian carcinoma must be individualized on the basis of surgical findings, gestational age, and the elective or emergent nature of the procedure. The patient's wishes in regard to continuation of the pregnancy should also be considered. As an example, two cases of Stage IIIC epithelial ovarian cancer have been reported in which conservative surgery consisting of ovarian cystectomy or oophorectomy plus partial omentectomy and peritoneal cytology at 15.5 and 16 weeks of gestation were followed by cyclophosphamide and cisplatin chemotherapy, and resulted in successful outcomes for mother and fetus [320,321]. The mothers underwent definitive surgery 4 and 6 weeks postpartum with residual papillary serous adenocarcinoma found in the remaining ovaries. Both infants developed normally without evidence of physical or mental impairment.

Routine palpation and direct examination of the adnexa at cesarean delivery are important.

Suspicious lesions found then can be biopsied or excised for histologic examination. Without histologic confirmation of malignancy and full reviews with the oncologist and the family, extensive surgery should not be performed at the time of delivery. It is best to review the clinical and histologic findings, consult with an oncologist, and plan a full staging laparotomy in the postpartum period.

### **Prognosis**

The prognosis for ovarian cancer is determined by the stage, the cell type, grade of the tumor, and the volume of residual tumor [322]. Whether other variables specific to pregnancy have an impact on the prognosis remains speculative. Increased blood flow to the pelvis, stimulation of gonadal stromal tumors by hCG, and the "immunosuppressed state of pregnancy" have been suggested but lack inherent supporting data.

The prognosis for borderline epithelial tumors is excellent, with 5- and 10-year survival rates of 90% to 100% with early-stage disease and 70% for advanced stages [323]. Invasive cancers have a less favorable prognosis, with a 5-year survival rate of 90% for adequately surgically evaluated Stage IA and IB, 65% for IC, 40% for Stage II, and less than 10% for Stages III and IV. Although the current approach of cytoreductive surgery combined with platinum-based chemotherapeutic regimens has improved survival rates at 2 years, the 5-year survival statistics have not improved significantly.

### **Management: Epithelial Cell Tumors**

Epithelial cell tumors represent the most common ovarian malignancies associated with pregnancy [323]. Most are tumors of low malignant potential or are Stage I. Conservative surgery is appropriate when conservation of fertility and pregnancy is desired, provided the tumor has been properly staged as IA, is well differentiated, and well encapsulated. Although there is debate over the need to biopsy the contralateral ovary, the high incidence of bilaterality in the serous morphologic type (20%–24%) compared with the mucinous type (3%) argues in favor of a biopsy of the opposite ovary in cases of serous tumors. The importance of adequate staging is again stressed even for patients with

tumors of low malignant potential, because 15% to 20% of these patients are found to have disease spread beyond the ovaries.

If the disease is greater than Stage IA or is histologically poorly differentiated, total hysterectomy with bilateral salpingo-oophorectomy, omentectomy, and tumor debulking are indicated, along with postoperative adjuvant therapy. In selected cases, hysterectomy can be deferred to allow completion of pregnancy. Patients who are treated conservatively need close follow-up, with the uterus and other adnexa removed when childbearing is completed.

### **Management: Benign Cystic Teratomas and Germ Cell Tumors**

Benign cystic teratomas (dermoid cysts), definitively the most common neoplastic cysts in pregnancy, are easily managed by simple cystectomy. The germ cell tumors – dysgerminoma, endodermal sinus tumor, immature teratoma, choriocarcinoma, embryonal carcinomas, and mixed types – occur predominantly in the second and third decade of life and therefore are common ovarian malignancies to coexist with pregnancy. Patients with these tumors often present with acute abdominal pain owing to rapid growth and a propensity for torsion. As a group, they are characteristically unilateral at diagnosis and usually can be managed with a unilateral salpingo-oophorectomy; they are usually Stage IA and the prognosis is not improved with more radical pelvic surgery [324].

### **Management: Dysgerminoma**

Dysgerminomas account for 30% of ovarian malignancies accompanying pregnancy and are unilateral in 85% of the cases. Obstetric complications and emergency surgical intervention are common in patients with dysgerminomas. Karlen and coworkers reviewed 27 cases of dysgerminoma associated with pregnancy [325]. Torsion and incarceration were common in these patients, who had rapidly enlarging neoplasms averaging 25 cm in diameter. Obstetric complications occurred in nearly one half of the patients and fetal demise in one of four of the reviewed cases. Unilateral salpingo-oophorectomy is adequate as long as the tumor is encapsulated and

the contralateral ovary is grossly normal and tumor free on biopsy. Ipsilateral pelvic lymphadenectomy and periaortic lymphadenectomy are especially important with this tumor owing to its propensity for lymphatic spread. Patients having Stage IC or higher disease should be treated with chemotherapy during or after completion of the pregnancy. Irradiation is an alternative for patients who have completed childbearing. Patients should be followed closely, particularly for the first 2 to 3 years, which is when 90% of recurrences present. DePalo and coworkers reviewed dysgerminomas and reported the 5-year relapse-free survival rate to be 91% for Stage I, 74% for Stage III disease by virtue of positive lymph nodes, and 24% for Stage III peritoneal disease [326].

### **Management: Endodermal Sinus Tumors**

Endodermal sinus tumor is a more aggressive malignancy than dysgerminoma and fortunately is rare during pregnancy, with only 15 cases reported in the literature up to 1991 [327–337]. Although this tumor grows rapidly and 50% of patients have symptoms for less than 1 week's duration, 50% to 70% are still Stage I at the time of presentation. Unilateral salpingo-oophorectomy is therefore adequate in many patients. In contrast to dysgerminoma, involvement of the contralateral ovary is not seen in the absence of widespread disease; hence, biopsy of the contralateral ovary is not indicated. Surgery alone, even in Stage I disease, is associated with significant recurrence rate and poor survival. Unlike dysgerminoma, these tumors are not radiation sensitive. Combination chemotherapy, including VAC, or vinblastine, bleomycin, and cis-platinum (VBP), or bleomycin, etoposide, and cisplatin (BEP), has greatly improved the prognosis. Whereas the disease was previously fatal, and 95% of the patients were dead within 2 years, the 2-year survival rate now approaches 100% for Stages I and II, and 50% for Stages III and IV disease [336–338]. Because of the rapid growth of this tumor, chemotherapy should be started within 2 weeks of surgery.

If the diagnosis of endodermal sinus tumor is made during the first or second trimester, the patient must decide whether to 1) permit the pregnancy to continue to viability before instituting adjuvant chemotherapy, 2) start chemotherapy with the fetus

in utero, or 3) terminate the pregnancy and start chemotherapy after the procedure. The patient with an endodermal sinus tumor, or the biologically similar embryonal carcinoma and immature teratoma, is faced with a choice for which little information is available.

Some case reports have described patients with endodermal sinus tumors who underwent conservative surgery between 13 and 20 weeks of pregnancy followed by two to six cycles of chemotherapy with the VAC regimen during pregnancy [329–331]. All babies and two of the three mothers had a good outcome; the third mother died of cancer 1 week postpartum.

Therapeutic decisions for patients with advanced stages of these highly malignant tumors are difficult and controversial. Many such patients are cured by early adjuvant chemotherapy after surgery, and thus delays in withholding chemotherapy are not warranted. As in earlier stages, the uterus and opposite ovary can be preserved if free of metastatic tumor. Some clinics are preserving the uterus and opposite ovary under all conditions in the hope that postoperative chemotherapy will sterilize those organs as well. No long-term follow-up of this approach is available. In many cases, patients request pregnancy termination for fear of potential teratogenic effects of chemotherapy.

### **Management: Rare Tumors**

There are few reports of ovarian immature teratomas, embryonal carcinomas, choriocarcinomas, or mixed germ cell tumors diagnosed during pregnancy. These tumors are frequently unilateral, and conservative surgery is appropriate. They are not radiation sensitive, and patients should be treated with postoperative chemotherapy, except for low-grade immature teratoma. The VAC regimen or a combination of etoposide and cisplatin is currently recommended [338–342].

### **Management: Gonadal Stromal Tumors**

Malignant gonadal stromal tumors are extremely rare during pregnancy. Granulosa cell tumors account for 3%, and Sertoli-Leydig cell tumors for less than 1% of reported cases of ovarian malignancy during pregnancy. These tumors are often initially



misdiagnosed in pregnancy. Young and coworkers identified four reasons for misdiagnosis: 1) the young age of the patients, 2) an alteration in the histologic appearance of the tumors during pregnancy owing to intracellular edema, 3) a decreased frequency of associated endocrine manifestations, and 4) pregnancy-induced changes in the ovary that simulate sex cord–stromal tumors morphologically and hormonally [343]. Of 36 cases in this series, 6 were unclassifiable sex cord–stromal tumors as a result of pregnancy-related changes. The actual incidence of these tumors during pregnancy is therefore uncertain.

These tumors frequently rupture during pregnancy. Of 36 cases reported by Young and coworkers, 10 (28%) ruptured before surgery, most commonly during or just after labor [343].

Because most gonadal stromal tumors are unilateral, conservation of the contralateral ovary and uterus is possible in patients desirous of preserving fertility after proper staging. Contralateral ovarian involvement is uncommon, and wedge biopsy is not recommended if the ovary appears grossly normal. Postpartum chemotherapy should be strongly considered in those patients with tumor rupture treated conservatively.

The prognosis for granulosa cell tumors is good for Stage I disease, but late recurrences are possible. The 10-year survival rate ranges from 86% to 96% for Stage I versus 26% to 49% for higher-stage tumors [343,344]. Rupture adversely affects prognosis, with an 86% 25-year survival rate for unruptured tumors versus 60% for ruptured Stage I tumors [345].

The prognosis in Sertoli-Leydig tumors is also difficult to determine [343,344,346]. Well-differentiated tumors generally behave benignly. Even those with poor or intermediate differentiation appear to have a good prognosis. It is recommended that they be managed conservatively in young nonpregnant patients, because these are neoplasms of low malignant potential.

## Fallopian Tube Disease

### *Incidence*

*Fallopian tube cancer* is the rarest of all gynecologic cancers, with an incidence of less than 1%. The mean age of women with cancer of the fallopian tube is 55

to 60 years and reports in pregnancy are extremely rare [347–349].

### *Pathology*

Fallopian tube cancer associated with pregnancy is usually unilateral and most often an adenocarcinoma [350]. Torsion of the fallopian tube usually occurs in a tube that is the site of the disease, such as tumor or pyosalpinx, but this can occur in a normal organ. Torsion is very uncommon during pregnancy [351,352]. Of 201 cases of fallopian tube torsion reported by Blum and Sayre [353], 12% occurred during pregnancy.

### *Diagnosis*

The clinical presentation of carcinoma of the fallopian tube is variable and nonspecific. The classic watery, blood-tinged vaginal discharge is not seen in pregnancy because at 12 weeks of gestation the communication between the uterine cavity and the fallopian tube is obliterated. In most instances, the diagnosis is established at incidental laparotomy. In instances of tubal torsion, acute lower abdominal pain with a palpable tender mass lateral to the uterus suggests this condition. Tubal torsion is almost always misdiagnosed as ovarian torsion.

### *Staging*

Staging of fallopian tube cancer is surgical. Fallopian tube cancer during pregnancy is staged using the same criteria as in nonpregnant women. Readers are referred to standard reference works [223].

### *Management*

The treatment for fallopian cancer is total abdominal hysterectomy with bilateral salpingo-oophorectomy, pelvic node and paraaortic node dissection, multiple peritoneal biopsies, and infracolic omentectomy [347,348]. The operative findings and the residual disease after surgery determine the use of postoperative radiation therapy or chemotherapy. Carcinoma in situ of the fallopian tube has been found in the portions of the tube submitted after postpartum tubal ligation [350]. In these unique cases, a total abdominal

hysterectomy with bilateral salpingo-oophorectomy is recommended; however, simple removal of the fallopian tubes can be a reasonable alternative.

For simple torsion of the fallopian tube, treatment is operative removal of the tube. The ovary should be preserved unless necrosis develops. Delay in diagnosis, inability to distinguish strangulation from necrosis, and fear of embolization have made adnexectomy the accepted method of management of adnexal torsion.

New techniques could assist in adnexal salvage, however. McHutchinson and coworkers [354] described the use of 5 ml of 10% fluorescein injected intravenously in cases of torsion. They observed the involved untwisted adnexa under ultraviolet light to determine tissue viability; 72% of their patients had preservation through the use of this technique. Detorsion of the tube might be attempted if there are no signs of infarction in cases of early diagnosis and intervention or with incomplete torsion. The pregnancy should be left undisturbed, and continuation to term is likely.

DiSaia and coworkers have also urged less than radical treatment for selected gynecologic malignancies among patients in the early reproductive years [355].

## LEGAL COMMENTARY

Medicolegal vulnerability for surgical complications is based on three basic theories. First, if the decision to do a procedure is improper, then the decision maker is responsible for all injuries and damages resulting from a surgical complication. Such liability arises regardless of whether the surgery itself was performed appropriately. Second, when the decision to perform surgery is appropriate, but substandard surgical technique causes a complication, liability is attached. Finally, when a complication arises in the absence of culpable conduct, liability nonetheless arises when further injury results from delayed or improper management of the complication. See also Chapter 21, Legal Commentary III and Appendix I, Appendix of Legal Principles.

### **Improper Surgical Recommendation**

A physician has the responsibility to exercise reasonable care in recommending a surgical procedure, meaning that there must be a reasonable basis in the

history, physical, or laboratory findings to warrant the recommendation. As in all medical considerations, the recommendation of surgery necessitates a risk-benefit analysis. The surgeon must write a note in the medical record documenting the decision-making process. Liability rarely attaches when the surgical recommendation requires the weighing of competing patient interests.

The decision to recommend a procedure is often complicated by pregnancy. In a patient with suspected carcinoma of the cervix, endocervical curettage that results in ruptured membranes can have medicolegal implications. The same is true with conization of the cervix. Although this procedure provides optimal diagnostic accuracy, its recommendation during pregnancy must be carefully considered.

An ancillary aspect of an appropriate surgical recommendation is the concept of informed consent. A patient is entitled to make an informed decision whether to undergo surgery. This decision requires knowledge of the potential complications of the procedure as well as the reasonable alternatives, including no surgery at all.

### ***Surgical Technique***

The rare frequency of a complication leads to the argument of preventability with appropriate surgical technique. The occurrence of a surgical complication does not automatically translate into legal liability, however. The patient must prove that the complication occurred because of the failure of reasonable surgical care. Such proof usually focuses on the failure of the surgeon to take some recognized precautionary measure – a measure intended to minimize the risk of the complication.

In areas of developing surgical techniques, the most significant area of legal exposure falls in the lag between actual practice and the establishment of accepted safeguards. During the infancy of a new procedure, more than one school of thought often exist about how to minimize the risk of a procedure. Under these circumstances, it is difficult to attach fault for allegiance to one school or another. As time passes, however, and more experiences are evaluated, the number of approaches dwindles, leaving what is considered the accepted standard of care. The failure to use what has evolved into the standard approach can result in liability. In the field of

developing surgical techniques, therefore, it is incumbent on the surgeon to keep abreast of journals and manufacturer's publications, as well as what is reported at local and national meetings.

### Improper Treatment of Complications

Finally, when a totally blameless complication arises but additional injury results from the failure to manage the complication in a timely and proper manner, a liability issue will arise. The surgeon must begin his or her vigilance while in the operating suite and before closing the surgical wound. Close observation should continue during the postoperative period, with appropriate examinations, tests, and instruction to the nursing and house staffs, all of which is documented.

The surgeon's duty, however, does not end at the threshold of the operating suite or even at the point of discharge from the hospital. The surgeon has a duty to instruct the patient appropriately on the necessary safeguards to avoid postoperative complications after discharge. From a medicolegal standpoint, failing to provide instructions to a patient who subsequently develops a postoperative infection will probably result in legal liability. The instructions should be provided to the patient in writing, not only to promote compliance but also to document the content of the instruction provided. At the time of discharge, there should be a notation on the record to evidence that the written instructions were provided.

Many surgeons choose to have a physician assistant, resident, or even a member of the nursing staff provide home-going instructions. Such delegation might not insulate the physician from ultimate responsibility for improperly given instructions, however. The person providing the instructions is considered an agent of the physician, or the responsibility of providing instruction is deemed a "nondelegatable" duty of the operating surgeon. Either conclusion pulls the operating surgeon back into the chain of culpability.

### REFERENCES

1. Beck WW: Intestinal obstruction in pregnancy. *Obstet Gynecol* 1974;43:374-378.
2. Sharp HT: The acute abdomen during pregnancy. *Clin Obstet Gynecol* 2002;45:405-413.
3. Spira IA, Rodrigues R, Wolff WI: Pseudo-obstruction of the colon. *Am J Gastroenterol* 1976;65:397-408.
4. Baer JL, Reis RA, Arens RA: Appendicitis in pregnancy with changes in positions in areas of normal appendix in pregnancy. *JAMA* 1932;98:1359-1364.
5. Baker DP: Trauma in the pregnant patient. *Surg Clin North Am* 1982;62:275-289.
6. Moise KJ, Belfort MA: Damage control for the obstetric patient. *Surg Clin North Am* 1997;77:835-852.
7. Fildes J, Reed L, Jones N, et al: Trauma: The leading cause of maternal death. *J Trauma* 1992;32:643-645.
8. Franger AL, Buchsbaum JH, Peaceman AM: Abdominal gunshot wounds in pregnancy. *Am J Obstet Gynecol* 1989;160:1124-1128.
9. Howard BK, Goodson JH, Menger WF: Supine hypotensive syndrome in late pregnancy. *Obstet Gynecol* 1953;1:371-376.
10. Pearlman MD, Tintinalli JE, Lorenz RP: A prospective controlled study of outcome after trauma during pregnancy. *Am J Obstet Gynecol* 1990;162:1502-1510.
11. Dahmus MA, Sibai BM: Blunt abdominal trauma: Are there predictive factors for abruptio placentae or maternal fetal distress? *Am J Obstet Gynecol* 1993;169:1054-1059.
12. Brent RL: The effect of embryonic and fetal exposure to x-ray, microwaves and ultrasound: Counseling the pregnant and nonpregnant about these risks. *Semin Oncol* 1989;16:347-368.
13. Oto A, Ernst RD, Shah R, et al: Right-lower-quadrant pain and suspected appendicitis in pregnant women: Evaluation with MR imaging - initial experience. *Radiology* 2005;234:445-451.
14. Birchard KR, Brown MA, Hyslop WB, et al: MRI of acute abdominal and pelvic pain in pregnant patients. *AJR Am J Roentgenol* 2005;184:452-458.
15. Tera H, Aberg C: Tensile strengths of twelve types of knots employed in surgery, using different suture materials. *Acta Chir Scand* 1976;142:1-7.
16. Hendrix SL, Schimp V, Martin J, et al: The legendary superior strength of the Pfannenstiel incision: A myth? *Am J Obstet Gynecol* 2000;182:1446-1451.
17. Ellis H, Coleridge-Smith PD, Joyce AD: Abdominal incisions: Vertical or transverse? *Postgrad Med J* 1984;60:407-410.

18. Edgerton MT: In: *The Art of Surgical Technique*. Baltimore: Williams & Wilkins, 1988; pp. 1–21.
19. Hasselgren PO, Hagberg E, Malmer H, et al: One instead of two knives for surgical incision: Does it increase the risk of postoperative wound infection? *Arch Surg* 1984;119:917–920.
20. Ramon R, Garcia S, Combalia A, et al: Bacteriological study of surgical knives: Is the use of two blades necessary? *Arch Orthop Trauma Surg* 1994; 113:157–158.
21. Fairclough JA, Mackie IG, Mintowt-Czyz W, et al: The contaminated skin-knife: A surgical myth. *J Bone Joint Surg* 1983;65:210.
22. Cruse PJE, Foord R: The epidemiology of wound infection: A 10-year prospective study of 62,939 wounds. *Surg Clin North Am* 1980;60:27–40.
23. Johnson CD, Serpell JW: Wound infection after abdominal incision with scalpel or diathermy. *Br J Surg* 1990;77:626–627.
24. Franchi M, Ghezzi F, Benedetti-Panici PL, et al: A multicentre collaborative study on the use of cold scalpel and electrocautery for midline abdominal incision. *Am J Surg* 2001;81:128–321.
25. Edlich RF, Rodeheaver GT, Thacker JG: Technical factors in the prevention of wound infections. In: Simmons RL, Howard RJ (eds): *Surgical Infectious Diseases*. Norwalk, CT: Appleton & Lange, 1988; p. 340.
26. Cruse PJE, Foord R: A five-year prospective study of 23,649 surgical wounds. *Arch Surg* 1973;107:206–210.
27. Mangram AJ, Horan TC, Pearson ML, et al: Guideline for Prevention of Surgical Site Infection, 1999: Centers for Disease Control and Prevention (CDC) Hospital Infection Control Practices Advisory Committee. *Am J Infect Control* 1999;27:97–132.
28. Buckman RF, Buckman PD, Hufnagel HV, et al: A physiologic basis for the adhesion-free healing of deperitonealized surfaces. *J Surg Res* 1976;21:67–76.
29. Tulandi T, Hum HS, Gelfand MM: Closure of laparotomy incisions with or without peritoneal suturing and second-look laparoscopy. *Am J Obstet Gynecol* 1988;158:536–537.
30. Duffy DM, diZerega GS: Is peritoneal closure necessary? *Obstet Gynecol Surv* 1994;12:817–822.
31. Edlich RF, Tsung MS, Rogers W, et al: Studies in the management of the contaminated wound: I. Technique of closure of such wounds together with a note on a reproducible model. *J Surg Res* 1968;8: 585–592.
32. Stone IK, von Fraunhofer JA, Masterson BJ: The biomechanical effects of tight suture closure upon fascia. *Surg Gynecol Obstet* 1986;163:448–452.
33. Israelsson LA, Jonsson T: Suture length to wound length ratio and healing of midline laparotomy incisions. *Br J Surg* 1993;80:1284–1286.
34. Fagniez PL, Hay JM, Lacaine F, et al: Abdominal mid-line incision closure: A multicentric randomized prospective trial of 3,135 patients, comparing continuous versus interrupted polyglycolic acid sutures. *Arch Surg* 1985;120:1351–1353.
35. Orr JW Jr, Orr PF, Barrett JM, et al: Continuous or interrupted fascial closure: A prospective evaluation of No. 1 Maxon suture in 402 gynecologic procedures. Part I. *Am J Obstet Gynecol* 1990;163: 1485–1489.
36. Hodgson NC, Malthaner RA, Ostbye T: The search for an ideal method of abdominal fascial closure: A meta-analysis. *Ann Surg* 2000;231:436–442.
37. deHoll D, Rodeheaver GT, Edgerton MT, et al: Potentiation of infection by suture closure of dead space. *Am J Surg* 1974;127:716–720.
38. Laufman H, Rubel T: Synthetic absorbable sutures. *Surg Gynecol Obstet* 1977;145:597–608.
39. Orr JW Jr: Sutures and closures: An update. *Ala J Med Sci* 1986;23:36–41.
40. van Rijssel EJC, Trimbos JB, Booster MH: Mechanical performance of square knots and sliding knots in surgery: A comparative study. *Am J Obstet Gynecol* 1990;162:93–97.
41. Trimbos JB, van Rijssel EJC, Klopper PJ: Performance of sliding knots in monofilament and multifilament suture material. *Am J Obstet Gynecol* 1986;68:425–430.
42. Qureshi A, Drew PJ, Duthie GS, et al: n-Butyl cyanoacrylate adhesive for skin closure of abdominal wounds: Preliminary results. *Ann R Coll Surgeons Engl* 1997;79:414–415.
43. Chen HH, Tsai WAS, Yeh CY, et al: Prospective study comparing wounds closed with tape with sutured wounds in colorectal surgery. *Arch Surg* 2001;136:801–803.
44. Edlich RF, Becker DG, Thacker JG, et al: Scientific basis for selecting staple and skin tape closures. *Clin Plast Surg* 1990;17:571–581.
45. Ranaboldo CJ, Rowe-Jones DC: Closure of laparotomy wounds: Skin staples versus sutures. *Br J Surg* 1992;79:1172–1173.

46. Frishman GN, Schwartz T, Hogan JW: Closure of Pfannenstiel skin incisions: Staples vs. subcuticular suture. *J Reprod Med* 1997;42:627-630.
47. Rush BF: Principles of operative surgery: Antisepsis, asepsis, technique, sutures and drains. In: Sabiston DC (ed): *Essentials of Surgery*. Philadelphia: WB Saunders, 1987; pp. 138-140.
48. Magee C, Rodeheaver GT, Golden GT, et al: Potentiation of wound infection by surgical drains. *Am J Surg* 1976;131:547-549.
49. Orr JW Jr, Hatch KD, Shingleton HM, et al: Gastrointestinal complications associated with pelvic exenteration. *Am J Obstet Gynecol* 1983;145:325-332.
50. Farrell MB, Worthington-Self S, Mucha P, et al: Closure of abdominal incisions with subcutaneous catheters. *Arch Surg* 1986;121:641-648.
51. Pearl ML, Valea FA, Chalas E: Panniculectomy and supraumbilical vertical midline incisions in morbidly obese gynecologic oncology patients. *J Am Coll Surg* 1998;186:649-653.
52. Gallup DG, Nolan TE, Smith RP: Primary mass closure of midline incisions with continuous polyglyconate monofilament absorbable suture. *Obstet Gynecol* 1990;76:872-875.
53. Devlin HB: Incisional hernia. In: Dudley H, Carter D, Russell RCG (eds): *Atlas of General Surgery*. London: Butterworth's, 1986; pp. 662-674.
54. Hemsell DL: Prophylactic antibiotics in gynecologic and obstetric surgery. *Rev Infect Dis* 1991; 13(Suppl10):S821-S841.
55. Cruse PJE: Wound infections: Epidemiology and clinical characteristics. In: Simmons RL, Howard RJ (eds): *Surgical Infectious Diseases*. Norwalk, CT: Appleton & Lange, 1988; pp. 327.
56. Robson MC, Lea CE, Dalton JB, et al: Quantitative bacteriology and delayed wound closure. *Surg Forum* 1968;19:501-502.
57. Simmons RL, Howard RJ (eds): Regional surgical infections. In: *Surgical Infectious Diseases*. Norwalk, CT: Appleton & Lange, 1988; pp. 404-407.
58. Stephenson H, Dotters DJ, Katz V, et al: Necrotizing fasciitis of the vulva. *Am J Obstet Gynecol* 1992;166:1324-1327.
59. Riseman JA, Zamboni WA, Curtis A, et al: Hyperbaric oxygen therapy for necrotizing fasciitis reduces mortality and the need for débridement. *Surgery* 1990;108:847-850.
60. Schulak JA, Corry RJ: Surgical complications. In: Sabiston DC (ed): *Essentials of Surgery*. Philadelphia: WB Saunders, 1987; pp. 201-204.
61. National Institutes of Health Consensus Development Conference: Prevention of venous thrombosis and pulmonary embolism. *JAMA* 1986;256:744-749.
62. American College of Obstetricians and Gynecologists: Deep vein thrombosis and pulmonary embolism. In: *Précis: An update in obstetrics and gynecology*. Obstetrics 3rd ed. 2005: 74-80.
63. Lockwood CJ: Inherited thrombophilias in pregnant patients: Detection and treatment paradigm. *Obstet Gynecol* 2002;99:333-341.
64. Macklon NS, Greer IS, Bowman A: An ultrasound study of gestational and postural changes in the deep venous system of the leg in pregnancy. *Br J Obstet Gynecol* 1997;104:191-197.
65. McColl MD, Ramsay JE, Trait RC, et al: Risk factors for pregnancy-associated venous thromboembolism. *Thromb Haemost* 1997;78:1183-1188.
66. Ros HS, Lichtenstein P, Belloco R, et al: Pulmonary embolism and stroke in relation to pregnancy: How can high-risk women be identified? *Am J Obstet Gynecol* 2002;186:198-203.
67. Girling JC, de Swiet M: Thromboembolism in pregnancy: An overview. *Current Opin Obstet Gynecol* 1996;8:458-463.
68. Levine JF, Branch DW, Rauch J: The antiphospholipid syndrome. *N Engl J Med* 2002;346:752-63.
69. Kakkar VV, Corrigan TP, Fossard DP, et al: Prevention of fatal postoperative pulmonary embolism by low doses of heparin: Reappraisal of results of international multicenter trial. *Lancet* 1977;1:567-569.
70. Clarke-Pearson DL, Coleman RE, Synan RS, et al: Venous thromboembolism prophylaxis in gynecologic oncology: A prospective controlled trial of low-dose heparin. *Am J Obstet Gynecol* 1983;145: 606-613.
71. Dahlman TC, Hellgren MSE, Blomback N: Thrombosis prophylaxis in pregnancy with use of subcutaneous heparin adjusted by monitoring heparin concentration in plasma. *Am J Obstet Gynecol* 1989;161:420-425.
72. Hull RD, Raskob GE, Garb M, et al: Effectiveness of intermittent leg compression for preventing deep vein thrombosis after total hip replacement. *JAMA* 1990;63:2313-2317.

73. Douketis JD, Ginsberg JS: Diagnostic problems with venous thromboembolic disease in pregnancy. *Haemostasis* 1995;25:58-71.
74. Hyers TM: Efficient workup and treatment for pulmonary thromboembolism. *Contemp Intern Med* 1990:13-24.
75. Dizon-Townson D: Pregnancy-related venous thromboembolism. *Clin Obstet Gynecol* 2002;45:363-368.
76. Winer-Muran HI, Boone JM, Brown HL, et al: Pulmonary embolism in pregnant patients: Fetal radiation dose with helical CT. *Radiology* 2002;224:487-492.
77. Ginsberg JS, Hirsh J, Turner DC, et al: Risks to the fetus of anticoagulant therapy during pregnancy. *Thromb Haemost* 1989;61:197-203.
78. Hall JHE, Paul RM, Wilson KM: Maternal and fetal sequelae of anticoagulation during pregnancy. *Am J Med* 1980;68:122-140.
79. Chong BH: Heparin-induced thrombocytopenia. *Aust NZ J Med* 1992;22:145-152.
80. Dahlman TC: Osteoporotic fractures and the recurrence of thromboembolism during pregnancy and the puerperium in 184 women undergoing thromboprophylaxis with heparin. *Am J Obstet Gynecol* 1993;168:12:1265-1270.
81. Chan WS, Ray JG: Low molecular weight heparin use during pregnancy: Issues of safety and practicality. *Obstet Gynecol Surv* 1999;54:649-654.
82. American College of Obstetricians and Gynecologists: Thromboembolism in pregnancy. ACOG Practice Bulletin No 19. Washington, DC: American College of Obstetricians and Gynecologists, 2000.
83. American Society of Regional Anesthesia: Recommendations for neuraxial anesthesia and anticoagulation. Richmond, VA: American Society of Regional Anesthesia, 1998.
84. Kearon C, Gent M, Hirsh J, et al: A comparison of three months of anticoagulation with extended anticoagulation for a first episode of idiopathic venous thromboembolism. *N Engl J Med* 1999;340:901-907.
85. Reimold SC, Rutherford JD: Valvular heart disease in pregnancy. *N Engl J Med* 2003;52-59.
86. Chan WS, Anand S, Ginsberg JS: Anticoagulation of pregnant women with mechanical heart valves: A systematic review of the literature. *Arch Intern Med* 2000;160:191-196.
87. Walker ID, Greaves M, Preston FE: Guideline investigation and management of heritable thrombophilia. *Br J Haematol* 2001;114:512-528.
88. Greer IA: The challenge of thrombophilia in maternal-fetal medicine. *N Engl J Med* 2000;342:424-425.
89. Ginsberg JS, Hirsh J: Use of antithrombotic agents during pregnancy. *Chest* 1998;114:524S-530S.
90. Barbour LA, Pickard J: Controversies in thromboembolic disease during pregnancy: A critical review. *Obstet Gynecol* 1995;86:621-633.
91. Krebs HB: Intestinal injury in gynecologic surgery: A ten-year experience. *Am J Obstet Gynecol* 1986;155:509-514.
92. Kracht M, Hay JM, Fagniez PL, Fingerhut A: Ileocolonic anastomosis after right hemicolectomy for carcinoma: Staple or hand-sewn? A prospective multi-centered randomized trial. *Int J Colorectal Dis* 1993;8:29-33.
93. Thomson JPS: LOOP colostomy. In: Dudley H, Carter DC, Russell CG (eds): *Atlas of General Surgery*. London: Butterworth's, 1986; pp. 458-461.
94. Alvarez RD: Gastrointestinal complications in gynecologic surgery: A review for the general gynecologist. *Obstet Gynecol* 1988;72:533-540.
95. Pitkin RM: Morphologic changes in pregnancy. In: Buchsbaum H, Schmidt J (eds): *Gynecologic and Obstetric Urology*. Philadelphia: WB Saunders, 1982; p. 476.
96. Symmonds RE: Ureteral injuries associated with gynecologic surgery: Prevention and management. *Clin Obstet Gynecol* 1976;19:623-644.
97. Wheelock JB, Krebs HB, Hurt WG: Sparing and repairing the bladder during gynecologic surgery. *Surg Tech Op* 1984;163-170.
98. Mattingly RF (ed): Anatomy of the female pelvis. In: *TeLinde's Operative Gynecology*. Philadelphia: JB Lippincott, 1977; pp. 34-36.
99. Donaldson JO: Neuropathy. In: *Neurology of Pregnancy*. Philadelphia: WB Saunders, 1978; pp. 47-48.
100. Low JA, Mauger GM, Carmichael JA: The effect of Wertheim hysterectomy upon bladder and urethral function. *Am J Obstet Gynecol* 1981;139:826-834.
101. Dessole S, Cosmi E, Balata A, et al: Accidental fetal lacerations during cesarean delivery: Experience in an Italian level III university hospital. *Am J Obstet Gynecol* 2004;191:1673-1677.

102. Smith JF, Hernandez C, Wax JR: Fetal laceration injury at cesarean delivery. *Obstet Gynecol* 1997; 90:344-346.
103. Weiner JJ, Westwood J: Fetal lacerations at cesarean section. *J Obstet Gynecol* 2002;22:23-24.
104. Diehl AK, Stern MP, Ostrower VS, et al: Prevalence of clinical gallbladder disease in Mexican-American, Anglo, and black women. *South Med J* 1980;73:438-443.
105. Kern F Jr: Epidemiology and natural history of gallstones. *Semin Liver Dis* 1983;3:87-96.
106. Lanzafame RJ: Laparoscopic cholecystectomy during pregnancy. *Surgery* 1995;118:627-633.
107. McKellar DR, Anderson CT, Boynton CJ, et al: Cholecystectomy during pregnancy without fetal loss. *Surg Gynecol Obstet* 1992;174:465-468.
108. Coleman MT, Trianfo VA, Rund DA: Nonobstetric emergencies in pregnancy: Trauma and surgical conditions. *Am J Obstet Gynecol* 1997;177:497-502.
109. Epstein FB: Acute abdominal pain in pregnancy. *Emerg Med Clin North Am* 1994;12:151-165.
110. Landers D, Carmona R, Crombleholme W, et al: Acute cholecystitis in pregnancy. *Obstet Gynecol* 1987;69:131-133.
111. Jamidar PA, Beck FJ, Hoffman BJ, et al: Endoscopic retrograde cholangiopancreatography in pregnancy. *Am J Gastroenterol* 1995;90:1263-1267.
112. Axelrad AM, Fleischer DE, Strack LL, et al: Performance of ERCP for symptomatic choledocholithiasis during pregnancy: Techniques to increase safety and improve patient management. *Am J Gastroenterol* 1994;89:109-112.
113. Lu EJ, Curet MJ, El-Sayed YY, et al: Medical versus surgical management of biliary tract disease in pregnancy. *Am J Surg* 2004;188:755-759.
114. Dixon NP, Faddis DM, Silberman H: Aggressive management of cholecystitis during pregnancy. *Am J Surg* 1987;154:292-294.
115. Swisher SG, Schmit PJ, Hunt KK, et al: Biliary disease during pregnancy. *Am J Surg* 1994;168:576-581.
116. Lee S, Bradley JP, Mele MM, et al: Cholelithiasis in pregnancy: Surgical versus medical management. *Obstet Gynecol* 2000;95:S70-S71.
117. Simon JA: Biliary tract disease and related surgical disorders during pregnancy. *Clin Obstet Gynecol* 1983;26:810-821.
118. Ghumman E, Barry M, Grace PA: Management of gallstones in pregnancy. *Br J Surg* 1997;84:1646-1650.
119. Perisset J, Collat D, Belliard R: Gallstones: Laparoscopic treatment – cholecystectomy, cholecystotomy, and lithotripsy. *Surg Endosc* 1990;4:1-5.
120. Graham G, Baxi L, Tharakan T: Laparoscopic cholecystectomy during pregnancy: A case series and review of the literature. *Obstet Gynecol Surv* 1998; 53:566-574.
121. Barone JE, Bears S, Chen S, et al: Outcome study of cholecystectomy during pregnancy. *Am J Surg* 1999;177:232-236.
122. Pucci RD, Seed RW: Case report of laparoscopic cholecystectomy in the third trimester of pregnancy. *Am J Obstet Gynecol* 1991;165:401-402.
123. Morrell DG, Mullins JR, Harrison PB: Laparoscopic cholecystectomy during pregnancy in symptomatic patients. *Surgery* 1992;112:856-859.
124. Arvidsson D, Gerdin E: Laparoscopic cholecystectomy during pregnancy. *Surg Laparosc Endosc* 1991;1:193-194.
125. Soper NJ: Laparoscopic cholecystectomy. *Curr Probl Surg* 1991;28:587-655.
126. Rollins MD, Chan KJ, Price RR: Laparoscopy for appendicitis and cholelithiasis during pregnancy: A new standard of care. *Surg Endosc* 2004;18:237-241.
127. Williams GR: Presidential address: A history of appendicitis. *Ann Surg* 1983;197:495-506.
128. Hancock H: Disease of the appendix caeci cured by operation. *Lancet* 1848;2:380-382.
129. Black WP: Acute appendicitis in pregnancy. *Br Med J* 1960;1:1938-1941.
130. Babaknia A, Parsa H, Woodruff JD: Appendicitis during pregnancy. *Obstet Gynecol* 1977;50:40-44.
131. Tamir IL: Acute appendicitis in the pregnant patient. *Am J Surg* 1990;160:571-576.
132. Mazze RI, Kallen B: Appendectomy during pregnancy: A Swedish registry study of 778 cases. *Obstet Gynecol* 1991;77:835-840.
133. Warfield GL: Acute appendicitis complicating pregnancy. *Postgrad Med* 1950;8:10-14.
134. Mahmoodian S: Appendicitis complicating pregnancy. *South Med J* 1992;85:19-24.
135. Cohen-Kerem R, Railton C, Oren D, et al: Pregnancy outcome following non-obstetric surgical intervention. *Am J Surg* 2005;190:467-473.
136. Cope Z (ed): The differential diagnosis of appendicitis. In: *The Early Diagnosis of the Acute Abdomen*. London: Oxford University Press, 1963; pp. 59-75.

137. DeVore GR: Acute abdominal pain in the pregnant patient due to pancreatitis, acute appendicitis, cholecystitis, or peptic ulcer disease. *Clin Perinatol* 1980;7:349–369.
138. Cunningham GF, McGubbin JH: Appendicitis complicating pregnancy. *Obstet Gynecol* 1975;45: 415–420.
139. Lim HK, Bae SH, Seo GS: Diagnosis of acute appendicitis in pregnant women: Value of sonography. *Am J Roentgenol* 1992;159:539–542.
140. Ames Castro M, Shipp TD, Castro EE, et al: The use of helical computed tomography in pregnancy for the diagnosis of acute appendicitis. *Am J Obstet Gynecol* 2001;184:954–957.
141. Cobben JP, Groot I, Haans L, et al: MRI for clinically suspected appendicitis during pregnancy. *AJR Am J Roentgenol* 2004;183:671–675.
142. Fatum M, Rojansky N: Laparoscopic surgery during pregnancy. *Obstet Gynecol Survey* 2001;56:50–59.
143. Al-Fozan H, Tulandi T: Safety and risks of laparoscopy in pregnancy. *Curr Opin Obstet Gynecol* 2002;14:375–379.
144. Barnes SL, Shane MD, Schoemann MB, et al: Laparoscopic appendectomy after 30 weeks pregnancy: Report of two cases and description of technique. *Am Surg* 2004;70:733–736.
145. Larsson G: Elective appendectomy at time of cesarean section. *JAMA* 1954;154:549–552.
146. Sweeney WA III: Incidental appendectomy at the time of cesarean section. *Obstet Gynecol* 1959;14: 528–592.
147. Douglas RG, Stromme W: *Operative Obstetrics*, 3rd ed. New York: Appleton-Century-Crofts, 1976.
148. Wilson EA, Dilts PV Jr, Simpson TJ: Appendectomy incidental to postpartum sterilization. *Am J Obstet Gynecol* 1973;116:76–81.
149. Parsons AK, Sauer MV, Parsons MT, et al: Appendectomy at cesarean section: A prospective study. *Obstet Gynecol* 1986;479–482.
150. Gilstrap LC III: Elective appendectomy during abdominal surgery. *JAMA* 1991;265:1736.
151. Ober WB, Fass RD: The early history of choriocarcinoma. *J Hist Med Allied Sci* 1961;16:479–482.
152. DiSaia PJ, Creasman WT (eds): *Gestational trophoblastic neoplasia*. In: *Clinical Gynecologic Oncology*, ed 6. St. Louis: Mosby-Year Book, 2002; pp. 185–210.
153. Li M, Hertz R, Spencer DB: Effects of methotrexate therapy upon choriocarcinoma and chorioadenoma. *Proc Soc Exp Biol Med* 1956;93:361–366.
154. Berkowitz RS, Goldstein DP: Gestational trophoblastic diseases. In Hoskins WJ, Perez CA, Young RC (eds): *Principles and Practice of Gynecologic Oncology*, ed 3. Philadelphia (PA): Lippincott-Williams & Wilkins, 2000; pp. 1117–1137.
155. Lurain JR, Brewer JI, Torok EE, et al: Natural history of hydatidiform mole after primary evacuation. *Am J Obstet Gynecol* 1983;145:591–595.
156. Curry SL, Hammond CB, Tyrey L, et al: Hydatidiform mole: Diagnosis, management, and long-term follow up of 347 patients. *Obstet Gynecol* 1975;45:1–8.
157. Berkowitz RS, Goldstein DP, Bernstein MR: Evolving concepts of molar pregnancy. *J Reprod Med* 1991;36:40–44.
158. Berkowitz RS, Im SS, Bernstein MR, et al: Gestational trophoblastic disease: Subsequent pregnancy outcome, including repeat molar pregnancy. *J Reprod Med* 1998;43:81–86.
159. Sand PK, Lurain JR, Brewer JI: Repeat gestational trophoblastic disease. *Obstet Gynecol* 1984; 63:140–144.
160. Soper JT, Lewis JL Jr, Hammond CB: Gestational trophoblastic disease. In: Hoskins WJ, Perez CA, Young RC (eds). *Principles and Practice of Gynecologic Oncology*, ed 2. Philadelphia, Lippincott-Raven, 1997; pp. 1039–1077.
161. Berkowitz RS, Goldstein DR, Bernstein MR: Natural history of partial molar pregnancy. *Obstet Gynecol* 1985;66:677–681.
162. Szulman AE, Ma HK, Wong LC, et al: Residual trophoblastic disease in association with partial hydatidiform mole. *Obstet Gynecol* 1981;45:1–8.
163. Morrow P, Nakamura R, Schlaerth J, et al: The influence of oral contraceptives on the postmolar human chorionic gonadotropin regression curve. *Am J Obstet Gynecol* 1985;151:906–914.
164. Lurain JR, Brewer JI, Torok EE, et al: Natural history of hydatiform mole after primary evacuation. *Am J Obstet Gynecol* 1983;145:591–595.
165. Kohorn EI: The new FIGO 2000 staging and risk factor scoring system for gestational trophoblastic disease: Description and clinical assessment. *Int J Gynecol Cancer* 2001;11:73–77.
166. Hammond CB, Borchet LG, Tyrey L, et al: Treatment of metastatic trophoblastic disease: Good and poor prognosis. *Am J Obstet Gynecol* 1973;115: 451–457.
167. World Health Organization Scientific Group: *Gestational Trophoblastic Disease*. Technical Report



- Series No. 692. Geneva: World Health Organization, 1983.
168. Bagshawe KD: Risk and prognostic factors in trophoblastic neoplasia. *Cancer* 1976;38:1373-1385.
  169. Song H, Wu B, Tang M, et al: A staging system of gestational trophoblastic neoplasms based on the development of the disease. *Chin Med J* 1984;97:557-566.
  170. Pettersson F, Kolstad P, Ludwig H, et al (eds): Annual Report on the Results of Treatment in Gynecologic Cancer, Volume 19. Stockholm: International Federation of Gynecology and Obstetrics, 1985.
  171. Goldstein DRP, Berkowitz RS: Nonmetastatic and low risk metastatic gestational trophoblastic neoplasms. *Semin Oncol* 1982;9:191-197.
  172. Osathanondh R, Goldstein DP, Pastorfide GB: Actinomycin D as the primary agent for gestational trophoblastic disease. *Cancer* 1975;36:863-866.
  173. Berkowitz RS, Goldstein DP, Bernstein MR: Ten years' experience with methotrexate and folinic acid as primary therapy for gestational trophoblastic disease. *Gynecol Oncol* 1985;23:111-118.
  174. Smith EB, Weed JC Jr, Tyrey L, et al: Treatment of nonmetastatic gestational trophoblastic disease: Results of methotrexate alone versus methotrexate-folinic acid. *Am J Obstet Gynecol* 1982;144:88-92.
  175. Baxter JF, Soong SJ, Hatch KD, et al: Treatment of nonmetastatic gestational trophoblastic disease with oral methotrexate. *Am J Obstet Gynecol* 1987;157:1166-1168.
  176. Homesley HD, Blessing JA, Rettenmaier M, et al: Weekly intramuscular methotrexate for nonmetastatic gestational trophoblastic disease. *Obstet Gynecol* 1988;72:413-418.
  177. Homesley HD, Blessing JA, Schlaerth J, et al: Rapid escalation of weekly intramuscular methotrexate for nonmetastatic gestational trophoblastic disease. *Gynecol Oncol* 1990;39:305-308.
  178. Goldstein DP: Prevention of gestational trophoblastic disease by the use of actinomycin D in molar pregnancies. *J Obstet Gynecol* 1974;43:475-479.
  179. Petrilli ES, Twiggs LB, Blessing JA, et al: Single-dose actinomycin treatment for nonmetastatic gestational trophoblastic disease. *Cancer* 1987;60:2173-2176.
  180. Schlaerth JB, Morros CP, Nalick RH, et al: Single-dose actinomycin D in the treatment of postmolar trophoblastic disease. *Gynecol Oncol* 1984;19:53-56.
  181. Hammond CB, Weed JC Jr, Currie JL: The role of operation in the current therapy of gestational trophoblastic disease. *Am J Obstet Gynecol* 1980;136:844-858.
  182. Mazur MZT, Kurman RJ: Choriocarcinoma and placental-site trophoblastic tumor. In: Szulman AE, Buchsbaum HJ (eds): *Gestational Trophoblastic Disease*. New York: Springer-Verlag, 1987; pp. 45-68.
  183. Rosenshein NB, Wijnen H, Woodruff JD: Clinical importance of the diagnosis of trophoblastic pseudotumors. *Am J Obstet Gynecol* 1985;65:527-534.
  184. Eckstein RP, Paradinas FJ, Bagshawe KD: Placental site trophoblastic tumor (trophoblastic pseudotumor): A study of four cases requiring hysterectomy including one fatal case. *Histopathology* 1982;6:211-226.
  185. Young RH, Scully RE: Placental-site trophoblastic tumor: Current status. *Clin Obstet Gynecol* 1984;27:248-258.
  186. Lewis J Jr, Ketcham AS, Hertz R: Surgical intervention during chemotherapy of gestational trophoblastic neoplasms. *Cancer* 1966;19:1517-1522.
  187. Lewis J Jr, Gore H, Hertig AT, et al: Treatment of trophoblastic disease with the rationale for the use of adjunctive chemotherapy at the time of indicated operation. *Am J Obstet Gynecol* 1966;96:710-722.
  188. American College of Obstetricians and Gynecologists: Diagnosis and treatment of gestational trophoblastic disease. *ACOG Practice Bulletin No. 53*. *Obstet Gynecol* 2004;103:1365-1377.
  189. Lurain JR, Brewer JI, Torok EE, et al: Gestational trophoblastic disease: Treatment results at the Brewer Trophoblastic Disease Center. *Obstet Gynecol* 1982;60:354-360.
  190. Bagshawe KD: Treatment of high-risk choriocarcinoma. *J Reprod Med* 1984;29:813-820.
  191. Newlands ES, Bagshawe KD, Begent RHJ, et al: Developments in chemotherapy for medium- and high-risk patients with gestational trophoblastic tumours (1979-1984). *Br J Obstet Gynaecol* 1986;93:63-69.
  192. DuBeshter B, Berkowitz RS, Goldstein DP, et al: Metastatic gestational trophoblastic disease: Experience at the New England Trophoblastic Disease Center, 1965-1985. *Obstet Gynecol* 1987;69:390-395.
  193. Theodore C, Azab M, Dorz JP, et al: Treatment of high-risk gestational trophoblastic disease with

- chemotherapy combinations containing cisplatin and etoposide. *Cancer* 1989;64:1824–1828.
194. Weed JC Jr, Woodward KT, Hammond CB: Choriocarcinoma metastatic to the brain: Therapy and prognosis. *Semin Oncol* 1982;9:208–212.
  195. Athanassiou A, Begent RHJ, Newlands ES, et al: Central nervous system metastases of choriocarcinoma: 33 years' experience at Charing Cross Hospital. *Cancer* 1983;52:1728–1735.
  196. Soper JT, Clarke-Pearson D, Hammond CB: Metastatic gestational trophoblastic disease: Prognostic factors in previously untreated patients. *Obstet Gynecol* 1988;71:338–43.
  197. Lurain JR, Casanova LA, Miller DS, et al: Prognostic factors in gestational trophoblastic tumors: A proposed new scoring system based on multivariate analysis. *Am J Obstet Gynecol* 1991;164:611–616.
  198. Surwit EA: Management of high-risk gestational trophoblastic disease. *J Reprod Med* 1987;32:657–662.
  199. Bolis G, Bonazzi C, Landoni F, et al: EMA-CO regimen in high-risk gestational trophoblastic tumor. *Gynecol Oncol* 1988;31:439–444.
  200. Schink HC, Singh DK, Rademaker AW, et al: EMA-CO chemotherapy: A well-tolerated, highly effective treatment for metastatic, high-risk gestational trophoblastic disease. Society of Gynecologic Oncologists, 23rd Annual Meeting, San Antonio, TX. March 15–18, 1992.
  201. Lurain JZR, Brewer JI: Treatment of high-risk gestational trophoblastic disease with methotrexate, actinomycin D, and cyclophosphamide chemotherapy. *Obstet Gynecol* 1985;65:830–836.
  202. Bagshawe KD: Treatment of trophoblastic tumors. *Ann Acad Med (GB)* 1976;5:565–570.
  203. Surwit EA, Alberts DS, Christian CD, et al: Poor prognosis gestational trophoblastic disease: An update. *Obstet Gynecol* 1984;64:21–26.
  204. Gordon AN, Kavanaugh JJ, Gershenson DM, et al: Cisplatin, vinblastine and bleomycin combination therapy in resistant gestational trophoblastic disease. *Cancer* 1986;58:1407–1410.
  205. DeBeshter B, Berkowitz RS, Goldstein DP, et al: Vinblastine, cisplatin, and bleomycin as salvage therapy for refractory high-risk gestational trophoblastic disease. *J Reprod Med* 1989;34:189–192.
  206. Azab M, Droz JP, Theodore C: Cisplatin, vinblastine and bleomycin combination in treatment of resistant high-risk gestational trophoblastic tumors. *Cancer* 1989;64:1829–1832.
  207. Choo YC, Chan SYW, Wong WC, et al: Ovarian dysfunction in patients with gestational trophoblastic neoplasia treated with short intensive course of etoposide (VP16–213). *Cancer* 1985;55:2348–2352.
  208. Jones WB: Gestational trophoblastic disease: The role of radiotherapy. In: Nori D, Hilaris BS (eds): *Radiation Therapy of Gynecologic Cancer*. New York: Alan R. Liss, 1987; p. 207.
  209. Rustin GJS, Newlands ES, Begent RHJ, et al: Weekly alternating etoposide, methotrexate, and actinomycin/vincristine and cyclophosphamide for the treatment of CNS metastases of choriocarcinoma. *J Clin Oncol* 1989;7:900–903.
  210. Evans AC Jr, Soper JT, Clarke-Pearson DL, et al: Gestational trophoblastic disease metastatic to the central nervous system. *Gynecol Oncol* 1995;59:226–230.
  211. Xu LT, Sun CF, Yang YE, et al: Resection of pulmonary metastatic choriocarcinoma in 43 drug-resistant patients. *Ann Thorac Surg* 1985;39:257–259.
  212. Weed JC, Hammond CB: Cerebral metastatic choriocarcinoma: Intensive therapy and prognosis. *Obstet Gynecol* 1980;55:89–94.
  213. Mutch DG, Soper JT, Babcock CJ, et al: Recurrent gestational trophoblastic disease: Experience of the Southeastern Regional Trophoblastic Disease Center. *Cancer* 1990;66:978–982.
  214. Garner EIO, Lipson E, Bernstein MR, et al: Hydatidiform mole: What happens during the next pregnancy? *Contemp Ob/Gyn* 2001;46:96–101.
  215. Barclay DL: Surgery of the vulva, perineum, and vagina in pregnancy. In: Barber HRK, Graber EA (eds): *Surgical Disease in Pregnancy*. Philadelphia: WB Saunders, 1974; pp. 320–323.
  216. Sturgeon S, Britton LA, Devessa SS, et al: In situ and invasive vulvar cancer incidence trends. *Am J Obstet Gynecol* 1992;166:1482–1485.
  217. Lutz MH, Underwood PB, Rozier JC, et al: Genital malignancy in pregnancy. *Am J Obstet Gynecol* 1977;129:536–542.
  218. Moore DH, Fowler WC Jr, Currie JL, et al: Case report: Squamous cell carcinoma of the vulva in pregnancy. *Gynecol Oncol* 1991;41:74–77.
  219. Regan MA, Rosenweig BA: Vulvar carcinoma in pregnancy: A case report and literature review. *Am J Perinatol* 1993;10:334–335.

220. Bakour SH, Jaleel H, Weaver JB, et al: Vulvar carcinoma presenting during pregnancy, associated with recurrent bone marrow hypoplasia: A case report and literature review. *Gynecol Oncol* 2002;87:207–209.
221. Ogunleye D, Lewin SN, Huettner P, et al: Recurrent vulvar carcinoma in pregnancy. *Gynecol Oncol* 2004;95:400–401.
222. Wilkinson EJ: Vulvar intraepithelial neoplasia and squamous cell carcinoma with emphasis on new nomenclature. In: Damianov I, Cohen AH, Mills SE, Young RH (eds): *Progress in Reproductive and Urinary Tract Pathology*, vol 2. New York: Field and Wood, 1990; pp. 1–20.
223. DiSaia PJ, Creasman ST (eds): Staging. In: *Clinical Gynecologic Oncology*, ed 6. St. Louis: Mosby- Year Book, 2002; pp. 613–620.
224. Monaghan JM, Lindegue A: Vulvar carcinoma in pregnancy. *Br J Obstet Gynecol* 1986;93:785–786.
225. Collins CG, Barclay DL: Cancer of the vulva and cancer of the vagina in pregnancy. *Clin Obstet Gynecol* 1963;6:927–942.
226. Gitsch G, Van Eukeren M, Hacker NF: Surgical therapy of vulvar cancer in pregnancy. *Gynecol Oncol* 1995;56:312–315.
227. Al-Kurdi M, Monaghan JM: Thirty-two years' experience in management of primary tumors of the vagina. *Br J Obstet Gynaecol* 1981;88:1145–1150.
228. Brinton LA, Nasca PC, Mallin K, et al: Case-controlled study of in-situ and invasive carcinoma of the vagina. *Gynecol Oncol* 1990;38:49–54.
229. Manetta A, Gutrecht EL, Berman ML, et al: Primary invasive carcinoma of the vagina. *Gynecol Oncol* 1990;38:49–54.
230. Senekjian EK, Hubby M, Bell DA, et al: Clear cell adenocarcinoma (CCA) of the vagina and cervix in association with pregnancy. *Gynecol Oncol* 1986; 24:207–219.
231. Fujita K, Aoki Y, Tanaka K: Stage I squamous cell carcinoma of vagina complicating pregnancy: Successful conservative treatment. *Gynecol Oncol* 2005;98:513–515.
232. Baruah N, Sangupta S: Primary carcinoma of the vagina complicating pregnancy. *J Ind Med Assoc* 1973;60:469–470.
233. Singhal S, Sharma S, De S, et al: Adult embryonal rhabdomyosarcoma of the vagina complicating pregnancy: A case report and review of the literature. *Asia Oceania J Obstet Gynaecol* 1990;16:301–306.
234. Ortega JA: A therapeutic approach to childhood pelvic rhabdomyosarcoma without pelvic exenteration. *J Paediat* 1979;94:205–209.
235. Stock RG, Chen ASJ, Seski J: A 30-year experience in the management of primary carcinoma of the vagina: Analysis of prognostic factors and treatment modalities. *Gynecol Oncol* 1995;56:43–52.
236. Davis KP, Stanhope CR, Garton GR, et al: Invasive vaginal carcinoma: Analysis of early-stage disease. *Gynecol Oncol* 1991;42:131–136.
237. Kiguchi K, Bibbo M, Hasegawa T, et al: Dysplasia during pregnancy: A cytologic follow-up study. *J Reprod Med* 1981;26:66–72.
238. Hacker NF, Berek JS, Lagase RLD, et al: Carcinoma of the cervix associated with pregnancy. *Obstet Gynecol* 1982;59:735–746.
239. Orr JW, Wilson K, Bodiford C: Corpus and cervix cancer: A nutritional comparison. *Am J Obstet Gynecol* 1985;153:775–779.
240. Winkelstein W: Smoking and cervical cancer – current status: A review. *Am J Epidemiol* 1990;131: 945–957.
241. Werness BA, Manger K, Howley PM: The role of the human papillomavirus oncoproteins in transformation and carcinogenic progression. In: DeVita VT Jr, Helhan S, Rosenberg SA (eds): *Important Advances in Oncology*. Philadelphia: JB Lippincott, 1991; pp. 2–18.
242. Powell JL: Pitfalls in cervical colposcopy. *Obstet Gynecol Clin North Am* 1993;20:177–188.
243. Averette HE, Nasser N, Yankow SL, et al: Cervical conization in pregnancy: Analysis of 180 operations. *Am J Obstet Gynecol* 1979;106:543–549.
244. Rubio CA, Thomassen P, Kock Y: Influence of the size of cone specimens on postoperative hemorrhage. *Am J Obstet Gynecol* 1975;122:939–944.
245. DiSaia PJ, Creasman ST (eds): Staging. In: *Clinical Gynecologic Oncology*, ed 6. St. Louis: Mosby-Year Book, 2002; pp. 613–615.
246. Greer BE, Easterling TR, McLennon DA, et al: Fetal and maternal considerations in the management of Stage IB cervical cancer during pregnancy. *Gynecol Oncol* 1989;34:61–65.
247. Koren G, Weiner L, Lishner M, et al: Cancer in pregnancy: Identification of unanswered questions on maternal and fetal risk. *Obstet Gynecol Surv* 1991;45:509–514.
248. Nevin J, Soeters R, DeHaecck K, et al: Advanced cervical carcinoma associated with pregnancy. *Int J Gynecol Cancer* 1993;3:57–63.

249. Saunders N, Landon CR: Management problems associated with carcinoma of the cervix diagnosed in the second trimester of pregnancy. *Gynecol Oncol* 1988;30:120–122.
250. McCall ML, Keaty EC, Thompson JD: Conservation of ovarian tissue in the treatment of carcinoma of the cervix with radical surgery. *Am J Obstet Gynecol* 1958;75:590–706.
251. Creasman WT, Rutledge FN, Fletcher GH: Carcinoma of the cervix associated with pregnancy. *Gynecol Oncol* 1970;36:495–501.
252. Sablinska R, Tarlowska L, Stelmochow J: Invasive carcinoma of the cervix associated with pregnancy: Correlation between patient age, advancement of cancer and gestation, and result of treatment. *Gynecol Oncol* 1977;5:363–373.
253. DiSaia PJ, Creasman WT (eds): Cancer in pregnancy. In: *Clinical Gynecologic Oncology*, ed 6. St. Louis: Mosby-Year Book, 2002; pp. 441–445.
254. Prem KS, Makowski EL, McKelvey JL: Carcinoma of the cervix associated with pregnancy. *Am J Obstet Gynecol* 1966;95:99–108.
255. Mikuta JJ: Invasive carcinoma in pregnancy. *South Med J* 1976;60:843–847.
256. Gustafsson DC, Kottmeier HL: Carcinoma of the cervix associated with pregnancy. *Acta Obstet Gynecol Scand* 1962;41:1–21.
257. Katz VL, Dotters DJ, Droegemueller W: Complications of uterine leiomyomas in pregnancy. *Obstet Gynecol* 1989;73:593–596.
258. Muram D, Gillieson M, Walters JH: Myomas of the uterus in pregnancy: Ultrasonographic follow-up. *Am J Obstet Gynecol* 1980;138:16–19.
259. Aharoni A, Reiter A, Golan D, et al: Patterns of growth of uterine leiomyomas during pregnancy: A prospective longitudinal study. *Br J Obstet Gynaecol* 1988;95:510–513.
260. Caterina E, Rosati P: Ultrasound diagnosis of uterine myomas and complications in pregnancy. *Obstet Gynecol* 1993;82:97–101.
261. Corscaden JA, Singh BP: Leiomyosarcoma of the uterus. *Am J Obstet Gynecol* 1958;75:149–153.
262. O'Connor DM, Norssis HJ: Mitotically active leiomyomas of the uterus. *Hum Pathol* 1990;21:223–227.
263. Burton CA, Grimes DA, March CM: Surgical management of leiomyomata during pregnancy. *Obstet Gynecol* 1989;74:707–709.
264. Kaymak O, Ustunyurt E, Okyay RE, et al: Myomec-tomy during cesarean section. *Int J Obstet Gynecol* 2005;89:90–93.
265. Hales HA, Peterson CM, Jones KP, et al: Leiomy-matosis peritonealis disseminata treated with a gonadotropin-releasing hormone agonist. *Am J Obstet Gynecol* 1992;167:515–516.
266. Karlen JR, Steinberg LB, Abbott JN: Carcinoma of the endometrium coexisting with pregnancy. *Obstet Gynecol* 1972;40:334–339.
267. Sandstrom RE, Welch RW, Green TH Jr: Adenocar-cinoma of the endometrium in pregnancy. *Obstet Gynecol* 1979;53:73S–76S.
268. Hoffman MS, Cavanagh D, Walter TS, et al: Ade-nocarcinoma of the endometrium and endometrioid carcinoma of the ovary associated with pregnancy. *Gynecol Oncol* 1989;32:82–85.
269. Suzuki A, Konishi I, Okamura H, et al: Adenocar-cinoma of the endometrium associated with intrauter-ine pregnancy. *Gynecol Oncol* 1984;18:261–269.
270. Itoh K, Shiozawa T, Shiiohara S, et al: Endometrial carcinoma in septate uterus detected 6 months after full-term delivery: Case report and review of the literature. *Gynecol Oncol* 2004;93:242–247.
271. Sivanesaratnam V: Management of the pregnant mother with malignant conditions. *Curr Op Obstet Gynecol* 2001;13:121–125.
272. Ayhan A, Gunalp S, Karaer C, et al: Endome-trial adenocarcinoma in pregnancy. *Gynecol Oncol* 1999;75:298–299.
273. Vaccarello L, Apte SM, Copeland LJ, et al: Endome-trial carcinoma associated with pregnancy: A report of the cases and review of the literature. *Gynecol Oncol* 1999;74:118–122.
274. Gallup DG, Cordray DR: Leiomyosarcoma of the uterus: Case reports and a review. *Obstet Gynecol Surv* 1979;34:300–312.
275. Di Saia PJ, Creasman WT (eds): Cancer in preg-nancy. In: *Clinical Gynecologic Oncology*, ed 6. St. Louis: Mosby-Year Book, 2002; pp. 445–446.
276. Barter JF, Smith EB, Szpak CA, et al: Leiomyosar-coma of the uterus: Clinicopathological study of 21 cases. *Gynecol Oncol* 1985;21:220–227.
277. Armenia CS, Shaver DN, Modisher MW: Decid-ual transformation of the cervical stroma simulating reticulum cell sarcoma. *Am J Obstet Gynecol* 1964; 89:808–816.
278. Elliot GM, Reynolds HA, Fidler HK: Pseudo-sarcoma botryoides of the cervix and vagina in preg-nancy. *Br J Obstet Gynaecol* 1967;74:728–733.

279. Prakash S, Scully RE: Sarcoma-like pseudopregnancy changes in uterine leiomyomas. *Obstet Gynecol* 1964;24:106–110.
280. Boyce CR, Buddhdev HN: Pregnancy complicated by metastasizing leiomyoma of the uterus. *Obstet Gynecol* 1973;42:252–258.
281. Lowe MP, Bender D, Sood AK, et al: Two successful pregnancies after conservative treatment of endometrial cancer and assisted reproduction. *Fertil Steril* 2002;77:188–189.
282. Gotlieb WH, Beiner ME, Shalmon B, et al: Outcome of fertility-sparing treatment with progestins in young patients with endometrial cancer. *Obstet Gynecol* 2003;102:718–725.
283. Mitsushita J, Toki T, Kato K, et al: Endometrial carcinoma remaining after term pregnancy following conservative treatment with medroxyprogesterone acetate. *Gynecol Oncol* 2000;79:129–132.
284. Ferrandina G, Zannoni GF, Gallotta V, et al: Progression of conservatively treated endometrial carcinoma after full-term pregnancy: A case report. *Gynecol Oncol* 2005;99:215–217.
285. Di Saia PJ, Creasman WT (eds): Adenocarcinoma of the uterus. In: *Clinical Gynecologic Oncology*, ed 6. St. Louis: Mosby-Year Book, 2002; pp. 137–171.
286. Barakat RR, Grigsby PW, Sabbatini P, et al: Corpus: Epithelial tumors. In: Hoskins WJ, Perez CA, Young RC (eds): *Principles and Practice of Gynecologic Oncology*, ed 3. Philadelphia: Lippincott Williams & Wilkins, 2000; pp. 919–959.
287. Malfetano JH, Goldkrand JW: Cis-platinum combination chemotherapy during pregnancy for advanced epithelial ovarian carcinoma. *Obstet Gynecol* 1990;75:545–547.
288. Eastman NJ, Hellman LM: Ovarian tumors in pregnancy. In: Eastman NJ, Hellman LM (eds): *Williams Obstetrics*, ed 13. New York: Appleton-Century-Crofts, 1966; pp. 916–917.
289. Grimes WA Jr, Bartholomew RA, Calvin ED, et al: Ovarian cysts in pregnancy. *Am J Obstet Gynecol* 1954;6:594–605.
290. Marino T, Craig SD: Managing adnexal masses in pregnancy. *Contemp Ob/Gyn* 2000;45:130–138.
291. Beral V, Fraser P, Chilners C: Does pregnancy protect against ovarian cancer? *Lancet* 1978;1:1083–1088.
292. Casagrande JT, Pike MC, Ross RK, et al: "Incessant ovulation" and ovarian cancer. *Lancet* 1979;2:170–173.
293. Centers for Disease Control: Cancer and steroid hormone study: Oral contraceptive use and the risk of ovarian cancer. *JAMA* 1983;249:1596–1599.
294. Cramer DW, Hutchinson GB, Welch WR, et al: Factors affecting the association of oral contraceptives and ovarian cancer. *N Engl J Med* 1982;307:1047–1051.
295. Rosenberg L, Shapiro S, Slone D, et al: Epithelial ovarian cancer and combination oral contraceptives. *JAMA* 1982;247:3210–3212.
296. Weiss NS, Lyon JKL, Liff LM, et al: Incidence of ovarian cancer in relation to the use of oral contraceptives. *Int J Cancer* 1981;28:669–671.
297. Beischer NA, Buttery BW, Fortune DW, et al: Growth and malignancy of ovarian tumors in pregnancy. *Aust NZ J Obstet Gynaecol* 1971;11:208–220.
298. Struyk AP, Treffers PE: Ovarian tumors in pregnancy. *Acta Obstet Gynaecol Scand* 1984;63:421–424.
299. Thornton JG, Wells M: Ovarian cysts in pregnancy: Does ultrasound make traditional management inappropriate? *Obstet Gynaecol* 1987;69:717–721.
300. Bromley B, Benacerraf B: Adnexal masses during pregnancy: Accuracy of sonographic diagnosis and outcome. *J Ultrasound Med* 1997;16:447–452.
301. Hill LM, Connors-Beatty DJ, Nowak A, et al: The role of ultrasonography in the detection and management of adnexal masses during the second and third trimesters of pregnancy. *Am J Obstet Gynecol* 1998;179:703–707.
302. Ueda M, Ueki M: Ovarian tumors associated with pregnancy. *Int J Gynecol Obstet* 1996;55:59–65.
303. Wheeler TC, Fleischer AC: Complex adnexal mass in pregnancy: Predictive value of color Doppler sonography. *J Ultrasound Med* 1997;16:425–428.
304. Ashkenazy M, Kessler I, Czernobilsky B, et al: Ovarian tumors in pregnancy. *Int J Gynecol Obstet* 1988; 27:79–83.
305. Butter BW, Beischer NA, Fortune DW, et al: Ovarian tumors in pregnancy. *Med J Aust* 1973;1:345–349.
306. Chung A, Birnbaum SJ: Ovarian cancer associated with pregnancy. *Obstet Gynecol* 1973;41:211–214.
307. Novak ER, Lambrose CD, Woodruff JD: Ovarian tumors in pregnancy: An ovarian tumor registry review. *Obstet Gynecol* 1975;46:401–406.
308. Geist SH (ed): *Ovarian Tumors*. New York: Hoeber Publishing Company, 1942.

309. Bast RC Jr, Klug RL, St John, et al: A radioimmunoassay using a monoclonal antibody to monitor the course of epithelial ovarian cancer. *N Engl J Med* 1983;309:883–887.
310. Jacobs I, Bast RC Jr: The CA-125 tumor-associated antigen: A review of the literature. *Hum Reprod* 1989;4:1–12.
311. Parker WH, Childers JM, Canis M, et al: Laparoscopic management of benign cystic teratomas during pregnancy. *Am J Obstet Gynecol* 1996;174:1499–1501.
312. Reedy MB, Kalen B, Kuehl TJ: Laparoscopy during pregnancy: A study of five fetal outcome parameters with use of the Swedish Health Registry. *Am J Obstet Gynecol* 1997;177:673–679.
313. Moore RD, Smith WG: Laparoscopic management of adnexal masses in pregnant women. *J Reprod Med* 1999;44:97–100.
314. Piver MS: Importance of proper staging in ovarian carcinoma. *Clin Obstet Gynecol* 1983;10:223–234.
315. Sonnendecker EWW: Is appendectomy mandatory in patients with ovarian carcinoma? *S Afr Med J* 1982;62:978–980.
316. Malfetano JH: The appendix and its metastatic potential in epithelial ovarian cancer. *Obstet Gynecol* 1987;69:396–398.
317. Powell JL, Donovan JT, Moran TJ, et al: Is appendectomy essential for the treatment of ovarian cancer? *The Female Patient* 1991;16:64–68.
318. Burghardt E, Girardi F, Lahousen M, et al: Patterns of pelvic and paraaortic lymph node involvement in ovarian cancer. *Gynecol Oncol* 1991;40:103.
319. Munnell EW: Primary ovarian cancer associated with pregnancy. *Clin Obstet Gynecol* 1963;4:983–993.
320. King LA, Nevin PC, Williams PP, et al: Treatment of advanced epithelial ovarian carcinoma in pregnancy with cis-platinum-based chemotherapy. *Gynecol Oncol* 1991;44:78–80.
321. Malfetano JH, Goldkrand JW: Cis-platinum combination chemotherapy during pregnancy for advanced epithelial ovarian cancer. *Obstet Gynecol* 1990;75:545–547.
322. Einhorn N, Nilsson B, Sjovall K: Factors influencing survival in carcinoma of the ovary. *Cancer* 1985;55:2019–2025.
323. Halmo SD, Nisher JA, Allen HH: Primary ovarian cancer in pregnancy. In: Allen HH, Nisher JA (eds): *Cancer in Pregnancy: Therapeutic Guidelines*. Mount Kisco, NY: Futura Publishing, 1986; pp. 274–275.
324. Stanhope CR, Smith JP: Germ cell tumors. *Clin Obstet Gynecol* 1983;10:357–364.
325. Karlen JR, Bari A, Cook WA: Dysgerminoma associated with pregnancy. *Obstet Gynecol* 1979;53:330–335.
326. DePalo G, Pilotti S, Kenda R, et al: Natural history of dysgerminoma. *Am J Obstet Gynecol* 1982;143:799–807.
327. Schwartz RP, Chatwani AJ, Stremel W, et al: Endodermal sinus tumor in pregnancy: Report of a case and review of the literature. *Gynecol Oncol* 1983;15:434–439.
328. Ito K, Teshima K, Suzuki H, et al: A case of ovarian endodermal sinus tumor associated with pregnancy. *Tohoku J Exp Med* 1984;142:183–194.
329. Malone JM, Gershenson DM, Creasy RK, et al: Endodermal sinus tumor of the ovary associated with pregnancy. *Obstet Gynecol* 1986;68:86S–89S.
330. Metz SA, Day TG, Pursell SH: Adjuvant chemotherapy in a pregnant patient with endodermal sinus tumor of the ovary. *Gynecol Oncol* 1989;32:371–374.
331. Kim DS, Park MI: Maternal and fetal survival following surgery and chemotherapy of endodermal sinus tumor of the ovary during pregnancy: A case report. *Obstet Gynecol* 1989;73:503–507.
332. Farahmand SM, Marchetti DL, Asirwatham JE, et al: Ovarian endodermal sinus tumor associated with pregnancy: Review of the literature. *Gynecol Oncol* 1991;41:156–160.
333. Perkins RL, Hernandez E, Miyazawa K: Ovarian endodermal sinus tumor during pregnancy. *Am J Gynecol Health* 1989;3:71–74.
334. VanDer See ACJ, DeBruijn HWA, Bouma J, et al: Endodermal sinus tumor of the ovary during pregnancy: A case report. *Am J Obstet Gynecol* 1991;164:504–506.
335. Weed JC Jr, Roh RA, Mendenhall HW: Recurrent endodermal sinus tumor during pregnancy. *Obstet Gynecol* 1979;54:653–656.
336. Kurman RJ, Norris HJ: Endodermal sinus tumor of the ovary: A clinical and pathologic analysis of 71 cases. *Cancer* 1976;38:2404–2419.
337. Gershenson DM, Junco GD, Herson J: Endodermal sinus tumor of the ovary: The MD Anderson experience. *Obstet Gynecol* 1983;61:194–202.

338. Schwartz PE: Combination chemotherapy in the management of ovarian germ cell malignancies. *Obstet Gynecol* 1984;64:564-572.
339. Gershenson DM, Del Junco G, Silva EG, et al: Immature teratoma of the ovary. *Obstet Gynecol* 1986;68:624-629.
340. Slayton RE, Park RC, Silverberg SG, et al: Vincristine, dactinomycin and cyclophosphamide in the treatment of malignant germ cell tumors of the ovary: A Gynecologic Oncology Group study (a final report). *Cancer* 1985;56:243-248.
341. Schneider J, Erasun F, Hervas JL, et al: Normal pregnancy and delivery two years after adjuvant chemotherapy for grade III immature ovarian teratoma. *Gynecol Oncol* 1988;29:245-249.
342. Lee RB, Kelly J, Elg SA, et al: Pregnancy following conservative surgery and adjunctive chemotherapy for stage III immature teratoma of the ovary. *Obstet Gynecol* 1989;73:853-855.
343. Young RH, Dudley AG, Scully RE: Granulosa cell, Sertoli-Leydig cell and unclassified sex-cord-stromal tumors associated with pregnancy: A clinicopathological analysis of thirty-six cases. *Gynecol Oncol* 1984;18:181-205.
344. Young RH, Scully RE: Ovarian sex-cord-stromal tumors: Recent progress. *Int J Gynecol Pathol* 1982;1:101-123.
345. Bjorkholm E, Silfverswerd C: Prognostic factors in granulosa cell tumors. *Gynecol Oncol* 1981;11:261-274.
346. Roth LM, Anderson MD, Goven AD, et al: Sertoli-Leydig cell tumors: A clinicopathologic study of 34 cases. *Cancer* 1981;48:187-197.
347. Adolph A, Le T, Khan K, et al: Recurrent metastatic fallopian tube carcinoma in pregnancy. *Gynecol Oncol* 2001;81:110-112.
348. Rosen AC, et al: Management and prognosis of primary fallopian tube carcinoma. *Gynecol Obstet Invest* 1999;47:45-51.
349. Sedlis A: Primary carcinoma of the fallopian tube. *Obstet Gynecol Surv* 1961;16:209-226.
350. Bider D, Mashiach S, Dulitzky M, et al: Clinical, surgical and pathological findings of adnexal torsion in pregnant and nonpregnant women. *Surg Gynecol Obstet* 1991;173:363-366.
351. Yalcin OT, Hassa H, Zeytinoglu S, et al: Isolated torsion of fallopian tube during pregnancy: Report of two cases. *Eur J Obstet Gynecol Reprod Biol* 1997;74:179-182.
352. Kushner DH, Rosenbaum M: Torsion of the fallopian tube complicating pregnancy. *Am J Obstet Gynecol* 1952;64:935-936.
353. Blum LL, Sayre BE: Torsion of fallopian tube in a virgin: Report of a case and review of the literature. *Arch Surg* 1937;34:1032-1048.
354. McHutchinson LLB, Koonings PP, Ballard CA, et al: Preservation of ovarian tissue in adnexal torsion with fluorescein. *Am J Obstet Gynecol* 1993;168:1386-1388.
355. DiSaia PJ, Townsend PE, Morris CP: The rationale for less than radical treatment for gynecologic malignancy in early reproductive years. *Obstet Gynecol Surv* 1974;29:581-593.

## John P. O'Grady

... the Forceps... As it is not sharper than the Hand, it may be introduced with all imaginable Safety, ... I can, from my own Experience, affirm it to be a most excellent Instrument, and so far from Hurting or Destroying, that it frequently saves the Mother's Life, and That of the Child, as will appear in the Course of this Treatise.

Edmund Chapman (1680?–1756)  
*Treatise on the Improvement of Midwifery*  
Third Edition, London 1759, John Brindley: pp. xxvii–iii

### ROLE OF INSTRUMENTAL DELIVERY

Since its inception in the seventeenth century, instrumental delivery has been controversial [1–5]. Although most practitioners employ methods of assisted delivery on occasion, there are substantial international and local variations, and the acceptability of certain techniques has changed rapidly in recent decades [6,7]. Although both forceps and the vacuum extractor continue in everyday use, vacuum extraction continues to gain in popularity [8–10]. In the United States, the prevalence of operative vaginal delivery is estimated at 10% to 15% [11]. Despite changes in practice and in the popularity of forceps versus vacuum extractor over recent decades, the important questions about assisted delivery remain the same: when to conduct operative deliveries, which instrument is best for specific indications, and what the associated short- and long-term risks are.

Previous generations of obstetricians were taught that the principal indications for instrumental delivery were two: prevention and rescue. *Prevention* involved saving both the baby and the mother from the potentially serious complications of prolonged second-stage labor, whereas *rescue* involved rapid fetal extractions in situations of presumed jeopardy. There was also the common use of what was termed *prophylactic* forceps. These were operations performed to shorten the second stage for the stated purpose of protecting the mother from injury to her pelvic support tissues while also reducing the risk of infant intracranial damage by cradling the infant's head. In experienced hands, both the forceps and the vacuum extractor are still well suited for at least the first two of these listed tasks. The issue of instrumental deliveries as a method of maternal or fetal *protection* is much more complex than originally thought. Support for the presumed benefits of these procedures has essentially disappeared after critical analysis of obstetric practices.

What is the best practice now? Obstetrics is certainly not the same as when Bolivar DeLee promoted protective elective forceps use in 1920 [12] and is profoundly different from that experienced



by the original developers of forceps in the eighteenth century. Management of labor and delivery has also substantially changed from what was practiced a decade or more ago. The introduction of fetal monitoring, increasing medicolegal concerns about maternal or fetal injuries, new data suggesting long-term maternal morbidity from childbearing, improvements in anesthesia, the availability of potent tocolytics and uterotonics, broad-spectrum antibiotics, and the increasing application of evidence-based medicine to critically evaluate traditional management protocols have collectively, profoundly, and permanently altered obstetric practice.

The safe alternatives for labor management and new concerns about potential long-term complications of vaginal birth have led to a progressively more selective and critical view of the role of instrumentation. Whereas in prior years an instrumental delivery would have been performed immediately once the second stage had reached an arbitrarily established period of time, alternatives in labor and delivery management are now routinely considered. In addition, some practitioners have chosen to restrict their practice to use only vacuum extraction, employ only outlet forceps, or to abandon assisted delivery procedures entirely.

This chapter discusses instrument design, technique of application, and the risks and benefits of assisted delivery. The principal controversies concerning instrumental delivery by both forceps and the vacuum extractor are reviewed, and recommendations are made about the use of these instruments. The focus of this presentation remains the desirability and safety of instrumental delivery and a critical analysis of what constitutes the best modern practice.

Finding unbiased and evidence-based data on which to base decisions about instrumental delivery is difficult. Many basic forceps and vacuum extraction techniques have not been subjected to systematic study. Much of the voluminous literature concerning instrumental delivery is anecdotal, uncontrolled, involves small samples, or is retrospective. As a result of the evolving nature of obstetric science, the author's guides in recommending best practice include information derived from the relatively small number of properly conducted, randomized prospective trials, outcome data from various prospective and retrospective clinical studies,

and, to a lesser degree, the collective experience and opinion of prior practitioners as reflected in textbooks, traditional practices, and various reviews.

### OVERVIEW: INSTRUMENTAL DELIVERY

An instrumental delivery is a major intervention, similar to other surgical operations. Both an appropriate indication and the consent of the mother are mandatory prerequisites. When labor process falters or ceases, the clinician must not simply resort to instrumentation or, for that matter, a cesarean but consider the alternatives appropriate for the specific obstetric setting and formulate a management plan. If assisted delivery is chosen, the basic requirements for the operation include careful choice of cases, patient consent, adequate operator training and experience, acceptable anesthesia, meticulous technique, and the willingness to abandon the attempt if it does not proceed easily [9,13–15].

### INDICATIONS FOR INTERVENTION

The indications for operative vaginal delivery are either fetal or maternal. These indications include a prolonged second stage of labor, shortening of the second stage for maternal benefit, and suspicion of immediate or potential fetal compromise.

Station, as used in this chapter, follows the American College of Obstetricians and Gynecologists (ACOG) guidelines and is reported in centimeters (+5/–5) [9]. *Station* is defined as the clinical estimate of the distance between the bony presenting part and the plane of the maternal ischial spines. Where station is discussed, two numbers are recorded (e.g., +2/5 cm). The first number is the estimated station, whereas the second number documents the reporting system used. In the example provided, the 5-cm scale for reporting station is the one intended and the station is +2. It should be noted that clinical stations reported in this 5-point scale do not entirely correspond to the stations recorded in the 3-point scale traditionally taught to practitioners (Table 17.1). Unfortunately, the determination of the station is subjective, and the correlation between examiners when serial examinations are performed or when ultrasound scanning is used to verify fetal positioning is not reassuring (see The Problem of Digital Examination).

**TABLE 17.1 Estimation of Station\* of the Presenting Part: Comparison between Methods**

Classic Three Station Scale	ACOG Centimeter Scale	Cranial Position
-3	-5	Pelvic inlet
-2	-4	
-1	-3	
	-2	Ischial spines (engagement)
0	0	
+1	+1	
	+2	On the perineum
+2	+3	
+3	+5	

\*Defined as the estimate of the distance from the bony presenting part to the plane of the ischial spines. See text for details.

Modified from Rosen MG: Management of labor. New York: Elsevier; 1990, with permission.

### Prolonged Second Stage

Arrested descent or prolongation of the second stage of labor is a common indication for assisted delivery. Severely prolonged or neglected labors, although now rare in Western practice, still occur with some frequency in less-developed parts of the world. Such cases predispose to fetal injury and in extreme cases to maternal pelvic tissue necrosis, leading to potentially serious complications such as vesicovaginal or rectovaginal fistulas [17]. By traditional definition, second-stage labor of more than 2 hours without a regional or epidural anesthetic, or 3 hours with such an anesthetic are considered prolonged for nulliparous women. For parous women, the acceptable intervals are 2 and 1 hours, respectively [15]. Clinical associations for a delayed second stage are multiple. These include, among others, nulliparity, fetal malpositioning, inadequate uterine activity, limited pelvic size, epidural anesthesia, a large mother or fetus, high station at complete dilation, advanced gestational age, hypertensive disorders, maternal diabetes, and a prolonged active phase [15,18–20].

Second-stage dystocia is an important clinical finding, suggesting the possibility of fetal malpresentation or malpositioning, inadequate uterine activity, or, less commonly, true disproportion. When second-stage progress is tardy, intervention might be required. Despite prior belief, there are now good data to indicate that the second stage can safely be extended beyond the classically established

limits as long as the maternal/fetal status remains good and progress continues. Although with modern monitoring fetal morbidity does not change with a longer second stage, maternal morbidity and rates of operative delivery clearly do increase when four hours or more of second stage have elapsed [15,21,22].

The established limits for the second stage are intended as markers or reminders that labor is prolonged, that maternal/fetal evaluation is needed, and that some action should be considered to expedite delivery. This action could be oxytocin stimulation, cesarean delivery, a forceps or vacuum extraction delivery, maternal encouragement, or some other action. How to proceed depends on the clinical examination, an evaluation of the maternal and fetal status, the skill and experience of the clinician, and the general clinical setting [6,23].

### Shortening of Second Stage

Elective shortening of the second stage is a *potential* indication for an instrumental delivery. Examples include the rare woman whose medical condition makes voluntary expulsive efforts either contraindicated or impossible. These situations include poor second-stage expulsive efforts from limited ability to cooperate, maternal exhaustion, excessive analgesia, or debility owing to a prior maternal injury or a musculoneurologic condition that limits the mother's strength. When epidural anesthesia is dense, maternal expulsive efforts can also falter in the second stage. (See Chapter 9, Obstetric Anesthesia.) In this setting, encouragement, repositioning, oxytocin administration, prolonging the labor, and instrumental delivery or a cesarean are possible treatments.

Previously, if an outlet procedure was possible and there was no evidence of disproportion, an elective instrumental delivery was frequently the choice. Such prophylactic instrumentation was part of standard American practice for many years but is now generally considered passé except in certain circumstances. Nonetheless, outlet procedures are safe. When studied in randomized trial, maternal or infant outcomes do not differ between elective low forceps and spontaneous deliveries [24–26]. With any instrumental delivery, however, the risk for maternal perineal injury does increase. The appropriateness of the use of prophylactic forceps in the delivery of premature infants remains unsettled.

### Suspicion of Fetal Compromise

The term *suspicion of immediate or potential fetal compromise* is now used for situations in which fetal well-being is uncertain and prompt delivery is required. Other terms frequently used in the past with similar clinical implications include *presumed fetal jeopardy*, *fetal distress*, *nonreassuring fetal heart rate tracing* (assuming that an electronic monitor is in use), and *nonreassuring fetal heart rate status* (based on auscultatory findings). Clinical examples include cases complicated by sudden maternal hemorrhage or cord prolapse, accompanied by an unremitting fetal bradycardia, or severe and recurrent late decelerations with poor return to the baseline. Examples of evidence in the medical record to substantiate the diagnosis of potential compromise include a combination of fetal heart rate–tracing data; a report of sudden maternal hemorrhage with an accompanying fetal bradycardia, determined by a hand-held external Doppler device or by direct auscultation; the observation of meconium passage; abnormal scalp or cord pH values; notation of abnormal transcutaneous O<sub>2</sub> data; or other similar data.

The limitation of standard electronic fetal monitoring (EFM) in the accurate diagnosis of fetal compromise/acidosis has been recognized for many years. Two major difficulties with EFM are a high incidence of tracings incorrectly interpreted as suspicious or abnormal and the relatively poor reproducibility of interpretations between various clinicians [27]. Recently, monitoring that combines conventional EFM tracings with new electronics for ST-segment analysis suggests that it could be possible to reduce the high false-positive rate for presumed jeopardy [28]. Whether these new techniques will prove clinically useful remains moot. Unfortunately, the history of fetal monitoring is notable for the enthusiastic introduction of various new methods of evaluation that ultimately failed to achieve their stated goal or proved no better than existing techniques. A good example is fetal pulse oximetry [29].

When continuous EFM is performed, late second-stage recurrent bradycardias are common, especially with maternal bearing-down efforts. These heart rate changes are usually of trivial clinical consequence as long as variability persists, the heart rate returns to baseline between contractions, and the decelerating patterns do not persist beyond approx-

imately 30 minutes. The technique employed for pushing is often a factor. Less athletic bearing down, efforts at intermittent pushing, or other techniques usually achieve equal progress while minimizing bothersome EFM patterns and maternal exhaustion. (See Chapter 22, Fetal Assessment.)

In contrast to what heretofore has been common practice, a recent randomized trial involving 320 nulliparous parturients concluded that the traditional method of coached second-stage expulsive efforts was not beneficial [30]. In this study, withholding coaching was also not harmful. Coached labors did have the advantage of a slight shortening of the second stage by a mean of approximately 13 minutes, but coached pushing was also associated with a significant higher incidence of meconium passage. Heroic pushing has other potential problems as well. In all likelihood, enforced bearing down increases the risk for injury to maternal pelvic support structures, but definitive data on this point are lacking.

Fixed bradycardias with prolonged loss of beat-to-beat variability or bradycardias accompanying a pattern of recurrent late decelerations are classic indications for an expedited delivery. In a previously normal infant with recurrent serious umbilical cord compression, the fetal pH falls approximately 0.02 pH units/minute, establishing a limit of several minutes before serious acidosis develops [31]. This should permit sufficient time for clinical examinations and a prompt review of options. If the pelvis is adequate, the baby well positioned and deep in the pelvis, and the surgeon skillful, prompt operative vaginal delivery by either forceps or vacuum extraction is often possible, avoiding a cesarean. Alternatively, having the mother stop extreme bearing-down efforts, placing her in left lateral recumbency, stopping any oxytocin infusion, administering oxygen and fluids, or other actions might permit the fetal heart rate to recover and, in some cases, a spontaneous delivery subsequently to occur.

Bradycardias at high station ( $\leq +1/5$  cm) because of cord prolapse, suspected abruptio placentae, difficult extraction of a second twin, or other problems are best managed by cesarean delivery. In certain settings, when the parturient is at full dilation and if the baby is small, the pelvis adequate, and the patient multiparous and both willing and capable of prompt bearing down, an assisted or at times even a spontaneous delivery is possible. The conduct

of such an attempted vaginal delivery requires an experienced surgeon who can act expeditiously and effectively command the efforts of both the mother and the other birth assistants. Because the outcome is uncertain, the mother should be moved to a site where a cesarean can be performed. The primary clinician remains at the perineum, helping to time the bearing-down efforts, encouraging the mother's progress, and perhaps applying an instrument while other personnel make simultaneous preparations for cesarean delivery. A vaginal delivery can occur if the presenting part descends rapidly and sufficiently far enough that an instrumental assist is possible. If for any reason an application is not possible, progress is slow, or the presenting part remains high, the obstetrician should perform a cesarean when the surgical preparations are complete. In these fluid situations, rapid clinical evaluation, recruitment of maternal assistance, experience, and sangfroid are the necessary components of successful management. Although operative heroics have no place in obstetric management, a role still remains for emergent instrumental vaginal delivery with either forceps or the vacuum extractor in carefully selected cases, managed by experienced accoucheurs. Specifically in this setting, if second-stage descent is not immediate and the instrumental delivery easy, a cesarean is best (see Trials and Failed Operations).

## EQUIPMENT

Delivery instruments are conveniently classified into eight types: five of forceps, two of vacuum extractors, and one for miscellaneous instruments [32–36].

1. *Classic outlet forceps*: The prototypes for this group are Elliot's and Simpson's cross-bladed, English lock designs, dating from the mid-nineteenth century (Figure 17.1) [3,14,32]. These instruments incorporate a pelvic and cephalic curve and are often used for outlet and low forceps operations including rotations. In experienced hands, these blades can perform virtually all forceps operations. In theory, the overlapping shanks and more pronounced cephalic curve of the Elliot instrument makes it a better choice than the Simpson design for use on relatively unmolded fetal heads.

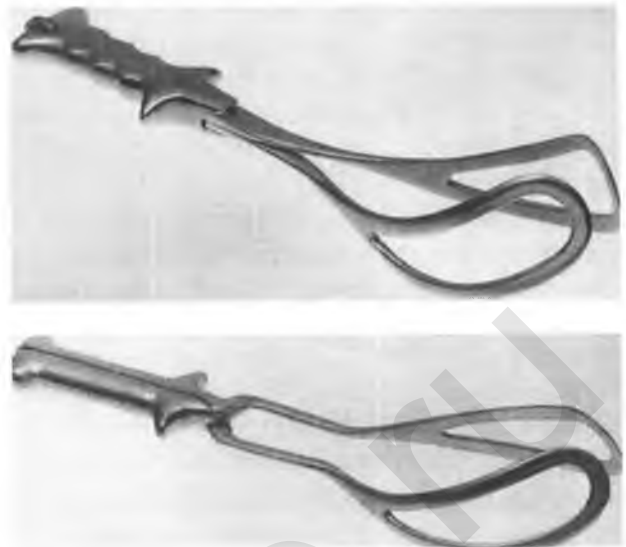


FIGURE 17.1. *Classic outlet type forceps. Elliot forceps (ca. 1858) are depicted above and Simpson forceps (ca. 1849) below.*

2. *Modified classic forceps*: Included in this group are a heterogeneous collection of forceps that are modifications of the Elliot or Simpson classic design [3,32]. These include instruments with overlapping, extended, or shortened shanks, varying cephalic and pelvic curves, and solid, fenestrated, or pseudofenestrated blades. Such instruments employ a variety of locks. The forceps of Tucker-McLane and Luikart-Simpson are examples. These forceps are usually applied as rotating instruments in low to midpelvic applications.
3. *Parallel or divergent blade forceps*: This designation includes types developed by Shute [37], Laufe [38], and others [14]. Their design avoids the cross-shank articulation of other forceps types, reducing fetal cranial compression. These instruments are designed for low or outlet applications, not specifically for rotations. Shute also proposed the use of his parallel blade instrument in shoulder dystocia cases as a method of rotating the fetal trunk and relieving the entrapment [39]. This unique forceps delivery technique has found few adherents, however.
4. *Axis-traction forceps*: Popular in the past, axis instruments are infrequently used today. Axis traction is incorporated in the DeWees, Hawks-Dennen, and Hays forceps designs, among others.

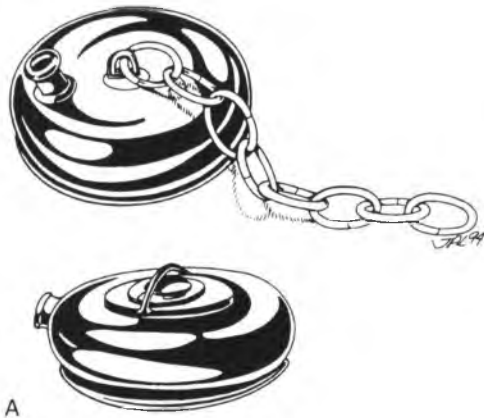


FIGURE 17.2.

A, Bird's rigid metal vacuum extractor cup. Note the eccentrically located vacuum port and the separate, permanently affixed traction chain. The regular model is illustrated. B, O'Neil rigid-cup design. Note the rotating central cuff and the arching traction bar. The occiput posterior model of this cup is illustrated. The laterally located vacuum port facilitates application to posteriorly positioned heads. For the O'Neil cup, a separate traction handle and cord (not depicted) are attached before the cup is applied.

- French or German lock designs are common in this group. In the now uncommon situation when axis traction is desired, it is easiest to attach a traction handle (i.e., Bill's) to a standard forceps.
5. *Specialized forceps*: This category includes a series of designs modified for specific obstetric problems. Examples include Kielland forceps for mid-pelvic rotation, especially where correction of asynclitism is necessary; Barton forceps for transverse arrests in a platypelloid pelvis; and Piper forceps for control of the aftercoming head in breech presentations.
  6. *Vacuum extractors – rigid cup*: This category includes the classic Malmström stainless steel rigid vacuum cup (Figure 17.2) as well as the various metal and newer hard plastic modifications of this basic design [40–41]. These cups vary in minor details of design, specifically in the arrangement of the traction attachment or vacuum port.
  7. *Vacuum extractors – soft cup*: Soft-cup instruments include various disposable polyethylene or combined polyethylene-silicone rubber elastomer cup designs from several manufacturers (Figure 17.3). The newly introduced,

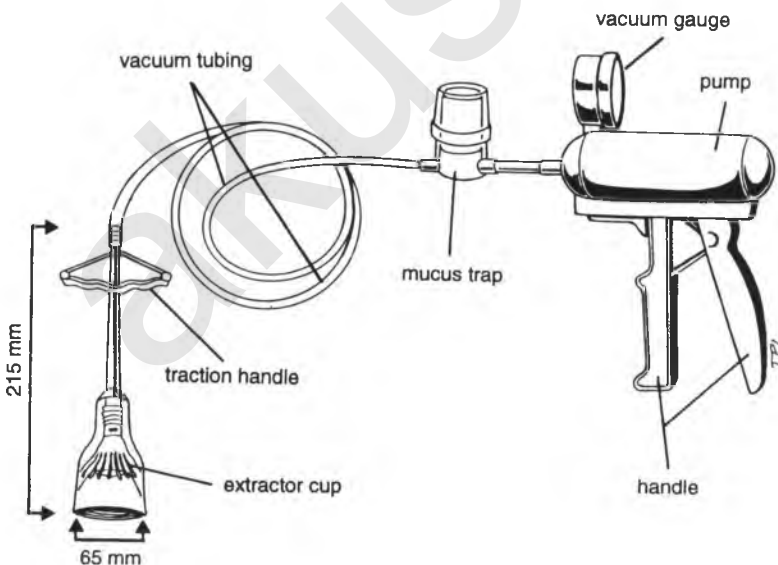


FIGURE 17.3.

A prototype soft-cup vacuum extractor. A hand vacuum pump, connecting tubing, and vacuum trap with an attached standard extractor cup are depicted. The combination of cup, tubing, trap, vacuum source, and pressure gauge is common to all extractors. Details vary by model. See text for discussion.



FIGURE 17.4.  
*Murless vectis blade.*

modified Malmstrom-design plastic extractors have hard cups. Because they function in essentially the same fashion as the original stainless steel extractors, they are included in the rigid-cup category.

8. *Other delivery instruments:* In this category are a variety of devices that are for the most part uncommonly available and rarely used. Included are vectis blades and a heterogeneous collection of other obstetric instruments, such as the bonnet of Elliot, or Thierry's spatulas, none of which gained popularity [42,43]. The most familiar of these specialized instruments, and the one most likely to be used by clinicians, is the Murless vectis blade, designed for cranial extraction during cesarean delivery (Figure 17.4) [44].
9. *Special features unique to vacuum extraction instruments:* All vacuum extractors, regardless of cup composition or design, incorporate several features. These include
  - A rigid or a flexible vacuum cup of varying composition (e.g., polyethylene, Silastic, or stainless steel)
  - A mushroom-shaped cup with a fixed internal vacuum grid or guard
  - A combined vacuum pump/handle or a vacuum port to permit a vacuum hose attachment
  - A handle, wire, or chain for traction
  - A gauge for the measurement of the generated vacuum

The vacuum instruments most popular among American practitioners are the disposable plastic

extractors. Their success is primarily due to their usefulness in most outlet or low extraction procedures, their low cost, preassembly, low incidence of fetal scalp injury, and disposability.

Soft cups have a reduced risk of scalp injury when compared with rigid cups but are limited by design in the traction force they can apply [9]. Soft cups are also not suitable for certain applications, particularly when the fetal head is occiput posterior or markedly asynclitic and in the midpelvis [41,45]. Overall, extraction failures are more common with soft cups, when compared with forceps. In routine outlet and many low operations, the differences between the various vacuum instruments and the forceps are of trivial clinical consequence. In midpelvic operations and especially in posterior presentations, however, such differences become much more important.

For a century, the limitations of various vacuum extractors in applying force due to spontaneous cup displacement (pop-off) have been discussed as a safety factor. The inherent limitation in traction available with extractors is a mixed blessing, however. Extraction failures are potentially dangerous. They tempt some accoucheurs to multiple unsuccessful and possibly traumatic extraction efforts, or lead the surgeon to replace the vacuum extractor with forceps, to attempt a sequential extraction. This argument for pop-offs as an inherent safety advantage of vacuum operations over forceps is misleading. An unsuccessful vacuum extraction with a number of pop-offs can actually *increase* the risk of fetal injury if clinicians do not recognize the limitations of the technique and cannot restrict their efforts. When any properly applied delivery instrument fails, there is a reason. The appropriate response is the exercise of caution, not an increased effort or a change of instrument in an effort to simply overcome the perceived difficulty.

## PROCEDURE CODING

### **Forceps Deliveries**

Forceps instrumental delivery procedures, following American College of Obstetricians and Gynecologists (ACOG) recommendations are described as *outlet*, *low*, and *mid*forceps operations. These designations depend on a clinical examination, which assesses the position and station of the fetal head

TABLE 17.2 ACOG Definitions: Forceps Operations\*

Type of Operation	Description of Classification
Outlet forceps	The scalp is visible at the introitus without spreading the labia. The fetal skull has reached the pelvic floor. The sagittal suture is in the anterior/posterior diameter or in the right or left occiput anterior or posterior position. The fetal head is at or on the perineum.
Low forceps	The leading point of the skull is at station $\geq +2/5$ cm. <sup>†</sup> There are two subdivisions. Rotation is 45 degrees or less. Rotation is more than 45 degrees.
Midforceps	The application of forceps when the fetal head is engaged but the leading point of the skull is above station $+2/5$ cm.
Highforceps	Not included in the classification.

ACOG, American College of Obstetricians and Gynecologists  
\*ACOG Practice Bulletin No. 17, June 2000, Operative Vaginal Delivery.

<sup>†</sup>Note: Station is defined as the distance from the bony presenting part to the plane of the ischial spines, recorded in centimeters ( $\pm 5$  cm). See text for details.

at the commencement of the procedure [9]. These ACOG guidelines were originally written for forceps procedures, but the same descriptions with minor modifications can be applied to vacuum extraction operations as well [Table 17.2].

Several retrospective analyses [46,47] and the prospective study of Hagadorn-Freathy and coworkers [48] indicate the utility of these recent ACOG coding criterion. Compared with the previous system, in which all operations that were not outlet procedures were coded as midforceps, this new coding better defines the type of procedure performed and more clearly identifies procedures that carry an increased risk for fetal/maternal morbidity.

### Vacuum Extraction Operations

As rotation occurs spontaneously with descent and is not imposed by the surgeon, the subclassifications established in the ACOG recommendations for forceps operations are of limited use when the vacuum extractor is applied. A simple distinction between occiput posterior and anterior positions is therefore proposed for vacuum extractions as a measure of potential difficulty (Table 17.3).

TABLE 17.3 Proposed Classification: Vacuum Extraction Operations

Type of Operation*	Description of Classification
Outlet vacuum operation	The fetal head is at or on the perineum. The scalp is visible at the introitus without separating the labia. The fetal skull has reached the pelvic floor.
Low vacuum operation	The position/station of the fetal head does not fulfill the criteria for an outlet operation. The leading edge of the fetal skull is at station $\geq +2/5$ cm. The fetal skull has not reached the pelvic floor. <ul style="list-style-type: none"> <li>• Occiput anterior (OA, LOA, ROA)</li> <li>• Occiput posterior (OP, LOP, ROP) or transverse (LOT, ROT)</li> </ul>
Midvacuum operation	Station is $\leq +2/5$ cm. The fetal head is engaged, but the criteria for outlet or low operations are not fulfilled. <ul style="list-style-type: none"> <li>• Occiput anterior (OA, LOA, ROA)</li> <li>• Occiput posterior (OP, LOP, ROP) or transverse (LOT, ROT)</li> </ul>
Vacuum-assisted cesarean delivery	This includes all vacuum-assisted cesarean deliveries; technique is not specified.
Special vacuum operation	This includes vacuum extraction operations when technique is not specified by usual criteria; full details are described in a dictated operative note.
High vacuum operation	Not included in the classification

OA, Occiput anterior; LOA, left occiput anterior; ROA, right occiput anterior; OP, occiput posterior; LOP, left occiput posterior; ROP, right occiput posterior; LOT, left occiput transverse; ROT, right occiput transverse.

\*The type of operation coded is determined by pelvic examination, noting the position and station of the fetal head prior to performing the extraction.

## Clinical Issues

### *Evaluation of Pelvic Adequacy*

In normal practice, the course of labor is followed by serial pelvic examinations. Cervical dilation and descent of the presenting part over time are recorded graphically in a *partogram* to help judge the process of the labor [50,51]. As suggested in the recent ACOG Practice Bulletin on dystocia [15], the most clinically useful distinctions in evaluating the labor course are between situations in which labor is slower than normal (*prolongation disorders*) and those when progress has ceased (*arrest disorders*).

Once it has been determined that progress has slowed or stopped, additional means of evaluation are needed. A simple review of the labor progress notes cannot diagnose the cause for dystocia or determine the correct treatment. It is possible that a complex mix of factors, including specific features of the passenger, passages, and powers, as well as prior obstetric management choices, predispose to poor progress. It is well to recall the past is often prologue to the present; that is, women tend to reproduce their prior reproductive performance. Gravidas who required assisted delivery in one parturition are more likely to have a similar requirement in another [52]. Fortunately, with close attention to fetal and maternal condition, the length of the labor, despite a prolonged active phase, an arrest in descent, or other common abnormalities, does not in and of itself result in long-term neurologic injury to the infant. However, malpositioning of the fetal head at full dilatation (occiput transverse/posterior) increases the risk of both instrumental and cesarean delivery, episiotomy, severe perineal laceration, and maternal blood loss [53].

An important and insufficiently emphasized role for instrumentation using either the forceps or the vacuum extractor is to assist progress by correcting minor degrees of fetal malpositioning. The application of a traction force to the fetal head, directed in a specific direction, can improve cranial deflection. Such small corrections in the attitude or positioning of the fetal head are often associated with a resumption of descent and a prompt, successful delivery. This observation emphasizes that the principal reason for the use of any delivery instrument is to safely assist but not necessarily replace the natural forces of labor. Furthermore, because force must be applied to assist delivery, instrumental delivery is

appropriate only when true fetopelvic disproportion has been excluded.

In most instances, failure to progress in labor results from inadequate uterine powers. Occasionally it is due to fetal malpositioning, and less commonly, to ineffectual maternal bearing-down efforts or maternal soft-tissue resistance. Dystocia infrequently involves an average-sized fetus in a truly contracted pelvis. The more common problems are macrosomic babies in average-sized pelvis, or average-sized but malpositioned infants in otherwise adequate pelvis. In many cases, dystocia occurs when adverse effects of anesthesia and positioning, minor degrees of malpresentation, subtle differences in pelvic architecture, and inadequate uterine stimulation all combine. For these reasons and because true pelvic deformity is rare, routine clinical pelvimetry is of little assistance in making clinical decisions, except in the rare case of absolute disproportion [54]. Also, although popular in the previous generation of practitioners, radiographic pelvimetry has little if any role in the modern management of dystocia [55]. To better identify cases at enhanced risk for dystocia, techniques combining ultrasonic and radiographic measures, or employing magnetic resonance imaging (MRI), have been reported [56–59]. None has entered routine practice, however.

When advancement of the fetal head ceases after adequate uterine stimulation, prolonging the second stage combined with pain relief, maternal encouragement, or repositioning have all been tried, spontaneous vaginal delivery is no longer an option, and intervention is required [5,15]. The clinician must then decide between a method of delivery – either a cesarean or a vaginal instrumental delivery. The accoucheur must proceed methodically in the evaluation, because poor progress is a strong marker for obstetric complications. If the problem is diagnosed as possible disproportion, a high presenting part, or an undeliverable position such as a brow presentation, cesarean delivery is best. Otherwise, assuming maternal and fetal status is acceptable, the obstetrician can consider the options of an instrumental delivery by forceps or vacuum extraction.

Although true cephalopelvic disproportion is not common, it does occur. Technically disproportion means that the size relationship between the fetal head and the maternal pelvis prevents vaginal delivery. As noted, the failure to achieve vaginal delivery either following labor or an instrumental trial



is often due to a combination of fetal size, maternal pelvic capacity, or malpresentation. Unfortunately, by clinical methods of evaluation, these problems can be indistinguishable from true disproportion. Careful consideration is therefore required whenever an assisted delivery is contemplated once progress has ceased. Clinical evidence for disproportion or a poor likelihood for success in a vaginal delivery includes 1) protraction or arrest disorders in labor, 2) marked cranial deflection, 3) progressive cranial molding unaccompanied by descent of the presenting part, 4) nonengagement of the presenting part despite adequate uterine activity, 5) a fetal head that overlies the pubic symphysis, or 6) other extreme malpresentations, such as transverse lies. These cases are not for instrumentation.

The first step in evaluating the appropriateness of an assisted delivery is an abdominal examination (Leopold's maneuvers; Table 17.4). A Hillis-Müller maneuver can also be performed by applying fundal pressure during a contraction, noting descent of the presenting part [60]. This assists the clinician in judging the degree of cranial molding and fetopelvic capacity. In addition, during pelvic examination the degree of cranial molding is estimated by judging the overlap of the bones of the fetal skull at the occipitoparietal and parietal-parietal junctions (Figure 17.5). The extent of this overlap and the ease of reduction by simple digital pressure are judged. If the bones are in tight apposition and cannot be easily separated by simple digital pressure, molding is advanced or extreme, and there is probably relative disproportion. In this setting, instrumental delivery must be approached with trepidation. Similarly, Leopold's maneuvers can indicate a high or unusually shaped presenting part, suggesting a brow/face presentation, a fetal anomaly, an oblique or transverse lie, or a multiple gestation. Rarely, the fetal head is palpated as overriding the pubic symphysis. Crichton describes another useful technique for estimating labor progress and relative disproportion [61]. He judges the desirability and feasibility of an instrumental delivery by estimating the degree to which the fetal head has descended into the pelvis, calculated in fifths, based on abdominal palpation. He argues that this method avoids the distractions of cranial molding and is a more reliable indication of station than pelvic examination. The author finds this technique difficult to apply,

TABLE 17.4 Leopold's Maneuvers

Maneuver	Procedure
First maneuver	The surgeon stands at the patient's side (traditionally the right) and palpates the contents of the uterine fundus. The fetal size is estimated, and the contents of the fundus are evaluated.
Second maneuver	Using both hands, the surgeon judges the contents of the midportion of the uterus. The fetal back versus small parts are distinguished by kneading the uterus back and forth gently, noting the contour of the fetus and the resistance to digital pressure.
Third maneuver	The surgeon grasps the lower uterine segment with the right hand and attempts to move it back and forth. This helps judge engagement and identify the presenting part, establishing the presentation.
Fourth maneuver	The surgeon turns toward the patient's feet and passes his or her hands longitudinally along the presenting part, noting whether the fingers diverge immediately suprapubically (suggesting engagement) or dip into the pelvic, displacing the presenting part (indicating lack of engagement). Unusual lateral masses (e.g., occiput) are also palpable during this examination.

save in notably slim women. Vacca also counsels to palpate for a prolapsed posterior fetal extremity, what he terms a sacral hand wedge, believing that such a compound presentation complicates a vacuum extraction effort [62]. In his series of cases, this fetal positioning resulted in an increased number of pulls and an increase in the traction force required for successful delivery. He suggests that it is best in this clinical setting to extract the hand entirely before attempting a vacuum operation.

In uncertain or difficult cases or whenever other than an outlet procedure is contemplated, bedside real-time transperineal or transabdominal ultrasonography is useful in management decisions. Scanning promptly identifies position, evaluates cranial

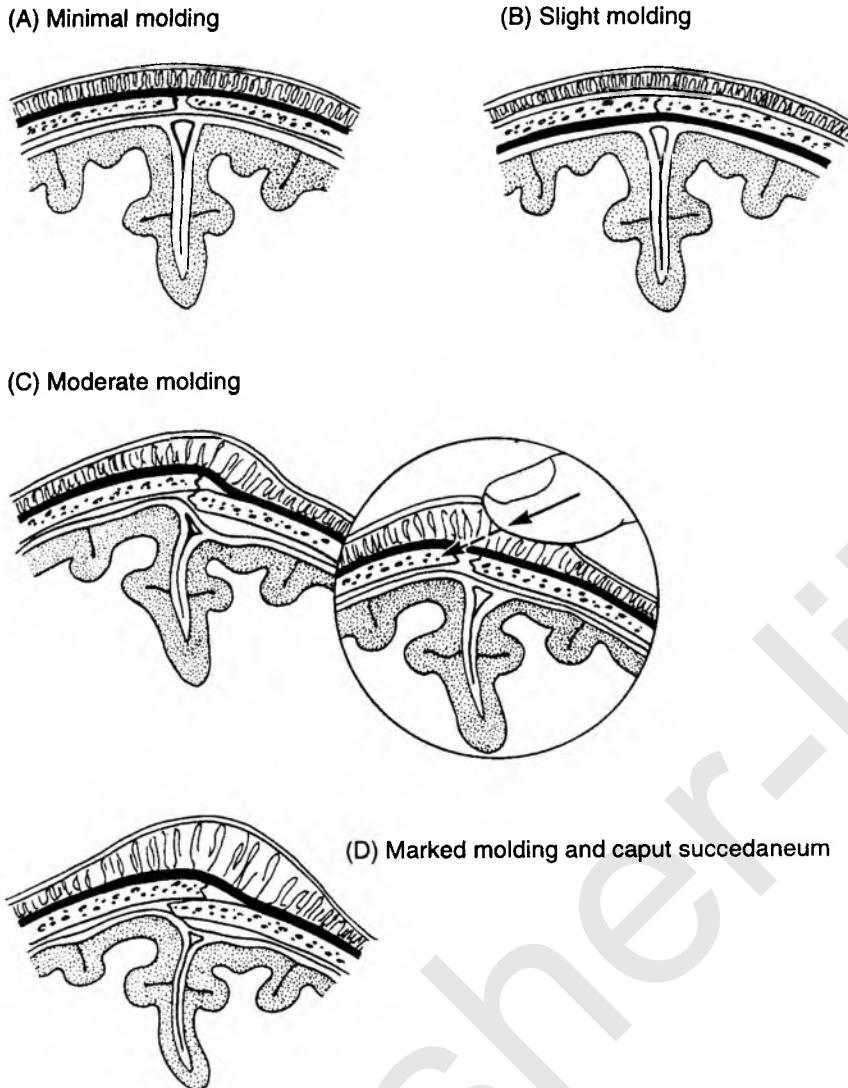


FIGURE 17.5.

*Clinical evaluation of cranial molding by digital examination. As molding progresses from minimal (A) through slight (B) to moderate (C) and finally to marked (D), the cranial bones progressively overlap and additional caput succedaneum is formed. Illustration (C) indicates how digital palpation judges the extent of cranial bone overlap and its reduction. (Redrawn from Vacca A, Handbook of Vacuum Extraction in Obstetric Practice. London: Edward Arnold; 1992; with permission.)*

deflection, and documents the extent of caput, and in expert hands, can estimate station. The use of ultrasonography in delivery management is discussed in greater detail later in this chapter and in Chapter 3, Ultrasonography.

In cephalic presentations, when disproportion is eliminated by best clinical estimate, a trial of labor under oxytocin stimulation is the best measure of pelvic adequacy. (See Chapter 10, Labor.) Progress is followed by serial pelvic examinations. Failure to progress after two hours or more of adequate uterine activity documents dystocia [15]. If progress resumes when oxytocin is administered for tardy progress, however, a subsequent instrumental assisted delivery with the possibility of a difficult delivery or shoulder dystocia is more likely.

### The Problem of Digital Examinations

It has long been recognized that determinations of fetal cranial position and station by digital examination is fraught with error. Even equally well-trained and experienced practitioners can disagree. The advent of real-time ultrasound examination provides a different type of reproducible data emphasizing the limitations of the traditional examinations [64]. Ultrasound studies for both position and station can be performed transvaginally, transperineally, or transabdominally. Interobserver agreements on fetal position are sufficiently accurate for clinical application [65–67]. Bedside real-time ultrasound examinations are helpful because the position of the fetal spine and of the fetal orbits can

be easily identified, especially when the true station is high and the occiput is posterior. Notably, digital vaginal examinations and the more objective ultrasound determination of cranial position are in agreement only approximately 50% to 70% of the time [67–70]. Of interest, in one report attending physicians proved to be no more accurate than residents in making estimates of cranial position [69]! Perhaps not surprisingly, when the presentation is occiput posterior, the error rate for digital examinations is highest [64,70]. Caput succedaneum also significantly diminishes the accuracy of digital examinations. In distinction from cranial position, determination of cranial engagement has a substantially better correlation between ultrasound and concomitant digital vaginal examinations [69,71].

The relative inaccuracy of clinical examinations, even when conducted by experienced personnel, is a striking finding. This throws into question many studies that depend on clinical examinations of fetal position and station as the basis for obstetric manipulations or procedures. As a result of these data, when possible, it is prudent to recheck the clinician's impression of cranial position by real-time scanning before proceeding with a major rotational or mid-pelvic procedure or any instrumental delivery perceived to be difficult.

Other types of ultrasound studies have proved less clinically useful. Disappointingly, ultrasound estimations of fetal weight or ratios between specific fetal measurements have not proved helpful in judging relative fetopelvic size, although several techniques have been suggested [72]. Whether clinically useful techniques for estimation of fetal bulk will prove useful in judging the likelihood for an instrumental delivery or risk of shoulder dystocia remains uncertain. Unfortunately, fetal weight is only one of several important variables when labor progress proves tardy.

### Sequential Instrument Use

Most data indicate that sequential operations (forceps operations followed by vacuum extraction, or vice versa) are associated with an increased risk for fetal intracranial hemorrhage (ICH), exceeding the risk when either forceps or vacuum extraction is used alone [8,9,73–75]. Towner's 1999 study,

the most frequently cited, involved 583,340 live-born singletons from a California database. The data included 59,344 vacuum-assisted deliveries and 15,945 forceps-assisted deliveries. Combined delivery (i.e., vacuum extraction plus forceps) occurred in 2,817 cases and was associated with significantly higher rates of subdural or cerebral hemorrhage, subgaleal hemorrhage, facial nerve injury, and brachial plexus injury than when vacuum extraction alone was performed. Furthermore, the rate of an intracranial hemorrhage when forceps and vacuum extraction were both used was 3.4 times greater versus when vacuum extraction was used alone. Similar data concerning an enhanced risk from combined procedures comes from review of the 1998 Food and Drug Administration (FDA) advisory paper on vacuum extraction, as well as from other sources [76]. It is fair to state that some reviewers disagree with the Towner conclusions, and it should be noted that these data were derived from birth certificate information and not clinical records [77].

The author believes that the risk is one of degree. When one type of instrument is applied and fails, there is no absolute prohibition to trying a different device [5,9]. When sequential applications are performed, however, the risk of maternal and fetal injury is likely increased. Case choice is critical. The most appropriate cases for changing instruments are those in which technical problems, such as a malfunctioning hand pump or a misapplied vacuum cup, are suspected. The least desirable cases are those in which the original vacuum extractor traction efforts resulted in minimal objective progress or several pop-offs after a correct vacuum cup application and appropriate traction, without descent of the presenting part. Injuries from multiple instrument use are mostly likely when a degree of unrecognized fetopelvic disproportion is present and, despite difficulty, the clinician cannot refrain from pursuing a vaginal operative delivery. Whenever a vaginal delivery becomes difficult and sequential instrument use is considered, the alternative of cesarean delivery must be entertained. Operative vaginal deliveries in which a second instrument is used after the failure of the first must be restricted to experienced physicians who have a clear understanding that the risk of birth injury is potentially increased in such operations.

## Documentation

All operative or assisted deliveries, whether forceps or vacuum extraction, require full documentation in the medical record by a computer-generated report, a detailed handwritten note, or dictation. The indications for the operation, the personnel involved, and the anesthesia/analgesia used, if any, and comments concerning the consent process are appropriate to include. The type of instrument chosen, difficulties in insertion, and the station, position, and deflection of the fetal skull are reported. It is also best to include a review of the clinical setting, including a statement of the evaluation of the fetopelvic relationship. Additional comments about the difficulty of the extraction, the number of traction efforts, any episodes of sudden cup displacement (for vacuum operations), whether an episiotomy was performed, and the resulting maternal or fetal complications or injuries, their repair, and estimate of blood loss should be included. In cases in which suspicion of immediate or potential fetal compromise (i.e., presumed fetal jeopardy, fetal distress) was the principal indication for the operation or there is an unanticipated poor neonatal outcome, it is prudent to obtain umbilical arterial and venous pH values and to submit the placenta for histologic examination. In documentation, stating *why* the procedure performed is as important as stating *how* it was actually conducted.

Such detailed summaries protect both the institution and the surgeon in event of subsequent claims of maternal or fetal injury. Bitter experience attests that accurate and complete documentation at the time of delivery saves a great deal of heartache and difficulty when the results of the delivery prove less than ideal and questions are asked years later about the operation or its indications.

## Training Deficiencies

Important questions have been raised about the adequacy of training in techniques of operative delivery. Survey studies of North American resident education programs and international studies of obstetric practice report both serious and concerning trends [11,78]. Virtually all programs (95%) in the most recent North American survey offered instruction in instrumental delivery. Reflecting the popularity of vacuum extraction, North American residency

programs now also provide such training. Plastic or silicone soft cups from various manufacturers were the choice of 86% of programs using vacuum extraction. Approximately 65% of training programs continue to teach midpelvic operative vaginal delivery, with two thirds of respondents favoring forceps use and one-third the vacuum extractor for these procedures. The training programs that no longer teach midpelvic procedures cite safety concerns (70%) and litigation risks (38%) as the principal reasons. In most areas of the country, vacuum extraction has now surpassed forceps in popularity [8].

As these data are more deeply analyzed, several bothersome trends emerge. There is a continued decline in the teaching of midpelvic operative procedures. In their 1990 survey, Ramin and coworkers reported that 14% of programs had abandoned instruction in midpelvic procedures [78]. In a 1995 survey, this number had risen to 36% [11]. In the 1995 data, only one half of responding programs would attempt a rotational forceps operation, with the remainder favoring either vacuum extraction (22%) or cesarean delivery (28%) in this setting.

What are the implications of these findings for the profession? The time-honored methods of teaching instrumental delivery in the operating suite must be changed. There are too few cases for all residents to gain good instruction in the less-frequent vaginal operations and too few qualified instructors. Classic teaching of instrumental delivery by conducting "educational procedures" without specific clinical indication are no longer acceptable. Unless instrumental delivery procedures are conducted with reasonable frequency within an institution, most young practitioners will never experience a sufficient number of cases either to feel comfortable with these operations or to perform them safely, especially in an emergency. As skilled practitioners are progressively lost by retirement or death, the opportunities for the education of obstetric residents also declines, lessening the chances that vaginal instrumental operations will either be considered or attempted by the next generation of accoucheurs. If the specialty wishes to retain instrumental delivery, new methods of teaching – whether by better use of existing cases, computer simulation, or other educational means – must be introduced. Otherwise, within a generation, the skill to perform potentially valuable obstetric

techniques could be lost. (See Chapter 25, Education and Certification.)

Unfortunately, the popularity and apparent simplicity of vacuum operations has fostered a less rigorous approach to these procedures than was traditionally accorded to forceps; however, both types of assisted delivery are surgical procedures. Furthermore, even experienced accoucheurs should not expect that skill in forceps operations automatically translates into success with the vacuum extractor, because the techniques are quite different. The first demand is to recognize that vacuum extraction requires specific training. For the neophyte, it is best to first review available instructional materials [41,45,63,79,80]. Then, with the assistance of an experienced accoucheur, the student can commence with simple outlet extractions where neither cranial malpositioning nor fetal jeopardy is at issue. Thereafter, progressive graduation to more complex procedures is possible. Study guides on compact disk or online are available for the review of basic vacuum extraction technique [81,82].

The data on training in all types of instrumental delivery raises issues for medical educators. Given the declining number of procedures and the recognized risk of instrumentation, which techniques should residents be taught? For most education programs, it is best to teach low and outlet forceps operations intensively, restricting true midpelvic procedures and most procedures requiring rotations of  $>45^\circ$  to trials of vacuum extraction. Residents should also receive instruction in both vacuum and forceps techniques for delivery from the occiput posterior position. This basic education should be accompanied by special emphasis on basic fetopelvic evaluation and correct instrument application. There are good data to document that formal education programs improve the safety of instrumental delivery in terms of both maternal and fetal morbidity [83]. Such choices in educational focus do limit training in forceps operations but will better ensure long-term fetal and maternal safety while retaining basic skills. Teaching of more complex operations must be restricted to programs with a sufficient number of cases and qualified instructors. With the advent of better simulations and training models, education in the more complex operations might improve, but such techniques are not yet in general use.

Midpelvic forceps procedures and major rotations need not necessarily be abandoned by the profession but must be limited to experienced obstetric surgeons in carefully chosen cases and never attempted by the neophyte without recourse to immediate expert instruction.

### Forceps versus Vacuum Extraction Operations

For many applications, the forceps and the vacuum extractor are interchangeable, but the choice of instrument often remains controversial. Certain circumstances and clinical situations favor the use of one device over the other. Initially, it is important to recall that the instruments do differ in the types of maternal and fetal complications associated with their use. In general, scalp injuries, including subgaleal hemorrhage and shoulder dystocia, are more strongly associated with vacuum extraction, whereas facial cosmetic injuries and maternal perineal trauma are more common following forceps operations [8,9,84–86]. Other important considerations in the choice of instrument include the surgeon's training and expertise, the extent of analgesia/anesthesia, the position and station of the presenting part, and the available assistance and equipment. As a practical matter, differences in training and experience are usually more important than the specific technical features of the available instruments in determining which device(s) are actually used. Nonetheless, there are special circumstances in which a specific instrument type is clearly best. For example, for the assisted delivery of the second twin or in applications when anesthesia is limited, the vacuum extractor has clear advantages over the forceps. In other settings, such as a breech delivery, there is no role for the extractor but occasionally an important one for forceps.

If there is clinical suspicion of immediate or potential fetal compromise (i.e., presumed fetal jeopardy, fetal distress) and the fetal head is positioned normally and advanced to a low station, traditionally trained practitioners sometimes prefer to apply forceps rather than the vacuum extractor. Regardless of which instrument is used, fetal and maternal outcomes in outlet operations are good. Such deliveries are associated with no difference in perinatal outcome when compared with simple, spontaneous deliveries. Especially if forceps are used, however, the clinician must exercise care to

avoid a perineal tear. Trauma to the rectum and other perineal structures constitutes the principal risk to the mother of any forceps operation.

When an emergent delivery is required, the surgeon should employ the most familiar instrument. Theoretical concerns aside, in difficult circumstances, greater success and less danger results when accustomed instruments are chosen.

In less pressing circumstances, there are other considerations. In outlet operations, assuming adequate analgesia, the vacuum extractor and the forceps are essentially equivalent instruments. As noted previously, however, a forceps delivery must be conducted with strict attention to the force is applied to the perineum to avoid a laceration. A pudendal block or another type of anesthesia is required if forceps are chosen; at times this can be omitted for a simple outlet vacuum extraction. Whenever a local anesthesia is used it must be accompanied by vocal reassurance, encouragement, and coaching, regardless of the instrument employed.

The most important advantages of vacuum extraction over forceps are in midpelvic procedures in which minimal cranial deflection is present or in trials of instrumental delivery when fetal jeopardy is not at issue [5,45]. True midpelvic vaginal instrumental operations are now uncommon, and many young obstetricians have little or no experience with such procedures. Although midpelvic forceps operations need not be abandoned by experienced practitioners, they must largely be restricted to them. The unaided, neophyte surgeon should never attempt these procedures. Even in experienced hands, the likelihood for maternal injury is greater in midcavity forceps operations than with properly conducted vacuum extractions [9,46,87–90].

Studies comparing forceps and vacuum extraction performance for similar indications reveal a consistent pattern [9]. Fetal scalp injuries and mild postnatal jaundice are more common following vacuum extraction operations, whereas maternal injuries to the perineum and rectum are more likely with forceps deliveries. Forceps procedures are also more likely to succeed, especially in midpelvic extractions or when the fetal occiput is other than occiput anterior [91]. If perineal lacerations occur and extend into the rectum, fistula formation accompanies a very small but clinically important percentage of cases. Injury to the rectal sphincter, with the potential for permanent rectal incontinence, is

another potential complication. Transitory neonatal jaundice and retinal hemorrhages are more frequent in vacuum-extracted neonates than in those delivered by forceps, but these complications are not of long-term consequence.

Compared with forceps, the vacuum extractor has several important technical advantages. The cup can be inserted and traction applied with minimal or no maternal analgesia. This is particularly useful when the patient refuses a major anesthetic, or when a regional anesthetic is unavailable, has failed, or has proved only partially successful. The vacuum extractor also minimizes the risk of vaginal vault injuries, especially in the more complex operations as cranial rotation accompanies descent of the presenting part. Third- and fourth degree perineal lacerations and fetal facial nerve injuries are also less likely with vacuum extraction, at least compared with classic forceps procedures [92]. Peculiarly, vacuum extraction operations are associated with an increased incidence of shoulder dystocia [8]. The reason for this association is not immediately apparent. Forceps can generate a greater force than the vacuum extractor, and in a borderline “fit,” the forceps might be anticipated to be more likely to draw down a large infant, leading to dystocia. This does not seem to be the case, however. Perhaps this association reflects the preferential use of vacuum extraction in cases of suspected fetopelvic disproportion and the greater ease in applying this instrument as opposed to the forceps. The difference could also be due to subtle differences in the physics of extraction between the two devices or in how they are actually used clinically [85]. Although this association between shoulder dystocia and vacuum extraction is repeatedly cited in the instrumental delivery literature, the mechanism for this unanticipated finding remains elusive.

If the vacuum extractor is chosen, the cup type must be tailored to the clinical requirements [41], because all vacuum cups are not equally successful in all applications. When outlet procedures are considered, there is a minimal difference between instruments and any cup type may be chosen. Rigid cups, however, risk the unsightly chignon. For asynclitic heads in transverse arrest, a rigid cup with a wire or flexible shank is the best choice, unless deflection is minimal. In cases of minimal cranial deflection, any soft-cup extractor may be applied. If extraction from an occiput posterior or

near-posterior position is attempted, a rigid cup is clearly superior. There is an important technical reason for this. If a posterior head is more than minimally deflexed, the usually employed plastic disposable extractors cannot be correctly applied. Their long, rigid handles preclude correct positioning of the midportion of the cup over the cranial pivot point, or if the correct application is possible, traction is by necessity oblique, leading to rapid cup displacement and an extraction failure [41,45].

### CONTRAINDICATIONS AND SPECIAL APPLICATIONS

There are settings in which an instrumental delivery is contraindicated [5,9,33,36,41,80]. The most important contraindications to vaginal delivery operations are operator inexperience and the inability to achieve a proper application. Other important issues include an inadequate trial of labor, uncertainty concerning fetal position or station, or the patient or her family are reluctant to undergo instrumentation.

There are also clinical settings in which specific instruments should not be used. Examples include vacuum extraction applications to the aftercoming head in breech presentations or to the fetal face. The vacuum extractor should also be used with caution in preterm pregnancies, because the data on safety are limited. There is no role for *elective* vacuum operations on premature infants, but the case for indicated procedures is less clear. Based on limited data and the author's experience, it is recommended that vacuum extraction operations not be performed on infants less than 32 to 33 weeks' gestation, and that a soft-cup extractor be preferentially employed if a vacuum procedure is performed on any fetus of less than 37 weeks' gestation [9].

In the evaluation of potential cases, several features predict difficulty, including the likelihood of an extraction failure (Table 17.5). It will come as no surprise that the larger, higher, and more molded the presenting part is, the less likely an instrumental delivery is to succeed, and probably the risk of concomitant maternal or fetal injury is increased. In such cases it is better to attempt to extend the labor if this can be safely accomplished. Otherwise, if progress has ceased or other problems are present, a cesarean is best.

**TABLE 17.5 Prognostically Poor Signs for Successful Instrumental Delivery by Either Forceps or the Vacuum Extractor\***

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Estimated fetal weight >4,500 g
Prolonged second stage of labor
Dysfunctional active phase of labor
Advanced cranial molding
Station above +2/5 cm
Position: occiput posterior, OT; especially if deflexed
Poor maternal expulsive efforts/exhaustion or an overly dense epidural anesthetic

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\*See text for details.

There is a risk of fetal hemorrhage if the vacuum extractor is used after either scalp sampling or the application of a spiral scalp electrode [93,94]. The magnitude of this risk is minuscule, however. Many successful and safe extractions have occurred despite prior scalp sampling or electrode placement. Now that scalp sampling is a rapidly disappearing procedure, the issue of the spiral electrode is the more important. A history of either scalp sampling or electrode placement is not an absolute contraindication to extraction operations but does require a prudent approach. In such cases, a vacuum cup is chosen based on routine criteria and applied in the usual manner. If the scalp electrode does not interfere with correct cap placement, it can simply be left in place on the fetal scalp. Clear vacuum tubing should always be used and occasionally checked during the procedure to be certain that the discharge is not bloody. The observation of a substantial amount of bloody discharge in the tube would be an indication to stop the procedure, reevaluate, and if necessary resort to forceps to complete the delivery. Obviously, were these types of complication to occur, the pediatrician would need to be informed to evaluate the neonate appropriately.

At cesarean delivery, either the vacuum extractor or the forceps can be used to assist cranial extraction [95]; however, this is often not necessary if an adequate incision has been made. When the fetal head is difficult to extract, the surgeon should immediately consider the reasons why. In most circumstances, extraction problems occur because of an initially inadequate incision or a deeply engaged presenting part. In these circumstances, one or more practical steps are appropriate to expedite the delivery. The

choice of how to proceed depends on the clinical circumstances. It is best to either extend the incision sufficiently to avoid struggling, relax the uterus by administering a tocolytic, request assistance in displacing the presenting part, or resort to an instrumentally assisted delivery. Vaginal displacement of the fetal head by an assistant; a vacuum, forceps, or vectis blade extraction; or, in oblique or transverse lies, uterine relaxation with a tocolytic and either a breech extraction or conversion to a cephalic presentation with application of a delivery instrument are both faster and less traumatic than continuing to struggle to extract the fetus manually.

When instrumental assistance is needed during a cesarean delivery and the fetus is cephalic and not too deeply engaged, a vectis blade such as a Murless or a classic forceps are the most convenient instruments. Several types of delivery forceps also can be used, and many operative delivery kits contain short or "baby" Simpson forceps, short Hale forceps, or a similar instrument for these applications. (See Chapter 18, Cesarean Delivery and Surgical Sterilization.)

A vacuum extraction during a cesarean delivery is most appropriate when the fetal head is positioned high in the uterus and difficult to grasp despite an adequate uterine incision. This situation often occurs in the delivery of twins or higher multiples. The application of a forceps or the vacuum extractor to such floating heads is usually easy and the subsequent delivery rapid and atraumatic. If the uterus has firmly contracted around the baby, particularly if the lie is oblique or transverse, uterine relaxation by the intravenous administration of a  $\beta$  mimetic such as terbutaline, or preferably, the more rapid onset tocolytic, nitroglycerin, usually permits an easy and safe instrumental extraction once the fetus is manipulated into a cephalic presentation.

Best practice is to identify these cases in advance and request the anesthesiologist to premix nitroglycerin before beginning the surgery. In the author's experience, a bolus of 150  $\mu\text{g}$  to 350  $\mu\text{g}$  of nitroglycerine IV as the vesicouterine fold is incised results in adequate uterine relaxation just in time for the subsequent extraction of the infant. During a cesarean for a multiple gestation, the author uses this drug routinely. The onset of nitroglycerin is rapid, the effect transient, and, in virtually all cases involving an initially normal mother and infant, it is well tolerated [96,97].

**TABLE 17.6 Prerequisites for Instrumental Delivery Operations**

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Informed consent and an acceptable indication
Application of vacuum extractor cup or forceps centered at the cranial pivot point
Analgesia (as clinically required)
• Local infiltration with vocal reinforcement
• Pudendal nerve block
• Saddle block
• Epidural anesthesia
Operator certain of fetal station and position. Pelvic examination to establish the station, position, and deflection of the fetal head
Empty maternal bladder (Credé maneuver, recent voiding, or catheterization)
Full cervical dilation
Ruptured membranes
Knowledge of fetal heart rate or pattern
The decision to abandon the procedure unless it proceeds easily

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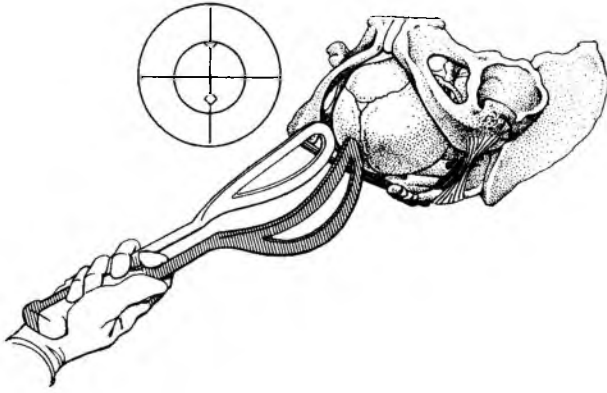
## **CONDUCTING AN INSTRUMENTAL DELIVERY**

The prerequisites for an instrumental delivery include a clear idea of the procedure to be undertaken, knowledge of the dynamics of the delivery (e.g., mechanism of labor, vector of force, required rotation), a favorable clinical setting (appropriate baby size, position, adequate maternal pelvic anatomy, anesthesia), and patient consent (Table 17.6).

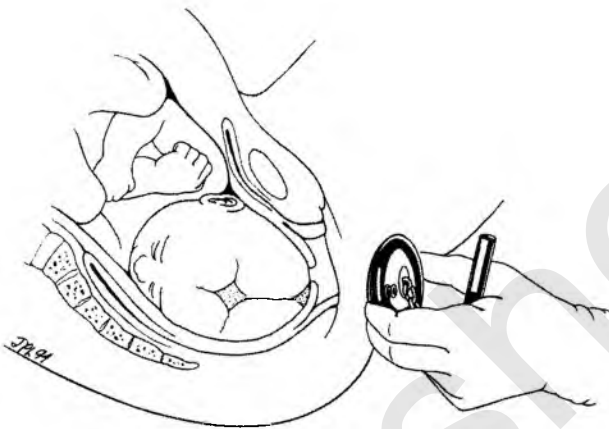
The procedure begins with a ghosting or phantom application (Figures 17.6 and 17.7). The surgeon holds the chosen delivery instrument in front of the maternal pelvis and rotates it to the position that it will occupy when the correct cephalic application is made. As this is performed, the surgeon reviews the proposed procedure and the direction of the vector of traction. The instrument is then introduced into the birth canal. Once the correct application has been made, traction is applied. After delivery, the appropriate documentation is prepared. (Pertinent details are discussed in the sections that follow.)

The dorsal lithotomy position is most common for instrumental delivery but is not absolutely necessary, especially if a vacuum extraction operation is performed. Voluntary voiding, a gentle Credé maneuver, or catheterization should empty the parturient's bladder. Bladder emptying is an important





**FIGURE 17.6.**  
*Occiput anterior position. Ghosting (phantom application) of Simpson forceps. The circular diagram indicates the relative positions of the posterior (triangle) and the anterior fontanelles (diamond). The left forceps blade is shaded. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*



**FIGURE 17.7.**  
*Ghosting or phantom application of a metal cup extractor (Malmström/Bird design cup is illustrated). Left occiput position, transverse (LOT) outlet vacuum extraction. The vacuum hose is not depicted in this illustration.*

prerequisite if a rotational or midpelvic procedure is contemplated, regardless of the type of instrument chosen. Because catheterization risks infection, it should be employed selectively, but when indicated it should be resorted to without hesitation if voluntary emptying is either impossible or believed to be incomplete.

Both forceps and vacuum extraction operations are associated with increased postoperative febrile morbidity. As a consequence, prophylactic antibiotics have been considered. As reported by a recent

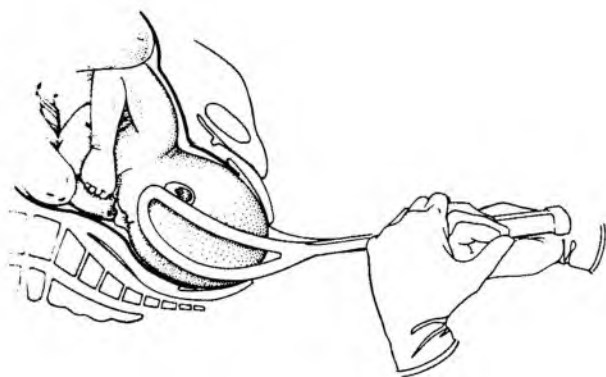
Cochrane Review, however, insufficient data are available on this point to permit practice recommendations [98]. The author does not routinely administer antibiotics in conjunction with instrumental delivery unless there is already a standard indication for treatment, such as an established maternal fever, prolonged membrane rupture, or a positive streptococcal culture.

With proper coaching and a cooperative patient, a pudendal block is often adequate for outlet forceps procedures and sufficient for most low vacuum extractions. Higher or rotational vacuum procedures and most forceps operations require spinal or epidural anesthesia. If the contemplated procedure involves a rotation, if the fetal head is mid-pelvic, or if there is any uncertainty about the likelihood of success, epidural analgesia/anesthesia is best.

In selected cases, outlet vacuum extraction operations can be performed with only local or no anesthesia; general anesthesia should always be avoided. General anesthesia denies the accoucheur the voluntary assistance of the mother, thus increasing the force that the instrument must apply to achieve delivery. In addition, a general anesthetic can unnecessarily depress the infant if the extraction is delayed for any reason.

Traction is timed to contractions. Tension on the blades or to the vacuum extractor handle mirrors the uterine contraction – a slow incremental pull builds to full pressure with a subsequent relaxation in force as the contraction abates. Traction applied without accompanying contractions or concomitant recruitment of maternal bearing-down efforts or jerking of the handles of the forceps or the vacuum extractor is inappropriate. The author favors a technique with only a single sustained traction effort accompanied by maternal bearing-down efforts during each uterine contraction.

When the forceps are used, the force for delivery is provided by the operator's arm, with the elbow bent at a right angle. If classic blades have been applied, some operators place a folded towel between the articulated blade handles to reduce compression of the fetal head. The other hand rests on the shank of the blades and presses downward (Saxtorph-Pajot or Osiander maneuver; Figure 17.8). This maneuver creates a vector of force guiding the fetal head through the pelvic curve (curve of Carus). As the delivery progresses, the angle of pull



**FIGURE 17.8.**  
*Occiput anterior position. Saxtorph-Pajot (Osiander's) maneuver. Paralleling the pattern of uterine contractions, one hand pulls horizontally while the second adds downward force over the lock. This ensures that the vector of force follows the natural pelvic curve (curve of Carus). (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

is modified either toward the symphysis, or alternatively in the direction of the perineum as resistance is felt and the presenting part descends.

The forceps handles should not be rocked up and down during the delivery because the posterior toe of the blade can injure the posterior vaginal vault as the fetal head descends. In a tight fit, some operators employ a slight side-to-side motion during traction, which is probably harmless. The fetal heart should either be auscultated or checked by real-time ultrasound scan, a hand-held Doppler device, scalp electrode recording, or direct auscultation before the operation begins, and between contractions/pulls. The blades are relaxed and disarticulated between contractions at the operator's discretion.

For vacuum extraction, once a correct cup application is established, full suction and traction immediately follow [99,100]. With soft-cup instruments, it is not necessary to wait an arbitrary period for the development of a chignon. Rapid applications of vacuum versus the traditional stepwise technique does not affect maternal internal or neonatal morbidity [99]. For rigid-cup instruments, either metal or plastic, the classic technique was to increase the vacuum by  $0.2 \text{ kg/cm}^2$  every 2 minutes once the cup was correctly applied until the full vacuum force was reached [101]. An alternative approach is to apply full vacuum within 2 minutes of the cup application and without additional delay for chignon formation. Both techniques are acceptable.



**FIGURE 17.9.**  
*Vacuum extractor traction technique using a Malmström type cup applied to a fetal head in the occiput anterior position is illustrated. Note position of the operator's fingers for the vaginal hand as traction is applied. (From O'Grady J, Gimovsky ML, McIlhargie CJ: Operative Obstetrics. Baltimore: Williams & Wilkins; 1995; with permission.)*

A two-handed technique for vacuum extraction is recommended (Figure 17.9). The vector of traction force should follow the pelvic curve in precisely the same fashion as for forceps. During the extraction, the surgeon places the nondominant hand within the vagina, palpating the fetal scalp with one or more fingers while placing a thumb and remaining fingers on the extractor cup to gauge the relative position of the cup edge to the scalp [102]. So positioned, the operator can better judge the appropriate angle for traction while detecting early cup separation. The bimanual technique reduces the risks from sudden cup displacement and is recommended for all vacuum extraction operations. Full vacuum ( $0.8 \text{ kg/cm}^2$ , 550 mm Hg–600 mm Hg,  $11.6 \text{ lb/in}^2$ ) can either be maintained or reduced to  $\leq 200 \text{ mm Hg}$  between contractions at the surgeon's discretion. When tested in randomized trials, both techniques prove similar in successful delivery and maternal/neonatal outcome [103]. For both forceps operations and vacuum extractions, an episiotomy is electively performed as the posterior perineum bulges, but only if maternal soft tissue impedes the descent of the presenting part. (See Chapter 23, Birth Injuries.)

As a general rule, vacuum cups should not be left applied to the scalp for longer than 20 to 30 minutes [41,80,104]. Prolonged extraction time risks shoulder dystocia and an increased risk for fetal scalp injury [85,105]. The level of risk for a scalp injury is probably lower with soft-cups than with the rigid-cup devices. Time limits for plastic or soft-cup applications have not been well established. Nonetheless, when one of these instruments is chosen, it is prudent not to exceed the proposed 20- to 30-minute limit. Twenty minutes is usually ample time for four or more tractions. If progress has not been easily made, and the child has not been successfully delivered or the presenting part drawn well down and very near delivery with four to five efforts, close reevaluation of the procedure is always necessary, and the operation may well need to be abandoned.

In consideration of these suggested time limits, the clinician must make reasonable choices. If progress is progressive but slow, he or she need not necessarily discontinue the extraction at the exact moment the 30th minute is reached. It is simply suggested that the majority of ultimately successful extractions will have occurred before this time. Thus, the 30th minute is an important marker. When this time is reached, the clinician needs to closely consider if true progress is being made or whether the extraction is doomed to failure.

Regardless of the instrument chosen, descent must begin with either the first or at least by the second traction effort. Failure to promptly make station as force is applied requires immediate reassessment [41,45,63,106]. In vacuum operations, repetitive episodes of what Bird termed *negative traction* must be avoided [106]. *Negative traction* is force that draws the fetal scalp away from the fetal skull but fails to result in descent of the presenting part. This is believed to result in rapid pressure fluctuations within the fetal cranium and can avulse bridging veins. These effects are suspected to predispose to intracranial or subgaleal hemorrhage and the formation of cephalohematomas.

When the fetal head fails to advance despite what is believed by the operator to be proper traction, there is a reason. There is possibly a technical problem in the vector of force, the cup is wrongly positioned, or a degree of disproportion is present. In this setting, the surgeon must carefully reassess the application, reconsider the vector of traction, and review the wisdom of further efforts.

TABLE 17.7 Checks Prior to Forceps Traction

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<i>Checks</i> for adequate anesthesia and correct maternal positioning
<i>Checks</i> to be certain that bladder distention is not present
<i>Checks</i> for cranial flexion
<i>Checks</i> mentally, rethinking the maneuvers necessary for the contemplated operation
<i>Checks</i> that the mother's assistance and attention are recruited
<i>Checks</i> for correct application:
• Midline position of the sagittal suture
• Only one finger insertion at blade fenestration
• 1.0–2.0 cm from the plane of the shanks to the posterior fontanelle edge
<i>Checks</i> fetal heart rate/rhythm
<i>Checks</i> by pelvic examination to ensure that nothing lies between the fetal head and the forceps (e.g., umbilical cord, cervix, membranes)

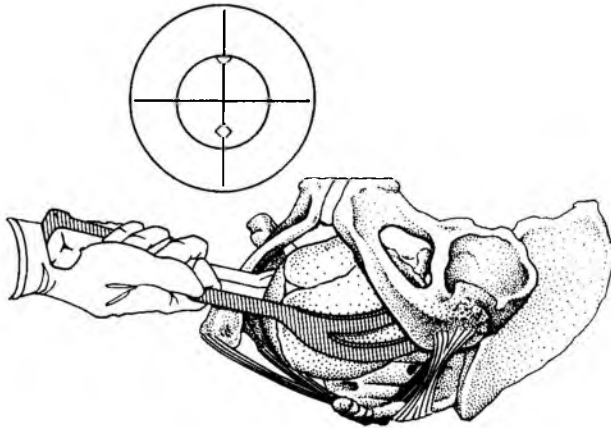
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### Applications

Proper application of both the forceps and the vacuum extractor is critical to safety and success [14,33,34,36,80,107]. A correct application for either the forceps or the vacuum extractor requires knowledge of fetal cranial anatomy and the ability to identify important landmarks by palpation. Traction must never be applied until the surgeon is convinced that the application is proper.

A correct forceps application (biparietal or bimalar) evenly distributes the compressive force generated by the blades over the fetal head. The fit between the fetal head and the fenestration of the blades, the location of the posterior fontanelle, in reference to the plane of the shanks, and the position of the sagittal suture are the components of the classic "checks" for forceps (Table 17.7). This is a *cephalic* application and distinct from a *pelvic* application. In the latter, the forceps are applied independently of knowledge of the exact position of the fetal head. The only currently acceptable pelvic application occurs when Piper or Kjelland forceps are applied to the aftercoming head in a breech presentation.

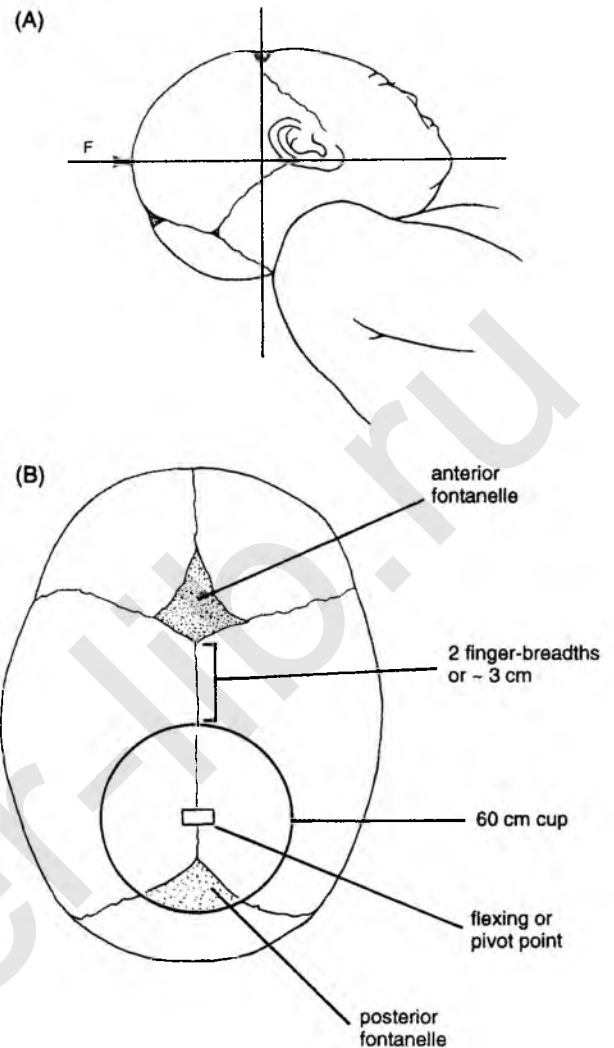
When the forceps blades are correctly applied, the tips of the forceps blades lie over the fetal cheeks, with the upper or concave border of the blade directed either toward the fetal occiput in anterior positions, or toward the face in posterior positions. The biparietal diameter of the fetal head



**FIGURE 17.10.**  
*Correct biparietal, bimalar, cephalic forceps application. Note that the plane of the shanks passes through the cranial pivot or flexion point. See text for details. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

fits in the center of the cephalic curve of the instrument (Figure 17.10).

For both the vacuum extractor and forceps, when the application is correct, the vector of traction force passes through the *flexing or pivot point* of the fetal skull (Figure 17.11) [5,45,108]. The pivot point is an imaginary site approximately 6 cm behind the edge of the anterior fontanelle or approximately 1.5 cm to 2.5 cm in advance of the posterior fontanelle centered over the sagittal suture. When the forceps are applied correctly, the pivot point lies in the middle of a plane that connects the center or widest diameter of the cephalic curve of the blades and the plane of the shanks. When traction is applied, if the pivot point of the head is not in the center of the blades, the fetal head is either overextended or alternatively excessively flexed when traction is applied. A correct forceps application also requires attention to how the blades fit to the fetal head. Normally, one can insert only one fingertip between the fenestration of the blade and the fetal head. If too much of the fenestration is palpable, the blade is not correctly applied or the fetal head is very small. If one blade is misapplied over the brow and the other over the occiput, the instrument cannot be locked or articulated, or, if somehow approximated, the blades usually slip off when traction is applied and could injure the infant. Correctly applied, the forceps fit easily and do not slip with normal traction, and fetal injury is avoided.



**FIGURE 17.11.**  
*As illustrated, the flexing or pivot point (F) of the fetal head is located midsagittally, approximately 6 cm from the center of the anterior fontanelle or 2 cm in advance of the posterior fontanelle. When a standard vacuum cup is applied, the cup edge will lie approximately 3 cm or two finger-breadths behind the anterior fontanelle. The posterior fontanelle is often covered by a correctly applied cup and is thus not useful as a landmark. B, This indicates the same site as viewed from above. Traction centered over this site by either a forceps or the vacuum extractor cup promotes cranial flexion and presents the smallest cranial diameter to the pelvis. See also Figure 17.12. (From O'Grady J, Gimovsky ML, McIlhargie CJ: Vacuum Extraction in Modern Obstetric Practice. New York: Parthenon Publishing Group Inc. 1995; with permission.)*

On molded heads, especially of larger infants, the best application is usually obtained with blades that have a long, tapering cephalic curve such as the Simpson forceps. Forceps with a short, full curve (Elliot-type) might not fit evenly and could result in points of increased pressure. Furthermore, blades that are too short for a heavily molded head might not be properly anchored below the malar eminences, risking laceration or slippage when traction is applied. The relative importance of these minor points of instrument choice in avoiding injury or ensuring success is unknown. There are no objective data on the issue, only various authors' opinions. Except for extreme cases, it is likely that these differences in blade construction and fit to the fetal head are only consequential in difficult pulls, exactly the type of procedures that should be avoided in modern practice.

When a vacuum extractor is employed, cranial traction is vectored through the pivot point of the fetal head by centering the cup in the midline, over this site [41,45,108]. The vector of force is thus oriented along the midline of the sagittal suture. Oblique traction to the fetal head simply increases the work of extraction, risks cup displacement, and increases the chance of fetal injury (Figure 17.12). When correctly placed, a standard 60-mm vacuum cup is positioned midline over the sagittal suture, with the edge of the cup lying approximately 3 cm from the edge of the anterior fontanelle. Thus in vacuum extraction operations, the *anterior fontanelle* becomes the reference point for checking instrument application. Depending on fetal anatomy, access to the posterior fontanelle is partially or entirely blocked by the extractor cup, making this familiar landmark unusable for judging the accuracy of cup placement.

### USE OF FORCE

Educating clinicians in the appropriate use of force in instrumental deliveries is a difficult task. In vacuum extraction operations, safety is best ensured by careful cup placement and by strictly limiting the surgeon's efforts in number of tractions, cup displacements, and the total period of cup application. For forceps, meticulous adherence to traction technique and limiting the total number of efforts are similarly appropriate steps. The use of force is a serious issue in instrumental delivery, because

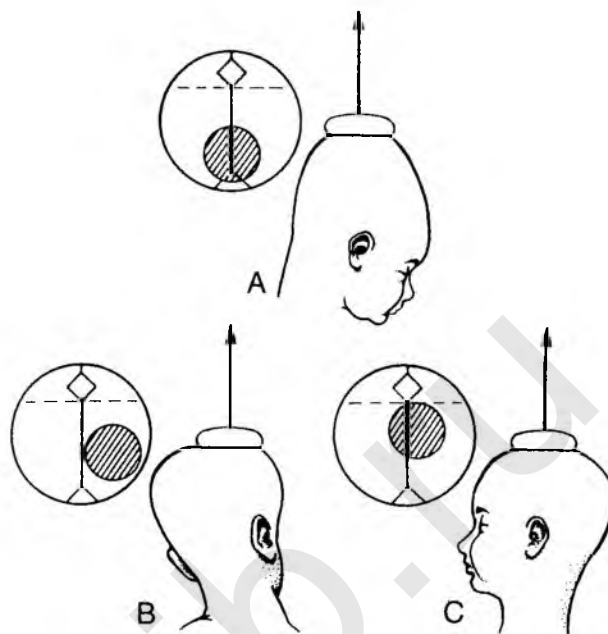


FIGURE 17.12. Relationship between the vacuum cup placement and flexion/deflection and asynclitism of the fetal head as traction is applied. A, Correct application over pivot point; B, oblique application; C, deflexing application. A rigid metal (Malmström) cup is depicted; however, the principles of placement are valid for all types of vacuum extractors. See text for details. (Modified from Bird, G. C., The use of the vacuum extractor. *Clin Obstet Gynaecol* 1982; 9:641-661; with permission.)

the degree of force applied has some correlation with the risk for fetal trauma and maternal injury [5,45,75,108].

The primary function of both obstetric forceps and the vacuum extractor is to *augment and not replace the natural forces of labor*. To reasonably ensure safety, several simple precautions are necessary to control the degree of force and ensure the correct vector of traction. For forceps, traction should never be greater than that accomplished by the operator's flexing his/her forearm. Operators should not brace their feet, and the force exerted should never be great enough to move the parturient's hips from the edge of the operating table or bed. While a firm pull is at times required, the surgeon is easily capable of successfully delivering an infant with forceps without ever taxing his/her strength. Periods of relaxation, corresponding to the intermittent rhythm of the uterine contractions, are important and permit the fetus to recover from the combined effects of traction and the uterine contraction.

**TABLE 17.8 Number of Traction Efforts Required to Achieve Delivery in 1,497 Cases of Assisted Vacuum Extraction and Forceps Delivery\***

No. of Traction Efforts	Malmström Vacuum Extractor (N = 433)	Forceps (Type Unspecified) (N = 555)
1-2	296 (68.4%)	213 (38.4%)
3-4	108 (24.9%)	270 (48.6%)
≥5	29 (6.7%)	72 (12.9%)

\*Neonates  $\leq 600$  g were excluded; other exclusions include breech presentations, cesarean deliveries, transverse lies, and multiple gestations. Twins were included if each weighed  $\geq 600$  g and if one was delivered spontaneously, in a cephalic presentation.

Modified from Sjostedt JE: The vacuum extractor and forceps in obstetrics: A clinical study. *Acta Obstet Gynecol Scand* 1967; 46(Suppl 10):3-206.

In theory, as long as station is continually reevaluated and the fetal heart rate and pattern are acceptable, there is no absolute limit to the period of time for a forceps application or to the number of traction efforts; however, the incidence of trauma and failure increases rapidly if the number of tractions exceeds four [75]. *In most cases, if delivery has not occurred or is not imminent following the fourth complete traction effort, careful reconsideration is mandatory.* If no descent occurs on the initial attempt or certainly by the second effort, assuming adequate traction in the correct vector of force with a correctly applied instrument, the procedure must immediately be reassessed (Table 17.8). As stated previously, the instrument is improperly applied, the vector of traction is incorrect, or a degree of disproportion exists.

A common problem in vacuum extraction operations is incorrectly oriented traction efforts. In vacuum procedures, if the angle of the vector of force applied by the operator too anterior or too posterior, descent is difficult or impossible, and cup displacement is likely. If traction is oriented too anteriorly, the operation fails as the fetal head is pulled against the unyielding resistance of the bony pelvis. Alternatively, a pull oriented too posteriorly is also unsuccessful or difficult due to perineal soft-tissue resistance. The risk of maternal or fetal injury is also increased by poorly directed traction efforts. A posterior angulation risks unnecessary maternal perineal injury. Oblique traction causes the vacuum

**TABLE 17.9 Vacuum Extraction: Clinical Applications**

Outlet and low procedures\* (OA, or rotation  $<45^\circ$ )

- Soft cup extractor
- Rigid cup extractor

Low and mid procedures (OP, OT)

- Rigid cup extractor
- Cesarean delivery
- Soft cup extractor
- Rigid cup extractor

OP, occiput posterior; OT, occiput transverse; OA, occiput anterior

\*Procedure coding per modified ACOG criterion, see Tables 17.2 and 17.3.

See text for details.

cup to lift on one edge, predisposing to cup displacement (pop-off) and an increased risk for fetal scalp or intracranial injury.

Instrumental vaginal delivery is no place for uncertainty, heroics, or tests of strength. If the surgeon cannot determine fetal position with accuracy or achieve proper cranial application of a forceps or vacuum extractor, or if the delivery proves difficult for any other reason, the procedure must be abandoned.

### Choice of Instrument

The choice of best delivery instrument requires 1) an understanding of the physics of the contemplated procedure, 2) consideration of the skill and experience of the accoucheur, and 3) review of the clinical circumstances, including maternal condition/acceptance, anesthesia, station, molding, and position. There are many different delivery instruments in the surgical armamentarium, of which some are better suited to certain applications than others. Throughout this text, the potential advantages or disadvantages of various designs of vacuum extractor or forceps have been discussed in detail. The current recommendations for instrument use reflect our clinical practice (Tables 17.9 and 17.10). In skilled hands, several forceps types can be used safely and successfully in different applications. Certain training programs often favor a specific forceps design or modified design in their training, thus permanently influencing the instruments chosen by their graduates. With some exceptions, operator skill is much more important a consideration than the instrument employed. For local reasons or physician

TABLE 17.10 Forceps: Suggested Clinical Applications\*

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Outlet and low procedures (OA <sup>†</sup> ; rotation $\leq 45^\circ$ )
• Classical forceps (any type)
Low and mid procedures (OP; no rotation)
• Classic forceps (any type)
• Axis traction forceps (use now rare)
Low and mid procedures (OT or OP with rotation $>45^\circ$ )
• Keilland <sup>‡</sup>
• Tucker McLane <sup>‡</sup>
• Classic forceps (any type) <sup>‡</sup>
• Barton (selected OT only) <sup>‡</sup>
Breech delivery
• Piper
• Keilland
At cesarean delivery
• Murless vectus blade
• Classic forceps

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\*See text for details.

<sup>†</sup>OP, occiput posterior; OT, occiput transverse; OA, occiput anterior.

<sup>‡</sup>These procedures restricted to the highly experienced only.

TABLE 17.11 Checks Prior to Vacuum Extraction Delivery

Checks for correct application:

- The vacuum port of a Malmström-design cup, or the handle of a soft-cup extractor, is directed to parallel the sagittal suture.
- No maternal tissue is included under the cup margin.
- The middle of the cup is positioned over the cranial pivot/point. This is midline over the sagittal suture with the cup margin 3 cm from the edge of the anterior fontanelle (see Figure 17.12).

Checks fetal heart rate/rhythm

Checks by pelvic examination to ensure that nothing lies between the fetal head and the vacuum extractor (e.g., umbilical cord, cervix, membranes).

Checks for adequate anesthesia and correct maternal positioning

Checks to be certain that bladder distention is not present

Checks for cranial flexion

Checks mentally, rethinking the maneuvers necessary for the contemplated operation

Checks that the mother's assistance and attention are recruited.

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preference, certain types of procedures are no longer be performed in certain obstetric services, rendering recommendations in certain categories inapplicable (e.g., some practitioners choose not to perform rotational or midpelvic instrumental deliveries).

For the purposes of this discussion, forceps can be divided into *traction designs* (e.g., classic forceps) and *rotators* (e.g., Keilland, Tucker McLane). Specialized forceps, such as the Piper or Barton, are now rarely applied but are useful in specific but restricted applications. Axis-traction instruments are mentioned only for completeness since they have become a *rara avis* in most obstetric services but are occasionally used by a traditionally trained and usually more senior obstetrician.

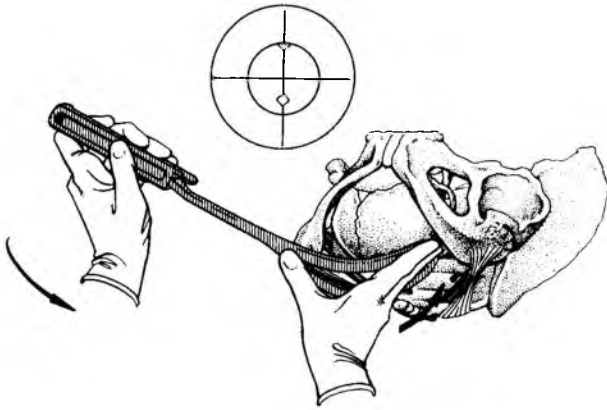
The case for vacuum extractors is in many ways similar; however, the important distinction in the choice of extractor is which instrument can be accurately applied in the specific clinical setting. The position and degree of deflection of the fetal head is critical. As discussed in more detail elsewhere in this text, for safety and success, the center of the vacuum cup must be positioned over the flexing point of the fetal skull. Extractors with rigid connectors between the cup and handle are precluded from a proper application involving occiput posterior or occiput transverse (OT) positions, especially when cranial deflection is present. When the head is malpositioned, a freely movable rigid-cup design is much more likely to achieve a correct application because it can be advanced either posteriorly or laterally, as required, to fit over the flexing point. Even with episiotomy, the cup portion of these extractors cannot be flexed or rotated sufficiently to achieve other than an imperfect and often oblique application. There are no scientific or clinical data to favor the use any of the available soft extractors distributed by the various manufactures over another. For these instruments, commercial consideration rather than clinical evidence for safety or efficacy has driven the proliferation of models. Clinicians should choose whichever design appeals to them, with cost as the major criterion for purchase.

## INSTRUMENT APPLICATION

### Forceps Operations

#### *Outlet Forceps Application to the Occiput Anterior Position*

The outlet operation is the basic forceps procedure. The techniques for the performance of this instrumentation have not changed appreciably in many years [34,35,110].

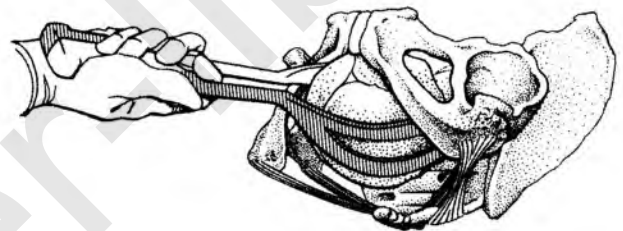


**FIGURE 17.13.**  
*Occiput anterior position; Application of left forceps blade. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*



**FIGURE 17.14.**  
*Occiput anterior position. The left blade is already in place. The right forceps blade is inserted. Articulation of the blades follows. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

1. Prior to forceps application, the operator performs a vaginal examination to assess position, station, and cranial molding. Adequate anesthesia is verified, and the operator ensures that the maternal bladder is empty. The contemplated procedure is briefly reviewed with the parents, and the mother's assistance in bearing down on request is recruited. If a pediatrician or other additional personnel for infant support and resuscitation are thought necessary, the surgeon ensures that they are already present or have been appropriately summoned.
2. The blades are ghosted prior to attempting insertion (see Figure 17.6). Regardless of the experience of the operator, or the speed demanded by the clinical circumstances, this step must never be omitted.
3. The left or posterior blade is selected first and lubricated. Between uterine contractions, the surgeon's right hand passes into the vagina, the fingers opening the potential space between the fetal head and the vaginal sidewall (Figure 17.13). The right hand then walks the blade between the fetal head and the pelvic sidewall, displacing the maternal soft tissue with firm but gentle finger pressure. The operator's first two fingers lie along the leading edge of the blade, with the thumb on the shank. The handle of the blade is swept gently down as it passes into the pelvis. Once the surgeon's hand is properly inserted into the vagina,



**FIGURE 17.15.**  
*Occiput anterior position. The forceps application is checked for accuracy prior to traction (see Table 17.9). (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

the blade will advance almost by its own weight, with minimal force.

4. Once the blade is introduced, its position can be readjusted between contractions as required. To readjust the blade position, it is important that the handle is held loosely and that the shank of the blade be manipulated only by finger pressure of the vaginal hand.
5. Next, the right blade is lubricated and inserted in the same fashion (Figure 17.14). The right-sided blade is introduced *above* the plane of the previously inserted left blade so that the lock can be easily articulated.
6. The forceps are then articulated. Immediately prior to traction, the operator checks for proper application (see Table 17.7, Figure 17.15). If the





FIGURE 17.16.

*Forceps delivery with a modified Ritgen maneuver. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

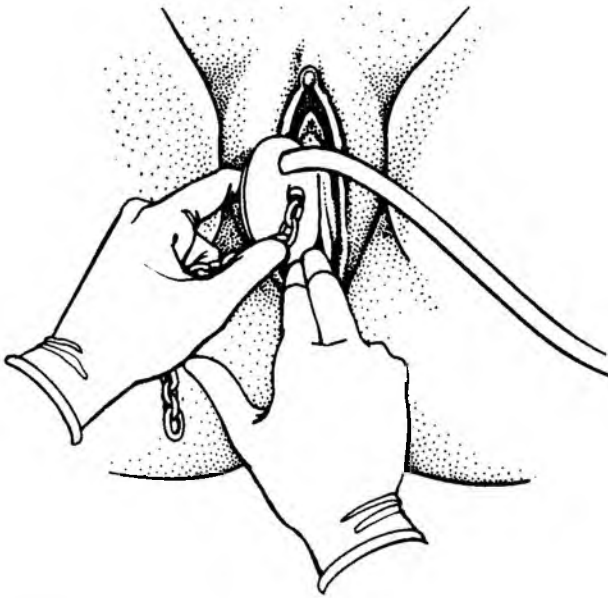
application is imperfect, the blades must be disarticulated and readjusted by wandering, using finger pressure along the lower curve of the blade. Cranial deflection is corrected prior to traction. Once correctly applied, electively a towel can be folded and placed between the handles to reduce cranial compression after the blades are articulated.

7. As the head crowns, an episiotomy can be performed, if required. A modified Ritgen maneuver secures the chin. The mother is next instructed not to push (Figure 17.16). The fetal head is then delivered *slowly* and the forceps removed. Restitution of the head and delivery of the infant's thorax and abdomen follow.
8. Following delivery of the placenta, the entire birth canal (i.e., cervix, vaginal vault, and perineum) should be carefully examined under good light. The episiotomy, if performed, and any perineal lacerations are then repaired. Finally, the rectum is digitally examined to ensure that both the mucosa and the external sphincter are intact.
9. A full operative note is then dictated and an appropriate notation made in the medical record. This completes the operation.

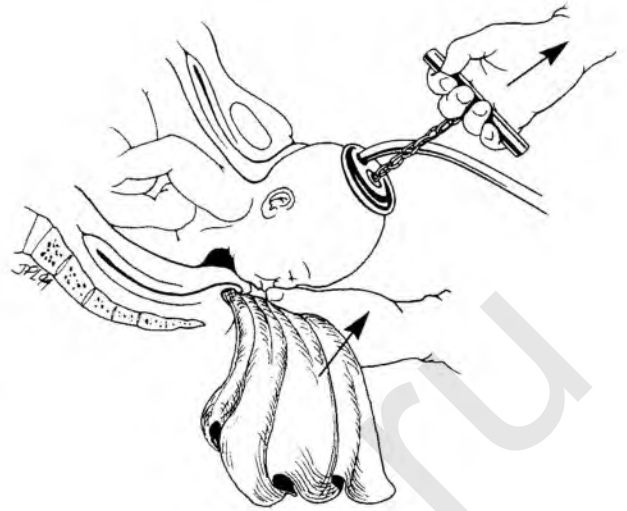
### Vacuum Extraction

A description of the outlet vacuum extraction procedure follows. As with forceps, the basics of the operation are well established [40,41,45,63, 80,108].

1. As the patient is prepared for delivery, a repeat pelvic examination is performed, noting position, station, and cranial molding. The degree of maternal discomfort is judged, and anesthesia/analgesia is administered as necessary. Bladder filling is judged, and catheterization, spontaneous voiding, or the Credé maneuver is performed as required for emptying. The contemplated procedure is briefly reviewed with the parents, and the mother's assistance in bearing down on request is recruited.
2. A ghost or phantom application of the vacuum extractor is then performed prior to the attempt at cup insertion in the same fashion as for forceps operations (see Figure 17.17). Regardless of the experience of the operator, or the speed demanded by the clinical circumstances, this step should never be omitted. Traditionally, either the vacuum port of a Bird-type cup or the handle of a rigid plastic extractor is positioned pointing toward the fetal occiput as a convenient marker of cranial rotation.
3. If an external pump is used, the operator (or assistant) first checks the function of the vacuum pump, and the vacuum hose (if required) is attached. The cup is generously lubricated with surgical soap or a lubricating gel. Soft cups are partially collapsed for introduction through the separated labia. A rigid cup is rotated laterally and slipped into the vagina (Figure 17.17). Once the cup is introduced, it is maneuvered until it is tentatively positioned on the fetal head in accordance with the established landmarks. Once the surgeon is certain that all maternal tissue has been excluded, an initial suction of 200 mm Hg or less is applied, just sufficient to fix the device to the scalp.
4. Prior to attempting traction, the operator performs the standard checks for proper application (see Table 17.4).
5. Full vacuum is applied, and two-handed traction then follows, timed to parallel the uterine



**FIGURE 17.17.**  
*Outlet vacuum extraction. Inserting metal cup vacuum extractor through the labia. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*



**FIGURE 17.18.**  
*Modified Ritgen maneuver and perineal management during a vacuum extraction operation using a Malnström/Bird vacuum extractor. The cup and chain assume a near 90° angle to the birth canal as the head extends.*

contractions. Traction in the pelvic curve accompanies instruction to the parturient when to bear down. The traction force is applied progressively, paralleling the uterine contraction, and released as the uterus relaxes. A single traction effort for each uterine contraction is recommended.

6. As the head crowns, an episiotomy is performed if required. A modified Ritgen maneuver secures the chin and the mother is instructed not to push (Figure 17.18). The head is then delivered *slowly* and the vacuum cup removed. Restitution of the head and delivery of the infant's thorax and abdomen follow.
7. Following delivery of the placenta, the entire birth canal (cervix, vaginal vault, and perineum) should be carefully examined under good light to exclude injuries. The episiotomy, if performed, and any perineal lacerations are then repaired. Finally, the rectum is digitally examined to ensure intactness.
8. If a pediatrician is not present for the delivery, he/she should be notified by either the surgeon or a designee that a vacuum extraction delivery has taken place.

9. A full operative note is then made in the medical record. This completes the operation.

#### **Forceps or Vacuum Extraction Operations with Rotation of 45° or Less**

In accordance with the ACOG guidelines (with modifications specific for vacuum operations), if the fetal head has reached the pelvic floor with the scalp visible at the introitus, instrumental delivery procedures from this position are coded as outlet forceps or outlet vacuum extraction operations respectively. If the head is at station +2/5 cm or more but the requirements for outlet forceps are not met, then the procedure is reported as a low forceps or low vacuum extraction operation.

#### ***Vacuum Extractor Operation***

Vacuum extraction technique is the same for cranial positions that normally require rotation (e.g., right occipitoanterior [ROA], left occipitoanterior [LOA], left occipitotransverse [LOT], right occipitotransverse [ROT]) as for OA positions. The cup is applied in the usual fashion, the standard checks are made, and traction is applied. In a successful extraction, the fetal head will spontaneously rotate to the usual OA position as the presenting part

descends [41,63]. Some operators assist this spontaneous rotation by combining the vacuum extraction with digitally applied cranial pressure to gently guide the head in the correct direction, but this is usually not necessary. Attempts to rotate the head by applying rotational force to the vacuum cup or handle should not be performed. These efforts simply promote cup displacement or scalp injury and are unnecessary.

### ***Forceps Operations***

If forceps are chosen for the delivery and the fetal head lies in an obliquity, the posterior blade should always be applied first because this part of the application is usually the most difficult. In the LOA position, for example, the left parietal bone is posterior and the left forceps blade is introduced first. For ROA, the reverse is the rule.

### **Low Forceps and Vacuum Extraction Operations with Rotation More Than 45° and Midforceps Operations**

The cardinal principles in forceps rotation, regardless of the number of degrees of rotation required, include correct application, minimal force, careful attention to the pelvic curve of the chosen instrument, and the willingness to reassess or abandon an apparently difficult operation. An important point is the construction of the forceps that are applied for the rotation. The pelvic curve of most forceps blades demands a substantial axis of rotation for the shanks and handles. The operator must account for this physical feature to avoid a maternal injury from the forceps blades as the rotation is performed. This is not a problem if a Keilland forceps is used because this instrument avoids this problem by lacking a pelvic curve.

If the fetal head is at station +2/5 cm or greater, and the proposed rotation is more than 45°, this procedure is considered a low forceps or vacuum extraction operation with rotation beyond 45°. When the fetal head is engaged but the criteria for a low forceps or vacuum extraction are not met, then the procedure is coded as a midforceps or midpelvic vacuum extraction operation.

The potential hazards of the higher extractions and major rotational deliveries are well known and include an increased danger of both maternal and

fetal trauma. Several potential management plans for deep transverse arrest exist, including both operative and nonoperative techniques. Initially, as long as progress continues and the maternal and fetal condition are good, the best plan is watchful expectancy and oxytocin stimulation. If prolonging the course of labor because of maternal or fetal reasons or administering oxytocin is not possible, or if progress has ceased, then vacuum extraction, the application of forceps, or a cesarean delivery are considered [15].

A transverse arrest is usually due to relative disproportion, inadequate uterine activity, overly dense epidural anesthesia, or some combination of these situations. Platypelloid and android pelvises predispose to transverse arrests. The fetal head can also be discovered at the transverse position during a spontaneous rotation from an originally posterior position, or in a delayed rotation commencing from the transverse. In a flat pelvis, the fetal head engages in the transverse position and descends through the midpelvis fixed in this position because of the anteroposterior narrowing. In the extreme platypelloid pelvis, spontaneous rotation occurs only at the outlet. Android pelvises predispose to transverse arrest because cranial rotation can be blocked by the sacrum and the narrow interspinous pelvic diameter.

If there is a transverse arrest and the fetal head is unengaged, especially if descent has not occurred after stimulation, cesarean delivery is indicated. If the head has descended below the level of the ischial spines, however, instrumental delivery either by a vacuum extraction or a forceps operation is a possibility, but only for experienced surgeons. Most midpelvic forceps rotations are easy and can be achieved with minimal maternal and fetal risk [46,111–113]. The operator must carefully assess pelvic capacity, the position and station of the fetal head, the maternal and fetal condition, and weigh the limits of his/her own skill prior to any attempted application, however. Cranial deflection or asynclitism, which is common in transverse arrests, should be corrected prior to either forceps rotation or traction.

As discussed previously, special care is required because misdiagnosis of the fetal station is common, especially if anterior or posterior cranial asynclitism is present. Descent of the anterior parietal bone can fool the examiner into believing that the fetal head lies at a lower station. Only with careful posterior pelvic examination and subsequent abdominal palpation or with real-time ultrasound scanning is it

found that the fetal head fails to fill the hollow of the sacrum and is actually higher in the pelvis than originally believed, or positioned in a different manner than the operator originally diagnosed.

Prior to performing an instrumental rotation, the obstetrician can attempt manual rotation. Because of its safety, simplicity, and occasional success, simple digital rotation is reasonable to attempt. On occasion, the effort corrects a transverse midpelvic procedure, changing it into a less difficult OA or oblique application, even if a full rotation is not possible. In a manual rotation, the surgeon inserts two fingers alongside the posterior parietal bone when the fetal head is in an oblique-to-transverse presentation, or along either parietal bone when the fetal head is in the occiput posterior position. Accompanying a contraction and voluntary bearing down, pressure is exerted against the lambdoid suture/parietal bone to rotate the head toward an OA or anterior oblique position. Occasionally, fundal pressure can assist in fixing the fetal head in the new position if the rotation is successful.

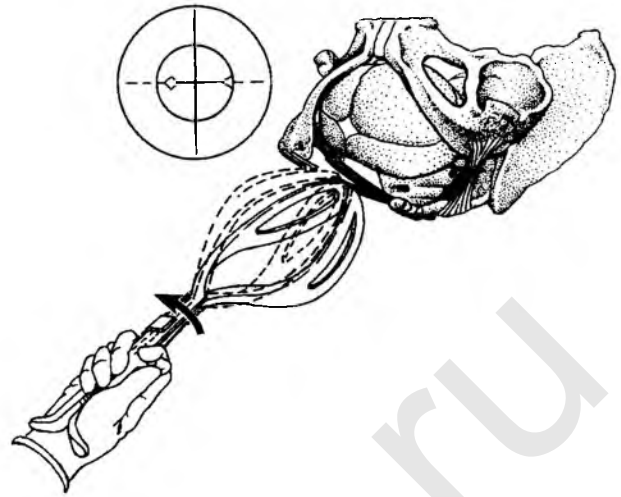
More complicated techniques for rotation exist, usually involving displacing the fetal head from the pelvis and then reintroducing it in a more favorable position. These manipulations almost invariably lose station, risk cord prolapse, and usually cannot be tolerated without anesthesia, however. For these reasons, such procedures are not recommended.

### **Transverse Arrests**

Forceps applications to a transverse head are usually performed with a modified classical forceps such as the Tucker-McLean or Kielland. For the unusual transverse arrest in a true platypelloid pelvis, the Barton forceps is the instrument of choice but is rarely used.

In a reversal of the usual rule for forceps applications, in transverse positions, it is the *anterior* blade, abutting the bladder and urethra, that is introduced first. This approach is taken because if the anterior application fails, the procedure will fail. Introduction of the posterior blade is usually easier, but this application can displace the head to a higher station, potentially compromising the more difficult anterior blade application.

When a Tucker-McLean or other classic forceps is applied to the occiput transverse position, the usual manner of blade insertion and wandering is



**FIGURE 17.19.** *LOT position. Ghosting Kielland forceps. The operator orients the buttons toward the occiput. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

followed. After ghosting, the anterior blade is initially inserted posterolaterally in the usual manner, then wandered into the correct position. The introduction of the posterior blade follows, and the blades are articulated. The application is checked for accuracy and adjustments are made, as required. Rotation follows between contractions with due attention to maintaining a fixed angle between the shanks/handles and the plane of the pelvis. This helps to avoid high birth canal lacerations from the tip of the blades as the forceps rotate through the pelvis. Once the head has reached OA, traction is applied and the delivery completed in the usual manner.

The Kielland forceps differs from the classic instruments in that this instrument lacks a pelvic curve. Although this makes rotation easier, it makes the Kielland an indifferent traction forceps, and modified technique is necessary. Initially, the Kielland forceps are ghosted against the perineum, with the buttons on the shanks oriented toward the fetal occiput (Figure 17.19). There are several techniques for the application of the anterior blade. Most often, the anterior blade is simply inserted posterolaterally and wandered into place. The anterior blade of the Kielland forceps can also be inserted by inversion or by the direct method (Figure 17.20). In inversion, the anterior blade is turned concave side upward and slowly advanced through the cervix into the uterus

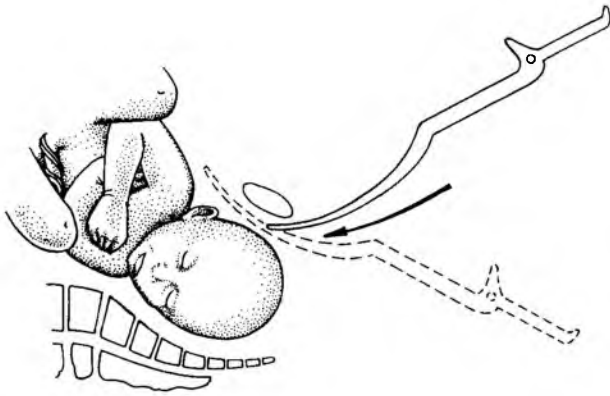


FIGURE 17.20.  
 LOT position. Kielland forceps, inversion technique, initial insertion of anterior (right) blade. See text for details. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)

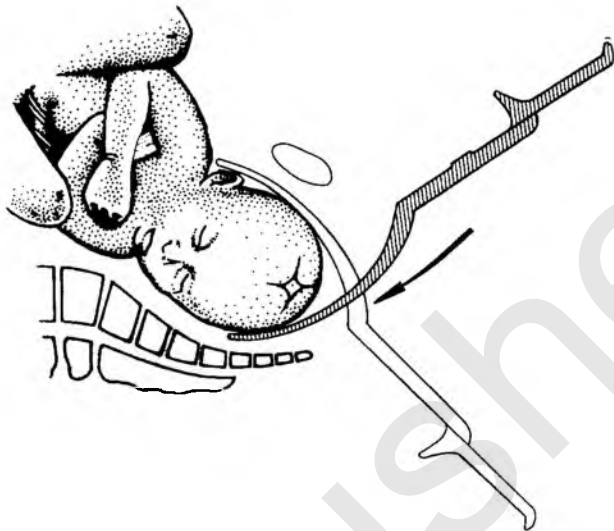


FIGURE 17.21.  
 LOT position. Kielland forceps, insertion of posterior (left) blade. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)

as the handle is gently swept downward. Between contractions, the blade is flipped over and then correctly positioned over the parietal bone. The posterior blade is subsequently introduced in the usual manner (Figure 17.21). The Kielland shanks and handles must never be raised from the horizontal because serious birth canal injuries can result from the sharp tips of the blades striking the posterolateral vagina. Because of the Kielland's inade-

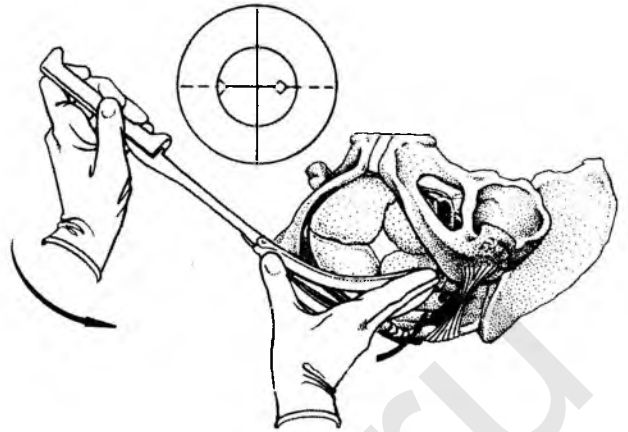
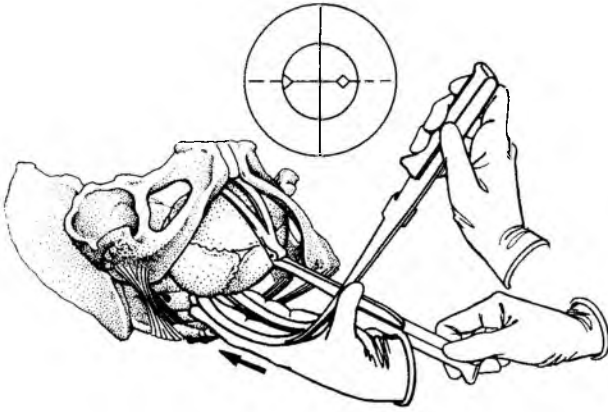


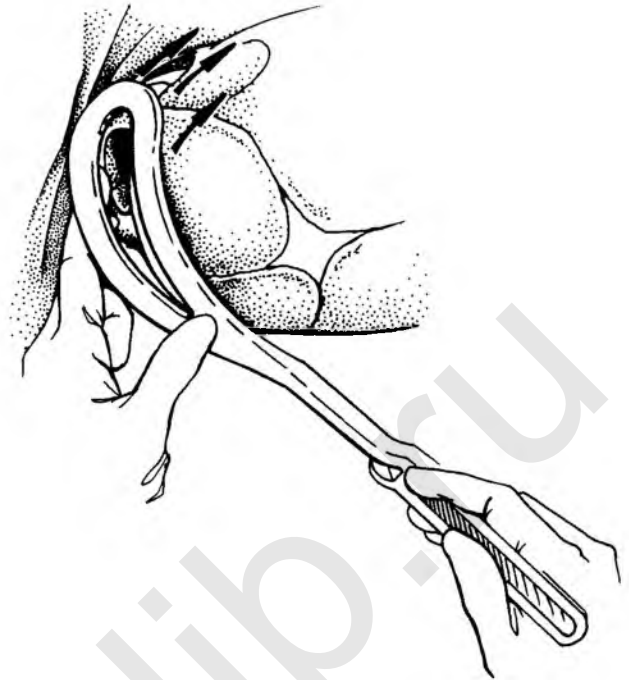
FIGURE 17.22.  
 ROT position. Insertion of the anterior blade of Barton forceps posterolaterally prior to wandering. Note that the blade is fully extended at the hinge. See text for details. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)

quacy as a traction blade, after a successful rotation and descent, a forceps with a standard pelvic curve, such as a Tucker-McLane, or a classic forceps can replace it. Owing to its long blades, the Kielland is contraindicated in platypelloid pelvis. Readers are referred to detailed texts for a more complete discussion of Kielland technique [14,34,36].

The Barton forceps has two parts: a markedly angulated posterior blade and a hinged anterior blade. In a Barton application, the anterior blade is introduced first by extending the blade on its hinge, inserting it posterolaterally as usual, and then wandering it into its final anterior position (Figure 17.22). The other blade is inserted directly posteriorly and progressively walked into the pelvis as the operator's hand intermittently elevates the presenting part to create a space for the blade (Figure 17.23). The blades are then articulated, and asynclitism corrected by adjusting the sliding lock before traction is attempted. Traction with the Barton forceps demands close attention to proper technique. The acute angle of the handles is unfamiliar to many practitioners and permits a potentially large mechanical advantage during a rotation. Normally, in platypelloid pelvis, rotation does not occur until the head reaches the perineum, and forceful rotations at a higher level should not be attempted. Neither the Barton nor the Kielland forceps should ever be applied by a neophyte without immediate expert instruction.



**FIGURE 17.23.**  
*ROT position. Insertion of posterior blade, Barton forceps (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*



**FIGURE 17.24.**  
*LOT position, wandering of anterior forceps blade. Force is applied with the index finger of the operator's vaginal hand to advance the blade. The thumb maintains the blade position, guarding against slippage. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

## Special Issues

### *Wandering*

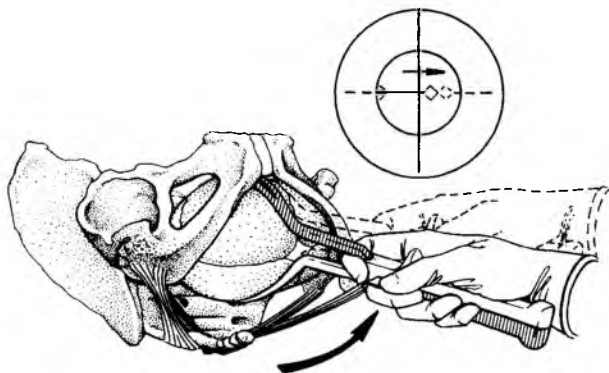
Adjusting an intravaginal forceps blade to an accurate application on the fetal head is termed *wandering* [14]. In wandering, the surgeon advances a forceps blade over the fetal head with digital pressure until the instrument reaches its correct site. When wandering is performed correctly, the presenting part is not displaced to a higher station, and an undesired rotation of the fetal head does not occur. Unfortunately, this ideal situation does not always happen. The force for wandering is applied solely by the operator's vaginal hand, pressing against the edge of the forceps blade. The hand supporting the handle twists the blade gently to maintain *minimal* pressure against the fetal head to help offset soft-tissue resistance from the vagina (Figure 17.24).

In many oblique or transverse applications, wandering is necessary. With an occasional exception, the blade is usually introduced posteriorly and then wandered over the fetal face or occiput to its final position. Often, wandering over the face is easiest and is safe when properly performed. A similar technique is used with oblique presentations when the check of instrument application indicates that the blades require readjustment. Advancement of the blade should never be difficult, and usually minimal force is required. Wandering is performed between contractions, while the gravida is not bearing down, and only after an anesthetic has been administered.

### Correction of Deflection and Asynclitism

Some degree of cranial deflection is common in occiput posterior and transverse positions. In contrast to a normally positioned calvarium, when the head is deflexed it presents a larger diameter to the maternal pelvis, increasing the difficulty of delivery. As previously discussed, marked deflection, as in brow presentation, can be due to a fetal anomaly or secondary to true disproportion. In selected cases and before attempts at forceps traction, the obstetrician can correct deflection by applying a forceps, grasping the fetal head, and repositioning it with a brief lateral motion (Figure 17.25). Several gentle repositionings and blade readjustments might be required to flex the head completely. Once normal flexion has been restored, the forceps application is carefully rechecked before full traction is attempted.

In vacuum extraction operations, deflection interferes with the accurate placement of the cup, at times precluding the use of many of the new plastic cups with long inflexible handles. If an accurate



**FIGURE 17.25.**  
**ROT position. Correction of cranial deflection. After flexion, the blades are readjusted, the position is rechecked, and rotation is performed, followed by traction. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)**

flexion point application with a Malmström-type rigid metal or plastic cup is possible, an initial traction in between contractions is occasionally used to reduce the deflection. In most instances, however, no specific manipulations are required, and the head spontaneously flexes during the extraction operation, assuming that the vacuum cup is correctly placed over the cranial pivot point. When the head is posterior, remember Aldo Vacca's admonition that it's always more posterior than you think!

*Asynclitism* is the oblique presentation of the fetal head to the pelvic curve (curve of Carus). Excessive cranial rotation either anteriorly to expose the posterior parietal bone (Litzmann's obliquity) or posteriorly involving the anterior parietal bone (Naegle's obliquity) is possible depending on the pelvic anatomy and the effects of uterine contractions. Application of a sliding lock forceps such as Barton's or Kielland's and repositioning of the blades reduces such asymmetry. In non-outlet forceps operations, neither rotation nor traction should be attempted until both cranial deflection and asynclitism have been corrected. Otherwise, excessive force might be required or improperly oriented force employed, increasing the risk of a fetal injury.

### **Occiput Posterior or Oblique Positions**

Occiput posterior positions are more difficult for both physician and patient [19], and the literature includes numerous schemes of management. With modern second-stage management, occiput poste-

rior presentations do not necessarily increase fetal morbidity or mortality. Nonetheless there remains an important relationship among occiput posterior presentation, instrumental or cesarean delivery, and maternal injury. This position is associated with maternal morbidity, including longer labors and the risk of significant perineal or sphincter trauma [53,114–117].

When studied radiographically and by ultrasound, 15% to 35% of fetuses are in an occiput posterior position at the onset of labor [118–120]. The incidence is higher in nulliparas [114,118,121–123]. At the time of delivery, however, only 2% to 8% of presentations are found to be occiput posterior [124,125]. There are interesting data in reference to occiput posterior presentation. In an ultrasound study of 408 singleton pregnancies, cranial position was documented at the onset of labor. Of the cases that presented in late labor in occiput posterior position, 62% were initially either occipitoanterior or transverse positions. This finding suggests that malrotation during descent rather than a persistence of an initially posterior position accounts for most cases of occiput posterior presentation at the time of final delivery.

It comes as no surprise that, as labor progresses, the likelihood of spontaneous cranial rotation out of occiput posterior progressively declines [121,123]. Thus, if the presentation is occiput posterior at 6 cm to 9 cm, it will remain so at delivery in 80% of cases [120]. Nonetheless, most infants who present early in labor with an occiput posterior head spontaneously rotate to an anterior position as labor progresses, apparently without a significant prolongation of the process [118,120,126].

There is concern over persisting posterior positions owing to their strong association with dystocia or tardy progress, fetal heart rate abnormalities, a low spontaneous vaginal delivery rate, and the potential for fetal and maternal and fetal injuries. Depending on the series and for a combination of reasons, only 15% to 50% of unselected occiput posterior presentations ultimately deliver vaginally [120,124,125].

There is also an increased frequency of uterine tachysystole, hypertonia, and tetanic contractions, as well as frequent abnormalities in electronic fetal heart rate tracings (especially variable-type decelerations) in occiput posterior as opposed to anterior presentations [127]. Presumably, increased fetal

cranial or intratracheal compression/pressure increases vagal tone, resulting in bothersome fetal heart rate alterations.

In comparison to an occiput anterior presentation, vaginal delivery from an occiput posterior position is strongly associated with both cesarean and instrumental delivery as well as increased maternal morbidity [122,125,128]. Obstetric problems and complications increase when a persisting occiput posterior presentation occurs. The likelihood of perineal lacerations, the need for oxytocin augmentation, a prolonged second stage, excessive blood loss, and the requirement for either cesarean or instrumental delivery are increased in this setting [53,120,122,125].

Some unique arrangement of pelvic anatomy, combined with features of the maternal abdominal wall and uterus, predisposes to a posterior cranial orientation and results in an increased risk for such positioning in subsequent pregnancies [123]. A shortened transverse diameter, narrow forepelvis, prominent ischial spines, straight sacrum, or convergent sidewalls probably increase the probability that the occiput will enter the hind pelvis preferentially either initially, or more likely, after spontaneous rotation as the head descends. Most reviews also find that epidural anesthesia is implicated in occiput posterior presentations. This is thought to be due to its effects on the tone of the pelvic musculature combined with an attenuation of maternal expulsive efforts and perhaps a reduction in the effectiveness of uterine contractions [124,129,130]. Of interest, in an Irish series reported by Fitzpatrick and coworkers in which active management of labor was performed, this association with epidurals was not confirmed [125]. Yancey's group reported similar results [131]. The Irish investigators actually observed a decline in the percentage of occiput posterior presentations from 3.8% to 2.4% over a 25-year period, while at the same time epidural use increased rapidly from 5% in 1975 to 70% by 1998. These data reflect the complexity of human labor and emphasize that much about the mechanism of labor and the potential efforts of various second-stage management choices remains unsettled.

### ***Occiput Posterior Management***

Intervention is not required in occiput posterior presentations until progress ceases. If progress

is tardy, attendants occasionally perform Puddicombe's maneuver, placing the woman in knee-chest position to promote rotation [132]. The efficacy of postural management is likely low; most initially posterior presentations spontaneously rotate to anterior [130].

Unfortunately, the randomized controlled trials of repositioning for *nonlaboring* women failed to find any evidence that periodic pelvic rocking exercises or hands-knee positioning (from 37 weeks to term) – manipulations similar to these proposed by Puddicombe – have any effect on occiput posterior positioning [121,133]. If the previously discussed mechanism for occiput posterior presentation is correct, most cases result from spontaneous rotations from other positions. Thus, antepartum repositioning attempts would be anticipated to have little impact on occiput posterior incidence at the actual time of delivery. Theoretically, maternal repositioning could assist the forces of gravity, displacing the fetal body away from the maternal spine and changing the orientation of the fetal mass in the maternal abdomen. These randomized trials involved repositioning women *before* the onset of labor, however. Thus, technically the issue of repositioning in labor to help change fetal position could be viewed as unsettled. In light of the existing data and with the previous inaccuracy of digital examination in the determination of position, the argument for a positive effect of intrapartum repositioning is weak.

Opinions concerning occiput posterior management vary from early operative intervention to watchful expectancy. Once progress ceases and the position remains occiput posterior, the remaining management alternatives are manual rotation, instrumental delivery, or a cesarean. The application of either forceps or the vacuum extractor to an occiput posterior position is problematic because there is a substantial risk for either failure or rectal sphincter injury [91,125]. Manual rotation can be attempted in an occiput posterior position but may fail. Some clinicians favor forceps delivery of posterior presentations directly, face to pubis ("sunny side up"), without an attempt at rotation. Unfortunately, this pull is often difficult and often result in a posterior tear (Figure 17.26). Vacuum extraction is a possibility, albeit with a substantial likelihood of failure [91]. Forceps rotational deliveries are also occasionally performed. These operations



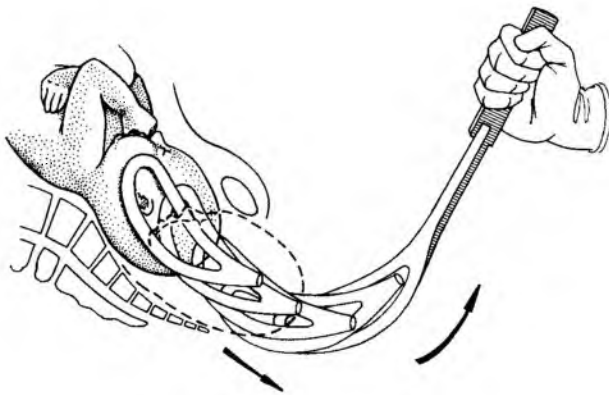


FIGURE 17.26.  
*Occiput posterior presentation. Delivery by classic forceps. Note the vector of traction with final delivery of the head by flexion. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

include the Scanzoni maneuver or one of its modifications, or a Kielland forceps rotation. As noted, many cases are delivered by a cesarean [124].

Vaginal delivery from the occiput posterior increases maternal and neonatal morbidity, especially if any delivery instrument is used. Episiotomy, followed by severe perineal or vaginal lacerations, are the principal maternal risks. Forceps deliveries from the posterior are more likely to result in tears than vacuum operations [128], but the use of the forceps as opposed to the vacuum extractor is substantially more successful. Failure of vacuum extraction from occiput posterior positions is common [91]. Because of the problems with occiput posterior presentation, if there are problems with the fetal tracing, the child is thought to be large, or if difficulty develops during the application of the blades or with the rotation, cesarean delivery is best.

There are some interesting clinical data concerning success in vaginal delivery from posterior positions. In a study of 1802 deliveries, 1438 of which were occiput anterior and 364 occiput posterior, Damron and coworkers reported that in the posterior midpelvic presentations, the failure rate for forceps was 16.7% against 71.4% for vacuum ( $p \leq 0.001$ ). When procedures from all pelvic stations were considered, the overall occiput posterior failure rate was 33% for vacuum operations against 13.6% for forceps. Rectal sphincter injuries were observed in 71.6% of forceps cases versus 33.1% of

vacuum extraction deliveries for occiput posterior presentations. These data must be compared with occiput anterior presentations, for which the failure rate was 6.3% for the vacuum extractor and 0.9% for the forceps.

If forceps are applied for a direct occiput posterior delivery, it often presents the surgeon with a long and stiff pull. As the fetal head descends, it cannot be elevated above the horizontal until the bulk of the calvarium has been extracted and the nose has passed beneath the symphysis pubis. A classic, long-bladed instrument such as a Simpson forceps is usually best for these deliveries. It is applied as if the fetal head was in the corresponding *anterior* presentation. Thus, the forceps is "upside down" on the fetal head. Deflection is common and might need to be corrected before traction is applied. Episiotomy is often necessary to permit the pull to occur in the correct vector and reduce the total force required for the extraction. Because of these features, posterior episiotomy extensions are common and often unavoidable.

An anterior forceps rotation by the Scanzoni rotation or one of its modifications is another possibility [112]. These procedures are not for the inexperienced physician, however. Approximately 25% of neonates subjected to such rotations exhibit discrete transient neurologic signs. Fortunately these resolve spontaneously, generally without sequelae.

In 1995, Menticoglou's group reported a series of 15 infants with spinal cord injuries that were associated with rotational instrumental deliveries [134]. They estimated that the magnitude of the risk was approximately 1 per 1,000 operations or less. These data require careful consideration by clinicians and reinforce the demand for the closest attention to detail and limits in effort whenever a major rotation such as a classic Scanzoni is proposed. Five of the cases in this series involved multiple instrument use (i.e., forceps *and* vacuum extraction), a congenital coagulopathy, or a "difficult" procedure. If these cases are excluded from the analysis, the estimated incidence drops to approximately 1 per 1,500 rotational deliveries of 90° or more. It is unclear whether these remaining cases represent the use of excessive force, improper technique, or an inherent procedural risk. Several points are important. Dennen emphasizes the importance of conducting major rotations, either clockwise or counterclockwise, *in a direction to transverse the smallest arc in reference to*

the occiput and the fetal spine [36]. Thus, if the fetal back is positioned to the mother's right, a rotation direct occiput posterior is best conducted in a clockwise direction. In the alternative situation, were the fetal spine positioned to the mother's left, cranial rotation from occiput posterior to OA should proceed counterclockwise. Such technique is presumed to reduce the risk of injury by minimizing torsion applied to the fetal neck. Unfortunately, the Menticoglou data are not detailed enough to ascertain the direction of rotation versus the position of the fetal body in the affected infants.

Rotations must proceed easily and should be performed independently of traction and between contractions. At times, the fetal head initially needs either a slight upward displacement or downward pull before the rotation is accomplished. It is also important to correct deflection before attempting the rotation because this reduces the force required and maintains the neck and head in a natural physical relationship.

Because these rotations are not commonly performed and carry some unique risks, clinicians should use all available assistance. As discussed in detail previously, the author recommends a preoperative real-time ultrasound examination when possible to verify the correct cranial position before conducting such major rotational deliveries.

In a classic *Scanzoni rotation*, after verifying the position by real-time scan and ghosting the blades, the obstetrician should apply an outlet-type forceps upside down as if to the corresponding anterior position (i.e., an occiput posterior position is treated as the corresponding OA; Figure 17.27). Once the application is made, cranial deflection is corrected if necessary. Thereafter between contractions the fetal head is simply rotated to OA or slightly beyond, without downward traction. On occasion the clinician might need to displace the head slightly upward to ease the subsequent rotation.

The posterior forceps blade is then left in place, but the anterior blade is removed (Figure 17.28). A second forceps blade is then reintroduced alongside the original and now upside-down splinting blade. The convex portion of the new blade is correctly oriented toward the fetal occiput; that is, this second blade is applied *right side up*. The splinting blade is then removed from below with a downward sweeping motion, and the remaining second forceps blade is introduced in the usual manner as for an OA pre-

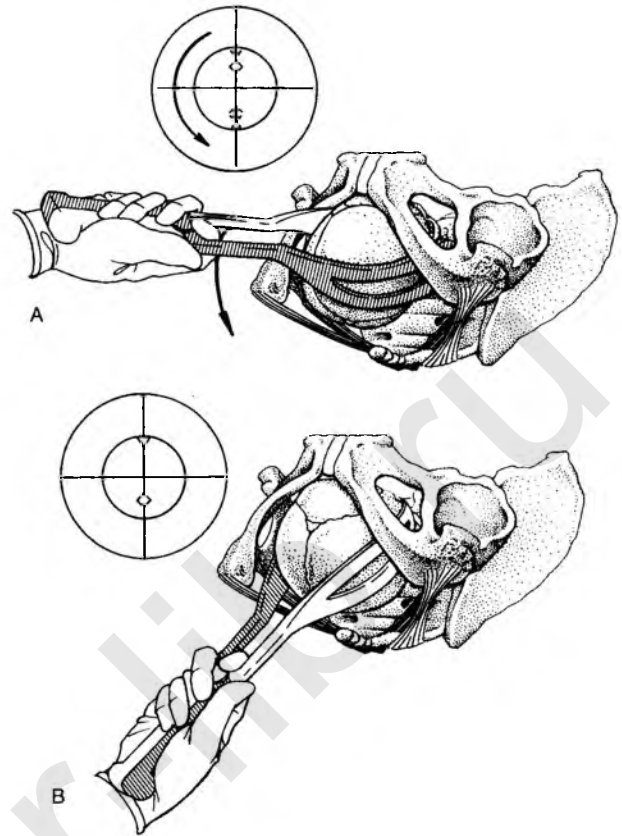


FIGURE 17.27. *Classic Scanzoni rotation. Application of a forceps (A) "upside down" and (B) initial station. See text for details. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

sentation. Following rechecking of the application and any additional correction of deflection, traction is applied and the delivery completed. This procedure can be done either with two sets of forceps or by rapid reversal of the original blades.

To avoid maternal injury, during rotational deliveries it is critical to maintain a fixed angle between an imaginary vertical plane passing through the pubic symphysis and the shanks of the forceps. If this angle is permitted to change, the tips of the blades can all too easily and rapidly lacerate the birth canal, usually in the upper segment near the cervix. Thus, care is necessary. An occiput posterior to OA rotation must never be forced. If the head will not easily rotate, the procedure must immediately be reassessed and usually abandoned. In this setting, either a direct occiput posterior forceps delivery or a cesarean could follow.

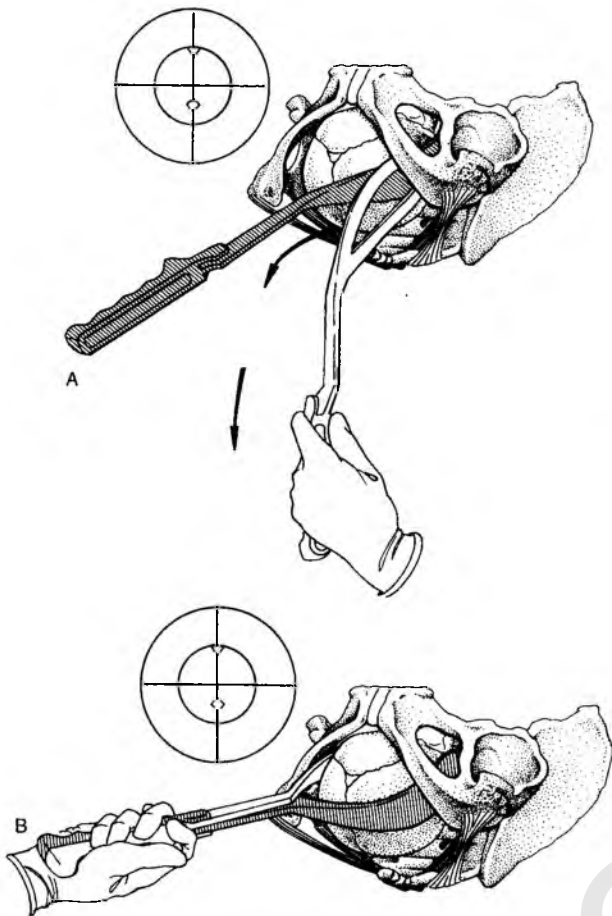


FIGURE 17.28. *Modified Scanzoni operation, occiput posterior presentation. Following rotation to OA, the splinting right Simpson forceps blade is removed (A) prior to reinserting the right Tucker-McLean blade in correct orientation on the other side of the pelvis (B). See text for details. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

Several variations of the original technique exist. In the *reverse Scanzoni maneuver*, the blades are initially inserted correctly oriented for the posterior occiput. In this procedure, the handle position of the forceps is the reverse of the usual orientation as the shanks point downward. The rotation is made to OA or slightly oblique, the application is rechecked, and traction for delivery is established without replacing or reversing the blades. In the *Haas maneuver*, the blades are initially applied as for a regular Scanzoni maneuver, but after rotation are not removed. The fetal head is simply delivered using the upside-down

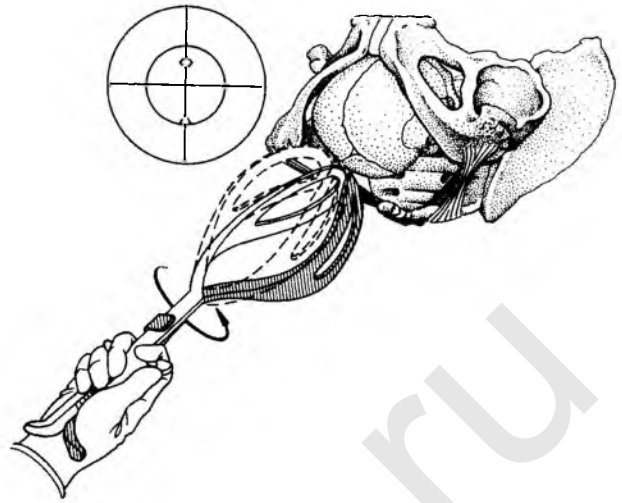


FIGURE 17.29. *Occiput posterior position, Kielland forceps. Ghosting of forceps. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)*

forceps. Due to their unfamiliarity neither of these techniques are recommended.

#### *Kielland Rotation*

In a *Kielland rotation* for occiput posterior, the forceps are ghosted as for the corresponding anterior application (Figure 17.29). The blades are then rotated 180° and thus appear upside down to the operator but are correctly aligned to the fetal cranium since the buttons are oriented toward the fetal occiput. Direct application from below follows. With the blades correctly applied, the fetal head is flexed and then rotated anteriorly as in the Scanzoni procedure (Figure 17.30). As noted earlier, the Kielland forceps lack a pelvic curve. To avoid laceration, it is therefore important that the plane of the shanks of the blades must never rise above the horizontal. In marked contrast to a forceps with a pelvic curve, rotation with the Kielland forceps is performed by simply rotating the operator's hand in the appropriate direction, maintaining the instrument at a near 90° angle to the perineum. Either raising or lowering the handles of this instrument risks laceration from the tips of the blades as they rotate within the birth canal. As noted previously, because the Kielland is an indifferent traction forceps, some operators replace it with a classic outlet-type blade

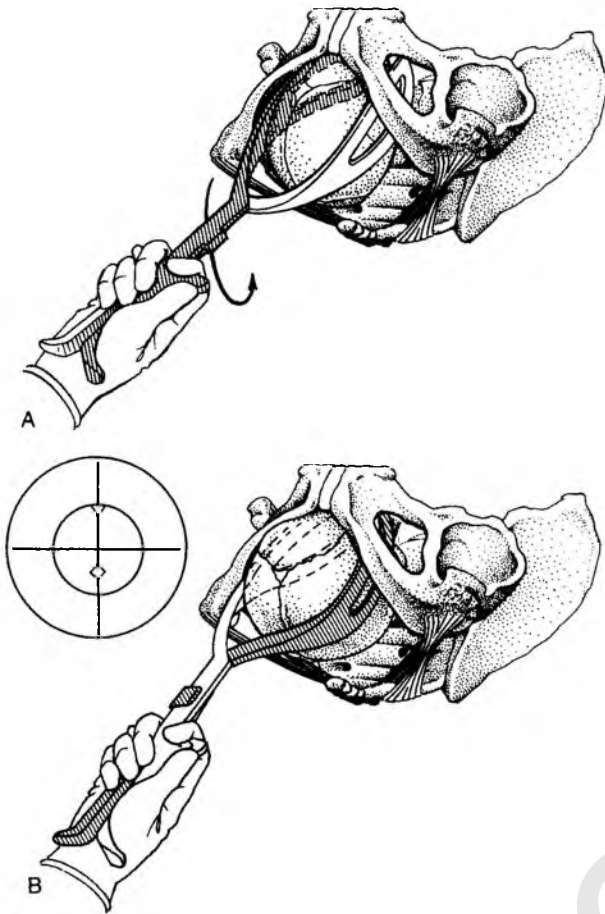


FIGURE 17.30. OP position, Kielland forceps. Rotation from occiput posterior (A) to occiput anterior (B). (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)

for the final delivery process after the rotation is completed.

### Special Applications

#### Aftercoming Head (Breech Presentation)

Application of Piper, Laufe, or Kielland forceps to the head of the aftercoming breech is an important part of intrapartum breech management (Figures 17.31 and 17.32). This application is the only pelvic – as opposed to cephalic – standard forceps operation. That is, the blades of the forceps are introduced in the same fashion regardless of the exact cranial position and not in a special relationship to the fontanelles. It is required that only the occiput be

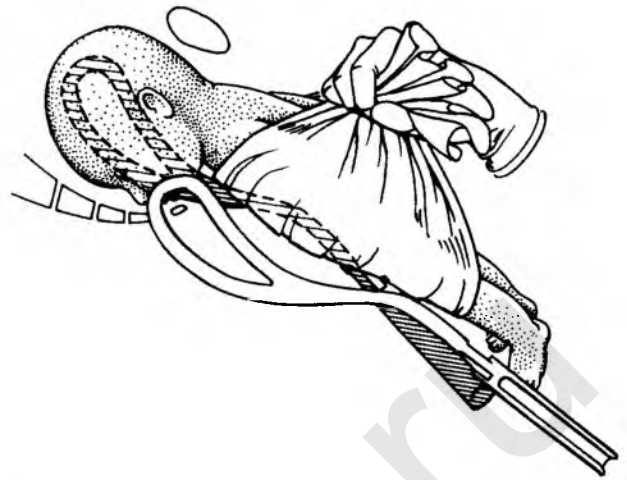


FIGURE 17.31. Breech presentation. Technique of insertion of Piper forceps from below; note that the infant has entirely delivered except for the head and that the application is pelvic. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)

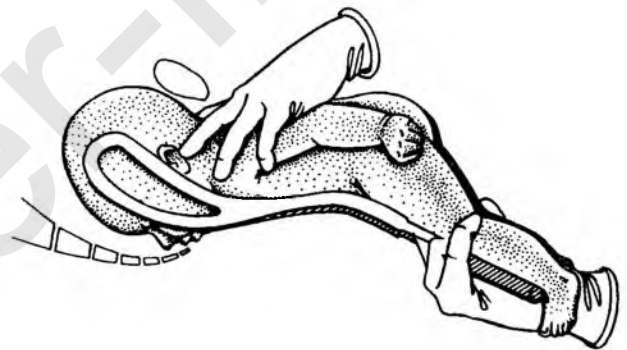


FIGURE 17.32. Breech presentation. Technique of traction with Piper forceps. (From O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins; 1988; with permission.)

anterior. Vacuum extraction has no role in assisted breech delivery. (See Chapter 12, Breech Presentation.)

### Risks and Benefits

Any instrumental delivery involves a potential risk for maternal and fetal injuries. The avoidance of trauma requires an understanding of the mechanisms for birth injury and expert knowledge of appropriate operative technique. Problems

associated with assisted delivery include 1) the maternal and fetal risks specific to the procedure or technique, and 2) the risks already present in the pregnancy or those caused by the complications compelling intervention. The latter risks are little influenced by the eventual mode of delivery and are thought to be the most important factor in most permanent infant abnormalities. Knowledge of both the specific technique and the situational risks is important. Education and refinement of technique do reduce the procedural risk but are powerless in changing an inherent risk of an already established injuries. (This subject is reviewed in detail in Chapter 23, Birth Injuries.)

Most injuries during labor and delivery are inconsequential; however, several highly uncommon central nervous system injuries or other complications such as subgaleal hemorrhages are potentially serious and sometimes fatal. The principal concern is that a potentially avoidable injury might result in permanent fetal damage or serious maternal and fetal morbidity. Despite previously held opinions, permanent neurologic impairment (e.g., cerebral palsy, intellectual deficits) are uncommon and in modern practice are rarely caused by mechanical birth trauma unless the original injury is combined with major birth asphyxia or complicated by prematurity. Many, if not most, serious or permanent fetal/neonatal neurologic abnormalities result from complex in utero problems that precede parturition and are not under the control of the accoucheur [137–140]. Common difficulties in labor, such as episodes of nonreassuring EFM tracings, can be the effect of occult injury rather than the cause of an abnormality.

Physicians in all specialties should hesitate before confidently ascribing an observed neonatal defect to an event at parturition unless the cause is obvious, such as a laceration. Review of all clinical data, careful examination of the neonate, consideration of pathophysiology and placental pathologic examination, if available, are often needed before a final determination is made concerning etiology [138,141,142].

Injuries that are the immediate consequences of the process of assisted delivery can be mostly avoided or lessened by case choice and surgical skill [143]. In judging risk, give consideration to the potential for both the maternal and the infant birth injuries accompanying instrumental delivery.

By far, the most common maternal injuries are episiotomy extensions and lacerations of the birth canal. Although both cervical and vaginal vault laceration can occur during spontaneous deliveries, these complications are more common and usually more extensive after instrumental delivery and can occasionally result in long-term sequelae. Risk generally increases with the complexity of the extraction and is proportional to the station of the fetal head at the beginning of the operation. In the following sections, several major risk factors are reviewed and their possible contribution to maternal or fetal injury is considered.

### **Large Infants**

Fetal *macrosomia*, variably defined as a birthweight of more than 4,000 g to 4,500 g or a weight greater than 90% for gestational age, is generally recognized as a risk factor for neonatal morbidity, obstetric injuries, and cesarean delivery [144–147]. Despite the fact that most large infants are delivered without complication, they remain a problem because of the associated risk of shoulder dystocia, other traumatic injuries, and potentially serious maternal complications [9]. Big babies are now common. Approximately 10% of infants weigh 4,000 g or more at birth, and 1% will reach or exceed 4,500 g. Factors including race, sex, the period of gestation, presence of diabetes, heavier mothers and poorly understood genetic factors are associated with large infants [148]. Problems with large infants and instrumental delivery occur when complete dilation is reached and descent of the fetal head proceeds far enough to entrap the unwary into attempting a delivery that proves difficult or traumatic [149].

Large infants are surprisingly difficult to diagnose, except in extreme cases. Physical examination, ultrasonic measurements at or near term, and various clinical parameters (e.g., fundal height, weight gain) are imprecise in the accurate prediction of macrosomia, however defined (Table 17.12).

It was hoped that the use of ultrasound weight estimates for elective induction or cesarean delivery could help to avoid the problem of excessive fetal size. Unfortunately, experience has proved disappointing [159]. What makes clinical choices difficult is the inherent inaccuracy of ultrasound weight estimates combined with the limitations in methods for evaluating pelvic capacity [160–162].

TABLE 17.12 Accuracy of Birthweight by Clinical Palpation, Sonographic Biometry, and Patient Self-estimate (Pregnancies >37 weeks)

Author (y)	Birthweight Estimates, by Technique					
	Clinical Palpation		Sonographic Biometry <sup>†</sup>		Patient Self-estimate <sup>‡</sup>	
	MA% Error*	BW ± 10% <sup>§</sup>	MA% Error	BW ± 10%	MA% Error	BW ± 10%
Watson (1988) [150]	7.9%	67%	8.2%	66%		
Chauhan (1992) [151]	9%	66%	15.6%	42%	8.7%	70%
Chauhan (1993) [152]	9.1%	65%	10.7%	56%		
Chauhan (1995) [153]	7.5%	65%			9.2%	67%
Chauhan (1995) [154]	9.9%	54%	11.4%	51%		
Sherman (1998) [155]	7.2%	73%	9.1%	69%		
Chauhan (1998) [156]	10.3%	61%	10%	60%		
Herrero (1999) [157]	9.5%	61%			9.5%	62%
Hendrix (2000) [158]	10.6%	58%	16.5%	32%		
<b>Range</b>	<b>7.2–10.6%</b>	<b>54–73%</b>	<b>8.1–16.5%</b>	<b>32–69%</b>	<b>8.7–9.5%</b>	<b>62–70%</b>

\*MA% error – Mean absolute percent error in fetal weight prediction.

<sup>†</sup>Based on algorithms employing combinations of fetal measurements including abdominal circumference (AC), femur length (FL), biparietal diameter (BPD), and head circumference (HC).

<sup>‡</sup>Parous women.

<sup>§</sup>BW ±10% – Percent of weights predicted to within ±10% of the actual birthweight.

Modified from Nahum GG: Estimation of fetal weight: Emedicine. <http://www.imedicine.com>, p. 1–61.

The central problem is what to do when a large infant is suspected. Although there is a clear association between fetal macrosomia and the likelihood of a birth injury, the incidence of persistent injury remains low [163]. Furthermore, as noted, current methods for the accurate diagnosis of fetal weight are lacking. For these reasons, neither trial of labor nor instrumental delivery is contraindicated in most instances when macrosomia is suspected. What is required in these cases, however, is a frank discussion with the mother and determination by the accoucheur not to prolong a trial of labor excessively, or to attempt difficult or extended extraction operations.

## EPISIOTOMY

A common obstetric procedure that is a risk factor for permanent maternal injury is episiotomy [164,165]. Despite prior beliefs concerning the benefit of episiotomy, no data support the traditional claims that this procedure, as routinely practiced, significantly protects the mother against either immediate or long-term birth canal injury or alters the risk of shoulder dystocia [166]. In fact, it is

now recognized that episiotomy *increases* the risk of third-degree (rectal sphincter) and fourth-degree (rectal mucosal) injuries. In its favor, episiotomy does provide limited protection against periurethral lacerations [144]. The effects of episiotomy in potentially reducing the force required for instrumental delivery or in shielding the premature fetal head from injury requires further study. Existing data are limited, largely anecdotal, and not compelling.

Episiotomy should not be performed routinely, even when an instrumental delivery is anticipated [167]. If maternal soft tissue interferes with instrument application or impedes the descent of the presenting part when traction is applied in the correct vector of force, an episiotomy can be considered. Extractions from the midpelvis or those involving cranial malpositioning (e.g., occiput posterior, deflexed position) require an angle of traction that applies heavy pressure to the perineum. This increases the need for episiotomy and can lead to perineal laceration [144]. Outside of the United States, when an episiotomy is required, mediolateral (ML) incisions are often preferred [168]. Although ML episiotomies are less likely than median

episiotomies (ME) to extend into the rectal sphincter or mucosa, the ML is harder to repair, is more likely to result in distortion of the perineum, results in more pain during the puerperium, and increases the likelihood of long-term dyspareunia. Best practice guidelines for whether to perform an episiotomy during an instrumental delivery, the type to employ, and the appropriate timing have yet to be established [9,169,170].

### **FETAL INJURIES: VACUUM EXTRACTION AND FORCEPS**

Only selected types of injury are discussed in this section. (For a more extensive review of fetal injuries see Chapter 24, Birth Injuries.) Fetal injuries from vacuum extraction relate to the physics of how the vacuum cup grasps the scalp and how force is applied to assist the parturition. In vacuum extraction, for traction to be applied, the fetal scalp is drawn into the cup. This process can produce the characteristic mound of scalp tissue and edema called the *chignon*. When traction follows, and force is applied to the handle of the vacuum extractor, the scalp is pulled upward. If a pop-off occurs, suction is suddenly lost, and the scalp recoils to the fetal head. When this occurs, the cup, which was under tension, is suddenly released and can even be projected from the birth canal. These events, with the associated disruption of small bridging veins, are believed to predispose to the common, but clinically unimportant, cephalohematomas and the relatively rare but potentially life-threatening subgaleal (SG) hemorrhages. Scalp bruising or lacerations and retinal hemorrhages are additional, usually insignificant risks of extraction procedures. The reported incidence of severe fetal injury or death from vacuum extraction is low and equal to that of forceps [8]. This risk is roughly estimated as 5 cases per 10,000 extraction procedures.

Fetal injuries from forceps are of several types. As with vacuum extraction, minor bruising/ecchymoses as a direct result of the blade lying against the fetal head are common. Rarely, direct injuries to the eye or facial nerve are possible. Forceps deliveries and perhaps less commonly vacuum extractions can also lead to intracranial hemorrhage [171]. Fetal skull fracture with or without brain contusion are additional rare risks. In the next section, several of the most important types of potential instrumental injuries are briefly reviewed.

(See Chapter 23, Birth Injuries, for additional data.)

### **Subgaleal/Subaponeurotic Hemorrhage**

Hemorrhage in the subgaleal (SG) or subaponeurotic space is due to rupture of the emissary veins bridging the gap between the aponeurosis and the underlying calvarium. The overall incidence of a clinically significant SG hemorrhage following a vacuum delivery is estimated as 2.6 to 4.5 in 10,000 procedures [172,173]. These rates could be overestimates and do not reflect the rates of injury in modern practice when soft-cup extractors are used and strict protocols for application are followed. The criteria for establishing this diagnosis are principally clinical, and thus there is a possibility of misdiagnosis, especially when small hemorrhages are present or the presentation is atypical. This condition is potentially life threatening, with the reported mortality rate varying from 11.8% to 22.8% [171]. Approximately one half of all SG hemorrhages are related to vacuum extraction, and most of the rest are associated with forceps operations [174]. Less commonly, SG bleeds follow spontaneous deliveries, or rarely even a cesarean.

Operative technique is an important variable in the development of these hemorrhages because the risk is thought to be proportional to the effort required for delivery. Thus, an SG hemorrhage is very uncommon or even rare unless excessive force, multiple vacuum extractor pop-offs, or serial instrumentation with the vacuum extractor and the forceps have occurred [41,104,108]. When a SG hemorrhage is diagnosed, some degree of accompanying intracranial bleeding is common [171]. These lesions include subarachnoid and subdural hemorrhages, and, to a lesser degree, intraventricular or intraparenchymal bleeds. Serious cases of SG bleeding have followed outwardly uneventful extractions, however. It is important to put this risk into perspective. Clinically significant SG bleeding was not observed in the large number of vacuum extraction cases included in recent meta-analyses [87]. This documents not only the rarity of severe scalp injuries but also emphasizes the importance of adhering to following strict technical guidelines when performing vacuum extraction operations.

Because of the small but significant risk of SG hemorrhage even when VE operations are performed properly, the author recommends notifying

pediatric personnel whenever an extraction occurs, regardless of the immediate condition of the neonate. Serial evaluation of the neonate is prudent because SG hemorrhages might not become clinically apparent until some hours postpartum.

### **Other Intracranial Injuries**

When potentially serious neonatal intracranial hemorrhages are observed, the mode of delivery, either cesarean or instrumental, might not be the risk factor [73,175]. Dysfunctional and prolonged labors and various degrees of cranial malpresentation could be the principal culprits. Not all reviewers share this conclusion, however [8]. This is not to suggest that the actual mechanism of delivery has no role in birth injury, because there are clearly individual cases in which this is so. Clinicians must always adhere to strict limitations in the choice of cases and in efforts to reduce maternal/fetal risk. It could be, however, that the greatest risk to the fetus is from abnormalities in labor rather than the final method of delivery. Thus, the choice of a cesarean instead of an instrumental delivery might not alter the risk for intracranial injury in cases involving significant dystocia from failure to progress or dysfunctional labor.

When delivery is required and circumstances are interpreted as difficult, the most common option is a cesarean. Cesareans, although largely safe, do have important long-term maternal consequences, including risks for subsequent scar rupture, abnormal placental adherence, and the potential for subsequent subfertility [176,177]. When progress is slow or stops and the usual maneuvers fail, the risks of a cesarean are weighed against those of prolonging the labor or attempting an instrumental delivery trial. In the author's opinion, a properly conducted trial of instrumental delivery retains a place in obstetric management when progress ceases, the clinical findings do not exclude the possibility of instrumentation, and the alternative is a cesarean [178].

### **Scalp Bruising and Lacerations**

Ecchymoses and rarely lacerations of the scalp or other major scalp injuries can follow a vacuum extraction. Localized scalp injury and quite uncommonly laceration can also follow a forceps delivery. Despite their initial appearance, these injuries usually spontaneously regress without sequelae. Again,

technique is a contributing factor. In a vacuum extraction, most injuries occur when the recommended 20- to 30-minute limit to total cup application is exceeded or cup manipulation is attempted. The ventouse is not primarily a rotating instrument, and attempts at manual cup rotation simply foster cup displacement and predispose to scalp injury. Under traction, the fetal head should rotate automatically as descent occurs.

### **MATERNAL INJURY**

Vacuum extraction has a low rate of maternal injury compared with forceps operations or cesarean delivery. Maternal injuries do occur with all vaginal delivery instruments, however, and must be considered in the evaluation of the procedure-associated risk.

### **PERINEAL AND OTHER BIRTH CANAL INJURIES**

Maternal perineal lacerations are common complications of all operative vaginal deliveries; most are associated with episiotomy. Electively incising the perineum predisposes to more serious perineal lacerations, and injuries to the rectal sphincter mechanism by direct extension [170]. The reported incidence of severe perineal lacerations, including third-degree and fourth-degree lacerations, during vacuum extraction procedures ranges from 5% to 30%. Forceps operations are more likely to result in anal sphincter trauma than vacuum extractions, however [91]. In pooled data from randomized trials studying maternal delivery trauma, a substantial decrease in anal sphincter trauma occurred when vacuum extraction and not forceps was employed [87,91,92]. Prior history is important. A previous perineal scar or difficult delivery predisposes to a repeat tear; thus, women who sustain vaginal lacerations in a previous delivery are at a significantly greater risk for repeat lacerations in subsequent deliveries. This is presumably due to perineal scarring and the loss of tissue elasticity. Women at greatest risk are those who experienced a laceration in the first delivery followed by another delivery combining both an instrumental delivery *and* an episiotomy.

### **Stress Urinary and Anal Incontinence**

Dystocia in labor, vaginal delivery, obstetric lacerations, multiparity, genetic factors, obesity, smoking, and age are risk factors in both reversible and



permanent injuries to the connective or support tissues of the maternal pelvis [179–181]. Some degree of injury to these support structures and to the rectum constitutes important and perhaps unavoidable risks of all types of instrumental delivery.

Anatomically, the female pelvic viscera are both *suspended* from above and *supported* from below. The proper function of the various support structures depends on the integrity of their muscular, fascial, and neuralgic constituents. Incompletely understood genetic and individual characteristics, such as obesity and inherent tissue strength, also influence the adequacy of pelvic support, especially as the person ages.

The upper suspensory structures of the pelvis are various pseudoligamentous connective tissues, loosely termed the pelvic ligaments. These ligaments are actually sheets of complex connective tissues that accompany vascular structures into the pelvis and surround the cervix. The lower supports for the uterus are musculofascial and include the urogenital and pelvic diaphragms. The pelvic diaphragm consists principally of the levator ani muscle. The urogenital diaphragm is a complex of small muscles and accompanying connective tissue that extends from the central perineal body radially to attach to various bony and ligamentous sites on the lower pelvis.

Labor, the process of passing the fetal body through the birth canal, and instrumental delivery distort and injure these various support structures and other pelvic tissues [182]. During parturition, portions of the pelvic ligaments and the muscles are simply torn or otherwise disrupted, and accompanying nerves are traumatized. Various spontaneous lacerations or episiotomy extensions account for additional pelvic injuries, especially to the rectal sphincter mechanism [144].

The issue is not whether vaginal delivery results in injuries to pelvic soft tissues. The question is the degree of the injury and the extent to which spontaneous postpartum healing or specific muscle strengthening exercises performed in the puerperium can ameliorate this damage. There are important and unresolved issues of management and best practice in instrumental delivery to avoid or reduce the likelihood of injury. Techniques that either reduce or avoid injury to pelvic supports and to the rectum are under study. Long-term follow-up studies controlling for prepartum pelvic support status (e.g., preexisting rectal dysfunction, urinary

incontinence) as well as length of labor, type of anesthesia, clinically observed perineal trauma, and delivery method are required before changes in current practice can be confidently recommended. (See Chapter 23, Birth Injuries, and Chapter 11, The Third Stage.)

### Trial and Failed Operations

All practitioners encounter potentially difficult second-stage management problems. Murphy reported that 4% of the women in a British population eventually went to a trial of instrumental delivery in the operating suite or to a cesarean at full cervical dilation [183]. When unsuccessful efforts at instrumental delivery are considered, it is important to distinguish trials from failed procedures. A *failed procedure* occurs when an instrument is applied under circumstances in which the surgeon does not anticipate failure, and no alternative preparations have been made. Maternal or fetal injuries can be associated with these delivery efforts. A *trial procedure* occurs when an instrumental delivery is attempted in the operating suite once all preparations for a cesarean have been completed. In the latter setting it is not clear to the clinician that the effort will prove successful. In a trial, the surgeon, birth attendants, and the parturient are prepared for the possibilities of failure. The application and traction are tentative, proceeding only if all goes easily.

There are several causes for failed procedures that have been discussed before [75,76,184,185]. Operator inexperience is a factor. Errors in the placement of the vacuum cup on the fetal head or an incorrect vector of traction with either this instrument or the forceps can lead to a failed extraction. Choice of instrument is also important. Instrumental procedures that are unsuccessful are more likely following attempted vacuum extractions than when forceps are applied (Table 17.13). These unfiltered numbers hide another important observation: the vacuum extractor is much more likely to fail if the fetal head is midpelvic or positioned as an occiput posterior [91]. Failures are also more common in certain clinical settings, such as after a prolonged second stage, if there is an inaccurate diagnosis of fetal station, if severe cranial molding is present, if there is a history of a prior cesarean, or if the fetus is macrosomic [41,91,184,186,187].

TABLE 17.13 Failed Instrumental Delivery: Selected Series

Study	Vacuum Extraction*	Forceps*
Lasbrey (1964) [188]	12/121	3/131
Ehlers (1974) [189]	13/107	0/99
Vacca (1983) [190]	19/142	15/144
Boyd (1986) [191]	–	53/6,524
Johanson (1993) [192]	35/130	13/130
Bofill (1996) [11]	18/319	25/305
Sheiner (2001) [184]	113/2,111	–
Al-Kadri (2003) [76]	129/1,723	13/905
<b>Totals</b>	<b>339/4,656 = 7.28%</b>	<b>122/8,238 = 1.48%</b>

\*Various instruments; failed/total procedures.

The setting for a trial of instrumental delivery is unique. In preparation, appropriate assistants, including an anesthesiologist, are summoned. After an informed consent, the parturient is moved to an area where it is possible to perform a prompt cesarean delivery if the attempt is unsuccessful. The difficulty inherent in moving the patient to an operating room is offset by the distress caused to all by a failed operation in the delivery suite, especially if an emergent cesarean is suddenly required. The consent process and the medical record documentation for these procedures must be meticulous.

In the operating room, the senior surgeon conducts a careful reexamination and decides whether to apply an instrument and attempt traction. If he or she judges that an application is inappropriate, and an instrument should not be applied, if the application proves difficult, or if an instrument is applied and with traction there is not immediate descent of the presenting part, the instruments are removed, any perineal injuries are sutured, and a cesarean is performed. If traction has been applied, and vaginal extraction is unsuccessful and must be abandoned for a cesarean, it is prudent to displace the head upward manually before proceeding with the cesarean. In this circumstance, the anesthesiologist should be requested to prepare 150  $\mu$ g to 350  $\mu$ g of nitroglycerine if the subsequent cranial extraction proves difficult and the surgeon should recruit an assistant for vaginal displacement of the fetal head, if this proves necessary.

It is difficult to interpret the extant clinical data on instrumental delivery trials; only failed procedures are reliably recorded. The distinction made

in this chapter between trial and failed operations is not usually reflected in the literature because the actual intraoperative management is imperfectly recorded. In reports in which the distinction between trial and failed instrumental delivery is made, the numbers of reported cases are limited, and the studies are retrospective. Although inherently limited, most but not all of these reports do not report serious increased maternal or fetal morbidity from failed operations [175,176,178,191].

Additional data helpful in understanding the potential risk of failed instrumental delivery have been provided by Bahl and coworkers [176]. They performed a study of 393 women who had reached full dilatation before going either to a cesarean or a non-routine instrumental delivery conducted in an operating suite. The operating room was principally used for rotational deliveries or for trials of instrumental delivery when either disproportion was considered or a difficult delivery was anticipated. These data document that failed instrumental delivery following a prolonged second stage, or those deliveries that required more than three traction efforts or the use of multiple instruments were associated with increased maternal trauma (OR 4.1) increased neonatal trauma (OR 4.2) and an increased likelihood of admission of the infant to a neonatal unit (OR 6.2). Of note, both the use of excessive traction efforts and multiple instrument use were associated with inexperienced operators in 52% and 45% of cases respectively. When fetal/neonatal death follows assisted delivery, a large percentage of the affected infants have evidence of fetal compromise prior to birth [143].

TABLE 17.14 Selected Studies of Long-term Follow-up after Instrumental Delivery

Author	Patients	Comparison Group	Follow-up (yr)	Outcome/Comment
McBride et al. (1979) [193]	188 low forceps 51 midforceps 57 forceps rotation	101 elective cesarean deliveries	5	Insignificant difference between groups
DeCosta (1982) [194]	127 vacuum extraction	127 "next spontaneous" delivery matched for parity/gestational age	≥2	Insignificant differences between groups
Friedman et al. (1984) [195]	70 midforceps 82 low forceps	70 spontaneous deliveries 82 spontaneous deliveries	7	In the midforceps group, IQ lower by $5.76 \pm 2.17$ Low forceps – no difference in IQ
Nilsen (1984) [196]	62 low, mid-, and high forceps (Kielland and Simpson forceps)*	38 low, mid-, and high Malmström vacuum extractor deliveries	18	Forceps deliveries associated with significantly elevated mean intelligence score than Norwegian mean
Dierker et al. (1985, 1986) [88,197]	110 midforceps	110 cesarean deliveries	≥2	Insignificant difference between groups; subjects matched for sex, age, weight, race, dystocia, and fetal distress
Seidman et al. (1991) [198]	567 forceps 1,207 vacuum	1,335 cesarean deliveries 29,136 spontaneous deliveries	17	Insignificant differences between groups in intelligence scores at age 17 <sup>†</sup>
Wesley et al. (1993) [199]	114 midforceps 1,078 low/outlet forceps	1,499 spontaneous deliveries	5	Insignificant differences between groups; forceps operations coded as low, low-mid, or mid-, but criteria were not specified
Ngan et al. (1990) [200]	295 vacuum extractions	302 spontaneous deliveries	10	Insignificant differences between groups
Bahl et al (2007) [205]	127 vacuum or forceps deliveries	64 immediate cesarean deliveries 74 failed instrumental deliveries followed by cesareans	5	All deliveries were at full dilatation. There was a 67% follow up of original cohorts. Rates of neurodevelopmental morbidity were comparable, irrespective of mode of delivery

*IQ*, Intelligence quotient.

\*By pre-1988–1989 American College of Obstetricians and Gynecologists definitions.

<sup>†</sup>6.6% of original cohort lost. Possible selection bias.

Although the judicious use of accepted protocols and routine exercises of clinical judgment would avoid many if not most failed operations, clinical judgment is imperfect. All surgeons will encounter a failed instrumental delivery at some time in their careers. Increased maternal and fetal morbidity appears to occur primarily when unanticipated failures are encountered; that is, cases in

which the instrumentation is commenced with full anticipation of success and without special preparation for a possible cesarean. After an unsuccessful trial of vaginal delivery with any instrument, and regardless of the extent of the effort, an internal fetal monitoring clip is attached, an external Doppler transducer is applied, and continuous heart rate monitoring commenced while the mother awaits

TABLE 17.15 Midforceps Procedures versus Cesarean Delivery: Selected Comparison Studies

Authors	No.		Outcome	
	Midforceps Delivery	Cesarean Delivery	Neonatal	Maternal
Bowes and Bowes (1980) [201]	40	37	4 × neonatal morbidity, including lacerations, asphyxia, meconium aspiration	No difference
Cardozo et al. (1983) [202]	65	127	Higher 5-min Apgar scores Fewer NICU admissions	N/A
Traub et al. (1984) [111]	132	101	No difference	N/A
Gilstrap et al. (1984) [203]	234	111	No significant differences in fetal acidosis, low 5-min Apgar scores, trauma, or neurologic defect at discharge when matched for indication	In forceps group: lower incidence of endometriosis and blood transfusion; higher incidence of perineal trauma
Dierker et al. (1985) [88]*	176	165	Increased incidence of cephalohematoma, low 1-min Apgar scores; with diagnosis of fetal distress or dystocia: equal neonatal morbidity present	In forceps groups: higher incidence of perineal trauma
Bashore et al. (1990) [46]	358	486	Minor and transient neonatal injuries with forceps; cord gases equal when cases matched by indication	Decreased postpartum febrile morbidity in forceps group
Robertson et al. (1990) [47]	505 Forceps 455 Vacuum	828	Increased incidence of pH ≤7.10, high base defect, birth trauma, and admission to NICU	Decreased postpartum hospital stay and blood transfusion in instrumented group
Cibils and Ringler (1990) [89]	274	106	Increased admission to NICU for cesarean babies	Increased incidence perineal lacerations (third- and fourth-degree); increased length of stay and febrile morbidity in cesarean group

NICU, neonatal intensive care unit.

\*Infants matched for weight, gestational age, dystocia, and heart rate abnormalities. Total population = 21,414 deliveries.

cesarean delivery. If this is not technically possible, the fetal heart should be auscultated after every contraction or every 5 minutes until the surgical skin preparation is begun. Bradycardias after traction are common. Normally, the fetal heart rate will return to the baseline after the combined contraction/traction effort is over. Failure of the fetal heart to resume a normal rate and for the bradycardia to persist alerts the surgeon that an emergency delivery, rather than a simple urgent delivery, is needed.

### LONG-TERM FOLLOW-UP STUDIES

All of the available long-term follow-up studies of instrumentally delivered infants have distinct limitations, including retrospective study protocols, non-random selection, and the loss of infants experiencing the most serious complications. In their defense, however, these studies also include large numbers and have been conducted in quite different populations over many years. This provides some

reassurance about the reliability of the findings. The extant data confirm the safety of instrumental deliveries. Because the type of delivery procedures involved in these series is heavily weighted toward the more common outlet/low operations, serious complications would be expected to be uncommon (Tables 17.14 and 17.15).

Several studies are worthy of special attention. Wesley and coworkers studied a cohort of 3,413 children from a prepaid health plan service at age 5 years, using a battery of cognitive tests [199]. No significant differences were detected between the 1,192 children delivered by forceps (including 114 delivered by midforceps) versus 1,499 who were delivered spontaneously.

McBride and coauthors studied a cohort of Australian children ages 4 to 5 years who were born between 1970 and 1974 [193]. In this group, there were no statistically significant intelligent quotient (IQ) differences between spontaneously delivered infants and those delivered by forceps.

Seidman's group retrospectively studied outcomes in 52,282 children born in Jerusalem between 1964 and 1972 [198]. The method of delivery and other birth events were correlated with intelligence testing administered at age 17 years. The author reported no demonstrable adverse effects from instrumental delivery.

Drawing from Collaborative Perinatal Project Data, Broman and coworkers [204] administered Stanford-Binet Intelligence tests to 26,760 children at age 4 years and correlated the results with perinatal events. In this analysis, the major variables found to affect IQ scores were not obstetric, indicating that in such a large population, the events of delivery were not the critical variables in cognitive function.

## CONCLUSION

Instrumentally assisted delivery by either forceps or vacuum extractor remains controversial. Neither instrument offers perfect safety or utility. Based on current data, properly conducted instrumental delivery procedures are safe and retain a legitimate role in modern obstetric practice [8,9,41,104]. The most important part of an instrumental delivery occurs *prior* to the actual instrumentation, when surgeons focus their knowledge, technical skill, and judgment on determining *if* an assisted

delivery should be attempted and *how* it is to be performed.

## REFERENCES

1. Speert, H., The obstetric forceps. *Clin Obstet Gynecol* 1960. 3:761–6.
2. Drinkwater, K., The midwifery forceps: Historical sketch. *Med Chir J* 1913. 64:451–65.
3. Hibbard, B., *The obstetrician's armamentarium*. 2000, San Anselmo, CA: Norman Publishing.
4. Patel, R.R., Murphy D.J., Forceps delivery in modern obstetric practice. *Br Med J* 2004. May 29; 328 (7451):1302–5.
5. O'Grady, J., Instrumental Delivery: A critique of current practice. In *Gynecologic, Obstetric, and Related Surgery*, Second Edition, Nichols, D., Clarke-Pearson D.L., eds. 2000, St. Louis: Mosby-Year Book, p. 1081–1105.
6. Guillemette, J., Fraser, W.D., Differences between obstetricians in caesarean section rates and the management of labour. *Br J Obstet Gynaecol* 1992. Feb; 99(2):105–8.
7. Chang, A., Noah, M.S., Laros, R.K., Jr., Obstetric attending physician characteristics and their impact on vacuum and forceps delivery rates: University of California at San Francisco experience from 1977 to 1999. *Am J Obstet Gynecol* 2002. Jun; 186(6): 1299–303.
8. Demissie, K., Rhoads, G.G., Smulian, J.C., Balasubramanian, B.A., Gandhi, K., Joseph, K.S., Kramer, M., Operative vaginal delivery and neonatal and infant adverse outcomes: Population-based retrospective analysis. *Br Med J* 2004. 3 July; 329 (7456):24–9.
9. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin Number 17, June 2000: Operative Vaginal Delivery.
10. Roberts, C., Algert, C.S., Carnegie, M., Operative delivery during labour: Trends and predictive factors. *Paediatr Perinat Epidemiol* 2002. Apr; 16(2): 115–23.
11. Bofill, J., Rust, O.A., Schorr, S.J., A randomized prospective trial of the obstetric forceps versus the M-cup vacuum extractor. *Am J Obstet Gynecol* 1996. Nov; 175(5):1325–30.
12. DeLee, J., The prophylactic forceps operation. *Am J Obstet Gynecol* 1920. 1:34–44.
13. Society of Obstetricians and Gynaecologists of Canada, Guidelines for operative vaginal birth.

- Number 148, May 2004. *Int J Gynaecol Obstet* 2005. Feb; 88(2):229–36.
14. O'Grady, J.P., *Modern Instrumental Delivery*. 1988, Baltimore, MD: Williams & Wilkins.
  15. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin Number 49, December 2003: Dystocia and Augmentation of Labor.
  16. Rosen, M., *Management of Labor*. 1990, New York: Elsevier.
  17. Hilton, P., Vesico-vaginal fistulas in developing countries. *Int J Gynaecol Obstet* 2003. Sept; 82(3): 285–95.
  18. Piper, J., Bolling, D.R., Newton, E.R., The second stage of labor: Factors influencing duration. *Am J Obstet Gynecol* 1991. Oct; 165(4 Pt 1):976–9.
  19. Fraser, W., Cayer, M., Soeder, B.M., Turcot, L., Marcoux, S., for the PEOPLE (Pushing Early or Pushing Late) and W.E.S. Group, Risk factors for difficult delivery in nulliparas with epidural anesthesia in second stage of labor. *Obstet Gynecol* 2002. Mar; 99(3):409–18.
  20. Feinstein, U., Sheiner, E., Levy, A., Hallak, M., Mazor, M., Risk factors for arrest of descent during the second stage of labor. *Int J Gynaecol Obstet* 2002. Apr; 77(1):7–14.
  21. Menticoglou, S., Manning, F., Harman, C., Perinatal outcome in relation to second-stage duration. *Am J Obstet Gynecol* 1995. Sept; 73(3 Pt 1):906–12.
  22. Cheng, Y., Hopkins, L.M., Caughey, A.B., How long is too long: Does a prolonged second stage of labor in nulliparous women affect maternal and neonatal outcomes? *Am J Obstet Gynecol* 2004. Sept; 191(3):933–8.
  23. Hansen, S., Clark, S.L., Foster, J.C., Active pushing versus passive fetal descent in the second stage of labor: A randomized controlled trial. *Obstet Gynecol* 2002. Jan; 99(1):29–34.
  24. Carmona, F., Martinez-Roman, S., Manau, D., Cararach, V., Iglesias, X., Immediate maternal and neonatal effects of low-forceps delivery according to the new criteria of the American College of Obstetricians and Gynecologists compared with spontaneous vaginal delivery in term pregnancies. *Am J Obstet Gynecol* 1995. Jul; 173(1): 55–9.
  25. Yancey, M., Herpolsheimer, A., Jordan, G.D., Benson, W.L., Brady, K., Maternal and neonatal effects of outlet forceps delivery compared with spontaneous vaginal delivery in term pregnancies. *Obstet Gynecol* 1991. Oct; 78(4):646–50.
  26. Niswander, K., Gordon, M., Safety of the low-forceps operation. *Am J Obstet Gynecol* 1973. 117:619–630.
  27. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin: Number 70, December, 2005: Intrapartum fetal heart rate monitoring.
  28. Ross, M., Devoe, L.D., Rosen, K.G., ST-segment analysis of the fetal electrocardiogram improves fetal heart rate tracing interpretation and clinical decision making. *J Matern Fetal Neonatal Med* 2004. Mar; 15(3):181–5.
  29. McNamara, D., C-section numbers unaffected by fetal pulse oximetry: Neonatal outcomes also failed to improve. *Ob Gyn News* 2006. Feb 15; 41(4): 1, 8.
  30. Bloom, S., Casey, B.M., Schaffer, J.I., McIntire, D.D., Leveno, K.J., A randomized trial of coached versus uncoached maternal pushing during the second stage of labor. *Am J Obstet Gynecol* 2006. Jan; 194(1):10–3.
  31. Wood, C., Ng, K.H., Hounslaw, D., Benning, H., Time: An important variable in normal delivery. *J Obstet Gynaecol Br Commonw* 1973. Apr; 80(4): 295–300.
  32. Das, K., *Obstetric Forceps: Its History and Evolution*. 1929, Calcutta: The Art Press.
  33. Dill, L., *The Obstetrical Forceps*. 1953, Springfield, IL: Charles C Thomas.
  34. Laufe, L., *Obstetric Forceps*. 1968, New York: Harper & Row.
  35. Dennon, E., *Forceps Deliveries*. 1964, Philadelphia: FA Davis.
  36. Dennen, P., *Dennen's Forceps Deliveries*, Third Edition. 1989, Philadelphia: F.A. Davis Company.
  37. Shute, W., An obstetrical forceps which uses new principle of parallelism. *Am J Obstet Gynecol* 1959. 77: 442–6.
  38. Laufe, L.E., A new divergent outlet forceps. *Am J Obstet Gynecol* 1968. Jun 15; 101(4):509–12.
  39. Shute, W., Management of shoulder dystocia with the Shute parallel forceps. *Am J Obstet Gynecol* 1962. Oct 1; 84:936–9.
  40. Putta, L., Spencer, J.P., Assisted vaginal delivery using the vacuum extractor. *Am Fam Phys* 2000. Sept; 62(6):1316–20.
  41. Vacca, A., Vacuum-assisted delivery: Improving patient outcomes and protecting yourself against litigation. *Obstet Manage* 2004. Feb; Suppl:S1–S12.

42. Mazouni, C., Bretelle, F., Collette, E., Heckenroth, H., Bonnier, P., Gamerre, M., Maternal and neonatal morbidity after first vaginal delivery using Thierry's spatulas. *Aust N Z J Obstet Gynaecol* 2005. Oct; 45(5):405-9.
43. Elliott, B., Ridgway, L.F., Berkus, M.D., Newton, E.R., Peairs, W., The development and testing of new instruments for operative vaginal delivery. *Am J Obstet Gynecol* 1992. Oct; 167(4 Pt 1):1121-4.
44. Murless, B., Lower segment cesarean section: A new head extractor? *Br Med J* 1945. 1:1234.
45. O'Grady, J., Gimovsky, M.L., McIlhargie, C.J., Vacuum Extraction in Modern Obstetric Practice. 1995, New York: Parthenon Publishing Group Inc.
46. Bashore, R., Phillips W.H. Jr, Brinkman C.R. 3rd, A comparison of the morbidity of midforceps and cesarean delivery. *Am J Obstet Gynecol* 1990. Jun; 162(6):1428-35.
47. Robertson, P., Laros, R.K., Jr., Zhao, R.L., Neonatal and maternal outcome in low-pelvic and midpelvic operative deliveries. *Am J Obstet Gynecol* 1990. Jun; 162(6):1436-42.
48. Hagadorn-Freathy, A., Yeomans, E.R., Hankins, G.D., Validation of the 1988 ACOG forceps classification system. *Obstet Gynecol* 1991. Mar; 77(3):356-60.
49. O'Grady, J., Gimovsky, M.L., McIlhargie, C.J., Operative Obstetrics. 1995, Baltimore: Williams & Wilkins.
50. O'Driscoll, K., Meagher, D., Boylan, P., Active Management of Labor, Third edition. 1993, Ayelbury, England: Mosby-Year Book Europe, Ltd.
51. Philpott, R., The recognition of cephalopelvic disproportion. *Clin Obstet Gynaecol* 1982. 9:609-24.
52. Lau, T., Leung, T.N., Repeat instrumental delivery: How high is the risk? *Aust N Z J Obstet Gynaecol* 1998. Feb; 38(1):31-3.
53. Senecal, J., Xiong, X., Fraser, W.D., PEOPLE (Pushing Early or Pushing Late with Epidural Study Group), Effect of fetal position on second-stage duration and labor outcome. *Obstet Gynecol* 2005. Apr; 105(4):763-72.
54. Blackadar, C., Viera, A.J., A retrospective review of performance and utility of routine clinical pelvimetry. *Fam Med* 2004. 36(7):505-7.
55. Pattinson, R., Farrell, E., Pelvimetry for fetal cephalic presentations at or near term (Cochrane Review). *Cochrane Database Syst Rev* 2004 (Issue 2).
56. Ferguson, J.N., Newberry, Y.G., DeAngelis, G.A., Finnerty, J.J., Agarwal, S., Turkheimer, E., The fetal-pelvic index has minimal utility in predicting fetal-pelvic disproportion. *Am J Obstet Gynecol* 1998. Nov; 179(5):1186-92.
57. Morgan, M., Thurnau, G.R., Efficacy of the fetal-pelvic index in nulliparous women at high risk for fetal-pelvic disproportion. *Am J Obstet Gynecol* 1992. Mar; 166(3):810-4.
58. Sporri, S., Hanggi, W., Braghetti, A., Vock, P., Schneider, H., Pelvimetry by magnetic resonance imaging as a diagnostic tool to evaluate dystocia. *Obstet Gynecol* 1997. Jun; 97(6):902-8.
59. Sporri, S., Thoeny, H.C., Raio, L., Lachat, R., Vock, P., Schneider, H., MR imaging pelvimetry: A useful adjunct in the treatment of women at risk for dystocia? *Am J Roentgenol* 2002. Jul; 179(1):137-44.
60. March, M., Adair, C.D., Veille, J.C., Burrus, D.R., The modified Mueller-Hillis maneuver in predicting abnormalities in second-stage labor. *Int J Gynaecol Obstet* 1996. Nov; 55(2):105-9.
61. Crichton, D., A reliable method of establishing the level of the fetal head in obstetrics. *S Afr Med J* 1974. Apr 17; 48(18):784-7.
62. Vacca, A., The 'sacral hand wedge': A cause of arrest of descent of the fetal head during vacuum-assisted delivery. *Br J Obstet Gynaecol* 2002. Sept; 109(9):1063-5.
63. Vacca, A., Handbook of Vacuum Extraction in Obstetric Practice. 1992, London: Edward Arnold.
64. Zahalka, S., Sadan, O., Malinger, G., Liberati, M., Boaz, M., Glezerman, M., Rotmensch, S., Comparison of transvaginal sonography with digital examination and transabdominal sonography for the determination of fetal head position in the second stage of labor. *Am J Obstet Gynecol* 2005. Aug; 193(2):381-6.
65. Weber, T., Hertzberg, B.S., Bowie, J.D., Transperineal US: Alternative technique to improve visualization of the presenting fetal part. *Radiology* 1991. Jun; 179(3):747-50.
66. Akmal, S., Tsoi, E., Nicolaides, K.H., Intrapartum sonography to determine fetal occipital position: Interobserver agreement. *Ultrasound Obstet Gynecol* 2004. Sept; 24(4):421-4.
67. Akmal, S., Tsoi, E., Kametas, N., Howard, R., Nicolaides, K.H., Intrapartum sonography to determine fetal head position. *J Matern Fetal Neonatal Med* 2002. Sept; 12(2):172-7.

68. Souka, A., Haritos, T., Basayiannis, K., Noikokyri, N., Antsaklis, A., Intrapartum ultrasound for the examination of the fetal head position in normal and obstructed labor. *J Matern Fetal Neonatal Med* 2003. Jan; 13(1):59–63.
69. Sherer, D., Miodovnik, M., Bradley, K.S., Langer, O., Intrapartum fetal head position I: Comparison between transvaginal digital examination and transabdominal ultrasound assessment during the active stage of labor. *Ultrasound Obstet Gynecol* 2002. Mar; 19(3):258–63.
70. Dupuis, O., Ruimark, S., Corinne, D., Simone, T., Andre, D., Rene-Charles, R., Fetal head position during the second stage of labor: Comparison of digital vaginal examination and transabdominal ultrasonographic examination. *Eur J Obstet Gynecol Reprod Biol* 2005. Dec 1; 123(2):193–7.
71. Dietz, H., Lanzarone V., Measuring engagement of the fetal head: Validity and reproducibility of a new ultrasound technique. *Ultrasound Obstet Gynecol* 2005. Feb; 25(2):165–8.
72. Nahum, G.G. Estimation of fetal weight. *Emedicine*. <http://www.imedicine.com>, p. 1–61.
73. Towner, D., Castro, M.A., Eby-Wilkens, E., Effect of mode of delivery in nulliparous women on neonatal intracranial injury. *N Engl J Med* 1999. Dec 2; 341(23):1709–14.
74. Gardella, C., Taylor, M., Benedetti, T., Hitti, J., Critchlow, C., The effect of sequential use of vacuum and forceps for assisted vaginal delivery on neonatal and maternal outcomes. *Am J Obstet Gynecol* 2001. Oct; 185(4):896–902.
75. Murphy, D., Liebling, R.E., Patel, R., Verity, L., Swingler, R., Cohort study of operative delivery in the second stage of labour and standard of obstetric care. *Br J Obstet Gynaecol* 2003. Jun; 110(6):610–5.
76. Al-Kadri, H., Sabr, Y., Al-Saif, S., Abulaimoun, B., Ba'Aqeel, H., Saleh, A., Failed individual and sequential instrumental vaginal delivery: Contributing risk factors and maternal-neonatal complications. *Acta Obstet Gynecol Scand* 2003. Jul; 82(7):642–8.
77. Ezenagu, L., Kakaria, R., Bofill, J.A., Sequential use of instruments at operative vaginal delivery: Is it safe? *Am J Obstet Gynecol* 1999. Jun; 180(6 Pt 1):1446–9.
78. Ramin, S., Little, B., Gilstrap, L.C., Survey of operative vaginal delivery in North America in 1990. *Obstet Gynecol* 1993. Feb; 81(2):307–11.
79. McQuivey, R., Vacuum-assisted delivery: A review. *J Matern Fetal Neonatal Med* 2004. Sept; 16(3): 171–80.
80. Laufe, L., Berkus, M.D., *Assisted Vaginal Delivery: Obstetrical Forceps and Vacuum Extraction Techniques*. 1992, New York: McGraw-Hill.
81. <http://www.vaccaresearch.com>.
82. [rwn@clinicalinnovations.com](mailto:rwn@clinicalinnovations.com): physician education workshop.
83. Cheong, Y., Abdullahi, H., Lashen, H., Fairlie, F.M., Can formal education and training improve the outcome of instrumental delivery? *Eur J Obstet Gynecol Reprod Biol* 2004. Apr 15; 113(2):139–44.
84. Caughey, A., Sandberg, P.L., Zlatnik, M.G., Thiet, M.P., Parer, J.T., Laros, R.K., Jr., Forceps compared with vacuum. *Obstet Gynecol* 2005. Nov; 106(5):908–12.
85. Mollberg, M., Hagberg, H., Bager, B., Lilja, H., Ladfors, L., Risk factors for obstetric brachial plexus palsy among neonates delivered by vacuum extraction. *Obstet Gynecol* 2005. Nov; 106(5 Pt 1):913–8.
86. Robinson, J., Norwitz, E.R., Cohen, A.P., McElrath, T.F., Lieberman, E.S., Episiotomy, operative vaginal delivery, and significant perinatal trauma in nulliparous women. *Am J Obstet Gynecol* 1999. Nov; 181(5 Pt 1):1180–4.
87. Johanson, R., Menon, V. Vacuum extraction versus forceps for assisted vaginal delivery. *Cochrane Database Syst Rev*, 2005.
88. Dierker, L.J., Rosen, M.G., Thompson, K., Debanne, S., Linn, P., The midforceps: Maternal and neonatal outcomes. *Am J Obstet Gynecol* 1985. May 15; 152(2):176–82.
89. Cibils, L., Ringler, G.E., Evaluation of midforceps delivery as an alternative. *J Perinat Med* 1990. 18(1):5–11.
90. O'Driscoll K, M.D., MacDonald, D., Geoghegan, F., Traumatic intracranial haemorrhage in firstborn infants and delivery with obstetric forceps. *Br J Obstet Gynaecol* 1981. Jun; 88(6):577–81.
91. Damron, D., Capeless, E.L., Operative vaginal delivery: A comparison of forceps and vacuum for success rate and risk of rectal sphincter injury. *Am J Obstet Gynecol* 2004. Sept; 191(3):907–10.
92. Johnson, J., Figueroa R, Garry D., Immediate maternal and neonatal effects of forceps and vacuum-assisted deliveries. *Obstet Gynecol* 2004. Mar 103(3):513–8.



93. Thiery, M., Fetal hemorrhage following blood samplings and use of vacuum extractor [letter]. *Am J Obstet Gynecol* 1979. May 15;134(2):231.
94. Roberts, I., Stone, M., Fetal hemorrhage: Complication of vacuum extractor after fetal blood sampling. *Am J Obstet Gynecol* 1978. Sept 1; 132(1):109.
95. Boehm, F., Vacuum extraction during cesarean section. *South Med J* 1985. Dec; 78(12):1502.
96. Walka, D., Schmid, B., Sinha, P., Veit, S., Lichtenegger, W., Nitroglycerin application during cesarean delivery: Plasma levels, fetal/maternal ratio of nitroglycerin, and effects in newborns. *Am J Obstet Gynecol* 2000. Apr; 182(4):955-61.
97. Clark, K., Viscomi, C.M., Lowell, J., Chien, E.K., Nitroglycerin for relaxation to establish a fetal airway (EXIT procedure). *Obstet Gynecol* 2004. 103(5):113-15.
98. Liabsuetrakul, T., Choobun, T., Peeyananjarassri, K., Islam, M., Antibiotic prophylaxis for operative vaginal delivery. *Cochrane Database Syst Rev*, 2004.
99. Lim, F., Holm JP, Schuitemaker NW, Jansen FH, Hermans J, Stepwise compared with rapid application of vacuum in ventouse extraction procedures. *Br J Obstet Gynaecol* 1997. Jan; 104(1):33-6.
100. Svenningsen, L., Birth progression and traction forces developed under vacuum extraction after slow or rapid application of suction. *Eur J Obstet Gynecol Reprod Biol* 1987. Oct; 26(2):105-12.
101. Malmström, T., The vacuum extractor: An obstetrical instrument and the parturiometer, a topographic device. *Acta Obstet Gynecol Scand* 1957. 36(Suppl):7-87.
102. Malmström, T., Jansson, I., Use of vacuum extractor. *Clin Obstet Gynecol* 1965. 8:893-913.
103. Bofill, J.A., Rust, O.A., Schorr, S.J., Brown, R.C., Roberts, W.E., Morrison, J.C., A randomized trial of two vacuum extraction techniques. *Obstet Gynecol* 1997. May; 89(5 Pt 1):758-62.
104. O'Grady, J., Pope, C.S., Patel, S.S., Vacuum extraction in modern obstetric practice: A review and critique. *Curr Opin Obstet Gynecol* 2000. 12:475-480.
105. Bofill, J., Rust, O.A., Devidas, M, Roberts, W.E., Morrison, J.C., Martin, J.N., Jr., Neonatal cephalohematoma from vacuum extraction. *J Reprod Med* 1997. Sept; 42(9):565-9.
106. Bird, G., The use of the vacuum extractor. *Clin Obstet Gynaecol* 1982. 9:641-61.
107. Dennen, E., *Forceps Deliveries*. 1964, Philadelphia: FA Davis.
108. Vacca, A., Vacuum-assisted delivery. *Best Pract Res Clin Obstet Gynaecol* 2002. Feb; 16(1):17-30.
109. Sjostedt, J., The vacuum extractor and forceps in obstetrics: A clinical study. *Acta Obstet Gynecol Scand* 1967. 46(Suppl 10):3-208.
110. Bayer-Zwirello, L., O'Grady, J.P., Patel, S.S., ACOG's 1999 VBAC guidelines: A survey of western Massachusetts obstetric services. *Obstet Gynecol* 2000. 95(4 Suppl):S 73.
111. Traub, A., Morrow, R.J., Ritchie, J.W., Dornan, K.J., A continuing use for Kielland's forceps? *Br J Obstet Gynaecol* 1984. Sept; 91(9):894-8.
112. Healy, D., Quinn, M.A., Pepperell, R.J., Rotational delivery of the fetus: Kielland's forceps and two other methods compared. *Br J Obstet Gynaecol* 1982. Jul; 89(7):501-6.
113. Krivak, T., Drewes, P., Horowitz, G.M., Kielland vs. nonrotational forceps for the second stage. *J Reprod Med* 1999. Jun; 44(6):511-7.
114. Fitzpatrick, M., O'Herlihy, C., The effects of labour and delivery on the pelvic floor. *Best Pract Res Clin Obstet Gynaecol* 2001. Feb; 15(1):63-79.
115. Wu, J., Williams, K.S., Hundley, A.F., Connolly, A.M., Visco, A.G., Occiput posterior fetal head position increases the risk of anal sphincter injury in vacuum-assisted deliveries. *Am J Obstet Gynecol* 2005. Aug; 193(2):525-9.
116. To, W., Li, I.C., Occipital posterior and occipital transverse positions: Reappraisal of the obstetric risks. *Aust N Z J Obstet Gynaecol* 2000. Aug; 40(3):275-9.
117. Benavides, L., Wu, J.M., Hundley, A.F., Ivester, T.S., Visco, A.G., The impact of occiput posterior fetal head position on the risk of anal sphincter injury in forceps-assisted vaginal deliveries. *Am J Obstet Gynecol* 2005. May; 192(5):1702-6.
118. Gardberg, M., Laakkonen, E., Salevaara, M., Intrapartum sonography and persistent occiput posterior position: A study of 408 deliveries. *Obstet Gynecol* 1998. May; 91(5 Pt 1):746-9.
119. Caldwell, W., Moley, H.C., D'Espito, D.A., A roentgenologic study of the mechanism of engagement of the fetal head. *Am J Obstet Gynecol* 1934. 28:824.
120. Akmal, S.K.N., Tsoi, E., Howard, R., Nicolaides, K.H., Ultrasonographic occiput position in early labour in the prediction of caesarean

- section. *Br J Obstet Gynaecol* 2004. Jun; 111(6): 532–6.
121. Karimnia A., Chamberlain, M.E., Keogh, J., Shea, A., Randomised controlled trial of effect of hands and knees posturing on incidence of occiput posterior position at birth. *Br Med J* 2004. Feb 28; 328; Epub 2004. Jan; 26(7438):490.
  122. Ponkey, S.E., Cohen, A.P., Heffner, L.J., Lieberman, E., Persistent fetal occiput posterior position: Obstetric outcomes. *Obstet Gynecol* 2003. May; 101(5 Pt 1):915–20.
  123. Gardberg, M., Tuppurainen, M., Persistent occiput posterior presentation – a clinical problem. *Acta Obstet Gynecol Scand* 1994. Jan; 73(1):45–7.
  124. Sizer, A., Nirmal, D.M., Occipitoposterior position: Associated factors and obstetric outcome in nulliparas. *Obstet Gynecol* 2000. 96:749–752.
  125. Fitzpatrick, M., McQuillan, K., O’Herlihy, C., Influence of persistent occiput posterior position on delivery outcome. *Obstet Gynecol* 2001. Dec; 98(6):1027–31.
  126. Akmal, S., Tsoi, E., Kametas, N., Howard, R., Nicolaides, K.H., Investigation of occiput posterior delivery by intrapartum sonography. *Ultrasound Obstet Gynecol* 2004. Sept; 24(4):425–8.
  127. Ingemarsson, E., Ingemarsson, I., Solum, T., Westgren, M., Influence of occiput posterior position on the fetal heart rate pattern. *Obstet Gynecol* 1980. Mar; 55(3):301–4.
  128. Pearl, M., Roberts, J.M., Laros, R.K., Hurd, W.W., Vaginal delivery from the persistent occiput posterior position: Influence on maternal and neonatal morbidity. *J Reprod Med* 1993. Dec; 38(12):955–61.
  129. Turcot, L., Marcoux, S., Fraser, W.D., Multivariate analysis of risk factors for operative delivery in nulliparous women. Canadian Early Amniotomy Study Group. *Am J Obstet Gynecol* 1997. Feb; 176(2): 395–402.
  130. Lieberman, E., Davidson, K., Lee-Parritz, A., Shearer, E., Changes in fetal position during labor and their association with epidural analgesia. *Obstet Gynecol* 2005. 105:974–82.
  131. Yancey, M., Zhang, J., Schweitzer, D.L., Schwarz, J., Klebanoff, M.A., Epidural analgesia and fetal head malposition at vaginal delivery. *Obstet Gynecol* 2001. Apr; 97(4):608–12.
  132. Puddicombe, J., Maternal posture for correction of posterior fetal position. *J Int Coll Surg* 1955. Jan; 23(1 Pt 1):73–7.
  133. Hofmeyr, G., Kulier, R. Hands and knees posture in late pregnancy or labour for fetal malposition (lateral or posterior). *Cochrane Database Syst Rev*, 2005.
  134. Menticoglou, S., Perlman, M., Manning, F.A., High cervical spinal cord injury in neonates delivered with forceps: Report of 15 cases. *Obstet Gynecol* 1995. Oct; 86(4 Pt 1):589–94.
  135. Milner, R., Neonatal mortality of breech deliveries with and without forceps to the aftercoming head. *Br J Obstet Gynaecol* 1975. Oct 8; 2(10):893–913.
  136. Piper, E., Bachman, C., The prevention of fetal injuries in breech deliveries. *JAMA* 1929. 92:217–21.
  137. Illingworth, R., Why blame the obstetrician? A review. *Br Med J* 1979. 1:797–801.
  138. Illingworth, R., A paediatrician asks – why is it called birth injury? *Br J Obstet Gynaecol* 1985. 92:122–30.
  139. Nelson, K.B., Ellenberg, J.H., Antecedents of cerebral palsy: Multivariate analysis of risk. *N Engl J Med* 1986. Jul 10; 315(2):81–6.
  140. Nelson, K., Grether, J.K., Potentially asphyxiating conditions and spastic cerebral palsy in infants of normal birth weight. *Am J Obstet Gynecol* 1998. Aug; 179(2):507–13.
  141. American College of Obstetricians and Gynecologists and The American Academy of Pediatrics: Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology. 2003, Washington, DC: American College of Obstetricians and Gynecologists.
  142. Phelan, J., Martin, G.I., Korst, L.M., Birth asphyxia and cerebral palsy. *Clin Perinatol* 2005. Mar; 31(1):1–17.
  143. O’Mahony, F., Settatee, R., Platt, C., Johanson, R., Review of singleton fetal and neonatal deaths associated with cranial trauma and cephalic delivery during a national intrapartum-related confidential enquiry. *Br J Obstet Gynaecol* 2005. May; 112(5):619–26.
  144. McLeod, N., Gilmour, D.T., Joseph, K.S., Farrell, S.A., Luther, E.R., Trends in major risk factors for anal sphincter lacerations: A 10-year study. *J Obstet Gynaecol Can* 2003. Jul; 25(7):586–93.
  145. Raio, L., Ghezzi, F., DiNaro, E., Buttarelli, M., Franchi, M., Durig, P., Bruhwiler, H., Perinatal outcome of fetuses with a birth weight greater than 4500 g: An analysis of 3356 cases. *Eur J*

- Obstet Gynecol Reprod Biol 2003. Aug 15; 109(2): 160–5.
146. Oral, E., Cagdas, A., Gezer, A., Kaleli, S., Aydinli, K., Ocer, F., Perinatal and maternal outcomes of fetal macrosomia. *Eur J Obstet Gynecol Reprod Biol* 2001. Dec 1; 99(2):167–71.
  147. Boulet, S., Alexander, G.R., Salihu, H.M., Pass, M., Macrosomic births in the United States: Determinants, outcomes, and proposed grades of risk. *Am J Obstet Gynecol* 2003. May; 188(5):1372–8.
  148. Okun, N., Verma, A., Mitchell, B.F., Flowerdew, G., Relative importance of maternal constitutional factors and glucose intolerance of pregnancy in the development of newborn macrosomia. *J Matern Fetal Med* 1997. Sept–Oct; 6(5):285–90.
  149. Acker, D., Gregory, K.D., Sachs, B.P., Friedman, E.A., Risk factors for Erb-Duchenne palsy. *Obstet Gynecol* 1988. Mar; 71(3 Pt 1):389–92.
  150. Watson, W., Soisson, A.P., Harlass, F.E., Estimated weight of the term fetus: Accuracy of ultrasound vs. clinical examination. *J Reprod Med* 1988. Apr; 33(4):369–71.
  151. Chauchan, S., Lutton, P.M., Bailey, K.J., Guerrieri, J.P., Morrison, J.C., Intrapartum clinical, sonographic, and parous patients' estimates of newborn birth weight. *Obstet Gynecol* 1992. Jun; 79(6): 956–8.
  152. Chauhan, S., Lutton, T.C., Bailey, K.J., Morrison, J.C., Intrapartum prediction of birth weight: Clinical versus sonographic estimation based on femur length alone. *Obstet Gynecol* 1993. May; 81(5 Pt 1):695–7.
  153. Chauhan, S., Sullivan, C.A., Lutton, T.C., Magann, E.R., Morrison, J.C., Parous patients' estimate of birth weight in postterm pregnancy. *J Perinatol*, 1995. May–Jun; 15(3):192–4.
  154. Chauhan, S., Cowan, B.D., Magann, E.F., Bradford, T.H., Roberts, W.E., Morrison, J.C., Intrapartum detection of a macrosomic fetus: Clinical versus 8 sonographic models. *Aust N Z J Obstet Gynaecol* 1995. Aug; 35(3):266–70.
  155. Sherman, D., Arieli, S., Tovbin, J., Siegel, G., Caspi, E., Bukovsky, I., A comparison of clinical and ultrasonic estimation of fetal weight. *Obstet Gynecol* 1998. Feb; 91(2):212–7.
  156. Chauchan, S., Hendrix, N.W., Magann, E.F., Morrison, J.P., Kenney, S.P., Devoe, L.D., Limitations of clinical and sonographic estimates of birth weight: Experience with 1034 parturients. *Obstet Gynecol* 1998. Jan; 91(1):72–7.
  157. Herrero, R., Fitzsimmons, J., Estimated fetal weight: Maternal vs. physician estimate. *J Reprod Med* 1999. Aug; 44(8):674–8.
  158. Hendrix, N., Grady, C.S., Chauhan, S.P., Clinical vs. sonographic estimate of birth weight in term parturients: A randomized clinical trial. *J Reprod Med* 2000. Apr; 45(4):317–22.
  159. Rouse, D.J., Owen, J., Goldenberg, R.L., Cliver, S.P., The effectiveness and costs of elective cesarean delivery for fetal macrosomia diagnosed by ultrasound. *JAMA* 1996. Nov 13; 276(18):1480–6.
  160. Ben-Haroush, A., Yogev, Y., Bar, J., Mashiach, R., Kaplan, B., Hod, M., Meizner, I., Accuracy of sonographically estimated fetal weight in 840 women with different pregnancy complications prior to induction of labor. *Ultrasound Obstet Gynecol* 2004. Feb; 23(2):172–6.
  161. Chauhan, S., Hendrix, N.W., Magann, E.F., Morrison, J.C., Kenney, S.P., Devoe, L.D., Limitations of clinical and sonographic estimates of birth weight: Experience with 1034 parturients. *Obstet Gynecol* 1998. Jan; 91(1):72–7.
  162. Smith, G., Smith, M.F., McNay, M.B., Fleming, J.E., The relation between fetal abdominal circumference and birthweight: Findings in 3512 pregnancies. *Br J Obstet Gynaecol* 1997. Feb; 104(2):186–90.
  163. Kolderup, L., Laros, R.K., Jr, Musci, T.J., Incidence of persistent birth injury in macrosomic infants: Association with mode of delivery. *Am J Obstet Gynecol* 1997. Jul; 177(1):37–41.
  164. Carroli, G., Belizan, J. Episiotomy for vaginal birth (Cochrane Review). *Cochrane Database Syst Rev*, 1999.
  165. Weber, A.M., Meyn, L., Episiotomy use in the United States, 1979–1997. *Obstet Gynecol* 2002. Dec; 100(6):1177–82.
  166. Youssef, R., Ramalingam, U., Macleod, M., Murphy, D.J., Cohort study of maternal and neonatal morbidity in relation to use of episiotomy at instrumental vaginal delivery. *Br J Obstet Gynaecol* 2005. Jul; 112(7):941–5.
  167. Eason, E., Labrecque, M., Wells, G., Feldman, P., Preventing perinatal trauma during childbirth: A systematic review. *Obstet Gynecol* 2000. Mar; 95(3):464–71.
  168. Sheiner, E., Levy, A., Walfisch, A., Hallak, M., Mazor, M., Third-degree perineal tears in a university medical center where midline episiotomies are not performed. *Arch Gynecol Obstet* 2005. Apr; 271(4):307–10.

169. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin: Number 10, November, 1999: Induction of Labor.
170. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin Number 71, April 2006: Episiotomy. *Obstet Gynecol* 2006. Apr; 107(4):957–61.
171. Kilani, R., Wetmore J., Neonatal subgaleal hematoma: Presentation and outcome – radiological findings and factors associated with mortality. *Am J Perinatol* 2006. Jan; 23(1):41–8.
172. Boo, N., Subaponeurotic haemorrhage in Malaysian neonates. *Singapore Med J* 1990. Jun; 31(3):207–10.
173. Govaert, P., Defoort, P., Wigglesworth, J.S., Cranial haemorrhage in the term newborn infant. *Clin Dev Med* 1993. 129:1–223.
174. Macleod, C., O'Neill, C., Vacuum assisted delivery – the need for caution. *Ir Med J* 2003. May; 96(5):147–8.
175. Revah, A., Ezra Y., Farine D., Ritchie K., Failed trial of vacuum or forceps – maternal and fetal outcomes. *Am J Obstet Gynecol* 1997. Jan; 176(1 Pt 1): 200–4.
176. Bahl, R., Strachan, B., Murphy, D.J., Outcome of subsequent pregnancy three years after previous operative delivery in the second stage of labour: Cohort study. *Br Med J* 2004. Feb 7; 328(7435): 311; Epub 2004 Jan 14.
177. Murphy, D., Stirrat, G.M., Heron, J., ALSPAC Study Team, The relationship between caesarean section and subfertility in a population-based sample of 14 541 pregnancies. *Hum Reprod* 2002. Jul; 17(7):1914–7.
178. Lowe, B., Fear of failure: A place for the trial of instrumental delivery. *Br J Obstet Gynaecol* 1987. Jan; 94(1):60–6.
179. Sultan, A., Kamm, M.A., Hudson, C.N., Thomas, J.M., Bartram, C.I., Anal-sphincter disruption during vaginal delivery. *N Engl J Med* 1993. Dec 23; 329(26):1905–11.
180. Buschbaum, G.M., Chin, M., Glantz, C., Guzik, D., Prevalence of urinary incontinence and associated risk factors in a cohort of nuns. *Obstet Gynecol* 2002. Aug; 100(2):226–9.
181. Wei, J., DeLancey, J.O., Functional anatomy of the pelvic floor and lower urinary tract. *Clin Obstet Gynecol* 2004. Mar; 47(1):3–17.
182. Christianson, L., Bovbjerg, V.E., McDavitt, E.C., Hullfish, K.L., Risk factors for perineal injury during delivery. *Am J Obstet Gynecol* 2003. Jul; 189(1): 225–60.
183. Murphy, D., Liebling, R.E., Verity, L., Swingler, R., Patel, R., Early maternal and neonatal morbidity associated with operative delivery in second stage of labour: A cohort study. *Lancet* 2001. Oct 13; 358(9289):1203–7.
184. Sheiner, E., Shoham-Vardi, I., Silberstein, T., Halkak, T., Katz, M., Mazor, M., Failed vacuum extraction: Maternal risk factors and pregnancy outcome. *J Reprod Med* 2001. Sept; 46(9):819–24.
185. Gopalan, S., Bennett, K., Critchlow, C., Factors predictive of failed operative vaginal delivery. *Am J Obstet Gynecol* 2004. Sept; 191(3):896–902.
186. Mola, G., Amoa, A.B., Edilyong, J., Factors associated with success or failure in trials of vacuum extraction. *Aust N Z J Obstet Gynaecol* 2002. Feb; 42(1):35–9.
187. Saropala, N., Chaturachinda, K., Failed instrumental delivery: Ramathibodi Hospital, 1980–1988. *Int J Gynaecol Obstet* 1991. Nov; 36(3):203–7.
188. Lansbrey, A., Orchard, C.D., Crichton, D., A study of the relative merits and scope for vacuum extraction as opposed to forceps delivery. *S Afr J Obstet Gynaecol* 1964. 2(1).
189. Ehlers, N., Jensen, I.K., Hansen, K.B., Retinal haemorrhages in the newborn: Comparison of delivery by forceps and by vacuum extractor. *Acta Ophthalmol (Copenh)* 1974. 52:73–82.
190. Vacca, A., Grant, A., Wyatt, G., Chalmers, I., Portsmouth operative delivery trial: A comparison of vacuum extractor and forceps delivery. *Br J Obstet Gynaecol* 1983. Dec; 90(12):1107–12.
191. Boyd, M., Usher, R.H., McLean, F.H., Normal, B.E., Failed forceps. *Obstet Gynecol* 1986. Dec; 68(6):779–83.
192. Johanson, R., Rice, C., Doyle, M., Arthur, J., Anyanwu, L., Ibrahim, J., Warwick, A., Redman, C.W., O'Brien, P.M., A randomised prospective study comparing the new vacuum extractor policy with forceps delivery. *Br J Obstet Gynaecol* 1993. Jun; 100(6):524–30.
193. McBride, W., Black, B.P., Brown, C.J., Dolby, R.M., Murray, A.D., Thomas, D.B., Method of delivery and developmental outcome at five years of age. *Med J Aust* 1979. Apr 21; 1(8):301–4.
194. DeCosta, C., The vacuum extractor – a re-appraisal. *Ir J Med Sci* 1982. Dec 12; 151(4):105–8.

195. Friedman, E., Sachtelben-Murray, M.R., Darouge, D., Neff, R.K., Long-term effects of labor and delivery on offspring: A matched-pair analysis. *Am J Obstet Gynecol* 1984. Dec 15; 150(8):941-5.
196. Nilsen, S., Boys born by forceps and vacuum extraction at 18 years of age. *Acta Obstet Gynecol Scand* 1984. 63(6):549-54.
197. Dierker, L.J., Rosen, M.G., Thompson, K., Lynn, P., Midforceps deliveries: Long-term outcome of infants. *Am J Obstet Gynecol* 1986. Apr; 154(4):764-8.
198. Seidman, D., Laor, A., Gale, R., Stevenson, D.K., Mashlach, S., Danon, Y.L., Long-term effects of vacuum and forceps deliveries. *Lancet* 1991. Jun 29; 337(8757):1583-5.
199. Wesley, B.D., van den Berg, B.J., Reece, E.A., The effect of forceps delivery on cognitive development. *Am J Obstet Gynecol* 1993. Nov; 169(5):1091-5.
200. Ngan, H., Miu, P., Ko, L., Ma, H.K., Long-term neurological sequelae following vacuum extractor delivery. *Aust N Z J Obstet Gynaecol* 1990. Mar; 30(2):111-4.
201. Bowes, W., Bowes, C., Current role of the mid-forceps operation. *Clin Obstet Gynecol* 1980. Jun; 23(2):549-57.
202. Cardozo, L., Gibb, D.M., Studd, J.W., Cooper, D.J., Should we abandon Keilland's forceps. *Br Med J (Clin Res Ed)*, 1983. Jul 30; 287(6388):315-7.
203. Gilstrap, L.C., Hauth, J.C., Schiano, S., Connor, K.D., Neonatal acidosis and method of delivery. *Obstet Gynecol* 1984. May; 63(5):681-5.
204. Broman, S., Nichols, P.L., Kennedy, W.A., *Preschool IQ: Prenatal and Early Developmental Correlates*. 1975, Hillsdale, NJ: Lawrence Erlbaum Associates (John Wiley & Sons).
205. Bahl, R., Patel, R.R., Swingler, R., Ellis, M., Murphy, D.J., Neurodevelopmental outcome at 5 years after operative delivery in the second stage of labor: A cohort study. *Am J Obstet Gynecol* 2007. Aug; 197(2):147.e1-6.

## Chapter 18 CESAREAN DELIVERY AND SURGICAL STERILIZATION

John P. O'Grady

Timothy K. Fitzpatrick

*... my Aim in this Piece being not so much to inform those who are altogether ignorant, by giving them Instructions for their first setting out in Practice, as to add something to what is already published, ... which ... may conduce to the Benefit of the less knowing, and not prove altogether unworthy the Notice of those, who, being already arrived to the highest Pitch of Knowledge and Art, do Honour to their Profession, and Service to the World, by proving the happy Instruments (under Providence) of assisting and preserving the Fair in the Time of their greatest Danger ...*

Edmund Chapman (1680?–1756)

*Treatise on the Improvement of Midwifery*

Third Edition, London, 1759; John Brindley; pp. 67–68

Cesarean delivery has a long and complex history. In antiquity, what we would now consider or term *cesarean* operations began as unanticipated and hurried peri- or postmortem surgeries performed in the effort to salvage a child when the mother was either newly dead or believed to be dying. Cesareans were also rarely performed on living women but usually only as a last resort in the effort to save the mother's life.

Most often these procedures were only attempted when all other methods of delivery had failed. As surgical procedures became increasingly safe, beginning late in the nineteenth century, cesarean delivery progressively evolved from a dreadfully dangerous and desperate procedure to its status today as simply an alternative to vaginal delivery, often performed electively. At present, one infant in three in the United States is delivered by a cesarean operation [1]. Despite this very common resort to cesareans and likely in good measure because of it, lively debate persists concerning the appropriate role for abdominal delivery in obstetric practice.

This chapter discusses and critiques the current practice of cesarean delivery, focusing on the indications for the operation, the performance of the surgery, and its potential complications. A detailed description of the performance of a cesarean is included as well as a brief description of surgical methods of sterilization and the technique for symphysiotomy. After forceps and vacuum extraction procedures, symphysiotomy is the principal alternative to the cesarean operation. For a review of the history of cesarean delivery and of operative delivery in general, see Chapter 1.

Perhaps the best place to begin is with terminology. The question of how to term abdominal surgical delivery operations correctly is still debated [2]. By formal definition, a *cesarean delivery* is a surgical procedure that delivers a fetus, placenta, and membranes through an incision that passes through both the mother's abdomen (laparotomy) and uterus (hysterotomy). Technically, this definition

does not include operations performed to deliver either an extrauterine abdominal pregnancy or a fetus extruded from the uterus after a rupture. In common usage, however, the former procedure is often not considered a cesarean, whereas the second might. The term is also not applied to the removal of previsible ectopic pregnancies, or to hysterotomies performed at gestational ages of less than 20 weeks, although usage in the latter situation is again inconsistent. Another ambiguous situation exists when a very low abdominal incision inadvertently cuts through the anterior vaginal wall during the delivery, as opposed to the lower uterine segment. Such *laparoelytrotomy* procedures technically should not be classified as cesarean deliveries either but usually are, either because the vaginal site of the incision goes unrecognized or because appropriate coding is not available. Despite these and other technical quibbles, the definition employed in this text is that any surgical delivery that extracts a fetus of more than 20 weeks' gestation, living or dead, by an abdominal incision in the mother is considered a cesarean.

### Cesarean Delivery Rate

The rate of cesarean delivery has increased dramatically over the last 20 years, not only in the United States, but also around the world [450,451]. In the United States, the mean cesarean rate remained under 10% until 1965, and then in the following decade, the rate more than doubled. By 1989, the cesarean rate reached approximately 24%; since then it has varied between 25% and 30% and now is beyond that in many institutions. There are substantial differences in the performance of cesarean delivery depending on institution, practitioner, and the region of the United States where the care is provided. There are also wide variations in cesarean rates when international comparisons are made [1,3,4,450,451].

It is not known what the appropriate cesarean rate should be [5, 6]. Current rates in the United States are well above the 15% rate originally proposed by the Public Health Service in their Healthy People 2000 project. Overall, it is clear that the cesarean delivery rate can be reduced without altering perinatal mortality, but potential morbidity is another issue. Long labors and complex or difficult vaginal deliveries contribute to serious compli-

cations, such as fetal intracranial hemorrhage and other injuries [7]. Difficult vaginal deliveries are also believed to be an important factor in maternal pelvic/perineal injury resulting in long-term maternal problems with rectal sphincter function, the integrity of pelvic support and fertility.

Data concerning the cesarean delivery mortality varies depending on several factors: the medical system where the surgery takes place, the preoperative condition of the mother, the expertise of immediate postoperative care, the availability of properly trained anesthesia personnel, and the existence of a blood bank. In Western industrialized countries, the crude death rate associated with cesarean delivery is approximately 4 to 6 in 100,000 procedures. These data must be placed in context. In the United States, the overall maternal mortality is approximately 8 deaths per 100,000 live births [8]. This means that for the United States, the impact of cesarean delivery on overall maternal mortality is relatively low. In the nonindustrialized world, maternal losses do occur from cesarean deliveries, but many also happen owing to the inability of the birth attendants to perform indicated operations. In some parts of the world, maternal mortality is remarkable. In sub-Saharan Africa, maternal mortality rates as high as 920 per 100,000 are reported [9,10]. The average mortality for developing regions of Africa, Asia, and Latin America is 450 per 100,000 procedures. Particularly high rates occur in settings when there has been major blood loss, as can be associated with uterine rupture or other serious obstetric complications such as abruptio placentae or placenta previa. The risk is especially high in settings when the birth attendants have inadequate experience, safe anesthesia is not available, or there are limited facilities for the provision of supportive care. Most maternal deaths are not immediate but occur within the first three postoperative days.

International statistics and data about maternal morbidity and mortality are fraught with problems, among them incomplete reporting and the use of varied criteria for establishing an association between a delayed mortality and pregnancy. Commonly understated or poorly reported causes of maternal mortality include trauma from either accidents or criminal activity, and delayed pregnancy-related deaths from cardiac disease, pulmonary embolism, and other medical causes. An important tool in the accurate identification of delayed

maternal mortalities is the computer linkage of birth certificates to death certificates. This association enables proper identification of late deaths when the connection to pregnancy has become remote. Specific to the issue of the risk of a cesarean against that of a vaginal delivery also requires that the cases chosen for comparison have comparable gestational age, indication, and maternal status. For all these reasons, international comparative maternal mortality and morbidity data for both general obstetric care and cesarean delivery should be interpreted with caution.

## ASSOCIATED RISK FACTORS

### **Repeat Procedures**

In 1985, 36% of cesarean deliveries in the United States were repeat procedures, and the rate of vaginal birth after cesarean (VBAC) was 6.6%. That same year, the American College of Obstetricians and Gynecologists (ACOG) issued guidelines to promote VBAC. Subsequently, the VBAC rate increased to 12.6% by 1988. It was originally anticipated that the VBAC rate would continue to rise to approximately 50%, blunting the rise in overall cesarean deliveries. The popularity of VBAC trials has now been largely reversed because of concern about the occasional catastrophic complications of such trials, problems with physician coverage, and associated legal risks. Collectively, these complications have forced many smaller delivery services not to offer VBAC trials and dissuaded many practitioners from offering them [5].

### **Dystocia**

*Dystocia* is abnormal labor resulting from problems related to the powers, the passenger, or the passage [11]. This term encompasses a group of often loosely assigned diagnoses that includes *fetopelvic* or *cephalopelvic disproportion (CPD)*, *failure to progress*, *obstructed labor*, *dysfunctional labor*, *poor progress*, and *second-stage arrest*, among others. Dystocia accounts for approximately one third of the total cesarean delivery rate and is responsible for 30% or more of the increase in cesareans in the United States over the past 20 years. A small percentage of dystocia cases involve true CPD; that is, a condition in which there is an anatomic discrepancy between

the size of the fetal cranium and that of the maternal bony pelvis. Most dystocias occur because of various combined problems including fetal malpositioning accompanied by various abnormalities in labor. These problems are often described under the term *failure to progress*. In most cases, when the baby simply "won't come out" (WCO), the infants are normal sized and in demonstrably normal-sized pelvis. The clinical problems associated with poor progress in these cases most often includes cranial malpositioning (e.g., deflexed, asynclitic, occiput posterior, and so forth) combined with an element of poor or uncoordinated uterine activity. The important point is that 50% to 70% of women with a diagnosis of dystocia or CPD as an indication for an initial cesarean delivery can undergo successful vaginal delivery with a subsequent pregnancy. These observations emphasize that failure to progress, CPD, and dystocia are often diagnoses of poor labor function combined with minor degrees of fetal malpositioning rather than a marker for anatomic incapacity of the pelvis. (See Chapter 10, Labor.)

### **Electronic Fetal Heart Rate Monitoring**

In the United States, electronic fetal monitoring (EFM) is the routine type of surveillance for laboring women. As currently practiced, EFM is an imperfect method for fetal evaluation. The technique usually restrains women to laboring in bed and is associated with an increased rate of cesarean delivery [12,13]. Despite limited predictive value, abnormal fetal heart rate patterns are still relied on as the major method for the identification of cases of presumed fetal jeopardy (i.e., fetal distress) and are often the principal basis for obstetric intervention. Contributing factors to the aggressive interpretation of EFM signals include uncertainties and variations in pattern recognition, the current legal climate concerning medical practice, and the limitations of other methods for evaluating fetal condition. In its favor, although EFM has had little effect on the overall incidence of permanent neurologic abnormalities (e.g., cerebral palsy [CP]), it does reduce the risk of intrauterine death and can reduce the likelihood of some types of neonatal seizures [12]. There are additional techniques for fetal evaluation, including acoustic and scalp stimulation, fetal scalp pH measurement, and determination of transcutaneous fetal scalp oxygen tension, but all of these



have their own problems with practicality, reliability, and predictability. (See Chapter 2, Fetal Assessment.)

Despite the recognized limitations of intrapartum monitoring, *antepartum* EFM often combined with ultrasound (e.g., the biophysical profile [BPP]) is the major method for the evaluation of fetal well-being prior to the onset of labor [13]. A reactive non-stress test (NST), with or without other ultrasonically derived biophysical data such as a BPP, predicts a very low risk for intrauterine fetal death in the several days following the test, assuming the absence of an acute event, such as an accident, an abruptio placentae, or a cord prolapse [14–16].

### Breech Presentation

Approximately 10% to 25% of the overall cesarean delivery rate is ascribed to fetal malpresentation, principally breech. The incidence of breech presentation varies with gestational age. At 27 to 28 weeks' gestation, 30% to 40% of fetuses present by breech, whereas only 3% to 4% of fetuses do so at term. The current near-universal practice of cesarean delivery for the breech-presenting infant follows controversial reports of adverse outcomes for these infants if they are delivered vaginally [17]. (See Chapter 12, Breech Presentation.) External version, when performed, safely converts an average of approximately 60% of breech-presenting infants to cephalic presentation, resulting in a reduction in the frequency of breech presentation in labor and thus a modest reduction in cesarean delivery [18,19]. Unfortunately, even after successful version to a cephalic presentation, the rate of cesarean delivery for labor dystocia is increased for this cohort of women compared with cases in which version was not required. This is presumably reflective of poorly understood features of uterine activity or labor dynamics that initially predisposed to malpresentation and result in dystocia [20]. Other uncommon malpresentations, such as fixed transverse lies, funic presentations, compound presentations, or twins in collision are additional but uncommon indications for cesarean delivery.

### Demographic Factors

A host of demographic factors have been linked to the increase in the rate of cesarean delivery.

### Maternal Age

The frequency of cesarean deliveries increases for women 30 years of age or older [21]. As more women in the United States start their families after 30 years of age, they contribute to the rise in cesarean rate. An increased incidence of medical complications and dysfunctional labor in this patient group is frequently cited as the explanation. Social factors, such as the concept of "the premium baby," also probably influence operative delivery rates in these "elderly nulliparas," who often became pregnant by assisted reproductive techniques.

### Socioeconomic Factors

Paradoxically, women with low family income are less likely to be delivered by cesarean delivery and have fewer reported pregnancy complications than middle-class women [22]. They are also more likely to have a successful VBAC trial. Women who seek care through various clinics and public hospitals, where midwives, house officers, and hospital attending physicians usually provide services, experience lower cesarean rates

### Practice Style and the Medicolegal Environment

Concern about malpractice litigation has doubtless contributed to the rise in cesarean delivery rate, but this effect is difficult to gauge. The individual style of a physician's practice and original training does strongly influence the rate of primary cesarean delivery [23,24]. The unique set of circumstances that influences a specific clinician to decide for or against operative delivery in a given setting is often difficult to ascertain, complicating the task for quality assurance reviewers. Peer pressure, the climate of opinion about less compelling or borderline indications for surgery, as well as other factors constitute important but difficult-to-measure variables.

### Operative Vaginal Deliveries

The 1996 survey of ACOG fellows reported rates for operative vaginal deliveries by either forceps or by vacuum extraction of 10% to 15% [25]. In Demisse's 2004 review of more than 10 million deliveries [26], the overall instrumental delivery rate was 11.8% (4.4% forceps; 7.4% vacuum extraction). Over the past 15 years in the United States, Canada, and England, the rate of operative vaginal

deliveries declined at approximately the same time the rate of cesarean deliveries increased; however, it is not clear that these events are linked. Physician unwillingness to attempt some instrumental deliveries is a only a partial explanation for the increased cesarean delivery rate. The situation is far too complex to assume a simple cause-and-effect relationship between declining instrumental delivery rates and the rapidly rising number of cesareans, however. Unfortunately, assisted vaginal delivery has a poor lay reputation with its potential benefits decried and its risks overemphasized. In modern practice, significant maternal or fetal trauma from assisted vaginal delivery is quite uncommon. Furthermore, despite high rates of cesarean delivery and declines in most traditional obstetric procedures, there has been little or no reduction observed in perinatal morbidity due to permanent neurologic injury. These and other data indicate that antepartum events are substantially more important in the etiology of permanent neurologic injury than intrapartum occurrences, including the method of delivery. Thus, although it should not be assumed that the willingness to perform at least some instrumental deliveries will have much impact on current cesarean delivery rates, a role still remains for operative vaginal delivery in properly selected cases. (See Chapter 17, Instrumental Delivery.)

### **EPIDURAL ANESTHESIA**

Epidural anesthesia is justly popular owing to its efficacy in the relief of pain, low incidence of side effects, and patient acceptability. A controversy continues about the potential adverse effects of epidural blockade in increasing rates of operative vaginal and cesarean delivery. Epidurals do prolong the second stage of labor and increase the use of oxytocin to maintain progress. Meta-analysis suggests that whereas the newer techniques employing low-concentration local anesthetics are still associated with an increase in instrumental delivery rates, the rate of cesarean delivery is unaltered, however [27]. (See Chapter 9, Obstetric Anesthesia, for further discussion of epidurals.)

### **INDICATIONS FOR CESAREAN**

As the morbidity associated with cesarean delivery remains low, and the risks associated with elective

**TABLE 18.1 Potential Indications for Cesarean Delivery**

#### **Maternal**

Obstruction of the birth canal by a pelvic mass  
 Invasive carcinoma of the cervix  
 Previous vaginal or perineal surgery (e.g., fistula repair, prior rectal injury\*)  
 Cerebral aneurysms or arteriovenous malformations  
 Connective tissue disorders (e.g., Marfans syndrome, Ehlers-Danlos syndrome)  
 Pelvic malformation or pelvic bony inadequacy  
 Severe hypertension\*  
 Prior cesarean delivery\*  
 Multiple gestation (e.g., twins with malpresentation, triplets, or greater multiples)  
 Prior abdominal or Shirodkar cerclage placement  
 Failure to progress in labor\* (i.e., dystocia, cephalopelvic disproportion)  
 Prior transmyometrial uterine surgery or a Müllerian anomaly\*  
 Uterine rupture  
 Antepartum hemorrhage (e.g., placenta previa, abruptio placentae, vaso previa)  
 Suspected placenta accreta/increta/percreta

#### **Fetal**

Malpresentation (e.g., fixed transverse lie, compound presentation)  
 Fetal distress (e.g., presumed fetal jeopardy, nonreassuring fetal monitoring)  
 Fetal growth disorders (including IUGR)\*  
 Fetal anomalies (e.g., neural tube defect, conjoined twins)  
 Active genital herpes  
 Fetal macrosomia\*  
 Fetal thrombocytopenia\*  
 Maternal HIV infection\*  
 Other

\*These indications are relative, and in all instances vaginal delivery is not automatically precluded. Management depends on the unique circumstances of each case. See text for details.

operations are better appreciated, indications for cesarean operations have progressively increased. Although the indications for a cesarean can be categorized as either maternal or fetal, in many cases the indications are combined (Table 18.1).

#### **Placenta Previa**

Placenta previa is a potentially serious obstetric complication. The prevalence is approximately 4 in 1,000 live births [28–30]. The occurrence rate

depends on several clinical factors. In modern practice, with the virtual universal use of ultrasound scanning except in the small number of women who escape prenatal care, it is now rare that an abnormally implanted placenta escapes antepartum detection. Risk factors for placenta previa include multiparity, advancing maternal age, prior cesarean delivery, habitual abortion, male fetus, infertility treatment, cocaine use, and smoking [31,32]. Multiple gestations are also usually believed to be a factor. In a study of almost 39 million pregnancies drawn from the United States natality files between 1989 and 1998, the rate of placenta previa was 40% higher in twin (3.9/1,000 live births) than in singleton pregnancies (2.8/1,000 live births) [33]. It is also fair to state that not all studies have agreed with this association between multiple gestation and previa.

Important comorbidities for an abnormally sited placenta include malpresentation, a history of mid-trimester bleeding, abruptio placentae, congenital uterine malformations, low 5-minute Apgar scores, placenta accreta, increta, and percreta, postpartum hemorrhage or anemia, and perinatal mortality.

When characteristic painless vaginal bleeding occurs in the third trimester, prompt abdominal and vaginal ultrasound studies are indicated. A placenta previa or other serious obstetric complication such as abruptio placentae could be the cause. Uncommonly, a succenturiate lobe could present as a previa and be detected by transvaginal ultrasound even when a concomitant abdominal study cannot visualize this ectopic placental mass. Such succenturiate lobes are easily missed during routine abdominal scanning and might not be detected until unanticipated hemorrhage occurs.

This emphasizes the importance of conducting transvaginal studies when third-trimester bleeding is the reason for scanning. For experienced ultrasonographers, combined abdominal, transvaginal, and occasionally translabial scanning provides complementary data that are useful in clinical decision making whenever a problem with the position of the placenta is raised. Employing transvaginal ultrasonography in study of 100 suspect cases, Leerentveld and coworkers [34] reported a positive predictive value (PPV) of 93.3% and a negative predictive value (NPV) of 97.6% for the diagnosis of previa. Sensitivity of 87.5% and a specificity of 98.8% were also calculated. Also important to clinicians is

TABLE 18.2 Gestational Week of Placenta Previa Diagnosis vs. Confirmed Previa at Term\*

Gestational Week Previa Diagnosed	Present at Term
15–19	12%
20–23	34%
24–27	49%
28–31	62%
32–35	73%

\*940 examinations, 714 pregnancies.

From Dashe J, McIntire DD, Ramus RM, Santos-Ramos R, Twickler DM: Persistence of placenta previa according to gestational age at ultrasound detection. *Obstet Gynecol*, 2002. May;99(5 Pt 1):692–7; with permission.

the observation that in this study, the performance of the transvaginal studies did not worsen the bleeding in any case.

Not all low-lying or suspected previas identified before the third trimester are verified at the time of delivery. Although placental "migration" is a factor in this observed decline in the occurrence of previa, various technical problems also contribute, and the erroneous diagnosis of previa artificially increases the rate of apparent migration [35]. Technical problems in establishing the correct diagnosis of previa by ultrasonic scanning are largely but not entirely associated with abdominal studies. Artifacts are produced by overfilling of the maternal bladder, misidentification of segmental uterine contractions as placenta, excessive compression of the maternal abdomen during scanning, reliance on abdominal scanning only to establish the diagnosis, confusion of placenta tissue with an extramembranous hematoma in cases of abruptio placentae, and failure to consider the possibility of an accessory or succenturiate lobe if the cervix cannot be visualized.

Once artifacts are excluded, there is the issue of *migration*. A general observation that has been repeatedly verified is that the earlier in gestation the diagnosis of previa is made, the less likely that a true previa will be found at delivery [35–39].

Dasche has presented interesting data on previa persistence in a large retrospective series (Table 18.2) [39]. The observed incidence of previa changed with two factors: the week of gestation when the diagnosis was made and the extent of coverage of the internal os. At each gestational age interval when the placental edge was observed to

cover the os, a previa was more likely to persist at term than when only a partial placenta previa was observed. Prior cesarean delivery proved to be an additional risk factor for persistence of previa when the diagnosis was first made in the second trimester. Similar findings were reported by Mustafa, who also noted that the greater the degree of placental overlap over the internal cervical os, the higher the likelihood of a true previa at the time of delivery [35]. Not unexpectedly, when the ultrasound diagnosis of complete previa is made in the third trimester, the likelihood of finding a previa at delivery is higher and the chances for migration are poorer than among those pregnancies diagnosed with partial or marginal previas [40].

Placental migration has been the subject of considerable interest to ultrasonographers. The physiology of normal gestation involves the progressive enlargement of the uterus with advancing gestation. As the lower segment elongates, the placenta implantation site rises with it. This process progressively increases the distance from the placental edge to the internal os. Oppenheimer and coworkers estimated the rate of upward migration to be 0.54 cm a week in the third trimester [41]. This physiology explains why the later in gestation the previa or partial previa is diagnosed, the less effective this growth-related process becomes in converting a previa to a less dangerous type of implantation. This is reflected in the inverse relationship between the gestational age of the previa diagnosis and the declining percentage of previas found to have apparently migrated "late" in the third trimester. The most useful distinction for management purposes is to separate cases with a reasonable likelihood of vaginal delivery from those for whom a cesarean is best.

The distinction between a placenta previa and a low-lying placenta is usually based on the ultrasonographer's impression of whether the placental edge either meets or crosses over the internal os. Although this anatomic relationship can be determined by direct observation at a cesarean, presently it is much more likely to be an ultrasound diagnosis. Thus, in most cases of apparent previa diagnosed before the onset of labor, the ultrasound findings determine the final mode of delivery. There are two questions of importance with a placenta located at or near the internal os. First, will a hemorrhage ensue either spontaneously or with the onset

of labor? Second, is vaginal delivery possible? Not all previas are symptomatic prior to the onset of labor. When the diagnosis of previa is based on clinical signs and symptoms only, approximately 30% to 40% of women with a true previa do not experience bleeding prior to labor [42].

It has been repeatedly noted that some women who have experienced even severe antepartum hemorrhages from what was diagnosed as a previa have been able subsequently to deliver vaginally. In these women a subsequent vaginal delivery might be possible due to one of three well-known processes: progressive placental migration, tamponade of the placental edge after membrane rupture and descent of the presenting part, or an erroneous initial diagnosis. As discussed previously, errors are possible due to several factors. As examples, an intrauterine hematoma from undiagnosed abruptio placentae might be present or bladder overfilling could have distorted the appearance of the lower segment of the uterus. Because of these features, close and serial observation of women with low-lying placenta/partial previa with the avoidance of acute early intervention except in instances of severe hemorrhage constitutes the best plan of management. Delay permits the fetus to mature, does not risk the mother, and with the effects of migration, in some cases eventually results in vaginal delivery.

In the past, a double set-up examination was used to determine the possibility of vaginal delivery when placenta previa was suspected. When a double set-up was performed, all preparations for a cesarean were completed, and a cervical examination was conducted in the operating suite by the most senior clinician. If the membranes were palpable and not the placenta, amniorrhexis was performed and induction of labor was attempted; otherwise a cesarean was performed. With the current use of translabial, transperineal, and transvaginal ultrasound, such procedures are rarely necessary. In the unusual case, if the ultrasonic and clinical data are difficult to interpret, a hematoma is suspected at the os, or a succenturiate lobe that is difficult to identify is possible, a double set-up is still appropriate. An alternative means to establish the correct diagnosis in some of these situations might also be magnetic resonance imaging (MRI).

The best available data indicate that the distance from the internal os to the placental edge is

important in making a clinical choice about attempting a vaginal delivery. If the placental edge is  $>3.5$  cm from the os, observed antepartum bleeding is unlikely to be due to previa, and a vaginal trial is appropriate. If the edge is  $>2.0$  cm from the os, a labor trial is still possible because approximately 60% of these cases deliver vaginally [41–43]. If the edge is closer than 2 cm or if the placental tissue crosses the os, a cesarean is best.

Prior cesarean delivery is a major risk factor for both previa and abnormal placental adherence. Because an abnormally located placenta is likely to persist in its location, follow-up studies are obviously indicated in cases in which the cervical os is seen to be covered by placenta during mid-trimester scanning. Also, whenever a low-lying placenta is observed to migrate, one should evaluate the cord insertion by color Doppler study to the exclude the possibility of a vasa previa. Placenta accreta/increta/percreta is a possible serious complication of a low-lying placenta or placenta previa that should also be considered, especially in women with a history of a prior cesarean delivery, prior gynecologic operations, or who have experienced prior difficulties with placental removal. Ultrasound diagnosis of abnormal placental adherence is discussed in the following section.

### **Placenta Accreta, Increta, and Percreta**

These conditions are due to abnormal placental adherence with varying degrees of trophoblastic invasion of the myometrial wall. The incidence of such abnormal placentation has increased markedly in recent decades. Wu and coworkers in a recent review [443] reported finding an abnormally adherent placenta in 1 of 533 of deliveries. The principal cause of this disorder is believed to be injury to the endometrial lining of the uterus secondary to either infection or surgical scarring (e.g., cesarean delivery). Advanced maternal age, cigarette smoking, and noncesarean types of uterine surgery are additional predisposing factors.

When abnormal myometrial invasion by the placenta is suspected, real-time ultrasound imaging with color Doppler study is best in establishing the correct diagnosis [440,441]. Warshak [440] and coworkers also suggest a role for MRI in difficult cases. Complete placental evaluation at MRI might require the maternal administration of dye that

crosses the placenta with uncertain fetal effect, however. Even Warshak's paper did not find that MRI was helpful save in the small number of equivocal cases that ultrasound scanning could not resolve. Obstetric experience with MRI is also quite limited in many centers. For these reasons, reliance on MRI studies may or may not be prudent in an individual institution. Therefore, ultrasonic examinations are recommended as the best evaluation tool for the large majority of cases when unusual placental adherence is suspect.

Several ultrasonic findings are reported to be predictive of deep placenta/myometrial invasion. These findings include hypervascularity by color Doppler study, attenuation of the usual "clear" space at the placental base myometrial interface, changes in intraparenchymal placental lacunar flow, and the observation of a substantial number of discrete hypoechoic spaces ("lakes") within the substance of the placenta [440–442]. Also of importance, especially when a percreta is present, are various abnormalities in the appearance of the interface between the lower uterine wall and that of the adjacent bladder. Not all of these markers are of equal importance. The absence of the "clear zone" at the placental base is the least useful sign for predicting an accreta, principally because it is often absent in otherwise normal anterior placentas. Failure to demonstrate this zone is a common cause of false-positive diagnoses or of the identification of "possible cases" where concern is raised unnecessarily. Studies of either color Doppler flow or evaluation of the continuity of the bladder/myometrial serosal interface are the most predictive. The presence of multiple fusiform ("tornado-shaped") placental lakes (lacunae), especially those positioned close to the maternal surface of a low-lying placenta or placenta previa, is the best single marker for an accreta. The lakes predictive of abnormal placental adherence are those easily demonstrated by standard color Doppler flow studies. Normal or innocuous vascular lakes are more circular in appearance than the pathologic ones. Although swirling flow can be demonstrated within the benign lakes, the rate of flow is so low in these areas that the color Doppler studies usually do not demonstrate any flow. It is presumed that the increased flow, Doppler-positive, and pathologic lakes reflect fetal circulation, which is at high velocity. In contrast, the otherwise benign Doppler-negative lakes that the color studies cannot

record reflect maternal flow, which is of substantially lower peak velocity.

All of these unusual disorders of placental adherence are potentially serious, especially if adjacent tissues have been invaded by placental growth (placenta percreta). At surgery, heavy blood loss is common, and partial excision of adjacent structures (e.g., bladder) or hysterectomy is sometimes required to control bleeding. If the diagnosis is suspected preoperatively, special preparations with the anesthesiology staff and the blood bank are prudent, and competent assistants must be recruited. The possibility of preoperative placement of intravascular catheters for intraoperative embolization should also be considered but might not actually result in a lower blood loss. In some cases it is better to leave the placenta in situ and close the abdomen rather than attempt an immediate removal. Postoperatively, the patient can be transferred, plans can be made to return under more favorable circumstances, or an antimetabolite such as methotrexate can be administered. These cases are complex, serious complications are common, consultation is necessary, and care must be individualized.

### Vasa Previa

In *vasa previa*, unsupported fetal vessels traverse the fetal membranes in advance of the presenting part [43–47]. With either spontaneous or induced membrane rupture, one or more of these fetal vessels can rupture, potentially leading to rapid fetal exsanguination. Historically, the fetal mortality rate from vasa previa has varied between 20% and 100% [44]. Vasa previa at term is closely associated with low-lying placentas or partial previas and is more common in IVF-related or multiple gestations or following placental migration from an originally low placental implantation site [45]. Fortunately, establishing the diagnosis by ultrasonic scan prior to the onset of labor is possible, if the possibility of the condition is suspected [46]. In routine scanning, whenever a placenta is noted to be low lying, in twin gestations, or when a case is under study for placental migration, the placental cord insertion site should be verified by color Doppler studies. Known vasa previas diagnosed prior to acute symptoms present the clinician with management issues. For fetal protection, delivery must occur prior to membrane rupture, but the pregnancy must also advance suffi-

ciently far to avoid the risks of prematurity. Management of such cases must be individualized, balancing the risk of vessel rupture against that of the risk of prematurity [47]. Delivering by an elective cesarean at or about the 35th to 36th week following steroid treatment is ideal, but early cervical change or demonstrated uterine activity could force the issue earlier. It is also apparently possible to coagulate abnormal vessels by laser in utero [454]. The impact of this exotic technology remains to be established in terms of overall management.

### Birth Canal Obstruction

Rarely, various maternal or fetal tumors obstruct the birth canal. A cervical myoma or a mobile ovarian tumor can either fill the true pelvis or occupy the lower uterine segment and obstruct the presenting part. A pelvic kidney is another possibility to be considered when an intrapelvic mass is palpated. Rarely, a fetal tumor such as a sacrococcygeal teratoma or the unique situation of conjoined twins renders the fetus or fetuses undeliverable. Conversely, an ovarian mass or pedunculated leiomyoma that fills the pelvis early in pregnancy can be spontaneously displaced abdominally by the second or third trimester, leaving the birth canal free and permitting unobstructed vaginal delivery.

If a tumor fills the pelvis, bulges into the cul-de-sac, or otherwise prevents engagement of the fetal presenting part (a tumor previa), then cesarean delivery prior to the onset of labor and usually removal of the mass are indicated, depending on the clinical findings. Attempting to dissect out large intramural leiomyomas during a cesarean is usually imprudent. The blood loss can be extensive and control difficult. The better plan is to avoid the myoma on surgical entry, deliver the child, close the abdomen, and then reassess for additional treatment in the puerperium or later. If the diagnosis of a “suspect” pelvic tumor is made early in pregnancy and surgical removal is contemplated, surgery is usually reserved until the second trimester. Pelvic masses obstructing labor might not be uterine or ovarian tumors. A pelvic kidney, a prolapsed spleen, an entrapped bowel loop, and a bladder tumor, among other possibilities, are rare culprits. In all of these cases, management must be individualized. If ultrasonic scan cannot identify the source or type of mass with high reliability, then either MRI, or less

frequently, a computerized tomography (CT) scan can be performed to assist in identification.

### **Invasive Carcinoma of the Cervix**

When cervical cancer is diagnosed during pregnancy, both the patient and the clinician face difficult decisions. Techniques for evaluating cytology, conducting culposcopy, and performing cervical biopsies are generally the same in pregnancy as in the nonpregnant state [48]. During pregnancy, however, conization is avoided when possible and endocervical curettage is not performed. Overall, approximately 1 in 2,000 pregnancies is associated with cervical cancer, and about 3% of cervical cancers occur in pregnant women [49–51].

As in nongravid patients, 80% to 90% of cervical neoplasias diagnosed during pregnancy are squamous or adenosquamous [52]. Approximately 70% are early tumors, not of advanced stage (Stages Ia, Ib, or IIa). Cervical cancer diagnosed during pregnancy is staged using the standard International Federation of Gynecologists and Obstetricians (FIGO) classification [53]. Because most pregnant women with cervical tumors are asymptomatic, the diagnosis is usually initially suspected or diagnosed following the performance of routine Papanicolaou smears [54]. If symptoms do exist, the most common is vaginal bleeding [55]. Whenever a suspect Pap study is reported, prompt culposcopic examination with biopsy of suspect lesions is performed. Fortunately, complete visualization of the transformation zone is usually possible. Conization is to be approached with trepidation because of the risks of hemorrhage, abortion, membrane rupture, or premature labor [56]. The risk in conization is probably proportional to the size of the excised tissue mass [57]. Nonetheless, a cone biopsy is indicated if the colposcopy is unsatisfactory and the cytology strongly suggests invasive cancer.

If invasive cancer is diagnosed, the therapeutic options depend on the period of gestation, the advance of the tumor, and the wishes of the affected woman. Most studies agree that there is no difference in survival between pregnant and nonpregnant individuals when matched for age, year of diagnosis, and tumor stage [52,55,58,59]. Interruption of pregnancy with immediate treatment or a delay in delivery until pulmonary maturation is reached are the basic options. Theoretically, there is some

increased maternal risk when definitive therapy is delayed by several weeks to permit fetal maturation, but available data do not suggest an increased maternal risk if this plan of management is chosen in women with Stage I tumors [48,60].

Traditionally, patients with early-stage carcinoma of the cervix (Stages I, IIa) have been delivered by a classic cesarean with radical hysterectomy and pelvic node dissection. This surgery is associated with low morbidity, 80% to 95% long-term survival, and preservation of ovarian function [61,62]. Usually it is argued that the more advanced stages of cervical cancer require cesarean delivery to avoid hemorrhage that might accompany a vaginal delivery, or implantation of tumor in an episiotomy or laceration site. In selected cases, a classic cesarean can be followed by irradiation as the definitive therapy, if a bulky cervical tumor is present. The more advanced cases (Stages IIb and beyond) are treated by irradiation, with or without chemotherapy. If the fetus is viable, it is first delivered before therapy is initiated [55]. Appropriately evaluated cases of severe cervical dysplasia or carcinoma in situ without evidence of invasion are not indications for cesarean delivery. In this setting, vaginal delivery is appropriate, with postdelivery reevaluation and subsequent definitive treatment.

There are unresolved controversies in the management of pregnant women with cervical cancer, and no prospective or randomized studies exist comparing the appropriateness of vaginal versus cesarean delivery for most cases. Nonrandomized studies indicate no differences in overall survival rates, regardless of the mode of delivery [63]. The risks of possible obstructed labor, hemorrhage, and the potential for seeding and recurrence at episiotomy sites have led most observers to recommend cesarean delivery when truly invasive cancer is diagnosed, however [48,52,64,65]. The general recommendation is that vaginal delivery be reserved for women with preinvasive Stage Ia or some early invasive disease with planned postpartum and potentially fertility-sparing definitive treatment.

Proper management of cases involving malignant and premalignant cervical tumors requires a team effort between oncologist and obstetrician and often includes other practitioners. Complete evaluation and careful counseling are needed to reach a management plan acceptable to the affected woman and appropriate to the stage and grade of her disease.

Coordination among the anesthesiologist, oncologist, and obstetrician are required for proper treatment. (See Chapter 16, Surgery in Pregnancy.)

### Previous Vaginal Surgery

Previous repair of vesicovaginal or retrovaginal fistula is a potential, and for most practitioners, a strong indication for cesarean delivery [66]. The risk is that in another pregnancy, descent of the fetal presenting part or the method of delivery will disrupt the original repair. The alternative view is that abdominal delivery should be reserved for cases in which the original fistula repair was difficult (i.e., more than one operation), required colostomy, or when inflammatory bowel disease (i.e., Crohn's disease) or other complications are present.

When the problem is a prior rectal sphincter laceration, especially if residual rectal dysfunction or partial incontinence is present, care needs individualization. Here the magnitude of the risk of a vaginal trial is difficult to ascertain accurately. The available evidence suggests that additional damage does follow subsequent vaginal birth, however. In such cases, a frank discussion with the affected woman concerning management alternatives is mandatory. In practical terms, most clinicians and patients will not accept the risk of a repeat injury or damage to the original repair; if either a fistula or a severe perineal laceration involving the rectum followed the first delivery, a cesarean delivery before the onset of labor usually is planned in this setting.

There are other clinical situations in which a cesarean delivery is appropriate based on previous surgery. A prior abdominal cerclage or epithelialized Shirodkar cervical sutures are two possible indications. For such cases, when future pregnancies are desired and the original suture placement required laparotomy, laparoscopy, or a difficult vaginal surgery for insertion, a cesarean is favored for the subsequent pregnancy.

### Cerebral Aneurysm or Arteriovenous Malformation

If the mother is diagnosed with an uncorrected cerebral aneurysm or arteriovenous malformation, cesarean delivery is usually recommended, although the specifics of care are individualized [455–461]. A

vaginal delivery is appropriate following successful repair of an arteriovenous malformation, however, provided hypertension is not present and the second stage is assisted instrumentally by forceps or the vacuum extractor to limit voluntary bearing-down efforts. Such cases are best managed under regional or epidural anesthesia. A similar protocol can be considered in the management of patients with prior retinal or vitreous hemorrhage. The basic treatment remains the same during pregnancy as among the nonpregnant. Aneurysms are identified and controlled with either surgery or endovascular techniques [461]. It should be emphasized that few data support these recommendations; no prospective trials have been performed. All such rare cases require individualized management with an anesthesiologist and a neurosurgeon.

### Connective Tissue Disorders

*The Marfan syndrome* is a rare, autosomal dominant disorder of connective tissue involving a defect in the gene encoding fibrillin located on chromosome 15. The fibrillin-1 gene codes for a glycoprotein associated with the formation of normal elastin. Abnormalities in elastin are the principal abnormality in this disorder. The prevalence of the Marfan syndrome in the general population is estimated as 7 to 17 in 100,000, and it occurs equally in both males and females [67]. Clinical manifestations of this disorder involve the skeletal (e.g., pectus deformity, kyphoscoliosis), ocular (e.g., retinal detachment, lens dislocation, myopia), and most important, the cardiovascular systems. The prognosis for affected individuals is limited by the extent of cardiovascular involvement [68,69]. The cardiovascular risk is principally associated with progressive dilation of the proximal aorta, leading to dissection or rupture. Mitral valve incompetence or dysfunction is also possible.

In Groenink's series from the Netherlands, involving 125 subjects with the Marfan syndrome who were equally divided between male and female subjects, approximately 1 in 3 (34%) developed serious cardiovascular problems in the 10 years following their initial diagnosis [68]. Overall, cardiovascular complications account for more than 80% of the mortalities in Marfan's patients. Fully 80% of these deaths are secondary to aortic dissection and to various complications of cardiac failure



either involving mitral or aortic valve dysfunction [67,68].

Aortic root size, measured by ultrasound scan, is an important risk factor for valve dysfunction, and more important, aortic dissection. For the nonpregnant patient, *prophylactic* cardiac surgery is often recommended for specified aortic root diameter (usually 50 mm–55 mm, and in some series, less) [70], or for those with a family history of dissection or if rapid expansion of the ultrasonically measured root diameter is documented ( $\geq 2$  mm/year) [68,71]. If a prophylactic aortic replacement is possible, the five-year survival is 95%, but this figure drops to approximately 50% if the repair is performed as an emergency.

The pregnancy-associated risks of the Marfan syndrome are twofold. First is the potential to deliver a child with the syndrome. Second, and of more immediate importance to the clinician, is the potential for a catastrophic and potentially lethal aortic dissection or another cardiovascular complication occurring during or immediately after the pregnancy. There is also a substantial incidence of other obstetric difficulties with these pregnancies. As an example, similar to individuals with the Ehlers-Danlos syndrome (EDS), pregnant Marfan patients are at risk for cervical insufficiency and postpartum hemorrhage. Spontaneous pregnancy loss and preterm labor are additional frequent complications [72,73]. For pregnant women with the Marfan syndrome a *preconceptual* aortic root diameter of  $\geq 40$  mm (other diameters are sometimes recommended), observed progression of the root diameter, mitral valve dysfunction, and declining cardiac function are the principal risk factors [74]. When the aortic root diameters are  $\leq 40$  mm and the valve appears normal on echocardiographic study, the maternal mortality rate is 5% or less. Women with root diameter  $\geq 40$  mm or evidence of decompensation should be advised not to attempt pregnancy [75,76]. However, women with this disorder remain at some risk for spontaneous dissection even if a preconceptual aortic diameter is found to fall within the “acceptable” limits. Close observation of these patients is therefore always required.

In terms of general management, if a woman's cardiac function is evaluated as normal and her aortic root diameter is  $\leq 40$  mm (or perhaps 45 mm), she might tolerate gestation well. If no cardiovascular problems ensue, vaginal delivery under epidural

anesthesia is possible [72,77]. If cardiac or vascular complications do occur, they usually manifest themselves in the second or early third trimester, but occasionally problems have been reported both in the first trimester or postpartum [78]. During gestation, the administration of beta blockers is recommended in the effort to potentially retard aortic root dilation [79]. If progressive root dilation is observed during gestation, there is a family history of dissection, there are other cardiovascular symptoms, or the initial aortic root size exceeds  $>40$  mm to 45 mm, a cesarean delivery is preferred [78]. It should be noted that various investigators recommend slightly different critical values for aortic measurements and also vary in their other recommendations for evaluation and treatment of these unique and difficult cases.

For general antepartum management, hypertension and cardiac arrhythmias should be serially investigated and aggressively treated. Postpartum hemorrhage is common in these women and should be anticipated. The pediatrician should be notified for a careful examination of the neonate for the stigmata of the Marfan syndrome constitute. The anesthesiologist also must be closely involved in labor and delivery management.

If a major cardiac surgery is required in pregnant women with the Marfan syndrome, the combination of cardiopulmonary bypass and hypothermia is not tolerated by the fetus. In this situation, a cesarean delivery should be performed first and then followed by the cardiovascular procedure [80].

Included under the term EDS is a heterogeneous group of rare, principally autosomal dominant connective tissue disorders associated with various pregnancy complications ranging from increased bruising to maternal death [81,87]. The incidence of EDS in pregnant women is estimated as 1 in 100,000. The principal structural defects arise from abnormalities in genes encoding collagen or collagen-modifying enzymes [82,83]. There are at least ten recognized variations or types of EDS, based on either demonstrated clinical findings or biochemical defects. The likelihood and the potential severity of EDS during pregnancy depend on the type. Types I to III are the most common and collectively account for 60% of cases. Type IV occurs in approximately 10%, with all the other recognized types occurring much less frequently. Women with Type IV EDS who have mutations in the gene for type III

procollagen are at particular risk for maternal mortality. In this subgroup, maternal losses are reported to range from 11.5% to 20% or more per pregnancy [84–86].

All types of EDS are associated with an increased risk of a wide range of obstetric complications. These complications include premature membrane rupture, premature (and possibly precipitate) labor, cervical insufficiency, spontaneous pregnancy loss, postpartum hemorrhage, separation of the pubic symphysis, the formation of perineal hematomas, and impaired wound healing [85,87].

Clinically EDS is characterized by hyperflexibility of joints, easy bruisability, hyperelasticity of skin and, in the more severe types, weakness in both the arterial and bowel walls, predisposing to spontaneous vessel rupture and intestinal perforations [88]. Coagulation is usually normal, but in some EDS patients, various abnormalities, including those of platelet function, are possible.

In 2001, Schalkwijk and coworkers described a type of EDS that was genetically and clinically different from the previously described varieties [82]. The newly identified tenascin-X deficiency type of EDS is apparently inherited as a recessive trait. Tenascin X can be either an important structural component of collagen or affect collagen synthesis in a currently unknown manner. The potential obstetric risks due to this disorder are unknown.

Management of the severe Type IV EDS cases when abortion is not performed are based on avoidance of activities that might produce increased physical stress. Other normal events such as uterine contractions or maternal bearing-down efforts can risk uterine or vessel rupture. Instrumental delivery can result in increased perineal injuries, hemorrhage, or hematoma formation. Usually steroids are recommended in these cases, and an elective cesarean is planned for after the 32nd week. Even if a cesarean is performed, wound healing can be impaired, and there is an increased risk of wound dehiscence. These facts should lead the surgeon to plan the wound closure accordingly. Performing a running bulk closure of the abdominal wall with a long-lasting absorbable suture material is one potential option.

In at least one case, 1-desamino-8-D-arginine vasopressin (DDAVP) has been administered preoperatively in a woman with EDS suspected of having an increased bleeding tendency [89]. This drug

should *not* be routinely administered unless a consulting hematologist advises its use, however.

### Pelvic Malformations

True pelvic contracture is rare in modern practice. An anatomic defect in the bony birth canal usually results from a previous pelvic fracture. There can be subsequent malunion of the pubic rami, or it occurs secondary to callus formation in the process of healing. Pelvic deformity is also occasionally seen in persons with hereditary skeletal dysplasias such as achondroplasia. Pelvic abnormalities from rickets, which occupied much obstetric interest in the nineteenth century, have disappeared from modern practice. Other rare causes of pelvic contracture include patients with severe kyphoscoliosis, prior poliomyelitis, or chronic neurologic injuries. Most of these patients are also usually at an increased risk for cardiovascular and pulmonary compromise and require careful assessment by the obstetrician and anesthesiologist before any surgical procedure is considered. Rarely, and in selected cases, pelvimetry using CT or MRI might prove helpful in evaluating pelvic configuration and dimensions in the consideration of a vaginal trial. These unusual cases require individualized care and prelabor or predelivery evaluation to determine if a trial of labor is prudent.

### Hypertension

Uncomplicated mild-to-moderate maternal hypertension is not an indication for cesarean delivery; however, a cesarean becomes the treatment of choice when preeclampsia or other hypertensive disease is severe and rapidly progressive, the pregnancy is remote from term (32 weeks or less), or the cervix is unfavorable for induction and prompt delivery is indicated for maternal (or fetal) reasons. Depending on the clinical setting, eclampsia is another potential indication for a cesarean. Patients with severe hypertensive disorders are also more likely to develop other serious obstetric complications such as abruptio placentae or fetal distress, or to carry a growth-restricted fetus. These compounding risks frequently necessitate timely cesarean delivery for both maternal and fetal well-being either prior to the onset of labor or intrapartum, even if a vaginal delivery had been originally planned.

### Combined and Other Indications

Despite efforts to comply with standard indications, occasionally an obstetrician performs a cesarean delivery for combined reasons. There are complex maternal and fetal conditions for which data concerning the best mode of delivery are unavailable or the extant data are contradictory. Such cases might involve a woman with a history of a significant maternal or fetal injury after a vaginal birth, or one who is carrying an anomalous fetus. Patients request cesareans for simple preference, to avoid labor indications such as the potential avoidance of damage to pelvic support structures. The latter issue is further discussed in Chapter 17, Instrumental Delivery.

The risks and benefits of the method of delivery must be weighed against the severity of the underlying disease or condition. Whenever the clinical circumstances are atypical, however, a careful informed consent is necessary. The circumstances and the rationale that went into the decision about the mode of delivery and documentation of the consent process should be carefully noted in the medical record.

## FETAL INDICATIONS

### Abnormal Presentation

In a term gestation, abnormal presentations such as a transverse lie, persistent brow, or fixed mentum posterior are best managed by cesarean delivery. Such malpresentations represent only a small percentage of patients presenting in labor, however (fewer than 1/300 deliveries) [90]. In addition, some cases in which the fetal presentation is transverse, oblique, or unstable can be successfully converted to a longitudinal lie and cephalic presentation by external version. Achievement of a cephalic presentation does not guarantee a successful vaginal delivery, however, because the problem leading to the initial malpresentation appears to affect labor progress as well [20]. Conditions such as a fixed transverse lie do not present a clinical challenge. Such cases and other similar problems of extreme malpresentation are easily diagnosed, and there is consensus about the need for prompt cesarean delivery. The difficult cases are those involving minor degrees of cranial deflection or malrotation, when progress in labor is desultory, or in frank breech presentations

in multiparas, for which management remains controversial. In cephalically presenting infants, fetal disproportion often accompanies marked cranial deflection, especially if the deflection persists as the head reaches the midpelvis. Instrumental deliveries in these instances require careful consideration to avoid efforts that attempt to overcome true disproportion. A compound presentation (e.g., head/hand) is an uncommon indication for cesarean delivery unless complicated by umbilical cord prolapse or an arrest of dilation and descent. A prolapsed posteriorly positioned fetal extremity is most likely to lead to a cesarean if the small part does not recede as labor progresses. In occiput posterior presentations, if the fetus fails to rotate promptly following oxytocin stimulation or cannot be manually rotated, a cesarean is often performed, even at full dilation and low station. This is due to the unwillingness of many obstetricians to perform rotational instrumental vaginal deliveries, secondary to the general waning of forceps delivery skills and concerns of fetal/maternal injury. (See Chapter 17, Instrumental Delivery.)

Operations for breech presentation contribute about 2.5% to the overall cesarean delivery rate. One result of the nearly 100% operative rate for breech presentations has been a loss of the opportunity for obstetricians in training to acquire the skills necessary for conducting a vaginal breech delivery. Unfortunately, these same skills are needed for safe fetal extractions during a cesarean delivery and are especially important for growth-restricted or premature infants. (See Chapter 12, Breech Presentation.)

### Suspicion of Immediate or Potential Fetal Compromise

Perceived fetal jeopardy, classically termed *fetal distress*, or *nonreassuring EFM* or *abnormal auscultatory FHR in labor* or, more recently, *suspicion of immediate or potential fetal compromise*, is the cause for 1% to 3% of cesarean deliveries. There is wide and inconsistent international variation in the reported incidence of fetal stress/distress in labor, strengthening the suspicion of the accuracy of this diagnosis. Despite the efforts of various professional organizations, the criteria used for the diagnosis of fetal jeopardy or distress still vary widely among

institutions and between practitioners. (See Chapter 22, Fetal Assessment.)

Given the well-known limitations in the diagnosis of fetal jeopardy by current EFM methods, new techniques to reduce the false-positive rate have been sought. Recent interest has focused on electronic techniques combining ST-segment analysis with heart rate patterns. This approach has been reported to improve diagnostic accuracy [91]. A new heart-rate monitor incorporating this system (STAN 531, Neoventa Medical, Gothenburg, Sweden) recently won the approval of the Food and Drug Administration (FDA). First use in the United States began in October of 2007 [91b]. Of interest, ACOG is withholding their endorsement of this technique and the device until additional clinical data are available. Whether this combined technique of electrocardiographic and EFM tracing analysis proves any better at the diagnosis of fetal jeopardy than classic EFM pattern analysis remains moot.

Although cesarean delivery can clearly salvage fetal life in selected situations (e.g., abruptio placentae, cord prolapse, vasa previa, placenta previa, and other acute obstetric conditions), it is less certain in many other cases that fetal or neonatal morbidity is reduced or prevented by operative intervention. A healthy skepticism concerning the efficacy of many "rescue" operations for a perceived abnormality in EFM tracing is appropriate. It is now known that 75% or more of the permanent neurologic damage to neonates occurs because of events remote from labor and delivery [92]. Given current knowledge and diagnostic abilities, these injuries are for the most part neither recognized antepartum nor preventable, and heroics practiced in the delivery suite cannot change these facts. Unfortunately, during labor it is usually difficult and often impossible for a clinician to differentiate between an otherwise normal infant stressed or damaged by the process of labor and one whose observed deterioration is due to a chronic debilitating process that had its origin before the onset of labor. It is precisely this problem that is the source of many difficult medicolegal cases.

### Fetal Anomalies

With the recent advances in ultrasound technology, the list of congenital anomalies for which prena-

tal diagnosis is available has expanded rapidly. (See Chapter 3, Ultrasound Examination, and Chapter 20, Fetal Surgery.) This, with the improvements in neonatal surgery and survival rates of anomalous fetuses, has placed added responsibility on the obstetrician for determining the best mode of delivery when the infant is suspected to be abnormal. The decision-making process must take into account whether 1) the fetal anomaly can result in dystocia; 2) the likelihood of fetal morbidity or mortality is increased by vaginal delivery; 3) timely delivery would prevent further deterioration in the fetal condition; 4) the fetus is capable of survival; and 5) there is a need for immediate medical or surgical intervention at birth.

Clinicians should recall the limitations of current imaging techniques. Despite the ability to visualize major congenital malformations by ultrasound scan, an exact evaluation of fetal condition is usually not be possible before birth [93–95,95b]. Furthermore, associated or minor anomalies accompanying more major malformations are frequently missed. Because the prognosis of many congenital conditions is not entirely predictable despite the most expert antepartum evaluation, both physician and patient might favor an abdominal delivery for reasons of emotion and uncertainty. Despite these and other limitations, there are several conditions for which there is a reasonable consensus that a cesarean delivery reduces fetal trauma and has the potential to improve fetal condition. Fetuses that *might* benefit from cesarean delivery include those with 1) advanced hydrocephalus and breech presentation with or without open neural tube defect, 2) diaphragmatic hernia, 3) large sacrococcygeal teratomas, 4) cardiac arrhythmias that are refractory to medical treatment and preclude FHR monitoring, 5) some forms of osteogenesis imperfecta, 6) conjoined twins, and perhaps 7) selected infants with abdominal wall defects (e.g., gastroschisis and omphalocele). Clinicians should remain skeptical of the presumed benefits of cesarean delivery for various fetal anomalies no matter how reasonable the argument seems, unless there is good evidence from a randomized trial that a true benefit exists. Recent experience with meningomyelocele, for example, fails to document strong advantage from operative delivery. This is again a situation in which preexisting condition and limits on development rather

than the mode of delivery are the most important factors in the neonate's eventual outcome [96]. (For additional data and a review of this complex and rapidly changing issue, see Chapter 20, Fetal Surgery.)

The most important issue is potential fetal viability. Obstetric interventions performed before the period of potential fetal survival are only meddlesome heroics. Unfortunately, because obstetric management is imperfect, and true fetal weights or gestational ages are accurately known only in retrospect, honest errors in judgment are inevitable.

Some general guidelines are helpful, however. To the clinician, potential fetal viability is not a rigid marker but a working definition that varies among institutions and, over time, will reflect improvements in neonatal care. As a practical matter, fetal survival is *unlikely* if the pregnancy is less than 22 completed weeks and the fetal weight less than 350 g to 400 g. Fetal survival is increasingly possible with gestational ages beyond 23 completed weeks and 450-g to 500-g weight, but many of these very small survivors have serious morbidity and permanent injury. Reasonably likely and intact neonatal survival – defined as greater than 50% survival and greater than 50% normal – cannot be confidently anticipated unless the period of gestation is at least 24 (and preferably 25) completed weeks with an estimated fetal weight of approximately 500 g. Clinicians should be aware of the statistics for their own as well as their higher-level referral institutions. Counseling provided to families must be realistic and avoid providing false hope but also accurate, indicating what the true likelihood is for intact infant survival [97]. For management of these very premature infants it is prudent to discuss the case with a neonatologist and review the outcomes to be anticipated given the estimates of weight and gestational age. With this discussion the clinician can be certain of the most recent local data. Further, this consultation can help avoid an uncomfortable situation that could occur if either the obstetric or pediatric/neonatal personnel provide different statistics to the family, potentially leading to different recommendations. It is always important to be certain that the pediatrician understands that the fetal weights and gestational ages are simply the best available *estimates*, the accuracy of which could vary between cases.

## Genital Herpes

Herpes simplex viral infection is a possible indication for cesarean delivery. Genital infections during pregnancy from herpes simplex virus (HSV) type 1 or type 2 are potential serious clinical problems because of the risk for transmission to the fetus or newborn [98,99,100]. In the United States there are approximately 65 million people currently infected with genital herpes. Another one million new cases occur each year. Overall, it is estimated that herpetic viral shedding occurs in 0.35% to 0.65% of all pregnant women. Maternal herpetic infections during pregnancy are associated with an increased risk of abortion, prematurity, and neonatal congenital herpes.

Although the maternal carriage rate for herpes is high, the overall attack rate is low and depends on whether the maternal infection is primary or recurrent. If the mother has a *recurrent herpetic infection* at the time of delivery, 3% to 5% of newborns will develop neonatal infection. Conversely, 33% to 50% of newborns will become infected if delivered vaginally in the presence of a *primary infection*. The general management rule is that virus and baby should not meet. Because risk is significantly reduced by cesarean delivery, ACOG recommends cesarean delivery for women with *active* herpetic lesions present in the birth canal at parturition [102]. A cesarean is appropriate regardless of the elapsed time if a membrane rupture has occurred.

Fetal infection is an ongoing problem [99,100]. In the United States, approximately 1,600 to 2,000 neonates contract HSV yearly. In about one third of cases, these infections are due to the HSV type 1 serotype, the remainder to HSV type 2. HSV type 1 infections are less likely to result in severe symptoms. In contrast, HSV type 2 infections are responsible for herpes encephalitis, the neonatal infection with the highest morbidity and the greatest likelihood of permanent injury [99]. A primary maternal infection generally carries a higher risk of neonatal infection than does recurrent disease. The presence of maternal antibody from prior infection is apparently partially protective to the fetus, reducing the risk.

Systemic neonatal infection with HSV can be devastating, leading to seizures, psychomotor

retardation, blindness, or death [100]. Unfortunately, most neonatal infections occur in infants delivered to women who are either asymptomatic or have unrecognized disease. Despite prior beliefs, both primary and recurrent herpetic infection can result in clinical manifestations varying from asymptomatic viral shedding to multiple skin ulcerations with systemic symptoms. The clinical presentation can be deceiving. Severe episodes of herpetic infection first seen in the second and third trimesters of pregnancy, often thought to be primary, usually prove to be recurrent episodes when studied by viral isolation and serologic testing.

The diagnosis of HSV can be established by either viral isolation or by antigen testing from specimens directly obtained from suspect lesions. There are several laboratory tests for confirmation of HSV infection. The most common studies, other than viral isolation by culture, are ELISA-based tests that evaluate type-specific antibodies to HSV types 1 and 2. Newer and more accurate type-specific tests based on glycoprotein are now commercially available and have been proved to have high sensitivity and specificity. As the various new tests for HSV are brought to market, bedside testing will eventually become a reality [98]. There are also various fluorescent antibody methods for the detection of HSV from suspect lesions. The usefulness of these latter tests is limited by a high rate of false-negative results that are especially common in recurrent and healing lesions. In terms of test interpretation, no type-specific antibodies have been identified during an observed primary outbreak. The existence of IgG antibodies documented at the onset of lesions defines a recurrent infection; the older IgM tests for HSV are generally considered unreliable. Women with preexisting IgG antibodies tend to have milder clinical symptoms with fewer systemic symptoms than do those without, although clinical variation is wide.

There is a large pool of asymptomatic HSV carriers. The failure to identify these individuals is largely due to the fact that only approximately 20% of cases of primary infections become symptomatic. Of the remaining 80% of infected people, one third remain asymptomatic, and the remaining two thirds have clinical recurrences that might or might not be recognized as these patients are followed over

time. Most people with genital herpes shed the virus asymptotically, and those with herpetic antibodies but without symptoms shed the virus at the same rate as patients with antibodies and clinical outbreaks [101].

HSV poses several problems for the clinician. As noted, recurrent HSV disease can result in severe symptoms but is often erroneously diagnosed as a primary outbreak. Additionally, a true primary infection can be mild and on clinical impression recorded as an HSV recurrence. There are also issues with accurate diagnosis given the limitations of the tests currently in use.

When an initial HSV infection is diagnosed during pregnancy, the mother should be screened for other STDs. Treatment with an oral antiviral agent should be considered [102,103]. Acyclovir is the drug that has been used most extensively. Acyclovir is well tolerated in late pregnancy, and there are no laboratory or clinical data suggestive of significant maternal or fetal toxicity [103].

Similar to women infected with the AIDS virus, invasive obstetric procedures performed on women with known HIV infection can increase the risk for newborn infection and are best avoided. These risks include elective membrane rupture, application of scalp electrodes, scalp vacuum extraction, and probably, forceps or vacuum deliveries.

The most effective strategy for HSV management is the initial identification of women with either a prior history of herpes or current suspicious lesions. If the mother is infected, caregivers should be alerted to the potential risk, and third-trimester antiviral therapy is appropriate. With the newly developed tests, improved antepartum screening for susceptibility to HSV should soon become possible. If the mother is antibody negative and thus infection susceptible, counseling and evaluation of the male partner, if he is affected, becomes possible. Blocking maternal infection from the male partner is thought to present an opportunity to reduce the incidence of neonatal herpes substantially. A combination of viral suppressive therapy and condom use reduces the transmission risk for herpes between couples. Cesarean delivery is the appropriate management for women with clinically apparent herpetic lesions at the time of parturition. For general management principles, see Table 18.3.

**TABLE 18.3 Principles of Management: Herpes in Pregnancy**


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Oral antiviral therapy is indicated for pregnancies beyond 36 weeks in women at risk for recurrent disease.
Vaginal delivery is acceptable for women with a history of HSV recurrence if there are no active lesions observed at the time of labor and the gravida reports no prodromal symptoms.
Cesarean delivery should be performed if active genital lesions (or prodromal symptoms) are present at the onset of labor.
Condom use and viral suppressive therapy reduces male-to-female transmission of infection.

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### Human Immunodeficiency Virus

Cesarean delivery has an important role in the management of pregnant women infected with the human immunodeficiency virus (HIV) [104,105]. HIV is a worldwide problem, and millions of people are infected [106–108]. A particularly hard-hit area is sub-Saharan Africa, especially the East African countries of Uganda, South Africa, Zambia, and Zimbabwe. In recent years, the treatment of HIV has improved greatly, prolonging the survival of those infected. In addition, proper management has markedly reduced the likelihood of fetal infection. Coinfection with HIV and other sexually transmitted disease (STDs) is common [109].

The HIV virus is an enveloped RNA retrovirus possessing an RNA-dependent DNA polymerase reverse transcriptase. Once infection occurs, the HIV viral nucleocapsid fuses to a cell membrane, enters the cytoplasm, and reverse transcription of RNA to DNA occurs. The viral DNA then integrates into the host cell DNA by means of a viral endonuclease, leading to the development of new virions. In humans, the HIV virus binds to the CD4 receptor, resulting in progressive depletion of the T-cell population of the host, inhibiting critical functions of the immune system. Specifically, delayed type hypersensitivity, processing of foreign substances, and appropriate responses to viral infection and to abnormal or precancerous cells are disrupted.

The HIV virus has been isolated from blood, semen, vaginal and cerebrospinal fluids, breast milk, amniotic fluid, and other sources. Approximately two thirds of HIV infections are sexually transmitted. Of the remaining cases, most are associated

with intravenous drug use; a small number result from blood transfusion. The least common source of infection is attributed to miscellaneous causes such as contamination from surgery or needle accidents among medical personnel. Death from HIV is primarily due to AIDS, a syndrome that develops from advanced immunodeficiency and that is characterized by opportunistic infection, or less frequently, to cancer. People with HIV are specifically prone to develop cervical cancer, lymphomas, and Kaposi's sarcoma. AIDS develops when CD4 or T4-cell concentrations fall, and infection by bacteria, viruses, fungi, parasites, and other organisms is established. Infection primarily contributes to maternal mortality by increasing a woman's susceptibility to serious infection or parasite disease such as tuberculosis, malaria, and *Pneumocystis carinii* [110].

In recent years, the incidence of HIV/AIDS-related deaths has rapidly declined, reflecting the development and widespread use of potential antiretroviral drugs and aggressive treatment of opportunistic infections. New therapies have now resulted in a large population of chronically infected subjects who are sustained by active treatment. Treatment for individual women and avoidance of perinatal viral transmission depends on three tasks: identification of those infected, appropriate antenatal treatment, and careful delivery planning.

Current antiretroviral therapy suppresses viral titers and restores immune competence. The introduction of highly active retroviral therapy (HAART) in recent years has reduced the incidence of opportunistic infections and prolonged life. HAART treatment blocks viral replication and delays the advance of the infection. As the science of HIV advances and various new treatment protocols are tested, the recommendations for HIV prophylaxis change. Clinicians therefore must continuously update their information either from locally available HIV/AIDS experts or from one of the available websites. Unfortunately, not all HIV-infected people are able to tolerate HAART, and others have tried HAART regimes and failed. HAART and other available antiviral drugs are expensive, and many have unpleasant side effects. Active research is underway to develop new, less toxic antiviral drugs and a preventive vaccine. In individual cases, successful therapy is primarily gauged by following the trend of CD4 and T-lymphocyte counts. Low counts are associated with a high risk for superimposed

infection. Primary or secondary prophylaxis against these opportunistic infections can usually be stopped once the CD4 and T-lymphocyte counts exceed an established threshold (usually  $>200$  cells/ $\mu$ l).

HIV is transmitted from mother to child at the time of parturition by exposure to maternal blood and other body fluids. This fact has led to changes in general obstetric management for HIV women, most important, antenatal viral suppression and cesarean delivery [111]. HIV is also transmitted by breastfeeding. In developed countries in untreated cases, perinatal maternal to infant HIV transmission occurs at a rate of 14% to 25% [112]. The risk of transmission from mother to infant is markedly reduced by antenatal treatment of the mother with azidothymidine (AZT) or other antiviral agents when combined by treatment of the infant during its first six months of life. With universal prenatal HIV counseling and testing, antiretroviral prophylaxis, selected elective cesarean delivery, and avoidance of breastfeeding, the maternal-to-fetal transmission rate has fallen to  $\leq 2\%$ . Controversy remains whether in the case of a low maternal viral titer performance of a cesarean still is of any additional benefit in avoiding fetal infection [112–115].

Based on conflicting data and extrapolations from the presumed mechanism of perinatal transmission, several standard obstetric maneuvers are *not* recommended in the management of women known to be HIV infected. These maneuvers include invasive fetal monitoring, elective membrane rupture, amniocentesis, and forceps or vacuum-assisted delivery. These manipulations increase the potential for fetal exposure to the mother's blood and thus for infection (Table 18.4).

**TABLE 18.4 Principles of Management: HIV in Pregnancy**

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Antepartum and intrapartum drug therapy reduces the risk of maternal-child transmission and is without significant fetal risk.

Cesarean delivery reduces maternal-child viral transmission but the principal benefit occurs in cases involving elevated maternal viral titers.

Breastfeeding is to be avoided.

Obstetric maneuvers potentially admixing fetal and maternal blood are to be avoided.

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When the fetal membranes rupture, time until delivery is a factor in viral transmission. This effect is more marked in advanced maternal infections when the viral titer is high. In studies of membrane rupture among women diagnosed with AIDS, the estimated risk of vertical HIV transmission increased from 8% to 31% as the period of membrane rupture advanced from 2 to 24 hours [116].

Antiretroviral therapy to pregnant women does not generally carry a substantial risk for adverse pregnancy outcomes [115]. Recent studies monitoring prenatal exposure to antiretroviral drugs have demonstrated no increases in birth defects among infants exposed to standard drugs, including lamivudine, nelfinavir, nevirapine, stravudine, and zidovudine [117–119].

Elective cesarean delivery does reduce HIV transmission rates and has proven efficacy without significant increases in maternal morbidity [113,118,119]. Not surprisingly, elective operations carry less morbidity than emergency procedures, emphasizing the importance of case identification, counseling, and appropriate scheduling [120]. Maternal viral levels, CD4 and T-cell counts, mode of delivery, and gestational age are independent factors associated with HIV transmission. *The benefit of a cesarean is directly related to the maternal viral load at the time of delivery.* When pregnant women are aggressively treated antepartum and the viral load is either very low or undetectable, the risk of transplacental transmission falls to less than 2% [121–123]. In light of these very low viral titers and the reduced risk of maternal-to-fetal viral transmission, it is unlikely that a cesarean confers any additional benefit to the infant in this situation. Cesarean delivery is recommended for women known to be infected with HIV when the most recent viral load is either unknown or is  $\geq 1,000$  copies/ml [122,124]. Current data do not clearly demonstrate a benefit to elective cesarean delivery if the mother was treated antepartum with current regimens and the maternal HIV RNA levels are below 1,000 copies/ml [122].

Any proposed benefit to an operative delivery must be judged against the morbidity of a cesarean. Obviously, careful patient counseling is required. If vaginal delivery is chosen, the duration of ruptured membranes should be minimized if possible, and as noted, invasive obstetric procedures (e.g., instrumental delivery, scalp electrode, and so forth) avoided [122]. Intravenous ZDV should be



administered during labor, and the infant subsequently treated with ZDV for the first six weeks of life. Elective cesareans are scheduled for 38 weeks, and amniocentesis should be avoided. This timing for surgery reflects a balancing of risks and benefits. Delivery at the 38th week has a small associated risk for neonatal respiratory difficulty or transient tachypnea, necessitating either mechanical ventilation or other therapy. This risk should be weighed against the likelihood of the spontaneous onset of active labor or membrane rupture before the pregnancy reaches the 39th week, which is the gestational age now recommended by ACOG as the best time to schedule repeat cesarean deliveries for normal women if pulmonary maturity is not directly confirmed.

In terms of clinical management, ZDV should be administered intravenously beginning at least three hours before the cesarean and continued until cord clamping [122,125]. Perioperative antimicrobial prophylaxis, such as a first-generation cephalosporin, is also recommended, although no controlled studies of efficacy for HIV patients have been performed. Postsurgery, other antiviral medications are resumed per the usual schedule.

If women who were scheduled for cesarean delivery appear with ruptured membranes, the best management has not been established [122]. In this setting, it is unclear if a cesarean confers any benefit beyond that already provided by the prenatal maternal treatment.

A remaining international problem in HIV management is the development of safe, affordable, effective, and acceptable alternatives to the costly retroviral regimens used in modern industrialized nations. The effort is also underway to develop an effective HIV immunization. Even with current treatment methods, however, HIV testing and treatment of infected women and cesarean delivery are cost efficient [126,127].

### Hepatitis C

A hepatitis C virus (HCV) infection is a possible but controversial indication for cesarean delivery. As yet there is no clearly demonstrated benefit to cesarean delivery in avoiding viral transmission to the fetus [128–131].

HCV is an RNA virus of the Flaviviridae family. There are six genotypes and more than 50 subtypes.

Serotype I accounts for 70% to 75% of infections and is associated with a lower response rate to therapy. The virus is notable for a high rate of spontaneous mutation and its failure to provoke a vigorous T-lymphocyte response.

HCV infection is the most common chronic blood-borne infection and the leading cause of chronic liver disease in the United States. Nearly 3 million Americans are thought to be infected, with more than 2.5 million having chronic infection. Death certificate data suggest that 10,000 or more deaths occur annually from hepatitis C or its complications. HCV transmission occurs secondary to organ transplantation from infected donors, occupational exposure to infected blood, blood transfusions received before 1992, sexual intercourse with infected persons, intravenous drug use, or birth to an infected mother. In declining order of incidence, the highest levels of seropositivity for HCV are found among hemophiliacs infused with clotting factors prior to 1992, injection drug users, the incarcerated, and the homeless. HCV cases not related to injection drug use are attributed principally to sexual contacts or occupational exposure to infected blood or blood products. Accidental needle stick injuries are often a concern to healthcare personnel. The transmission risk is low, however, estimated at 2% or less. Rarely, body piercing and tattooing with contaminated equipment are additional causes of infection.

Many current HCV-infected people have occult disease and remain to be identified. Recognition of at-risk behaviors and blood testing have clearly reduced the incidence of new infection. Nonetheless, owing to the large number of unrecognized or asymptomatic cases that remain in the population, it is estimated that there will be a fourfold increase in diagnosed cases by 2015, principally by the diagnosis of those already infected. At present, most HCV-positive diagnoses are made in people 40 to 59 years of age, with the prevalence highest in African Americans.

The estimated seroprevalence of HCV is 2% to 3% among partners of HCV people in monogamous relationships, but rises to 4% to 6% among those with a history of multiple sex partners. For monogamous heterosexual couples, the likelihood for cross-infection of HCV is estimated to be very low, between 0% and 0.6% [132]. Interpreting these data on sexual transmissibility is complicated by concomitant intravenous drug use or HIV infection,

which tend to be common comorbidities among those studied.

Infection is characterized by a high rate of chronicity. Those chronically infected are at substantial risk for cirrhosis of the liver. The cirrhosis risk is increased among the immunosuppressed (e.g., HIV infection) and is associated with male sex, more advanced age at the time of infection, and chronic alcohol use. Some 55% to 85% of people developing acute hepatitis C will remain chronically HCV infected. A further 5% to 20% will develop cirrhosis over periods of up to 25 years.

The rate of spontaneous cure after an acute HCV infection is estimated at 20% to 25%. The likelihood of spontaneous viral clearance is associated with female sex, age of less than 40 years, and icteric illness [133]. Infants with HCV infection exhibit spontaneous viral clearance at a rate of 75% to 100%. Those with acute hepatitis C who recover with disappearance of the virus on serologic testing do not develop long-term complications and do not require additional treatment [134].

At present, new HCV infection in children is primarily due to perinatal or vertical transmission [135]. It is estimated that 240,000 American children carry hepatitis C antibodies. Most of these cases arise from blood transfusions received before 1992 or from birth to HCV-positive mothers. Postpartum transmission is believed to be rare. Children are more likely to have spontaneous improvement, display a slower rate of disease progression, and have normal or near-normal aminotransferase levels despite chronic infection. Unfortunately, little is known concerning the lifetime risk for prenatally infected children.

Best obstetric management has not been established [128–129]. There are no data indicating that maternal antiviral treatment reduces prenatal transmission. Furthermore, both of the common treatment drugs, interferon and ribavirin, are contraindicated during pregnancy. There are also no prospective data documenting that the rate of transmission is reduced by cesarean delivery [128,129,130,131]. It has been claimed, however, that cesarean deliveries *prior* to the onset of labor might reduce the risk of HCV transmission. Additional study of this point is needed.

Several steps to reduce fetal exposure to maternal blood, such as the avoidance of scalp sampling and prolonged labor after membrane rupture, are sug-

**TABLE 18.5 Principles of Management: Hepatitis C**

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Hepatitis C-positive women should be investigated for other STDs, including HIV.
Hepatitis C antiviral treatment is contraindicated during pregnancy.
Obstetric procedures potentially resulting in the admixture of maternal and fetal blood are to be avoided.
Breast feeding is acceptable.
There is no demonstrated benefit to cesarean delivery beyond the usual obstetric indications.

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gested as reasonable precautions until more about viral transmission is known. Perhaps what should be added is the avoidance of elective membrane rupture, amniocentesis, and instrumental delivery. Such practice restrictions are based on theoretic considerations, as opposed to established risk factors based on prospective study (Table 18.5).

The overall risk for perinatal HCV transmission is approximately 2% when the mother is anti-HCV seropositive. In the 1998 multicentered study of 403 HIV-negative mothers infected with hepatitis C, it was observed that all cases of vertical transmission occurred when the mother was HCV-RNA positive [130]. Thus, it appears that the risk of vertical transmission is low when maternal HCV-RNA is not detected. Surprisingly, the measured HCV-RNA titer proved unrelated to the risk of transmission. If the parturient is HCV-RNA positive at the time of delivery, the transmission risk is 4% to 7%. If mother is infected with *both* the HCV and HIV viruses, the transmission rate for HCV increases to as much as 20%.

In contrast to HIV, breastfeeding does not transmit HCV and need not be avoided. Furthermore, horizontal transmission of the virus from child to child is rare.

Current therapy for HCV infection is based primarily on the administration of pegylated interferon combined with ribavirin. *Pegylated interferons* are compounds produced by bundling polyethylene glycol to the interferon molecule, which reduces renal clearance and increases the half-life of the drug. The effectiveness of therapy is usually determined by qualitative HCV-RNA assays. Treatment is usually recommended to those with HCV-RNA levels greater than 50 IU/ml and a liver biopsy showing fibrosis as well as some degree of inflammation/necrosis. Most of these people also

have persistently elevated ALT liver enzyme levels. Therapeutic problems occur in determining the best treatment for patients with cirrhosis or advanced liver fibrosis and those failing to respond to optimal therapy with interferon/ribavirin.

### Multiple Gestation

A multifetal pregnancy is a potential but not absolute indication for cesarean delivery [136]. The rate of successful vaginal delivery for twin pregnancies is high, assuming the leading twin is in a cephalic presentation. If the leading twin is in a breech or transverse lie, cesarean delivery is best. When the leading infant is cephalic and delivers vaginally, delivery of the second infant depends upon presentation. A cephalic second twin is rarely an obstetric challenge unless a compound presentation occurs or the cord prolapses. When the second twin is noncephalic, cesarean delivery for the second infant is increasingly frequently performed especially if there is a major discrepancy in fetal weights. Such cesareans become more likely as the interdelivery interval increases [137]. If lie is not longitudinal, the second twin can be delivered vaginally either by external version or by internal version with breech extraction. These procedures are best performed under real-time ultrasound guidance and after the administration of nitroglycerine or some other potent tocolytic [138]. In instances of higher multiples, data concerning the outcome of vaginal trials compared with cesarean delivery are not available, and cesarean delivery is common. Prematurity, significant size discrepancy among infants, and malpresentation are common in these higher multiple gestations and when present usually render a vaginal trial imprudent. (See Chapter 13, Multiple Gestation.)

### Fetal Macrosomia

Controversy exists regarding both the definition and management of large, or macrosomic, fetuses [140,141]. The weight limit used to define macrosomia varies in the literature, with the most common modifier being maternal diabetes. If the mother is an insulin-requiring diabetic, a birthweight of >4,000 g is often considered evidence of macrosomia. In the absence of diabetes, the term *macrosomia* is applied to infants weighing >4,500 g.

There are both maternal and fetal problems in the deliveries of large infants. First- and second-stage

dystocia is the principal risk. The incidence of shoulder dystocia is also increases from approximately 3% for infants with a birth weight of 4,100 g to 4,500 g, to 8.2% for those over 4500 g. If poor second-stage progress accompanies a midpelvic instrumental delivery of a large infant, the incidence of shoulder dystocia is increased several-fold. The peculiar phenotype of the infant of a diabetic mother predisposes to shoulder and body dystocia. In the fetus of a diabetic mother, fat is disproportionately distributed to the abdomen and back. As the infant increases in size this increases the difference between the cranial and abdominal circumferences. This is a factor in both shoulder and body dystocia at delivery (Figure 18.1). Although shoulder dystocia is the most feared complication associated with larger infants, other fetal injuries and maternal injuries are also possible. The cesarean delivery rate, especially if labor has been induced, is higher for the macrosomic infant as opposed to those of lesser size [139]. Nonetheless, obstetric intrapartum management can result in excellent results, since most large infants are delivered vaginally without difficulty [140]. (See Chapter 14, Shoulder Dystocia.)

The decision to perform a cesarean delivery for a large infant often is based on an ultrasound estimate of fetal weight. Unfortunately, such ultrasound studies have distinct limitations [141b]. The mean absolute error of an ultrasound fetal weight estimate in the third trimester is  $\pm 6\%$  to  $12\%$  of actual birth weight, with 40% to 75% of estimates falling within  $\pm 10\%$  of the actual birthweight. Owing to these inherent limitations in the method, ultrasonically derived estimates of fetal weight should not be used as the sole basis for reaching obstetric management decisions. Except in extreme cases, ultrasound weight estimates should be used only in conjunction with other clinical data (i.e., pelvic architecture and capacity, position, station, estimated gestational age, Leopold's maneuvers, and course in pregnancy) in making clinical decisions. Eventually, it will most likely be found that various methods for estimation of *relative* fetal dimensions such as ratios between head/abdomen or other body part, perhaps in conjunction with one or more measures of "fatness" such thigh or cheek thickness, will be better predictors of risk for injury than simple weight estimates.

Given the limited accuracy of estimates of fetal weight by ultrasonography, depending on scanning

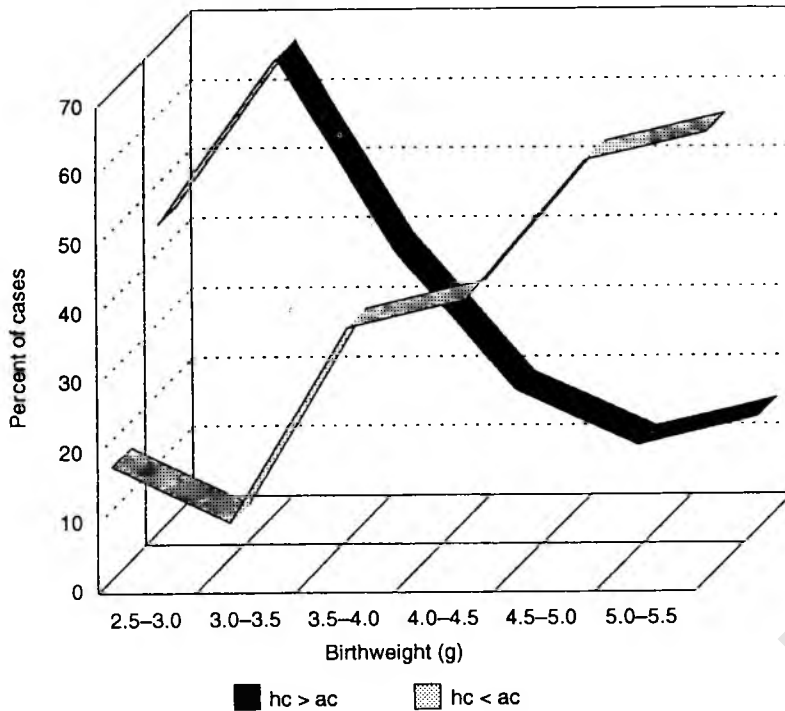


FIGURE 18.1.  
Head circumference (hc) to abdominal circumference (ac) plotted against birth weight ( $n = 137$ ). Plot graphed from data derived from Seigworth GR: Shoulder dystocia: Review of 5 years' experience. *Obstet Gynecol*, 1966. Dec;28(6):764-7; with permission.

to determine whether an individual case should go to primary cesarean delivery is problematic. Recognition of the possible at-risk case, consideration of options, and patient counseling are the clinician's responsibilities when vaginal delivery of the suspected macrosomic fetus is contemplated. Given the uncertainties, patient counseling should precede either an elective cesarean or a trial of vaginal delivery when a large infant is suspected. A written or dictated note should be placed in the medical record detailing the reasons why either a vaginal trial or a cesarean delivery was chosen in that individual case, when the decision is predicated on the risk of presumed or suspected macrosomia.

### Thrombocytopenia

Platelets are small, non-nucleated cells arising from marrow megakaryocytes that circulate in peripheral blood. Platelets have an important role in primary hemostasis. They adhere to sites of endothelial injury and, after clumping and activation, platelets act to arrest bleeding and stimulate the coagulation cascade to convert fibrinogen to fibrin, which then stabilizes the initial platelet plug.

The normal range for maternal platelet counts is 150,000  $\mu/l$  to 400,000  $\mu/l$  [142]. Counts less

than 150,000 but greater than 100,000 define mild thrombocytopenia (TTP). Counts from 50,000 to 100,000 indicate moderate TTP, whereas counts of less than 50,000 indicate severe TTP. Spontaneous bleeding is quite uncommon and even rare unless the platelet count falls below 10,000. Coagulation in surgery is usually normal until the platelet count drops well below 50,000.

When the platelet count is low, there are several potential causes, namely, increased platelet utilization or destruction, decreased platelet production, or platelet sequestration. During pregnancy, increased destruction and consumption are the principal causes for low platelet counts. Microangiopathies, including hemolytic uremic syndrome, thrombotic thrombocytopenic purpura (TTP), disseminated intravascular coagulation (DIC), and the syndrome of hemolytic anemia, elevated liver enzymes, and low platelet count (HELLP), are additional and potentially serious but uncommon causes of low platelet counts.

After anemia, TTP is the most common hematologic problem encountered during pregnancy. The incidence is approximately 7% to 10% [143-146]. Pregnancy-related TTP is principally due to increased platelet destruction. When the mother's platelet count is low, concomitant fetal TTP is found

in about 1.3% of cases; this finding is compared with 0.4% when the maternal platelet count is normal [144].

*Gestational thrombocytopenia* (GTTP) is a mild form of TTP with platelet counts usually less than 70,000  $\mu\text{L}$ . GTTP can be associated with one of the pregnancy-associated hypertension syndromes (e.g., HELLP, eclampsia/preeclampsia) or be spontaneous. When women with such obstetric or medical conditions are excluded, the incidence of GTTP is approximately 5% of all pregnancies. This disorder accounts for 70% or more cases of TTP during pregnancy and is thought to be due to spontaneous increased platelet consumption. Normally, the mother is asymptomatic, and there is no history of either bleeding or thrombocytopenia predating the pregnancy. Preconception platelet counts are normal. Platelet counts return to normal postpartum, with most recovering within several weeks of delivery. GTTP is a diagnosis of exclusion. Unfortunately, platelet antibody tests are often not reliable in distinguishing this disorder from the more dangerous immune TTP [147]. Although GTTP can recur in subsequent pregnancies, it poses no threat to either mother or infant. This condition in of itself is not an indication for cesarean delivery [148,149], and no specific management is required beyond periodic monitoring of maternal platelet counts [143].

Among the potentially serious forms of TTP in pregnancy are those with an immune cause, such as immune (idiopathic) thrombocytopenia purpura (ITTP) or autoimmune thrombocytopenia purpura (ATTP). *Immune thrombocytopenia* is a much different disorder than GTTP. This condition can result in fetal thrombocytopenia because of the transplacental passage of antiplatelet antibodies. In ITTP, IgG antibodies are developed against a woman's own platelet membrane antigens. This leads to platelet destruction in the reticuloendothelial system. When platelet loss exceeds replacement, thrombocytopenia develops with a decline in the platelet count. In this condition, there is some risk for spontaneous maternal hemorrhage if the platelet count drops to less than 20,000. Maternal IgG antibodies cross the placental barrier and can result in fetal TTP. Approximately 12% to 15% of infants of affected mothers have platelet counts at birth of less than 50,000; 3% experience bleeding problems [149], and fewer than 1% of infants develop from immune

TTP mothers experience intraventricular hemorrhage [143]. Fortunately, severe fetal or neonatal TTP is quite uncommon, and the principal method for determining the fetal platelet count, cordocentesis, carries a high risk of fetal loss and is no longer recommended as a routine test for fetal evaluation. As fetal risks are low and scalp sampling in labor unreliable, the method of delivery is best based on the usual obstetric indications with the avoidance of instrumental delivery.

*Alloimmune thrombocytopenia* (ATTP) is a potentially serious disorder that develops because of platelet antigen incompatibility between fetus and mother. In this condition, the mother remains asymptomatic but produces antiplatelet antibodies against fetal platelet markers. These antibodies subsequently cross the placenta and result in fetal thrombocytopenia [150,151]. Intracranial bleeding can occur in to 10% to 20% of affected infants, with 25% to 50% of these cases occurring prenatally. The incidence of ATTP is estimated as 1 per 800 to 1,000 live births [150].

Pregnancy-related alloimmune disease is usually not diagnosed until after the birth of an affected child. Unfortunately, in one half of cases, this disorder affects the first pregnancy, thus precluding any opportunity for therapy. While treatment is possible, recurrences are not preventable, and the risk of a recurrence is high. Intracranial hemorrhage occurring in utero has been identified by antenatal ultrasound in a few cases. Prenatal hemorrhages can result in either hydrocephalus or a porencephalic cyst owing to destruction of brain tissue. In subsequent pregnancies when prenatal treatment is possible, the clinical outcome for the fetus from alloimmune TTP is usually but not invariably good.

When treatment was possible because of diagnosis in a prior gestation, in the past therapies included maternal treatment with corticosteroids or parenteral immunoglobulins. On occasion, fetal platelet counts were attempted by cordocentesis, and PLAI-negative irradiated platelets were transfused as needed [150,152,153]. In a recent report of a ten-year experience based on pooled data, however, Overton and colleagues [154] estimated the per pregnancy loss rate from cordocentesis and transfusion as approximately 6%. Because of this high risk of fetal loss, new, less invasive management protocols have been suggested, principally consisting of corticosteroids and maternal immunoglobulin

treatment alone, avoiding the invasive procedures with high fetal loss rates [155,156].

Current recommendations are that cesarean deliveries for women with thrombocytopenia remain reserved for obstetric indications. Efforts at obtaining fetal platelet counts by scalp sampling or cordocentesis are no longer considered appropriate to determine which cases might be delivered vaginally. These tests should not be routinely performed owing to the associated risk, unreliability of technique, and unproved efficacy of the associated interventions.

### **REPEAT CESAREAN DELIVERY AND VBAC ISSUES**

The performance of trials of vaginal birth after cesarean (VBAC) versus an elective repeat cesarean for subsequent pregnancies in women who have experienced a previous cesarean remains controversial [157,158,159]. As recently as the mid-1980s, over 90% of women with a prior cesarean delivery routinely underwent repeat cesarean operations. In the following decade, numerous published reports supported the safety and relative success of trials of vaginal birth after cesarean delivery [157,158]. As an example, in a 1991 meta-analysis of 31 studies on the morbidity and mortality of vaginal birth after cesarean, Rosen and coworkers found decreased maternal febrile morbidity after a successful trial of labor, no difference in the rates of uterine dehiscence or rupture, and no difference in maternal and perinatal death rates between VBAC patients and those undergoing elective repeat operative delivery [157]. Depending on the indication for the prior cesarean, the anticipated success rates for VBAC are reported as approximately 60% and 85%. Based on such data, in 1989 ACOG urged physicians to counsel and encourage women to undergo VBAC as a safe alternative to the repeat cesarean operation.

Despite the numerous and generally encouraging reports about VBAC, new concerns have emerged that have effectively reversed the trend toward VBAC deliveries [5]. These important issues include 1) the risk of uterine scar rupture, 2) uncertainty of best management in the face of an unknown uterine scar, 3) the role for uterine exploration if a vaginal trial is performed, 4) the risk of vaginal birth after more than one cesarean, 5) the use of epidural anesthesia, 6) oxytocin in VBAC trials,

7) misoprostol labor induction, 8) VBAC trials in multiple gestations, and 9) VBAC trials in a cases of suspected fetal macrosomia. Because of physician uncertainty, an unfavorable medicolegal climate, and practice restrictions imposed by recent ACOG pronouncements and by institutions, the VBAC rate has rapidly declined in recent years. A major factor has been the recent ACOG opinion that VBAC trials are appropriate only when the attending physician is *immediately* available [159]. As usually interpreted, this requirement for immediate availability demands the clinician to be physically present throughout the labor. After much uncertainty concerning how strictly to interpret this opinion, many institutions have decided to forgo VBAC trials, especially in smaller institutions when immediate 24-hour in-house staff and anesthesia coverage are not available [160].

### **Uterine Scar Separation: Dehiscence Versus Rupture**

When evaluating the maternal/fetal risks of a VBAC trial, clinicians must differentiate scar separation or dehiscence from frank uterine rupture [161]. A *scar separation* or *dehiscence* refers to an opening of the previous myometrial scar; however, the overlying visceral peritoneum is intact, and hemorrhage or expulsion of the uterine contents does not occur. Such defects in the uterine wall often remain entirely asymptomatic and are detected only as incidental findings during a laparotomy [162,163]. Because uterine exploration is not performed universally after successful VBAC trials, the true incidence of occult uterine scar separations (or "windows") is unknown. This fact accounts for the lower incidence of dehiscences reported after VBACs versus after elective repeat cesarean delivery, where it is possible to observe the lower uterine segment directly.

*Scar rupture* refers to the opening, usually acute, of an established uterine scar and overlying visceral peritoneum, often with the expulsion of the fetus or placenta into the peritoneal cavity. Whereas a dehiscence can be silent, true uterine ruptures rarely are. Most acute ruptures are associated with either retroperitoneal or intraperitoneal hemorrhage. Depending on the severity, acute maternal hemodynamic changes, complaints of pain, loss of station, fetal distress, or even fetal loss can occur. More than one half of uterine ruptures occur in prior

cesarean scars. An occasional rupture occurs spontaneously during labor in previously unscarred uteri. Cases of rupture during labor in unscarred uteri are virtually restricted to multiparas, since spontaneous uterine rupture before or during labor is at best unusual among nulliparas [164].

The available literature concerning trials of labor, VBAC, and its complications is voluminous but difficult to interpret. Differing definitions of uterine rupture and wound dehiscence are often used. Some studies rely on medical record reviews, others on ICD coding on discharge summaries. Whether a specific delivery was originally intended as a VBAC trial is also uncertain. Finally, owing to the variations in population and medical recording practices, very large numbers of cases are needed to permit accurate estimates of risk, because the incidence of some of these events is one in thousands. This inevitably means that either meta-analysis of selected studies or data drawn from very large populations is required. Finally, as the data about prostaglandin induction indicate, even large well-organized studies can reach varying conclusions. It is notable that the recent study by Chauhan collectively reviewed 929 published articles and eventually chose only 93 for inclusion in their analysis [165]. The others were rejected because of various methodologic, content, or quality issues.

The VBAC success rate is usually quoted as approximately 75% [167]. The associated risk for uterine rupture is variously reported in the literature as 1 to 23 in 1,000 [161,168–173]. Recent reviews by Guise [166,174] reported a rate of 2.7 ruptures per 1,000 labor trials, which compares well with the 2004 paper by Smith and coworkers [161], indicating an overall risk of 3.5 per 1,000. An important factor favoring success in a VBAC trial is a history of a prior vaginal delivery or a prior VBAC [163,175]. Vaginal delivery is more likely and uterine rupture less likely if the woman has experienced a prior vaginal birth [161,176] (Table 18.6).

The loss of the fetus secondary to a uterine rupture in a VBAC trial occurs in approximately 1 in 2,400 cases [161]. The risk of perinatal loss from uterine rupture is apparently higher in women induced with prostaglandins [161]. Whether this reflects an issue unique to the pharmacologic effects of the prostaglandins or to their preferential use in patients less favorable for labor induction (and thus presumably at risk for a longer or more dif-

**TABLE 18.6 Clinical Features Favorable to a Successful VBAC Trial**

Spontaneous onset of labor; advanced dilation at time of presentation
Nonrecurrent indication
Fetus $\leq 4,000$ g
More than 6 months since last delivery
Prior successful vaginal delivery

*Data from [159,161,176,190].*

ficult labor) is unclear. In the Smith data, the risk for uterine rupture for women without a history of a prior vaginal birth and who were induced with prostaglandin was 1 in 71. For women without prior vaginal birth in whom prostaglandins were not used, the incidence fell to 1 in 210. Conversely, if there was a history of prior vaginal birth the rupture rates were considerably lower, 1 in 175 for prostaglandin induction, and 1 in 514 for cases without prostaglandin administration. Another most interesting observation in this study was that the perinatal mortality rate was fully threefold greater if the uterine rupture occurred in an institution with  $\leq 3000$  births a year compared with larger services.

It is fair to say that the literature is inconsistent about the relationship between labor induction and uterine rupture [166,177–181]. In the comprehensive literature review conducted by Guise, when prospective cohort studies were reviewed, neither prostaglandin nor oxytocin administration increased the rate of uterine rupture [166]. Case-controlled studies cited in the same review yielded an estimate of a two- to fourfold increase. An increased risk was also reported independently by Lydon-Rochelle [182] and Smith [161].

Because of residual uncertainties, prostaglandin use in women induced for VBAC trials is *not* recommended. By extension, the issue of oxytocin administration for labor induction or augmentation in VBAC trials is also controversial [157,176]. As a practical matter, the authors hesitate to administer oxytocin for arrested labor in cases complicated by tardy progress, unless the most meticulous of clinical evaluations notes no evidence of disproportion, and the problem is thought to be uncoordinated uterine activity. When an oxytocic agent is administered for induction, the authors favor insertion of an intrauterine pressure catheter (IUPC) and careful

titration of the oxytocic effect. Failure to resume normal progress promptly under adequate oxytocin stimulation is an indication for immediate cesarean delivery. In contrast, the authors have administered oxytocin for labor *induction*, then reducing the dose of the uterotonic once an active contraction pattern develops and labor is established.

In terms of clinical risk, good data from composite literature reviews indicate that the chance of a low transverse scar or a scar of unclassified origin rupturing during an attempt at vaginal delivery is less than 1% [161,165,166,170,183]. In most cases of uterine scar rupture, repair of the uterus is feasible, and fertility is preserved. If there is extensive damage to the uterus or if a myometrial tear extends into the broad ligament, hysterectomy might be required. Although fertility can be lost from a catastrophic uterine rupture, associated maternal mortality is rare. Most ruptures of low transverse uterine incisions occur during labor. Fortunately, this most commonly occurs when the mother is under observation, but a classic scar or a scar invaded by a placenta percreta can also rupture without active labor and remote from term. Infrequently, ruptures occur in association with abdominal trauma, Müllerian anomalies, quite rarely, or severe abruptio placentae. Prior surgery invading the myometrium, such as for the removal of leiomyomas or a uterine unification operation, are also important risk factors for uterine rupture. Because of these risks and the fact that on occasion labor will begin early, even a plan for an elective repeat cesarean does not invariably prevent a uterine rupture in high risk cases.

The clinical consequences of rupture depend on the extent of the defect and the resultant hemorrhage. Fetal risk relates largely to the extent of placental separation and the accompanying maternal shock/vascular collapse. The classic clinical signs of uterine rupture include: otherwise unexplained abnormal fetal heart rate patterns, usually of sudden onset; the apparent cessation of labor; loss of station; vaginal bleeding; and maternal cardiovascular collapse. When a uterine rupture occurs, the perinatal mortality is about 5%. Furthermore, in approximately 15% of ruptures, hysterectomy is necessary.

These data need to be placed in perspective. Based on his review, Guise estimates that it requires 370 elective repeat surgeries to prevent a single uterine rupture associated with a labor trial [166,174].

As the risk of serious complications is low, to prevent a hysterectomy, 2,941 repeat procedures are required. To avoid perinatal death requires 7,142 procedures. Maternal mortality, although possible, is a remote risk, estimated at 2 per 100,000 labor trials. Of interest, Guise reported no maternal deaths in his review [166].

These data do not make the task of the clinician easier. The problem of VBAC becomes one of the willingness of pregnant women, physicians, and institutions to accept a small but finite risk of a potentially serious complication from both a repeat cesarean (maternal) and a labor trial (fetal) (Table 18.7) [184,185]. Morbidity in maternal failed trials is also an issue [170, 299]. Based on many clinical observations, the longer the period of membrane rupture and the longer the labor, the greater the risk for maternal febrile/infectious morbidity in a failed trial when a cesarean eventually becomes necessary. As is true for much of medical practice, there are few clearly correct answers in the VBAC controversy. What faces clinicians and gravid women are alternatives, each with its inherent risks and possible benefits [5,184,186].

Data pertaining to the risk of repeat rupture of the uterus following a rupture and repair in a previous pregnancy are limited. The best estimate for the risk for repeat rupture or dehiscence of a lower uterine scar is 6.4%; however, for a repeat scar rupture involving the upper segment of the uterus (classic scar), the risk is a very high 32%.

In view of the known complications, a woman with a history of rupture of a classic upper uterine scar should be advised that pregnancy involves a substantial risk. If already pregnant, she should be delivered prior to the onset of labor as soon as fetal pulmonic maturity is ensured. Because of lack of data regarding the fate of pregnancies after rupture of a lower segment transverse uterine scar, subsequent deliveries for patients with this history can be either by repeat cesarean delivery, or in selected cases, by vaginal delivery with close maternal-fetal monitoring. Thoughtful patient counseling and full medical record documentation are needed in such cases.

### Unknown Uterine Scar

The risk of uterine rupture is apparently the same for women undergoing a trial of labor with a uterine



TABLE 18.7 Potential Maternal Complications: Cesarean versus Vaginal Delivery

Maternal Outcomes Vaginal Delivery	Cesarean Delivery
Mortality: $\leq 1$ in 8,000	Mortality: $\leq 1$ in 2,000
Morbidity	Morbidity
Urinary incontinence*	Endometritis/febrile morbidity
Rectal incontinence	Longer recovery; wound infection; wound dehiscence
Hemorrhage: uterine atony, inversion, rupture	Operative injury; ureteral, bladder, GI injury; hemorrhage
Deep venous thrombosis	Pelvic infection/abscess/hematoma
Subjectively decreased pelvic tone	Deep venous thrombosis/pelvic vein thrombosis
Risk of emergency cesarean delivery in labor	Delayed breastfeeding/holding neonate
Rectal or perineal injury/laceration	Urinary tract infection
Birth canal laceration	Ileus
Secundines	Formation of adhesions
Endo/parametritis	Rehospitalization
Dyspareunia	Long-term complications:
	• Placenta previa
	• Placenta accreta/increta/percreta
	• Abruptio placentae
	• Endometritis/adenomyosis
	• Scar rupture
	• Infertility

\*These data remain controversial.

scar as that for women with a documented scar type [166]. This is due to the fact that most modern transverse abdominal scars are associated with original lower uterine segment transverse incisions. In contrast, if the stated indication for the previous cesarean delivery was one that was likely to require a classic incision – a transverse lie or breech presentation in a premature fetus – a repeat cesarean operation is prudent if the medical record is unavailable or proves incomplete, especially in cases in which the abdominal scar is vertical.

### Uterine Exploration

In the past, postpartum transvaginal manual exploration of the uterine cavity was often performed to palpate for a uterine dehiscence after a successful VBAC delivery. In practice, this recommendation was never universally followed and rapidly proved impractical. Often, the scar site could not be palpated or, if a defect were suspected, it was rapidly recognized that repair of an asymptomatic uterine dehiscence was unnecessary. For these reasons, manual exploration of the site of an original lower seg-

ment scar that can be reached by the examiner's finger on vaginal examination does not provide useful clinical information and should not be routinely performed. In the unusual case that excessive vaginal bleeding occurs or blood or vernix in the maternal urine is observed, or if the parturient becomes suddenly hypotensive or complains of persistent or an unusual pain, prompt examination is indicated to exclude a rupture. If a defect is found in a symptomatic woman, abdominal exploration is mandatory. At surgery, either repairs of the uterus or a hysterectomy can be performed, as the clinical situation requires.

### Vaginal Birth After More Than One Cesarean

Reports on vaginal birth after two prior cesarean deliveries indicate that the risk of uterine rupture is not substantially different from that after one prior cesarean [186]. The success rate for a vaginal delivery is 60% to 80% for both groups. Trial of labor in patients with two previous cesarean deliveries is therefore a reasonable option for selected patients. Data pertaining to vaginal delivery following three

or more cesarean deliveries are too scant to permit an estimate of safety, and management of such cases must be individualized.

### **Epidural Analgesia/Anesthesia**

The use of epidural anesthesia during VBAC trials does not increase the risk of either maternal or fetal morbidity. An issue commonly debated is whether epidural analgesia might mask the symptoms of uterine rupture. Several clinical signs and reported symptoms, such as vaginal hemorrhage, pain, signs of fetal distress, loss of station, or, rarely maternal cardiovascular collapse, alert the obstetrician to the probability of uterine rupture. These signs and symptoms associated with rupture are unchanged by regional blockade. Based on available data, it appears that pain related to uterine rupture is incompletely blocked by epidural analgesia in doses used to provide pain relief in labor. Nonetheless, it is always prudent to employ an epidural for analgesia during VBAC trial. Dense or anesthetic levels are unnecessary, predispose to poor progress, and should not be employed. (See Chapter 9, Obstetric Anesthesia.) If surgical anesthesia is necessary, the epidural can simply be reinforced, or, if required, a general inhalation anesthetic can be administered.

### **Twins, Breech Presentation, and Fetal Size**

Most studies pertaining to VBAC have, among their inclusion criteria, singleton gestations, and often require estimated fetal weights of 4,000 g or less. These restrictions presumably reflect a belief that overdistension of the uterus predisposes to uterine rupture. In view of the general reluctance of obstetricians to attempt vaginal delivery of a fetus in the breech presentation, and because only 38% of twins are in the cephalic-cephalic presentation, most patients carrying a twin gestation and who have had a prior cesarean delivery are delivered by a repeat cesarean. A prior cesarean delivery is not an absolute contraindication to a vaginal trial in twins, however, as long as the leading twin is cephalic and progress in labor is normal. Management of these cases is not for the neophyte, however. For those women undergoing a trial of labor, careful counseling and attentive intrapartum monitoring are necessary.

In singleton gestations when the fetus is in the breech presentation, Ophir and coworkers found

that 78% of a group of 47 such patients who were allowed to undergo VBAC subsequently delivered vaginally [187]. In this group, there was no increase in neonatal morbidity. The option of an attempt at gentle external cephalic version can be safely offered to patients with prior cesarean delivery and a singleton breech-presenting fetus [188]. The prudent clinician conducts such procedures at a site where prompt cesarean delivery is possible, if a complication results. As a matter of practicality, however, few clinicians are probably willing to be this aggressive in seeking a vaginal trial.

When diabetes has been excluded, suspected fetal macrosomia is not an absolute contraindication to a VBAC trial, but close attention to normal progress and prudent use of instrumental delivery is required, and the rate of success could well be less.

### **Conclusions**

Obstetricians know that Edward Cragin's 1916 adage "once a cesarean, always a cesarean" is no longer an accepted guide to management [189]. When a VBAC trial is agreed on, the obstetrician and the institution should be able to provide appropriate technical support to ensure a safe delivery, including the presence of an experienced physician and a plan for prompt emergency cesarean delivery if it becomes necessary. These labors also require close fetal heart rate monitoring, rapid access to a blood bank, and immediately available anesthesia services. As Table 18.6 notes, there are several historical features that predict a favorable labor trial. Perhaps it is more accurate to state that the mantra for the twenty-first century is "once a cesarean, always a carefully monitored pregnancy and labor."

### **SPECIAL ISSUES**

#### **Timing of Elective Procedures**

For several reasons, a cesarean delivery might need to be performed electively, prior to the onset of labor. If so, fetal pulmonic maturity should be determined before the procedure or, in uncomplicated cases, the procedure should be scheduled near to term to reasonably ensure fetal pulmonic maturity, following the general criteria used for any labor induction [191]. The two general methods for determining the timing for elective cesarean operations are

scheduling based on clinical and menstrual data, and ultrasonic analysis, or alternatively based on amniotic fluid sampling to confirm pulmonic maturity by specific testing. In uncomplicated cases of women with reliably recorded and regular menses and in the absence of gestational diabetes, elective scheduling after 38 *completed* weeks of gestation is now recommended. The requirements for scheduling based on combined clinical and biophysical findings include

- 20 weeks of documented fetal heart tones by a nonelectronic fetoscope or up to 30 weeks by a Doppler fetoscope
- 36 weeks or more have elapsed since a positive serum human chorionic gonadotropin pregnancy test result
- Ultrasonic measurements based on a crown-rump length obtained between 6 and 12 weeks of gestation, supporting a gestational age of  $\geq 39$  weeks
- An ultrasound scan at 13 to 20 weeks of gestation that is consistent with a gestational age of at least 39 weeks as verified by clinical history and physical examination

Determinations of the period of gestation based solely on maternal history assume that the data concerning menstrual history, early examination, heart rate auscultation, quickening, and fundal growth when extrapolated to the third trimester are reliable markers of term gestation. Unfortunately, this is often not the case, and substantial and potentially serious errors are possible.

In less certain circumstances, other specialized studies at or near term can prove helpful. In uncomplicated nondiabetic patients, the ultrasound documentation of a biparietal diameter (BPD) of  $>9.2$  cm or a femur length of  $>7.3$  cm are reasonably reliable indicators of fetal maturity [192,193]. A distal femoral epiphysis of  $>3$  mm also defines a fetus of greater than 36 weeks with a high likelihood of pulmonic maturity. Fluid obtained at amniocentesis also can be submitted for pulmonic maturity analysis [194,195–198]. The principal test for amniotic fluid surfactant in past years was the lecithin/sphingomyelin (L/S) ratio. Test results exceeding a 2:1 ratio confirmed maturity. In recent years, faster and more convenient tests have been introduced. Currently, a surfactant/albumin

ratio test (e.g., Abbott TDx-FLM II test) of 60 mg/g of surfactant/albumin or more, or positive phosphatidylglycerol (PG) slide test reliably predicts pulmonic maturity. Several other amniotic fluid tests exist, including studies for lamellar body analysis, determination of dipalmitoylphosphatidylcholine concentration, and variations of the older foam stability index, among others. None these tests has proved popular, however. Recently the faster, automated tests have become those most often employed, largely replacing the original L/S ratio. Tests such as the Abbott TDx-FLM II have relatively high false-negative rates but low false-positive rates. Thus, a positive test interpreted as indicating pulmonic maturity has a strong PPV and false positives are rare. In most cases, as a practical matter, booking an elective, repeat cesarean is based on a combination of physical examination, history, ultrasound information, and in selected cases, data from amniotic fluid sampling. In uncertain cases when amniotic fluid sampling is deemed imprudent or is impossible, and dating is otherwise uncertain or the data are contraindicating, simply awaiting the spontaneous onset of labor before repeating an operative delivery might be best.

### Elective Cesarean Delivery

In recent years, the accepted indications for cesarean delivery have increased, while the long-term controversy over the appropriate percentage for cesareans has become muted. Operative delivery rates now exceed 30% in many parts of the United States.

The principally accepted indications for cesarean delivery include two major categories: those arising as emergencies during the course of labor, and those identified before the onset of parturition. The latter indication often leads to surgery that is scheduled in advance. Conditions identified before the onset of labor that constitute indications for a scheduled cesarean include previous cesarean delivery, fetal malpresentation, placenta previa, and multiple gestations, among others. Conditions arising during labor that lead to operative intervention usually involve failure to progress or dystocia, or one of a variety of acute problems such as intrapartum hemorrhage, cord prolapse, or presumed fetal jeopardy due to unacceptable fetal monitoring data. The current levels of surgery are sustained because of

the perception by practitioners and institutions of increased fetal and thus legal risk in certain cases (e.g., VBAC trials, pelvic instrumental delivery, and so forth), to scientific advances indicating a benefit to the fetus from abdominal delivery (e.g., breech presentation, birth injury, or HIV transmission prevention), or simply to patient choice.

The prevailing wisdom in society, and to an alarming degree within the profession, is that a cesarean is the panacea for all obstetric difficulties. Certainly, the cynical observer would note that clinicians rarely face legal entanglements for performing a cesarean unless a major surgical complication such as a ureteric injury occurs. In contrast, the alleged failure to perform a cesarean or to perform one in a timely manner is the common theme in many medical legal claims.

A controversy has developed recently over what might be termed *purely elective cesarean deliveries* [199–203]. The various contributors to this controversy color the debate by their choice of terminology. The terms *cesarean on demand*, *designer deliveries*, *requested surgery*, and *patient choice cesarean* all appear in the literature. These terms are used to describe cesarean deliveries performed at a woman's request without the traditionally accepted medical indications for obstetric surgery (i.e., placenta previa, dystocia, malpresentation, or others). To best frame the argument, one must review the strength of the evidence supporting current practice, data concerning maternal and fetal birth injuries related to vaginal delivery, and consider the maternal-fetal risks associated with a cesarean. In addition, the possible adverse effect of a rising cesarean delivery rate on institutions and their obstetric services should not be forgotten. Philosophical discussions about intervention in a natural process and the psychological benefits or risks of vaginal delivery are important but must be considered separately.

Elective cesarean delivery remains controversial [200,202,204,205]; nonetheless, it appears that a consensus is slowly emerging that such procedures fall within acceptable practice. The situation is quite complex, however. When pregnant women are questioned, a large percentage of them state that they prefer to plan for vaginal delivery [206]. The rate of VBAC trials has substantially declined mostly because of the limitations in personnel, the medicolegal implications, and the resulting reluctance of both institutions and practitioners to accept the

associated risks. Current operative delivery rates hover around 30%. At the same time, the rate of primary elective cesareans performed at patient request has steadily risen. Depending on the series, 4% to 18% of all cesareans and 14% to 22% of elective cesareans are now conducted "on request" [207].

As Minkoff comments "elective cesarean delivery is no longer a marginal idea" [199]. The recent ACOG Committee Opinion No. 289 opined that elective cesareans are acceptable practice following full patient discussion and informed consent [201]. Surveys of opinion among international practitioners indicate that a substantial percentage of obstetricians would conduct or support elective surgeries, backing the concept that it is a women's right to have an elective operative delivery without a traditional medical indication. As an example of this literature, Wu and coworkers [208] conducted a web-based email questionnaire study of members of the Society for Maternal-Fetal Medicine (SMFM) and the American Urogynecologic Society (AUGS). The overall response rate was slightly above 50%. These data indicate that 80.4% of AUGS and 55.4% of SMFM respondents would be willing to perform a purely elective cesarean. A logistic regression model was used controlling for age, sex, specialty, years in practice, and whether the respondents had children themselves. These data indicate that AUGS members remained 3.4 times more likely to agree to perform a primary cesarean delivery (95% CI, 2.3–4.9,  $p \leq 0.001$ ) than their SMFM counterparts [209]. Bergholt and coworkers [209] conducted a similar study in 2004. In this investigation, an anonymous postal questionnaire was sent to 455 practitioners who were members of the Danish Society of Obstetrics and Gynecology. Practitioners were presented with a group of specific clinical scenarios involving specified gestational ages and estimates of fetal weight and asked if they favored an elective cesarean in each instance. In this series, 1.1% to 22.5% of the responding practitioners agreed with a cesarean depending on the parameters of the various hypothetical cases scenarios that were proposed in the questionnaire. Cotzias and coworkers conducted a similar postal survey in England and Wales [210]. In this study, nearly 70% of obstetric consultants were willing to agree with maternal request for a cesarean in the absence of the usual indications.

TABLE 18.8 Fetal Outcomes: Cesarean vs. Vaginal Delivery

Vaginal Delivery	Cesarean Delivery
Mortality: 1–3 in 4,000	Mortality: $\leq 1$ in 1,000
Common Morbidity:	Common Morbidity:
Shoulder dystocia	Transient mild respiratory acidosis
Intrauterine hypoxia*	Lacerations: face, buttocks, extremities
Fracture of clavicle, long bones, or skull	Fracture of clavicle, long bones, or skull
Intracranial hemorrhage 1 in 2,000	Intracranial hemorrhage 1 in 2,000
Facial nerve injury* 1 in 3,000	Facial nerve injury 1 in 2,000
Brachial plexus injury* 1 in 1,300	Brachial plexus injury 1 in 2,400
Convulsions* 1 in 1,560	Convulsions 1 in 1,160
CNS depression* 1 in 3,230	CNS depression 1 in 1,500
Feeding difficulty* 1 in 150	Feeding difficulty 1 in 90
Mechanical ventilation* 1 in 390	Mechanical ventilation 1 in 140
Persistent pulmonary hypertension* 1 in 1,240	Persistent pulmonary hypertension 1 in 270
Transient tachypnea of newborn* 1 in 90	Transient tachypnea of newborn 1 in 30
Respiratory distress syndrome* 1 in 640	Respiratory distress syndrome 1 in 470
	Long-term increased risk of unexplained stillborn

\*Difference statistically significant  $p \leq 0.05$ .

A recent German study of board-certified obstetricians and gynecologists [211] had similar findings. In this questionnaire investigation, when asked for the preferred mode of delivery for either themselves or a partner in a low-risk pregnancy, 90% of responding clinicians favored vaginal delivery; however, 59% of the physicians also approved of maintaining the opportunity for cesarean delivery on demand.

This issue is far from settled. Spirited discussion continues both in and out of the profession about elective cesarean surgery and its potential effects on practice. Despite the fact that substantial percentages of clinicians state that they are willing to accept or conduct elective operations, it would be incorrect to claim that obstetricians fully accept this concept [200]. There remains substantial opposition to elective cesareans from those within the specialty and among nonphysician groups interested in women's health issues [201,212–214]. Elective cesareans have both advantages and potential disadvantages (Tables 18.7 and 18.8). Detractors emphasize the potential risks, deny the possible advantages, and are generally displeased with “medicalization” of the birth process.

The immediate and long-term risks of a major operative procedure such as a cesarean require con-

sideration. The question becomes largely that of morbidity. The mortal risk for elective cesarean surgery in otherwise normal subjects is extremely low. Philosophical consideration aside, the mortal risks to mother and child are probably equal regardless of the method of delivery. In the estimation of risk, it is inappropriate to compare all cesarean deliveries to unselected vaginal deliveries. The unfiltered cesarean group includes many problem cases such as emergencies complicated by infection, hemorrhage, or hypertension. The proper comparison is a cohort of uncomplicated cesarean patients, who are similar in age and parity, operated on before the onset of labor against a group of normal women undergoing vaginal deliveries who required an unplanned surgery for various obstetric indications.

What this debate hinges on is societal demand, the willingness of third-party payers to accept elective procedures as reimbursable, and the strength of the data concerning the long-term outcome of vaginal delivery, related pelvic support, and perineal injury. It is the opinion of the authors that more elective cesareans will be performed, but that the rate will vary substantially in different locales. Because there is no correct answer for this conundrum, spirited controversy will persist.

TABLE 18.9 Postmortem Cesarean Deliveries: Elapsed Time and Infant Survival

Estimated Time of Maternal Death to Delivery (min)*	Number of Patients	Percent
0-5	42 (normal infants)	70
6-10	7 (normal infants) 1 (mild neurologic sequelae)	13
11-15	6 (normal infants) 1 (severe neurologic sequelae)	12
16-20	1 (severe neurologic sequelae)	1.7
21+	2 (severe neurologic sequelae)	3.3
Total	60	100

\*Estimated time from death of the mother until delivery (cases from 1900 to 1985).

From Katz VL, Dotters DJ, Droegmueller W: Perimortem cesarean delivery. *Obstet Gynecol.* 1986. 68:571-576; with permission.

### Perimortem Cesarean Delivery

Postmortem or perimortem abdominal deliveries have been reported for more than 400 years. It was early appreciated that a living child might rarely be salvaged late in pregnancy if the mother had died acutely and a delivery was performed promptly. In addition, early Christian religious requirements in at least some parts of northern Europe demanded a separate burial for both mother and child (and baptism for the latter), providing an additional reason for prompt postmortem delivery. Eventually, various laws incorporating “good Samaritan” provisions were developed, legally shielding those performing good faith postmortem deliveries from an accusation of criminal behavior. It is possible that the occasional success of postmortem abdominal deliveries led physicians to attempt cesarean deliveries on living women with the expectation of at least some maternal and infant survivals. (See Chapter 1, A History: Operative Delivery.)

New observations over the last decade have changed the approach to postmortem cesareans [215-219]. In several recently reported instances, despite the clinician’s belief that the mother was dead or moribund, women who could not be revived while the fetus remained in utero have been successfully resuscitated postdelivery. This eventual response to resuscitation after removal of the fetus is presumed to be because of the decompression of the obstructing uterus, leading to improvements in cardiovascular function and perhaps also in the mechanical aspects of respiration. Further, additional data have become available concerning fetal outcome versus the time interval from presumed

maternal death until delivery, establishing rough limits for clinicians to employ in making the choice to intervene. These observations have led to modification in standard procedures whenever perimortem operations are contemplated.

As in all clinical scenarios, there are unique or outlying cases. Unusual cases of intact fetal survival involving as much as 20 to 30 minutes after maternal cardiac arrest have been reported [218-221]. In most of these successful cases with prolonged times to the fetal extraction, the maternal-fetal condition was apparently physiologically normal in terms of placental oxygenation and fetal growth immediately before the mother’s fatal injury, and aggressive resuscitation efforts were begun early. The outcomes in these cases, however, are not reflective of the general experience with such cases as reflected in the medical literature.

The current approach to perimortem deliveries is necessarily limited because it derives from the analysis of case reports. These data are combined with a general understanding of maternal cardiovascular physiology to develop recommendations for practice. The principal report is that of Katz and coworkers, who surveyed the English literature concerning postmortem operations and reported 269 cases from which 188 infants survived (Table 18.9) [222]. In the analysis of these data, important variables in fetal survival included both the length of time from the apparent maternal demise and the extent and type of the maternal resuscitation efforts attempted.

The important variables in the successful salvage of an infant include the time interval between the maternal cardiopulmonary arrest and delivery, the

weeks of gestational age, any preexisting maternal or fetal problems, and the extent and effectiveness of the cardiopulmonary resuscitation efforts before the delivery [218,219,221–225]. With these general principles in mind, our proposed management concepts are derived from the available, albeit flawed information, in the effort to make reasonable choices supported by the preponderance of data.

Despite the publication of several unique cases with long intervals from maternal collapse to delivery, strong evidence suggests that the more rapid the delivery, the better the fetal outcome. Based on the Katz data, postmortem deliveries performed 15 minutes or more *after* maternal demise were less likely to result in a living or undamaged infant; however, all infants delivered within 5 minutes of maternal collapse proved normal. As noted, there are rare instances in which longer insult to injury times have been recorded but these cases are clearly atypical. As the available data are perused, it is important to remember the distinct limitations of individual case reports and collected case series. For the series, the data are collected from individual reports over several years and by necessity must be drawn from more than one institution. Methods of evaluation and treatment are obviously not constant. There are other distinct limitations to these data as well. The times reported in the various reports must remain suspect, both in terms of accurately recording the time of the maternal injury but more important in estimating when effective uterine blood flow ceased. In terms of physiology, the interval from actual maternal injury or collapse until the cessation of cardiovascular function varies and depends on the cause of death. Thus, an automobile accident with pelvic trauma, or a gunshot wound to an extremity that eventually proves fatal owing to exsanguination or to the presence of other injuries has a different physiology than a case involving a pulmonary embolism or a cardiac arrest that results in a maternal cardiovascular collapse with cessation of circulation.

When maternal resuscitation is attempted following collapse, including restoration of circulating volume, oxygenation, cardiac compression, and other measures, and it is judged that the mother has either sustained obvious lethal injury or the possibility of resuscitation is believed to be limited or poor, it is recommended that active efforts continue to point until actual delivery occurs. This is believed

**TABLE 18.10 Postmortem Cesarean Delivery: Important Clinical Questions**

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Has an adequate effort at maternal resuscitation been made (>5 minutes)?
Are continued efforts deemed either futile or quite unlikely to succeed?
Is the gestational age of the infant known or estimated as at least 25 weeks?
Are there facilities and personnel for immediate infant resuscitation and support?
Is the time from the maternal insult/injury more than 15 minutes? If so, when was active maternal resuscitation initiated?

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to maximize the chance for intact fetal survival. The fetus can survive only if maternal blood flow, specifically the oxygen supply, is maintained. In cases in which maternal exsanguination occurred secondary to lethal trauma or when the fetal condition was already precarious for any reason, the probability of intact neonatal survival is correspondingly lessened. Either the usual methods for maternal revival prove much less successful in supporting both circulation and oxygenation in these situations, or the fetus is less able to tolerate asphyxia. The critical variable in all cases is the timeliness and celerity of the resuscitation efforts.

Before a perimortem cesarean procedure is performed, there are several important considerations (Table 18.10). Unfortunately, the clinician is faced with the necessity of making a decision of potentially serious consequence with only a few minutes available for reflection.

Initially, when faced with the situation of an apparently moribund mother, clinicians must assure themselves that active maternal resuscitation has had a reasonable trial before proceeding to an operative delivery (Table 18.11). Sustained efforts for at least five minutes are indicated. There is at least one case of a mother who sustained a cardiopulmonary arrest at 15 weeks of pregnancy, was resuscitated, and subsequently carried successfully to term. During the resuscitation, as much left lateral tilt as possible should be used to sustain venous return to the heart and to support the mother's cardiac output.

Before performing a postmortem operation, the next most important concern is gestational age. Estimating the gestational age is difficult if there is no available history. An operation performed too early is only a gesture if it has no potential for either

**TABLE 18.11 Technique: Perimortem Cesarean Procedures**

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Operative decision by most experienced clinician available
Summon pediatric/nursing assistants.
Perform a midline abdominal incision and a vertical uterine incision.
Obtain cord blood and arterial/venous gas samples.
Administer a potent uterotonic intramyometrially and broad-spectrum antibiotics intravenously to the mother.
If mother is pronounced dead, perform a bulk abdominal wall closure for aesthetic reasons.
If resuscitation is to continue, perform a uterine closure in layers: a bulk abdominal wall closure follows.
Full medical record documentation
Consult with family.

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maternal or fetal benefit. Furthermore, if the best estimate for gestational age is *less* than 24 to 25 weeks, there could be minimal improvement in maternal cardiovascular physiology from emptying the uterus. Thus, in an early pregnancy, uterine wedging and aggressively pursuing active resuscitation efforts is therefore the best management. Further, it is unlikely that any profoundly premature infant will survive unless delivered in reasonable condition and provided with immediate expert care. For these reasons, when pregnancies known with reasonable certainty to be <25 weeks are encountered, it is best to persist with efforts at maternal treatment and not open the uterus. In contrast, in cases involving pregnancies of 25 weeks' gestation or more, postmortem cesarean delivery should be considered [226].

Regardless of the care taken in case choice, it is well to recall that even prompt and successful delivery might not result in a fetal survival or for that matter an intact survival. The fetus might prove to be smaller than anticipated and thus previsible or already irreparably damaged, despite being of a reasonable gestational age. Delivery also provides no guarantee that the mother's condition will improve sufficiently after removal of the products of conception to improve her chances of eventual survival. *Not to act, however, will result in the loss of both mother and child when there is some likelihood that one, and possibly both, could survive.*

In terms of practical management, if the mother has arrested, spending time searching for fetal heart tones either by a Doppler device or by auscultation is not relevant to the decision process. In the ordered

chaos of an acute maternal resuscitation, potentially using an unfamiliar ultrasound machine or handheld device for Doppler detection of cardiac activity is always difficult and can prove impossible, even if fetal cardiac activity is present. In addition, if the fetal heart is not moving, the clinician does not know how long the heart has been arrested. Potentially, real-time ultrasound scanning performed while the resuscitation continues might assist in the rapid estimate of gestational age and help to avoid egregious errors. Care must be taken since serious errors can occur if the uterus is larger than dates because of a multiple gestation, if the woman is obese but less than 24 weeks' gestation, or if hydramnios is present. Any fetus with a BPD  $\geq 62$  mm or a trans-cerebellar diameter of  $\geq 28$  mm should be considered a potential survivor. Such refinements in management might well prove impossible to introduce in an emergent situation, however. Often, only a quick abdominal palpation and a history of a due date (provided by others) are all that is available to the clinician.

In these difficult circumstances, the most senior obstetrician in attendance should make the final operative decision. In the absence of an obstetrician, the decision must be left to the attending physician with the greatest amount of obstetric experience. Maternal resuscitation efforts should be continued aggressively up to the very moment when the maternal abdomen is opened.

Time should not be lost in seeking the usual surgical equipment or in moving the mother to an operating theater. There is usually no time for abdominal preparation or to obtain the usual surgical equipment; often simply gloves, a scalpel, and a splash of an iodine solution are all that are provided. A midline incision is best; both abdomen and uterus are usually entered vertically, and the infant promptly delivered. A transverse uterine entry with digital separation of the wound can prove equally fast in experienced hands. Care must be taken to avoid a maternal intestinal injury or a fetal laceration. Both a cord blood sample and cord gas should be obtained for subsequent study. If there is a possibility of maternal survival, the uterus should be closed in layers with particular attention to close reapproximation of the uterine walls. Firm approximation of the myometrial incision is important. If and when maternal arterial perfusion returns to normal, vessels not bleeding at the low perfusion pressures characteristic of resuscitation



suddenly become active as the arterial pressure increases. A potent uterotonic such as methylergonovine (Methergine) or 15-methylprostaglandin F<sub>2α</sub> (Hemabate) is also injected directly into the myometrium, and a broad-spectrum first-generation cephalosporin should be administered intravenously to the mother. A bulk closure for the maternal abdominal wall is appropriate. As soon as possible after the fetus is removed, maternal resuscitation efforts should be resumed.

A particularly difficult management issue involves the unusual situation in which a pregnant woman sustains an injury that either leaves her in a persistent vegetative state or the woman has a disease soon to prove fatal [227]. In selected cases, active maternal support has been continued in the hopes of carrying the pregnancy to the point of potential viability. Such cases involve major issues, including ethical considerations, the best use of hospital resources, desires and consent of relatives, uncertainty of fetal condition or fetal damage from the initiating event, and complex dilemmas concerning the timing of delivery. Individualization of management, legal consultation, and the involvement of multiple consultants as well as the hospital administrators are required in these unfortunate situations.

A perimortem or postmortem cesarean is an emergency procedure. Obviously, obtaining consent from the mother is not possible. Attempting to locate or negotiate with family members should not take time away from this emergency operation. The surgeon should evaluate and proceed to delivery with celerity if the minimal prerequisites for a postmortem operation are present, or a fleeting opportunity can be irretrievably lost. In this setting, no other opinion is legally binding. *The greatest risk is the clinician's failure to act promptly, with the resultant loss of the chance for a fetal salvage.* Whatever the outcome of the procedure, a complete note must be included in the medical record, preferably by dictation or by a computerized record. No legal settlements related to postmortem cesarean procedures have been reported.

## PROCEDURES AND TECHNIQUE

This section reviews the operative technique for cesarean delivery, cesarean hysterectomy, and the surgical management of acute obstetric hemorrhage.

As always, proper choice of cases, accurate diagnosis, adequate light and exposure, an expert surgeon and assistants, appropriate anesthesia and staff, gentle handling of tissues, and meticulous attention to hemostasis are the most important components of success in obstetric surgery. General surgical issues, including type of abdominal entry, choice of suture material, and aspects of general surgical management, are discussed elsewhere in this text.

## **Anesthesia**

Most clinicians prefer epidural or spinal anesthesia for cesarean delivery, as the clinical circumstances permit. The parturient remains awake, members of the family can be present, and the potentially difficult problems of intubation and airway management are avoided. In modern practice, maternal morbidity related to regional anesthesia is rare [228]. Unfortunately, epidural anesthesia is neither always available nor appropriate, especially if emergency surgery is required. Issues involving anesthetic management are intensively discussed in Chapter 9, Obstetric Anesthesia.

Whatever the choice of anesthesia, the obstetric surgeon must review any special clinical circumstances leading to the cesarean or important maternal history with the anesthesiologist prior to the surgical procedure. Obviously, the anesthesiologist must be informed if the mother has any medical problems, such as hypertension, insulin-dependent diabetes, or other potentially serious medical conditions. In patients having preexisting medical problems and elective surgeries, a preoperative anesthesia consultation is best obtained remote from term. Conducted without the pressures of the labor suite and with sufficient time to perform indicated studies, this preoperative interview permits review of any medical conditions, ordering of indicated tests (e.g., coagulation studies, antibody screens), specialized consultations (e.g., echocardiography, ECG), and sufficient time for the patient and the anesthesiologist to meet and discuss the projected operation and the necessary anesthesia. Such preliminary visits are indicated for women with congenital or acquired cardiac disease, serious medical complications (e.g., advanced diabetes mellitus, severe chronic hypertension), hereditary disorders likely to result in complications (e.g., the Marfan syndrome or Ehlers-Danlos), patients receiving anticoagulants,

those with prior back surgery, those with a history of prior anesthesia complications, and for other complicated or unusual cases.

In selected cases, antepartum collection of maternal blood with subsequent perioperative retransfusion is an appropriate strategy [229]. Although methods of perioperative blood aspiration and autotransfusion are well established in general surgical practice, they have been little used in obstetrics. Limited data suggest that such techniques are safe [230].

### General Preparation

After the anesthetic has been administered, assuming either spinal or epidural, the woman is positioned on the operating table, her legs are restrained as per the surgical routine, and a disposable electrical ground pad for electrocautery is applied, if desired. A modern electrocautery unit combined with a disposable patient contact pad that makes good contact to the patient's skin presents minimal electrical risk. Electrocautery is ideal for control of small intraperitoneal bleeding sites or bleeding vessels encountered under the fascia. A hand-activated disposable cautery pen, with a narrow electrode tip that permits close control of both the site and the extent of cautery, is best. The potential patient risk from the use of a modern electrocautery unit is far outweighed by the benefit of this method of hemostasis.

After the spinal or epidural anesthetic has been induced an indwelling catheter (Foley) is inserted. When general anesthesia is administered, the catheter is inserted immediately before the onset of the anesthetic; in instances requiring extreme speed, the bladder can be decompressed perioperatively, as required.

Positioning is an important issue that should not be left to the surgical attendants alone. The authors favor as much left lateral tilt as is feasible, especially if there is a history of supine hypotension or a suspicion of fetal jeopardy. Lateral positioning is accomplished either by a tilt of the operating table, or preferably by the use of a pelvic wedge. In the authors' institution, one or more liter bags of Ringer's lactate are usually placed under the patient's right hip. Rotation to the left is best because this decompresses both the maternal vena cava and the aorta. If a woman in the third trimester

is positioned supine, venous return to the heart is impeded, and the cardiac output and resultant uterine blood flow can decline substantially. This effect varies greatly, and the extent of uterine hypoperfusion might not be reflected in the usual brachial arterial pressure determinations. Because the simple prophylactic measure of hip wedging is not harmful, easily performed, and potentially beneficial, the authors favor its use. The use of lateral tilt is extrapolated from the physiology of uterine vascular flow and is not based on data from prospective studies [231,232].

### Fetal Monitoring

The importance of continuing fetal monitoring in the interval between exiting the labor suite and the final delivery in the operating theater needs emphasis. If a fetal scalp electrode was placed in the labor suite to better evaluate heart rate patterns, it should be left in situ when the parturient is moved to the operating suite, and once there promptly attached to an electronic monitor. The electrode is left in place until immediately prior to draping. Alternatively, when time is of the essence, the child can be delivered with the electrode attached. The wire is simply cut and subsequently withdrawn from below. If an effort at vaginal instrumental delivery was made, regardless of the instrument or how gentle the manipulation or traction, an electronic monitor should be applied promptly, using either an external Doppler transducer or electrode. The FHR should be continuously recorded while preparations for cesarean are made. Occasionally, unexpected fixed fetal bradycardias ensue. If this occurs, cranial displacement or expedited surgery is appropriate. If the FHR was stable preoperatively and no FHR problems were diagnosed or suspected, periodic FHR auscultation by a handheld Doppler device or a fetoscope, at a frequency determined by hospital protocol, is appropriate. Alternatively, the external FHR Doppler transducer can be left in place until skin preparation has begun. The usual patient repositioning necessary for the various preoperative preparations frequently precludes uninterrupted monitoring by the abdominal Doppler technique. Intermittent monitoring by the handheld Doppler device or continuous monitoring by scalp electrode are the most practical methods in these instances.

It is not uncommon that a prolonged interval ensues between the last FHR determination and the actual delivery. When such delays before the actual procedure occur, they usually result from the cumulative effect of multiple small delays from patient transfer, positioning, administration of anesthesia, draping, and other procedures. The clinician is often surprised later at the total time consumed by such routine operating suite activities. Part of this delay is psychological. If a frankly ominous FHR pattern or acute jeopardy were not diagnosed in the labor room, and the decision has been made for a cesarean based on criteria of progress, tracing, and other factors, both the physician and the other birth attendants usually assume a "routine-case" attitude once the gravida is in the surgical suite. Simply moving the patient to the operating suite seems to instill a feeling among the staff that some definitive action has been taken. In the authors' experience, in these circumstances, rapid FHR deteriorations are not anticipated and therefore are not always promptly detected. Close attention to the FHR with the same diligence in the operating suite as in the labor suite is mandatory.

### Skin Preparation

Once the patient is correctly positioned on the operating table, any desired or necessary skin preparation (shaving) is performed if desired. Shaving results in microscopic nicks and tears of the epidermis and actually increases the risk for skin infection, unless performed immediately preoperatively [233,234].

The skin at the operative site is next prepared with an antiseptic solution. Various solutions are appropriate for use, following the established protocols of the individual institution. Draping for surgery follows.

The authors favor prepackaged disposable paper drapes that include an operating port with an circumferential adhesive backing and a fluid collection bag. As the drape is unfolded, the anesthesiologist grasps the anterior edges and fixes them to an ether screen, or other adjacent equipment. Once the drape is correctly positioned, the paper covering the adhesive portion is removed and the drape is pressed against the patient's skin, sealing the site around the incision. No other surgical drapes are routinely employed except the usual table and Mayo stand covers.

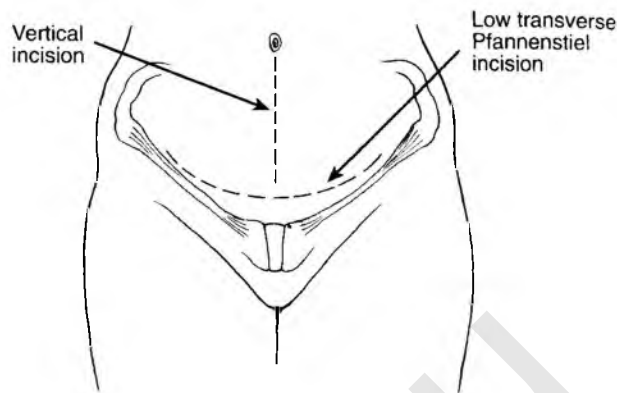


FIGURE 18.2.  
*Placement of common lower abdominal surgical incisions for cesarean delivery.*

### Incision

After the anesthesiologist has confirmed that the patient is ready, and the scrub nurse and the surgical assistant are poised, the surgeon prepares for the initial skin incision. The type of skin incision depends on the clinical circumstances. Usually the skin is entered by a low transverse Pfannenstiel-type incision that curves gently upward, placed in a natural skin fold (Figure 18.2). Classically, this incision is located two fingerbreadths above the pubic symphysis. If a Maylard or Cherney operation is contemplated, the initial incision can be placed higher or lower, at the surgeon's discretion. When a Joel-Cohen technique is chosen, a transverse incision is performed but positioned higher on the abdomen than the usual Pfannenstiel incision and without the usual upward curve at the ends. Occasionally, a midline vertical incision from umbilicus to symphysis is preferred. The vertical entry has the advantage of speed and can be easily extended as required, especially if exploration of the upper abdomen is necessary or further problems are likely. The low transverse incision is adequate for almost all cesarean operations and has cosmetic advantages, however. Transverse incisions also interfere less with postoperative respiratory function and result in less pain than vertical incisions.

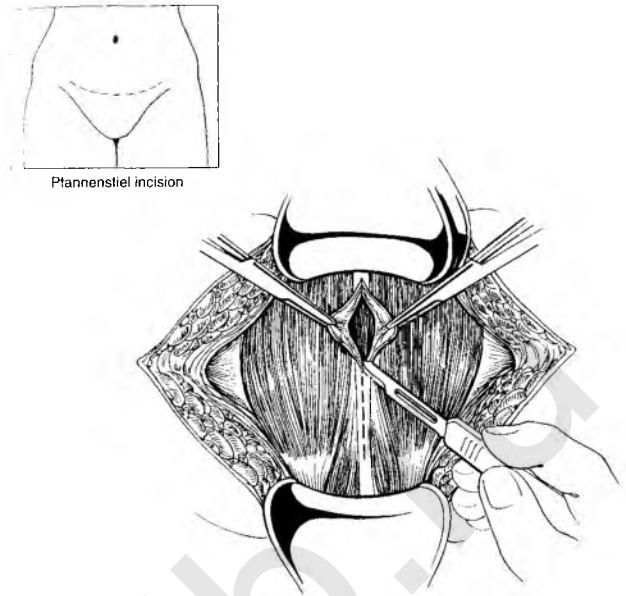
The long-presumed advantage of the Pfannenstiel or transverse-type abdominal incision over the vertical incision in avoiding the potentially serious complications of dehiscence or herniation has recently been questioned. Hendrix and coworkers reported a similar incidence of wound complications

for both incision types in a case-controlled study of 17,995 surgical procedures (8,950 cesarean; 9,405 gynecologic operations) [235]. Identified risk factors for wound disturbance in their obstetric cases included concomitant wound infection and a history of patient smoking.

A supraumbilical transverse incision is also possible but rarely performed. This incision is usually for specific indications such as extreme obesity or the presence of lower segment or adnexal masses. This incision is positioned just superior to the umbilicus. As the dissection is performed, both the underlying rectus muscles and the peritoneum are entered transversely as well.

The best abdominal incision for the morbidly obese is controversial [236,237]. If a large panniculus is present, a midline vertical incision or a high transverse incision as described previously often provides the simplest entry and the best exposure. Alternatively, the panniculus is drawn upward and a routinely placed transverse lower abdominal incision is performed. In the obese, the advantage of a transverse incision is the reputed (but now questioned) strength of the wound closure and the thinness of maternal fat at the site of suprapubic entry. Disadvantages include the wider incision required, the necessity to place the wound in a perpetually moist skin fold, and the steep Trendelenburg position and aggressive retraction necessary for adequate surgical exposure. As a practical matter, when the parturient is morbidly obese, the authors make a final decision about the type of incision after the woman has been prepared, positioned, and draped, depending on the unique features of her anatomy. In a case-controlled study of abdominal entry in the obese, there were similar rates of wound complications when either the high transverse (supraumbilical) or the low transverse (Pfannenstiel-type) incisions were performed [238]. Vertical abdominal incisions, although providing certain advantages in exposure, have more postoperative complications than do transverse incisions and also have a stronger association with postpartum endometritis [236].

The most common abdominal wall entry, a transverse Pfannenstiel-type incision, should be approximately 15 cm long. For cosmetic reasons, the authors prefer to place this type of incision in a natural skin fold (Figure 18.3). The size of the incision is about the length of a standard Allis clamp, hence, the Allis clamp test [239]. Shorter incisions are not recom-



**FIGURE 18.3.** *Pfannenstiel procedure. Note the transverse skin incision with sharp dissection used to separate the rectus abdominis muscles in the midline.*

mended, because they can lead to difficulty in exposure or fetal cranial extraction. Just before the cut is made, the authors' practice is to routinely grasp the patient's skin with two Allis clamps at the projected corners of the wound to define the outer limits of the skin incision. The correct curve for the incision is determined when the surgeon presses a hand downward on the lower abdomen, inducing folds in the skin. The curvilinear skin folds are then easily demonstrated. Keeping the incision within a natural skin fold (Langer's lines) improves the eventual cosmetic result. The midpoint of the Pfannenstiel incision is approximately two fingerbreadths above the palpated edge of the pubic symphysis.

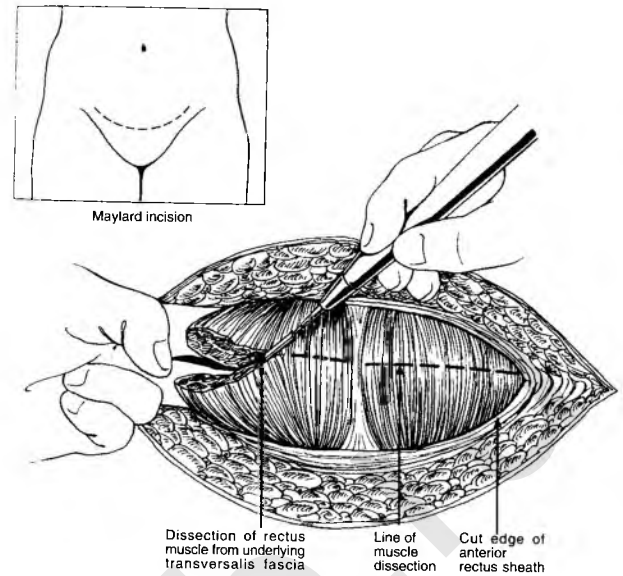
As the procedure begins, the Allis clamps are tensioned, and the incision is made at a right angle to the surface in the skin fold previously demonstrated. When the clamps are removed, the symmetry and placement of the incision are observed and any corrections are made. Careful centering of the skin incision and maintaining symmetry is particularly important when vertical incisions are made on obese patients. Fortunately, the linea nigra usually is sufficiently dark to assist the surgeon in identifying the midline. Nonetheless, in vertical entries, attention to detail is required to keep the incision from

deviating from the midline, especially in the lower portion of the wound.

Meticulous placement of the skin incision is too often neglected. This is the only portion of the operation ever seen by the patient or her family. If the scar is irregular, untidy, or asymmetric, the mother and her family are likely to assume that the rest of the surgery was performed with equally little attention to detail. An operation represents a serious medical intervention, and as such demands the closest attention to detail in all aspects of its performance.

After the skin is entered, the incision is rapidly carried through the subcutaneous tissue to the fascia, which is then nicked on either side of the midline. As an alternative, the subcutaneous tissue can be divided by blunt finger dissection. Finger dissection is part of the Joel-Cohen technique, which also includes a similar blunt separation of the uterine wound. When the incision technique is used, the tradition of using two scalpels – one for the skin and the second for subsequent, deeper dissection – is unnecessary. The one-knife method does not increase the infection risk. As the incision is made, minor bleeding vessels are most frequently encountered at the wound edges and corners. If entered, these bleeders are clamped with mosquito clamps or other fine hemostats and subsequently either tied or electrocoagulated. For many of these small vessels, small clamps once applied can be removed after a few minutes, with no other treatment required. Once the fascia is exposed, it is usually incised in layers, traditionally by curved Mayo scissors. Although it is also possible to open the fascia with the midline and separate it bluntly, this is not a technique favored by the authors.

In standard technique, the fascial edges are next grasped with heavy, toothed clamps such as Kochers, and elevated. Under continuous tension, the fascia is then separated from the underlying muscles by blunt and sharp dissection or by electrocautery. Perforating vessels are clamped and either electrocoagulated or suture ligated. Once the upper and lower fascial flaps have been dissected free and bleeding from any vessels controlled, attention is directed to the underlying rectus abdominis muscles (Figure 18.3). The muscle bodies are often easily separated with simple finger dissection, and if the muscles are adherent, they are promptly divided by sharp dissection; bleeding is usually minimal. The muscle



**FIGURE 18.4.** *Maylard procedure. Note the transverse skin incision and the transverse severing of the rectus abdominis muscles by use of electrocautery.*

midline is indicated by the attachment of the variable pyramidalis muscle.

Lateral tension on the middle of the muscle bodies indicates the degree of exposure to be expected once retractors are positioned in the wound. If the wound is inadequate, it is usually either the original skin incision or the rectus muscles that are restricting exposure. Less often, the original fascial entry proves inadequate. If the wound is too small, it is best to promptly incise the portion of the skin, muscle, or fascia restricting the exposure rather than waiting until there is difficulty with the subsequent fetal extraction. When exposure is markedly limited, the rectus muscle can be divided in the midbelly either sharply or by electrocautery (i.e., Maylard operation; Figure 18.4) [240]. Because this results in significant tissue damage, and because the underlying artery could be entered, the authors prefer the Cherney procedure, if considerations of time and exposure permit (Figure 18.5). In the Cherney operation, the lower fascia is reflected, exposing the tendinous attachments of the rectus abdominis muscle bodies to the fascia of the pubis. The tendinous and avascular lower portion of the muscle is then severed as low as possible. One or both muscles can be divided at the attachments as required. If time and exposure do not permit a Cherney

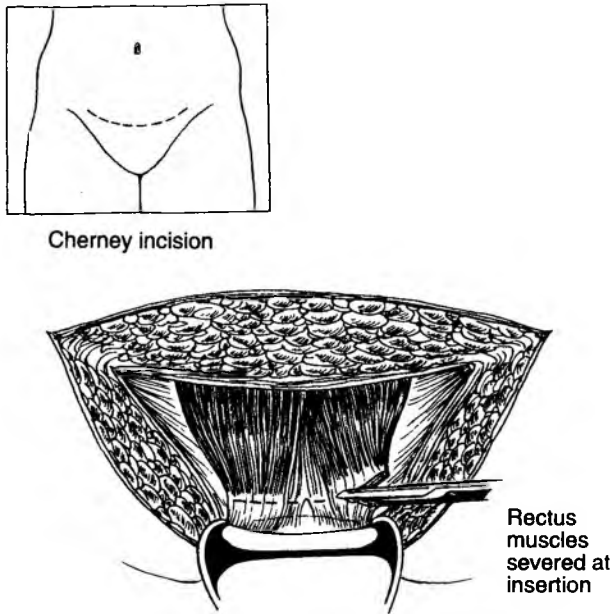


FIGURE 18.5.  
*Cherney procedure. Note the transverse skin incision and subsequent severing of the rectus abdominis muscles from their insertion at the pubic symphysis.*

approach, one or both rectus muscles can be divided by electrocautery to provide sufficient space for fetal extraction. (For additional discussion of problems with fetal extraction from the uterus, see *Difficult Cranial Delivery*.)

The peritoneum is then entered. Preperitoneal fat is bluntly dissected away, revealing the glistening peritoneum beneath. Traditionally, the peritoneum is grasped with forceps or fine clamps, tented to exclude bowel or omentum, and entered sharply. The peritoneum can also be entered bluntly, with simple finger dissection. Usually, the initial entry is then widened sharply with fine scissors, with attention to avoiding the bladder, edge, bowel, and omentum, thus exposing the intraperitoneal contents.

One or more retractors are then placed in the wound as needed for exposure, and the Trendelenberg position is requested. Packing of the gutters usually is not required at a cesarean delivery and should not be performed routinely. If the omentum or bowel continuously prolapses into the operative field, however, one or more appropriately tagged moistened laparotomy sponges can be introduced laterally into the gutters to keep the lower uterine segment free; however, this is an uncommon requirement. Some surgeons routinely prefer to use

a tubular plastic retractor with a rolled edge such as the Alexis wound retractor (Applied Medical, Rancho Santa Margarita, CA 92688) or a Mobius Elastic abdominal retractor (Apple Medical Corporation, Marlborough, MA 01752) (Figures 18.6 and 18.7). These plastic disposable devices reduce the clutter of instruments necessary for surgical exposure.

The surgeon's hand is then placed into the wound and the vesicouterine reflection identified. The rotation of the uterus and the fetal size and position are then also noted. If it has been decided to reflect the bladder, the visceral peritoneum at the vesicouterine fold is elevated and sharply entered transversely. The vesicouterine fold is then developed with blunt and sharp dissection, exposing the lower uterine segment. If there has been prior surgery, the fold could be adherent, and blunt dissection could prove either limited or impossible. Once the bladder flap has been developed, the surgeon prepares to make the uterine incision. The site for the incision is first palpated, to judge the thickness of the myometrium and the position of the fetus.

Several types of uterine entry are possible, but the most popular is the low transverse incision (Figure 18.8). This incision combines a low risk for complication and a reduced likelihood of blood loss compared with vertical incisions. Some type of extension of the original transverse myometrial incision is required in about 1% to 2% of all cesareans [241,242]. Extending the originally transverse incision upward on one side, the J incision, or incising upward in the center for a T usually is performed when there is a sudden need for additional room. This situation can occur after the surgeon's realization that the infant is larger than anticipated, a transverse lie has developed, or the lower uterine segment proves too narrow to permit fetal extraction. If additional space is required acutely, the authors favor either the J or the double J or trapdoor-type incision as opposed to a T incision. The T incision is the weakest and poorest healing of the myometrial wounds and should, when possible, be avoided. In the authors' experience, the insistence on an adequate entry incision, the occasional use of nitroglycerine tocolysis, or the use of a vacuum extractor or forceps intraoperatively substantially reduces the need for an emergent, secondary enlargement of the hysterotomy incision. Although maternal morbidity from an extension of an originally transverse myometrial incision exceeds that from the usual



**FIGURE 18.6.**  
*Without a self-retaining plastic retractor adequate exposure requires 3 retractors and assistant. For color reproduction, see Color Plate 2.*



**FIGURE 18.7.**  
*A and B. Without Mobius retractor, surgical field immersed in blood midline. If the myometrium is thick or recurrent bleeding restricts exposure, it is best to grasp the gaping myometrial wound edges with Allis clamps. Elevating and everting the hysterotomy incision improves visualization, restricts blood loss, and helps to avoid an inadvertent fetal injury. For color reproduction, see Color Plate 3.*



**B**

lower-segment transverse operation, it is still substantially less than that associated with the true classic operation.

The low vertical or Krönig incision is another possibility occasionally chosen when the lower uterine segment is either narrow or undeveloped [233,237]. Unfortunately, there are limitations to vertical incisions. They can prove difficult to restrict to the lower uterine segment, especially in cases of prematurity and malpresentation, when such entries are most frequently performed. Rupture risks during VBAC trials for low vertical incisions restricted to the lower uterine segment are similar to that for low transverse incisions. If the intraoperative dissection enters the upper segment, however, the scar can function like a classic incision, increasing the mother's risk for complication in any subsequent pregnancy [243,244].

A true classic cesarean operation is a longitudinal midline incision performed in the upper, muscular portion of the uterus. This incision occasionally results as an extension from a low vertical incision. The primary advantage of the classic operation is speed and unrestricted exposure. This incision occasionally is chosen if access to the lower segment is limited by adhesions, obesity, or other reasons. These facts account for the usual association of classic operations with emergency surgery, malpresentation, and other important obstetric complications. Disadvantages of a classic procedure include increased blood loss, a longer repair time, and an increased risk of delayed complications, including a high rupture rate in a subsequent pregnancy, especially if labor ensues [237,242,245]. Unfortunately, compared with transverse incisions, classic scar cesarean ruptures in a subsequent pregnancy are more likely to be catastrophic and to occur unpredictably in the third trimester, with or without the onset of labor.

In the usual low transverse operation, the curvilinear incision is placed in the midline of the lower uterine segment, several centimeters below the attachment of the vesicouterine fold. Placement of the myometrial incision varies owing to differences in the anatomy of the uterus that result from the process of labor and the extent of lower segment retraction. In the normal case, a short midline transverse incision is usually made, carefully dissecting through the layers of the myometrium until the chorioamniotic membrane bulges into the wound.

Bandage scissors and lateral wound retractors are then requested. The scissors are inserted extraamniotically (or intraamniotically if the membranes have been ruptured) into the initial entry wound and a curvilinear incision is made, extending sharply upward at the far edges. The presumed advantage of bandage scissors is to control the direction of the myometrial entry, specifically avoiding the uterine vessels and inadvertent fetal injury while keeping the incision in the lower uterine segment. Another acceptable technique is to open the uterus bluntly; this is performed by simply inserting two fingers into the initial myometrial incision and rapidly separating the wound. The blunt technique is as safe as the scissor technique in avoiding disruption of the lateral vessels. The authors usually do not employ this technique except in cases demanding speed, but it has become increasingly popular and could have some advantages. It is also possible to use an automatic stapling device (e.g. AutoSuture CS-57) for the uterine incision, as is discussed later, but except for specialty surgery, the use of this device is not recommended.

Once the myometrial incision is made, the membranes are ruptured if they have already not been entered, the instruments are removed, and the surgeon's hand is introduced into the wound to elevate the presenting part (Figure 18.9). Simple fundal pressure by the surgical assistant usually promptly completes the delivery. When fetal extraction is difficult; the authors have found that acute myometrial relaxation by administering an intravenous bolus of a dilute solution of nitroglycerine (150  $\mu\text{g}$ –350  $\mu\text{g}$ ) to the mother is often helpful. As mentioned previously, if the myometrial incision proves acutely inadequate, and there is no transversely available space, the authors prefer to extend one or both sides of the wound cephalad in a J rather than T incision. A T incision has the poorest healing characteristics and should be avoided unless clinical circumstances are pressing and lateral exposure to permit an upward extension of the corner of the original incision is inadequate or impossible.

When the presenting part is high and the fetus is in a cephalic presentation, delivery from the uterus with simple fundal pressure is usually easy. When a breech presentation is encountered, an extraction is often required. For a frank breech, a Pinard maneuver is first performed. Pressure on the popliteal space



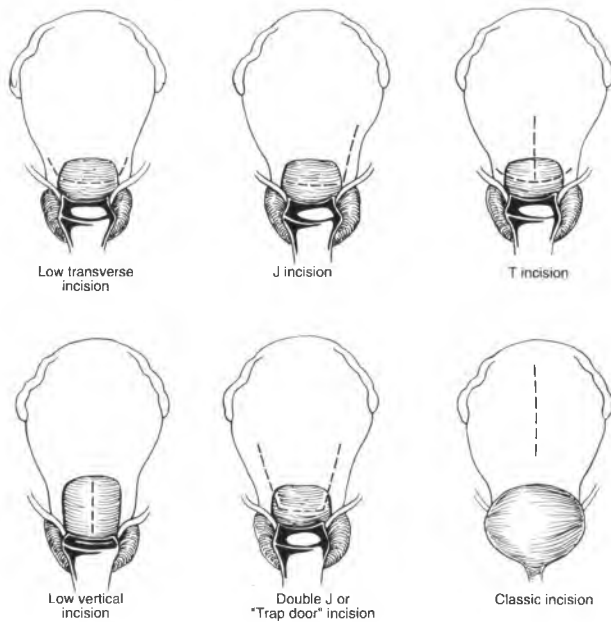


FIGURE 18.8.

*Possible myometrial incisions for cesarean delivery. The classical and T incisions are the least desirable. See text for details.*



FIGURE 18.9.

*Cesarean delivery: usual bimanual technique for manual extraction of the fetal head through the myometrial incision is depicted.*

will help flex the knee. The feet then are drawn down and the lower extremity securely grasped and brought down into the wound. The legs are maneuvered to rotate the fetal body to position the fetal sacrum anteriorly. The operator then positions his/her thumbs along the sacrum, pointing in the direction of the fetal head, and the infant's body is drawn smoothly into the wound. As with any extraction, nuchal arms are common. When the arms pass upward, the extraction is blocked and they must be

released. This is easily accomplished by rotation of the fetal body  $45^\circ$  to  $90^\circ$  as traction is applied. This will "wing" the anterior scapula. The ipsilateral arm is then swept across the chest and extracted. A similar rotation of the fetal body in the other direction follows with extraction of the remaining arm. A loop of cord is then gently pulled down to accompany the fetal body. A modified Mauriceau-Smellie-Viet maneuver follows because cranial extension is common. In this procedure, the surgeon's hand is passed into the uterus and across the fetal face. Pressure downward on the maxilla usually with a finger in the mouth is combined with a slight upward displacement of the fetal body to flex the head. To complete the delivery, a Credé-like maneuver over the lower uterine segment is performed with one hand while the surgeon maintains the flexion of the fetal head with the intravaginal hand. The head is then extracted from the uterus with an upward rotational motion.

For a transverse lie, if the head cannot easily be extracted, a breech extraction is often required. In this setting, a vertical or classic incision can be best but, a wide transverse incision might well suffice in many cases. Especially if the membranes are ruptured, the anesthesiologist should be requested to relax the uterus by administering an intravenous bolus of nitroglycerine, or a betamimetic such as terbutaline, or a halogenated inhalational anesthetic agent. Because nitroglycerine is potent, rapid in onset, and has a brief half-life, it is the authors' preference.

When an apparent fixed transverse lie is present and the membranes are intact, the surgeon should initially attempt to convert the presentation to longitudinal to bring a pole down into the lower uterine segment; however, this is not always possible or even prudent. If the lie proves immovable, the attempt should be made to incise the myometrium in layers, keeping the membranes intact. Of course, this is not always possible, or the membranes might have been previously ruptured. Once the myometrial incision is made, a method of extraction is decided upon. Often this will be a complete breech extraction. If so, the fetal feet are located and drawn down into the wound. The membranes are then ruptured (if not already so) and the extraction performed as previously described. At times, the fetal head will be immediately present and either a forceps or a vacuum extractor can be applied after membrane rupture, avoiding the extraction. In our experience,

the method of delivery is often decided only at the moment of uterine entry since shifts in fetal position or inadvertent membrane rupture or tardy administration of the uterotonic might have occurred. The important point is that the surgeon needs to be able to perform a number of delivery techniques when faced with a fixed transverse lie. The actual procedure that is performed is tailored to the immediate clinical findings and circumstances at the time of surgery. Obviously, flexibility is required because decisions need to be made promptly and the appropriate delivery procedure performed with celerity.

When perioperative tocolysis is thought to be a distinct possibility, the anesthesiologist should be informed *before* the start of the surgery so that the nitroglycerine can be located and premixed in advance. If the nitroglycerin bolus is requested as the vesicouterine fold is incised, tocolysis will become effective at just in time to facilitate fetal extraction. In the absence of serious internal disorders, complications from the intravenous bolusing of dilute solutions of nitroglycerine (150  $\mu\text{g}$ –350  $\mu\text{g}$ ) to otherwise normal and stable women are insignificant. The drug's half-life is only 30 to 50 seconds. Since the introduction of nitroglycerine, the authors have rarely performed classic incisions even for fixed transverse lies with limited fluid, because sufficient uterine relaxation is provided by one or more nitroglycerine boluses to permit an atraumatic and uncomplicated extraction. (See Difficult Cranial Delivery).

If the initial uterine incision inadvertently severs either (or both) uterine arteries, attempting to control the hemorrhage while the baby remains in utero is not the best choice. When the infant remains inside, the exposure is poor and access to the vessels is limited. Blind clamping in a restricted space is always an undesirable technique, risking injury to adjacent structures. Clamps placed in the corner of the uterine wound are also easily dislodged during the fetal extraction, possibly extending the original tear. The best management is simply to first expedite the delivery of the infant and then control the bleeding by clamping or direct vessel ligation, *after* the fetus has been extracted.

### Difficult Cranial Delivery

For the outwardly routine case involving a fetus in a cephalic presentation, although the usual method of cranial delivery at a cesarean is simple manual

extraction, this procedure is not always easy. A narrow lower uterine segment, a deeply engaged and molded head, or some unusual combination of factors can lead to difficulty in extracting the presenting part. There are several alternatives if the usual efforts at manual removal fail or prove difficult. When the problem is with delivery of the infant's head, it is usually because the fetal cranium is deeply engaged and molded into the birth canal, the original incision is too small or the head is positioned as an occiput posterior. What is needed is a methodical approach to this surgical difficulty.

The appropriate way to begin is to first evaluate the incision. Many difficult extractions result from an inadequate initial incision. This is a common error, and if the pelvis is deep, the abdominal wall thick, or the fetus large, the problem is compounded. The incision must be approximately 10 cm or greater at the site where the fetal head is extracted. Especially in an obese woman, this can require a substantially larger incision in the upper tissue planes to permit adequate room. The uterine incision is easily extended laterally to the vessels and upward in a "smile" on one or both sides, as necessary. If the rectus muscles interfere, a partial Maylard incision can be performed with division of one or both muscle bodies either by use of a scalpel or, preferably, by electrocautery. Alternatively, a Cherney-type dissection of the attachment of the muscle body from its attachment to the symphysis can be conducted. This latter procedure is uncommonly attempted, due to the combination of poor exposure and the desire for speed.

Once an adequate incision has been made, the clinical situation needs consideration. Is uterine tone increased and has the amniotic fluid escaped? If so, nitroglycerine tocolysis can facilitate the delivery by reducing uterine tone, making the extraction less difficult. Here is where thinking ahead is a great boon. If the possibility of difficulty was foreseen, the anesthesiologist can have drawn up a nitroglycerine dose and then simply inject it on request when the extraction problem becomes manifest.

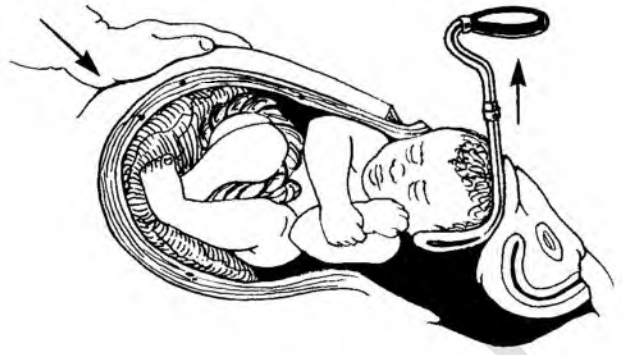
The next step is for the surgeon to introduce his or her hand into the uterus through the myometrial wound and position it below and in front of the fetal head. The technique outlined by Cho [438] is worth review. This paper outlines methods similar to those favored by the authors. Cho describes a method of approach he terms *ERR*, involving the basic elements of cranial *elevation*, *rotation*, and *reduction*. In

this technique, the surgeon's hand is kept straight with the wrist angle neutral as it is introduced in advance of the fetal head. Cho emphasizes the importance of maintaining the hand straight at the wrist such that the hand can be used as a scoop to both grasp and elevate the fetal head. Once the hand has been advanced along the calvarium sufficiently deep to break the suction, the fetal head is elevated and rotated to occiput anterior. An interesting refinement of this technique involves the fingertip reduction of the lower portion of the myometrial incision *below the head* prior to the attempting the upward extraction of the fetal head. Cho claims that this approach reduces the risk of an extension. Regardless of how the head is extracted, fundal pressure should not be exerted until the operator's fingers have both freed the head and positioned it properly to guide it upward and out of the pelvis.

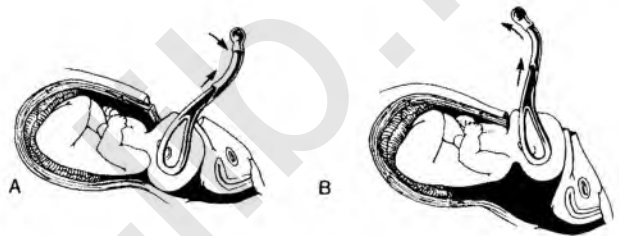
In the case of difficulty, once it is apparent that the cranial extraction of a deeply engaged head is not easy, the surgeon should cease efforts from above and request help. A gloved assistant then performs an immediate vaginal examination, pressing the fetal head upward from below (through the pelvic curve) as the surgeon's hand is progressively advanced downward until the cranium can be cupped and drawn into the uterine wound for extraction (Figure 18.10) [246,247]. This "passing-it-up" technique is suggested for routine use if there has been an unsuccessful vaginal trial of instrumental delivery or whenever labor has been prolonged and the fetal head is heavily molded and deeply engaged. In the authors' experience, manual vaginal



**FIGURE 18.10.**  
*Cesarean delivery: extraction of an impacted fetal head. An assistant elevates the fetal head from below during a vaginal examination. Fundal pressure assists delivery once the fetal head is secured.*



**FIGURE 18.11.**  
*Cesarean delivery: fetal cranial extraction using a Murless cranial extractor (vectis blade extractor).*



**FIGURE 18.12.**  
*A and B. Cesarean delivery: fetal cranial extraction with forceps for an infant in a deflexed occiput posterior position. Note the cranial flexion extraction.*

displacement is usually the easiest and the least traumatic procedure to assist the delivery of a deeply engaged head. The assistant can more easily judge the required angle of force from below than the surgeon can from above. Other alternatives for difficult delivery include delivery of the fetal body by upward traction or, once the head has been released, applying a classic or specially modified forceps, a vacuum extractor, or using a vectis blade such as the Murless (Figures 18.11 and 18.12) [246–249].

When faced with deep cranial entrapment, the surgeon must rely on uterine relaxation, upward cranial displacement by an assistant, delivery of the fetal body with secondary upward traction, or a unique combination of these techniques to release the infant's head and permit its upward extraction. If the fetal head is low and the surgeon's hand cannot be advanced to elevate it, no instrument can be applied. In this setting, in our experience, first breaking the suction between the fetal head and the pelvic tissues by the combination of upward pressure provided by a gloved assistant from below plus

the efforts of the surgeon from above is the critical step for an atraumatic extraction. Once the head is freed, an instrument can be applied and could help avoid an extension of the myometrial incision because in these cases the tissues are usually markedly friable and edematous, and the fetal head often heavily molded and distorted.

If instruments are used during a cesarean operation, clinicians must remember the physics of cranial displacement. The fetal head must remain flexed and be withdrawn slowly using limited force. If the fetal head is oblique or posterior it should be rotated to an occiput anterior position. This is usually performed manually without great fuss once the suction is released and the head extracted from deep in the pelvis. In unusual circumstances an instrument such as a vacuum extractor might be necessary to assist this process. The initial occiput anterior positioning of the head permits both an easier and more accurate application of forceps or a vectus blade and critically retains cranial flexion, reducing the risk of either an extension or a fetal injury.

The particular advantages of the forceps or the vacuum extractor at cesarean delivery are in the circumstance of an unengaged or high fetal head, and not for the deeply engaged presenting part. When the fetal head is high, it can usually be promptly and securely grasped by an instrument and easily directed toward and through the uterine wound, expediting the delivery. With an unengaged head, especially if the membranes have ruptured and labor has commenced, abdominal instrumental delivery is often easier than either version and extraction or forced fundal pressure with manual efforts at cranial delivery. Because the need for an instrumental extraction cannot invariably be predicted, the authors routinely include both a short classic forceps and a Murless blade in their cesarean delivery instruments. A vacuum extractor is also kept ready in the operating suite. These instruments are for the unusual and unpredictable situations when abdominal or combined abdominopelvic delivery manipulations fail or prove difficult.

### Placental Delivery and Myometrial Wound Closure

After delivery of the infant's body, the cord is then doubly clamped, and the child's mouth/pharynx is cleared with a suction bulb or a DeLee trap as

needed, following institutional protocol. The infant is then passed from the table to other birth attendants. The edges of the uterine incision are next promptly identified and grasped by Allis, Allis-Adair, or other atraumatic clamps, to restrict bleeding. For this purpose, the authors prefer a Pendington clamp, because its triangular wide blade grasps a substantial amount of tissue and controls hemorrhage without crushing the myometrium. A dilute solution of oxytocin is then rapidly infused to assist in firming the uterus (20 IU–40 IU in 1 liter of an isotonic salt solution). Uterine massage is often performed as well. The placenta is then permitted to separate and deliver spontaneously. The usual cord blood samples can be obtained in the interim while the placental delivery is awaited. Immediate manual placental removal is not recommended. If the placenta is manually removed prior to its spontaneous separation, the result is increased blood loss and an increased incidence of postpartum endometritis. Manual removal should not routinely be performed unless partial separation with immediate hemorrhage occurs or other clinical events require that the process be accelerated [250,251]. If the uterus is so poor at contracting that it cannot expel the placenta through the hysterotomy wound, atony is profound. In this setting, prompt therapy with massage, oxytocin, an ergot preparation [251,252], or a prostaglandin is indicated.

Once the placenta delivers and the uterus has firmed, a brief intrauterine exploration is then performed at the surgeon's discretion to ascertain that no secundines remain. The inner aspect of the myometrium should be visualized directly, and the adherent clot or membrane gently removed. In cases in which the membranes were intact at the time of surgery or if cervical dilatation was minimal, some clinicians pass an instrument, such as a ring forceps, from the uterine cavity down into the vagina through the cervix to ensure postoperative drainage. There are no data to show whether this procedure is helpful, and the authors' use of this technique in cases of initially intact membranes is cheerfully inconsistent.

Attention is next directed to the closure of the uterine wound. For uterine repair, exteriorization of the uterus is electively performed if the exposure is better. This procedure does not increase febrile morbidity, but maternal nausea is more common when externalization has been performed [253,254,452].

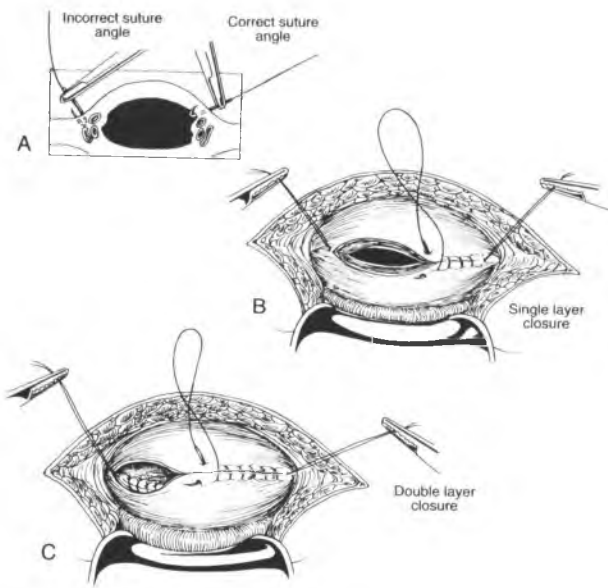


FIGURE 18.13.

*A–C. Cesarean delivery: standard two-layer myometrial closure. Note correct placement of angle sutures to avoid puncture of the uterine vessels.*

On review of these data, the evidence for clinically significant benefit or harm to this procedure is at best unconvincing. The surgeon should ask the simplest question. Does exteriorization improve visualization or access to the wound? If so, it should be performed; if not, the uterus is best left in situ and repaired in that position.

If access to the sidewalls is tight or exposure is otherwise limited, the authors place a figure-of-eight suture in each corner of the uterine wound before beginning the myometrial closure. This suture must be carefully inserted to avoid puncturing the lateral vessels (Figure 18.13). This technique permits easy ligation of lateral vessels if they have been injured. When a vessel laceration has not occurred, these sutures close the corners and help to avoid blind lateral suturing under conditions of limited exposure. Once angle sutures have been placed bilaterally and tied, the suture ends are grasped with a Kelly clamp and elevated. Lateral tension on the suture ends easily rotates and elevates the uterus, improves visualization, and facilitates the reapproximation of the myometrium. The uterus is then closed initially with a simple running suture in a single layer. The authors' preference is a polyglycolic acid or polyglactin suture for the closure. When this

suture line is complete, a second, embricating layer can be added, if required or desired.

### Uterine Closure

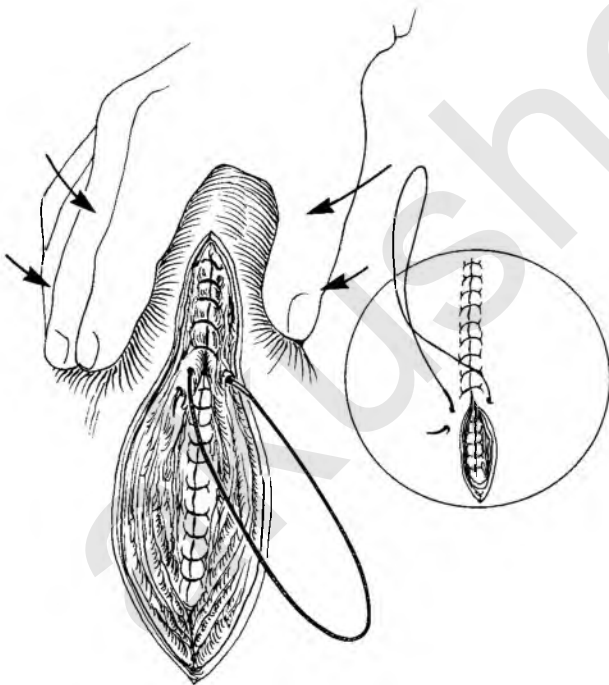
There is controversy about using the classic imbricating two-layer running-locking closure versus a single-layer closure for the myometrium at cesarean delivery [255,327,444–449]. There are as well no available data concerning the best suture material for use at cesarean delivery [266]. For many years we have preferred polyglactin suture at cesarean owing to its lack of tissue reactivity and favorable handling characteristics, strength, and persistence, and we have performed double-layer closures. Even in our own community, there is wide variation in practice, however. While double-layered closures are the norm, chromic suture is still popular. Some surgeons even routinely combine polyglactin sutures for one myometrial layer but use chromic for the second. This is yet another feature of standard practice that awaits systematic study.

Two recently published observational follow-up studies have evaluated the long-term outcome of single- versus double-layer uterine closure [448, 327]. Interestingly, they reached opposite conclusions. Bujol and coworkers [448] reported an increased risk of uterine rupture in subsequent gestations when the original closure was single layered. A later study by Durwald and Mercer [327] found no increase in risk for subsequent rupture when the original uterine closure included only a single layer, however. None of the other available reviews or studies of this subject has indicated major outcome differences between single-versus double-layer suturing of the myometrium [255,445–447,449]. Randomized trials with better controls such as those now underway will be required to properly evaluate this important issue.

There are technical concerns with a single-layer closure. Adequate hemostasis is more often a problem. Additional singleton or figure-of-eight sutures to the closure line are often required for control. Nonetheless, there are potential benefits to the single-layer closure in terms of operative time and blood loss. (This subject is discussed in greater detail in the section on alternative techniques.) Regardless of the number of layers in the repair, some

counsel against incorporation of the decidua in the closure of the myometrium, thinking that this results in a weaker scar because of poor healing or endometriosis [256]. The evidence for this is not compelling, however, and either a full-thickness or partial-thickness closure is acceptable [233]. The authors' usual practice is full-thickness suturing of the myometrium using a simple running or running/locking suture.

If the myometrial incision is made relatively high, or if the patient has not been in labor, the transverse incision might have been placed close to the retraction ring between the upper and lower uterine segments. This presents the surgeon with a thick upper segment but a considerably thinner lower segment. In this setting, wound reapproximation usually requires a two-layer closure. An upper segment or classic incision also requires a layered closure. During the reapproximation of a classic uterine incision, a large-caliber suture material (e.g., No. 1) is ordinarily employed to reduce the risk of tearing. Either running or interrupted sutures can be used as required (Figure 18.14).



**FIGURE 18.14.**  
*Layered reapproximation of a classic cesarean delivery wound. The surgeon's hand compresses the myometrium to ease closure. Running suture technique is depicted.*

### Auto Stapler

There is a unique uterine stapling device available for cesarean deliveries. The U.S. Surgical Corporation (Norwalk, CT) Auto Suture Poly CS-57 is a hand-activated device that both incises and staples the myometrium in one action. The instrument is preloaded with dissolving copolymer staples made of polylactic and polyglycolic acid. After a small midline incision is made in the uterus, the instrument is inserted into the wound, positioned, and then fired. The device drives two parallel lines of absorbable sutures into the myometrium while severing the muscle between them. When this is done bilaterally, a wide V-shaped incision is produced. The device has been claimed to reduce operating time, blood loss, and the potential for lacerations [257–261].

Unfortunately, analysis of controlled trials of this autostapler versus traditional uterine closure involving 526 cases reported in the Cochrane Review [262] fails to show any improvement in febrile morbidity, endometritis, or length of hospitalization when the uterine stapling devices are used. This expensive single-use instrument has no advantage over conventional methods of uterine closure, except perhaps during specialized fetal surgery procedures. When the fetus is exteriorized during fetal procedures, the line of staples inserted by the device controls bleeding while the myometrial wound is open. (See Chapter 20, Fetal Surgery.) This device is not recommended for routine obstetric use [262–264].

### Abdominal-Pelvic Exploration

If no major bleeding sites persist after the myometrial closure, the surgeon should next focus on the adnexa. As is required for this examination, the uterus is either delivered into the wound, if it was not already externalized, or simply rotated to permit direct visualization of the fallopian tubes and the ovaries. If tubal ligation is desired, it is performed at this time. If pedunculated hydatids are present, they can be electively removed.

The examination of the adnexa is mandatory, except in cases that either require extreme speed or involve multiple adhesions that bar easy access. In 40 years of practice, the senior author has identified two cases of early ovarian cancer and many benign

tumors by such routine examination. While finding a tumor is uncommon, establishing the diagnosis of ovarian cancer at the earliest possible and asymptomatic stage during a cesarean procedure has the potential to be life saving. Adnexal examination is therefore a step that should not be omitted.

If the cecum is easily accessible, the appendix is electively delivered and examined for the presence of liths, masses, or other pathology. If access to the appendix is not immediate or easy, its exposure should not be attempted.

The myometrial wound closure and the vesicouterine fold are then carefully reexamined for bleeding sites. If any are located, they are suture ligated or electrocauterized. If chorioamnionitis was not a clinical diagnosis preoperatively, and if the myometrial suture line is dry, the vesicouterine fold is not routinely reapproximated. Otherwise, the fold can be electively closed with a simple running 3–0 polyglycolic suture.

Assuming no problems are identified, the pelvic organs are replaced in the anatomic position, and the abdomen is electively lavaged with 1 to 2 liters of warm saline to remove clotted and free blood or vernix. Some clinicians prefer to avoid lavage and simply proceed immediately to closure of the abdomen.

For many years, obstetricians have routinely irrigated the pelvis and abdomen with 500 ml to 1000 ml of warm saline before closure of the abdomen. This procedure was based on two beliefs: first, a possible reduction in maternal morbidity, and second, a technique for the detection of small bleeders. There are good data that routine lavage does not reduce postoperative maternal morbidity when prophylactic antibiotics have been administered [265]. In routine cases, because lavage does no harm and has the advantage of helping to identify bleeding sites on the uterus or vesicouterine fold, the authors prefer to retain peritoneal irrigation as part of postuterine abdominal wall closure. This approach includes examination of the adnexal structures, a review of the adequacy of hemostasis, and attention to the instrument and sponge counts.

If recurrent ooze from the vesicouterine fold, a myometrial tear, or the original myometrial incision site persists despite compression, electrocautery, or resuturing, drainage must be considered. Subfascial drainage should also be considered if a Maylard incision was performed or if electrocautery was

employed to divide the muscles and persisting ooze is present. Only a soft vacuum drain of the Jackson-Pratt type should be inserted. Nonsuction-type drains are not advised. Drainage through the original skin incision is imprudent and should not be performed. For low transverse myometrial incisions, the drain should be positioned in the retroperitoneum behind the vesicouterine fold and passed out of the abdominal cavity through the extraperitoneal space, exiting the skin through a puncture wound separate from the abdominal incision. The drainage tube is subsequently sutured to the patient's skin. Once the bladder flap drain is in place, the vesicouterine fold is closed over it with a fine running polyglycolic suture. If intraperitoneal hemorrhage is also considered a risk, a suction drain can also be positioned in the cul-de-sac, again exiting the skin by means of a separate puncture wound. An intraperitoneal drain is for detecting continuous bleeding that necessitates reexploration. In pregnant postoperative patients, however, an intraperitoneal drainage is productive of a copious serosanguinous drainage that can be difficult to interpret. This type of drainage is quite uncommonly necessary and these drains are not recommended. The authors review the output of the drains closely in the first several postoperative hours. Drains should be removed once their output falls or becomes serous and always within 24 to 48 hours, unless the site drained is an abscess cavity.

The placement of any drain is controversial, but in selected situations of uncertain hemostasis, drainage can signal the need for reexploration. Drains also can reduce the risk of hematoma formation.

To complete the case, the bowel is placed behind the uterus, the omentum in front. Closure of the abdominal wall is then performed. The parietal peritoneum is not usually suture reapproximated unless continued bowel intrusion into the operative field occurs, or in the now-rare situation when a modified Smead-Jones closure is performed. Extrapolation from general surgical experience indicates that closing of the peritoneum does not improve the strength of the abdominal wound and actually can increase the rate of complications. Peritoneal closure remains controversial, however. (This issue is discussed in detail later.) If the rectus muscle was severed (*Maylard technique*), its ragged edges are either left free or electively sutured to the overlying fascia or, if the tendon was incised (*Cherney technique*), it

is resutured to its original pubic insertion or to the fascia overlying the bone, whichever is more convenient.

In the rare case when it is indicated to drain the plane of the muscle bodies, the peritoneum is first closed with a running suture. A Jackson-Pratt suction drain (or similar type) is then placed in the center of the wound and exited through the fascia and skin by a separate stab wound. The fascia should be carefully closed over the drain, with care not to incorporate it in the suture line.

There are few reliable data about techniques for appropriate fascial closure at a cesarean [266]; however, clinical investigations are likely to provide additional information within the next few years. Current major research studies focusing on basic surgical issues in cesarean technique include the CAESAR trial currently in progress in England [267] and the CORONIS trial being conducted in Argentina, Ghana, India, Kenya, Pakistan, and Sudan [439]. The following suggestions for closing fascia are derived from the authors' surgical experience and extrapolations from extant studies.

Closure of fascia requires attention to detail (Figure 18.15). The authors often initially suture ligate both corners of the fascia with separately placed figure-of-eight sutures. This isolates the corners, which can be difficult to access in some women, while providing a convenient site for traction. There are several acceptable techniques for fascial closure. The authors' preference is a simple running doubled looped suture of No. 1 polydioxanone that is tied at one end when the closure is complete. An acceptable alternative is to use two simple running sutures of 0 polyglycolic acid or polyglactin suture material beginning at each corner, then crossing at the midline. The sutures are tied separately. To better position the sutures to avoid dog-ears, a single simple stitch is occasionally placed in the midline to align the fascia prior to initiating the closure. As the layer is closed, fascial bites should include at least 1.5 cm of tissue, with the bites approximately 1 cm apart. The closure should be tensioned to be snug but not tight, and the surgeon should avoid damaging the running suture with either the forceps or the needle holder. Particularly when a looped suture is employed, the final knot should be secured with multiple square knots.

For a vertical incision, the closure is similar. The fascia is reapproximated using a looped suture of

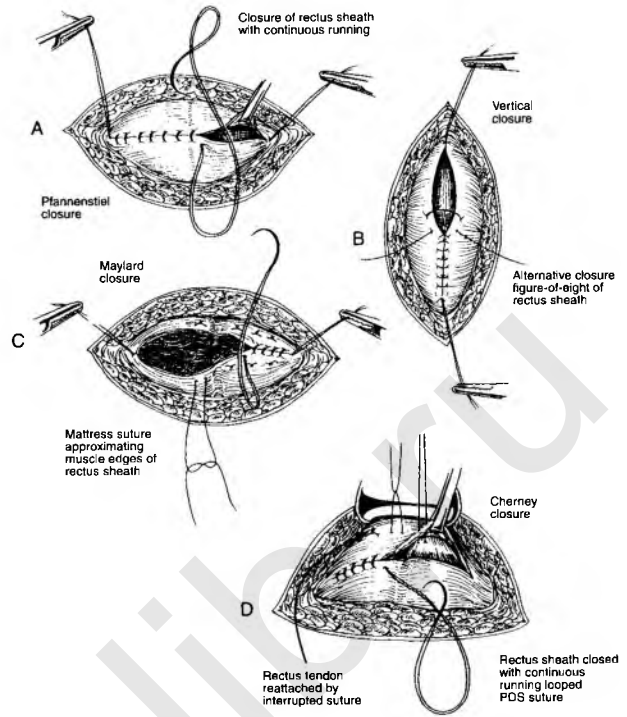


FIGURE 18.15. Details of fascia closure: Pfannenstiel (A), vertical (B), Maylard (C), and Cherney (D) operations.

No. 1 polydioxanone, run as a simple stitch and tied at one end. In obese patients or in cases with the potential for infection, a modified *Smead-Jones closure* is occasionally performed. In this procedure, the fascia is reapproximated with No. 1 polydioxanone, or alternatively, a nonabsorbable suture such as 0 or No. 1 nylon, using a far-far, near-near technique, incorporating the peritoneum and muscle in generous fascial bites. In recent years the authors have replaced the traditional interrupted *Smead-Jones* technique with the use of a looped absorbable suture as their standard bulk closure, employing a heavy, nonpermanent suture with good success. This modification of the original technique is faster and easier to perform. In grossly contaminated cases, after the fascia is closed, the wound is lavaged, and monofilament mattress sutures are inserted into the skin but not tied. This wound is then packed with sterile gauze. In these cases the skin is left open, and a delayed closure is planned.

Whether to reapproximate the fat and thin subcutaneous connective tissues (Camper's fascia) is one among those issues for which the



recommendation has continuously changed over the years. Most surgeons were originally trained to reapproximate this layer with interrupted sutures of fine plain gut or similar suture material. The stated reasons for closure were to reduce dead space, presumably avoiding the collection of blood or serum that might interfere with wound healing and promote infection. An additional reason given was to reduce the tension on the skin edge, thereby facilitating healing with a more cosmetic scar. Whether these traditional beliefs were correct has been the subject of various learned opinions over the years. (See Special Issues.) The current recommendation is to close the subcutaneous tissue if the layer is thick. The addition of routine drains when subcutaneous tissues are reapproximated has been shown in randomized trial not to reduce wound-related complications [268–270]. Such drainage is not recommended.

Once the fascia is closed, attention is directed to the subcutaneous tissues. Adherent clots are gently lavaged away, and the wound is closely explored to detect any sites of bleeding or ooze. Those noted are electrocoagulated or tied with small-diameter free ties of nonpermanent suture material. Electrocautery close to the skin edge is purposely avoided.

Usually the skin is next reapproximated with stainless steel staples, using a disposable stapling device. The staples are placed at a right angle to the incision and are inserted to reapproximate the skin without gaping. In applying the staples, the skin edges are grasped with fine-toothed forceps, and slightly everted and elevated to assist the closure. If the wound is large and the supporting tissues flaccid, the authors usually place the first staple in the center of the wound. Subsequently, additional clips are placed midway between this central staple and the end of the wound. Once this is complete, the authors return to one corner of the wound and complete the usual staple closure. Placing the midline and halfway staples helps to ensure a symmetric closure and avoids an unsightly dog-ear that occurs if the upper and lower portions of the incision are not evenly matched. Electively, a subcuticular skin closure, using either a fine absorbable or nylon suture, can also be performed for a transverse incision, at the surgeon's convenience. When a nonabsorbable suture is used, care is taken to place the stitches to avoid locking. This closure is occasionally reinforced with adhesive strips, as necessary, to achieve

a good skin reapproximation. A subcuticular closure is best avoided in potentially infected cases. Many surgeons prefer the subcuticular closure because its result is cosmetically appealing, and patients might complain of less postoperative pain [271]. Good data about the best skin closure techniques are lacking, however [272]. Surgeons should use techniques that seem to have the best results in their clinical experience. Once the skin closure is complete, a dressing is applied, completing the operation.

## SPECIAL ISSUES

### **Closure of Subcutaneous Layers**

Whether routinely to close the subcutaneous fat and associated fascia (*Camper's fascia*) as a separate part of closure of the cesarean wound is an abdominal surgery detail that has received varied recommendations. Data derived from a recent meta-analysis of six studies that included 875 patients favors closure of Camper's fascia if the depth of the subcutaneous fat exceeds 2 cm [273]. These studies also document that closure significantly reduces both the formation of wound seromas and spontaneous wound disruption. The best suture material for subcutaneous closure is not established. Traditionally, 3–0 plain was used. Currently, the authors' favor a fine (3–0, 4–0) interrupted or running polyglycolic acid suture. In the authors' experience it is easier to achieve even approximation of the tissues with the use of interrupted sutures than with a running stitch. The specific technique for closure is purely optional, however. Because there are no clinical trials to guide management, either for surgical technique (interrupted vs. running) or choice of suture material, surgeons should employ the method(s) that have produced the best results in their experience.

### **Peritoneal Closure**

Whether routinely to close either the visceral or the parietal peritoneum after a cesarean is subject to debate [274–282]. Surgeons trained in the past were instructed to close the peritoneum routinely whenever it had been incised [281]. The technique of closure was normally a simple running suture, and the suture material employed varied with the operator. Either fine (3–0 or 4–0) polyglycolic or gut suture (plain or chromic) were the most popular. The traditional reasons for peritoneal closure were

several, including maintaining the peritoneal barrier against infection (visceral peritoneum), reducing the likelihood of wound dehiscence, the restoration of normal anatomy, and the minimization of adhesion formation [282]. Additional reasons for closure included technical issues; for example, when the bowel continuously protruded between the rectus muscles and into the wound during the closure of the abdominal wall, or as part of a specific abdominal wall closure technique, such as the Smead-Jones. In addition, some surgeons favored closure simply for aesthetic appearance.

Nonclosure of the peritoneum was introduced into obstetric practice by extension from the experience of gynecologic and general surgeons. In favor of nonclosure, it was argued that because of the rapidity with which the peritoneum is spontaneously reconstituted, closure was simply unnecessary. Avoiding peritoneal closure was also reported to shorten surgical time and reduce short-term postoperative morbidity. Finally, data were either contradictory or nonexistent supporting the traditional reasons given for peritoneal reapproximation. In sum, no compelling data supported peritoneal closure, whereas several clinical and laboratory studies found evidence in favor of peritoneal nonclosure.

As noted, several benefits are claimed for nonperitoneal closure, including reduced adhesion formation, shorter operative times, reduced short-term maternal morbidity, and a more rapid return to normal bowel function [277,279,280,283,453]. Although there appear to be short-term benefits to nonclosure, postoperative or follow-up studies evaluating the long-term outcome of closure/nonclosure of the peritoneum are few, none are controlled, and most involve either small numbers or have other methodologic problems. It does not appear that this question has been definitively answered.

As an example of the extant literature, Bamigboye and Hofmeyr conducted a meta-analysis of nine trials of peritoneal closure versus nonclosure at cesarean delivery involving a total of 184 women [276]. They reported that there was a significantly reduced operative time (mean 7.33 minutes), less febrile morbidity, and reduced hospital stays when peritoneal closure was *not* performed. They also stated that the requirements for analgesia and data on the incidence of wound infection tended to favor nonclosure but were not significant. In this review, the collective data concerning the compli-

cation of endometritis varied, and the only follow-up study available reported no differences in measured outcome. The power of this latter study was interpreted as low, rendering the results uncertain. Another study by Weerawetwat and coworkers had findings that were quite similar [284]. Specifically, they noted no difference in postoperative complications, including adhesion formation, whether the parietal, visceral, or both peritoneal surfaces were closed or not.

The central issue of peritoneal closure is that of adhesions. Adhesion formation is promoted by ischemia, infection, and inflammation, rather than the simple existence of an open intraabdominal surface.

The basic physiology of peritoneal repair is pertinent in this closure/nonclosure debate. Experimental and observational data indicate that healing of the peritoneum differs from that of other tissues. First, healing is notably rapid. Animal models indicate that peritoneal injuries normally heal without scarring within five days. Furthermore, direct reapproximation of the incised edges of the peritoneum is not required for closure. Following peritoneal trauma, injury occurs to the underlying microvasculature. The subsequent healing process involves a pattern of sequential and specific cellular infiltration, followed by the rapid growth by local mesothelial cells. As spontaneous repair begins, metaplasia of the subperitoneal perivascular connective tissue occurs.

Within 48 hours of injury, these initial mesothelial cells are replaced by macrophages. Early in the process, polymorphonuclear cells are noted, and fibrin strands also develop at the injury site. By the third day, mesothelial cells, which arise from multiple sites along the peritoneal wound, begin to cover the macrophage sheet. After the fourth day, these mesothelial cells predominate. As the process continues, the fibrinolytic system is activated, and the fibrin strands originally laid over the wound are progressively lysed. The growing islands of mesothelial cells from the multiple repair sites then become confluent, closing the defect. Peritoneal fluid, which contains leukocytes, eosinophils, basophils, and macrophages, is an important actor in the normal physiology of the peritoneum and peritoneal healing. Various cellular mediators produced by mesothelial cells and these peritoneal fluid macrophages are thought to have an active but not yet entirely clear role in the healing of peritoneal

injuries [285]. Problems do occur in this complex healing process of the peritoneum. If the process of fibrinolysis is inhibited by one of several processes, the original fibrin strands covering a peritoneal defect can persist. Infiltration by fibroblasts and neovascularization follow, and an adhesion forms. Surgical peritoneal injury by cautery; excessive tissue manipulation, crushing, or other injury; or reapproximation by sutures or repair of defects by grafts apparently increases local tissue ischemia. This results in local tissue reaction and predisposes to adhesion formation [286]. The presence of blood in the peritoneal cavity apparently does not necessarily predispose to adhesion formation unless tissue ischemia is also present. In the healing process, the reactivity of various suture materials in promoting adhesion formation is also not clearly understood. Nonetheless, the consensus is that highly reactive materials such as surgical gut are less desirable than newer, synthetic, slowly resolving, and less reactive polyglycolic sutures for use within the peritoneal cavity.

As adhesions arise from sites of peritoneum injury, meticulous attention to basic surgical techniques is thought to reduce the risk of adhesion formation. This approach includes close attention to homeostasis, avoiding the use of highly reactive suture materials, minimalization of tissue manipulation, prophylaxis against infection in potentially contaminated cases, and perhaps avoidance of electrocautery on exposed peritoneal surfaces. Whether closing the peritoneum as the abdominal cavity is exited increases or reduces adhesion formation *specifically during pregnancy* is controversial and is reviewed later.

Intraabdominal adhesions from prior surgery are an important source of morbidity and increase the long-term healthcare costs of surgery [283]. Approximately 40% or more of bowel obstructions in industrialized nations are due to adhesions from prior surgery, and most involve the small bowel [287]. Operations potentially leading to adhesion formation and subsequent bowel complications include various common obstetric and gynecologic procedures, appendectomies, and primary bowel surgery [275,281,288]. In obstetric practice, adhesion formation can follow cesarean delivery and is reported usually to become progressively more severe as the total number of surgical deliveries increases [289]. Not surprisingly, the more exten-

sive the adhesions, the more a cesarean is delayed, and the greater the potential for adverse effects on the fetus.

The combined financial burden and morbidity associated with surgical adhesions focuses attention on the physiology of adhesion formation, the identification of high-risk procedures or techniques leading to adhesions, and the development of methods of prevention.

A potential method for adhesion prevention is the inhibition of cellular mediators involved in inflammation. After abdominal surgery and peritoneal injury, various inflammatory cytokines such as interleukin (IL-1, IL-6) and tissue necrosis factor are released into the abdominal cavity. These substances are probably involved in the process of adhesion formation [290]. Eventually, it might be possible to develop specific drug therapies to block their adverse effects on adhesion formation.

Physical agents or techniques to block or deter adhesion formation in recognized high-risk situations have been successfully developed and introduced into clinical practice in some areas of surgery. The most frequently used methods of adhesion avoidance include refinements in surgical technique, treatment with antibiotics, and the local application of various types of isolating films. Blocking films are intended to separate injured peritoneal surfaces from other intraabdominal structures during the first several days of peritoneal healing, and thus in theory block the process of adhesion formation. The most popular of these mechanical barriers are polytetrafluoroethylene (PTFE, Gore-Tex), hyaluronic acid with methylcellulose (Seprafilm), and oxidized regenerated cellulose (Interceed). The efficacy of all of these agents has to some degree been confirmed [291–294]; however, these films are rarely employed during cesarean delivery.

### Peritoneal Closure Issues in Obstetrics

Several claims have been made for avoiding routine peritoneal closure with cesareans. These arguments and observations concern postoperative pain, speed of the operator, and the degree of adhesion formation. Several studies have evaluated pain reduction when the peritoneum is not closed [295,296]. In terms of outcome of peritoneal closure, Roset and coworkers conducted a questionnaire study and followed up 144 of 280 women who had originally

been part of a peritoneal closure trial performed seven years before [297]. They were able to locate 69 women originally matched to nonclosure and 75 matched to closure and reported similar outcomes in both groups in terms of fertility, urinary symptoms, and abdominal pain. In the 29 total subjects who had gone on to subsequent surgery, the incidence of adhesion formation was statistically insignificant, although the numbers in each group were small. In addition, the selection of women in this study was nonrandom. Collectively, these limitations substantially restrict the importance of these data.

Tulandi and coworkers reported a series that included 333 infertility laparatomies [277]. In 120 of these 333, subsequent laparoscopies were performed, and the degree of adhesion formation was directly observed and recorded. All patients had originally received a 32% solution of dextran intraabdominally prior to peritoneal closure. There were no significant differences between the groups in adhesion formation (22% closure group vs. 15% nonclosure group). This study, like the one before, has distinct limitations, however. The peritoneal closure technique was not randomized, these women were not pregnant, and the assignment to closure or nonclosure groups depended solely on a change in surgical technique that was introduced during the year of the investigation when the original laparoscopies were performed. Thus, in this study there was no true randomization.

Meyers and coworkers conducted a retrospective record review of women undergoing repeat cesareans over an 18-month period [274]. In 191 total cases, they identified 58 instances (40%) in which intraabdominal adhesions were recorded in the operative note. In terms of technique, adhesions were recorded in 1 in 17 closure cases, but in 17 of 40 of the nonclosure cases, a difference that was found to be significant ( $p = 0.003$ ). This study suffers from being retrospective and nonrandomized, and it also depended on accurate and complete surgical operative reports again, involving only small numbers.

The recent papers by Lyell [275] and Myers [274] report fewer intraoperative abdominal and pelvic adhesions when closure of the parietal peritoneum *was* routinely performed at cesarean delivery. Furthermore, Lyell's group noted no differences of significance in surgical time, regardless of closure or nonclosure.

In the investigation, Lyell prospectively studied 173 cases of first repeat cesarean deliveries [275]. In the original surgery, 106 women in the series had the peritoneum left open, whereas 67 had a peritoneal closure. On the reopening of the peritoneum at the first repeat operation, fewer dense and filmy adhesions were noted in the closure group as opposed to the nonclosure group (52% vs. 73%), a significant ( $p = 0.0006$ ) finding. There were also significantly fewer total adhesions (30% vs. 45%,  $p = 0.043$ ) observed in the group with prior parietal peritoneum closure.

Given the limitations and inconsistencies in the available data, what should be recommended? Current information is contradictory in terms of whether peritoneal closure is consequential in long-term patient outcome. Not closing the peritoneum probably shortens operative time, but this improvement is clinically insignificant and is not reported by all observers. The issue of a difference in postoperative pain is complicated by different operative techniques and the administration of varying types of anesthesia and analgesia in the reported series. The data about reduced postoperative febrile morbidity and improved bowel function when the peritoneum has not been closed are statistically significant. These factors probably have some, albeit limited, clinical consequence; however, the overarching and major issue is that of adhesion formation. It is quite clear that adhesions encountered during surgery increase the risk for unintended injury to abdominal organs, predispose to other complications, and increase operating times. Although serious long-term problems are uncommon, they are important in a small but significant number of patients. Intraabdominal adhesions can result in serious complications apart from pregnancy, such as partial or complete bowel obstruction.

Unfortunately, long-term data involving sufficient numbers of properly randomized cases of peritoneal closure versus nonclosure in women undergoing cesareans are simply not available. Nonetheless, the extant data are interesting and hint that aspects of the physiology of peritoneal healing could differ in pregnant women.

There are at least theoretical reasons why the process of adhesion formation might differ between cesarean deliveries and other gynecologic or surgical operations performed when the patient is not gravid [275]. Some features of pregnancy favor adhesion

formation, whereas others do not. In contrast to routine surgery, both blood and amniotic fluid routinely contaminate the peritoneal cavity at cesarean operations. Much of this fluid is not removed by either suction or lavage prior to closure. Amniotic fluid has potent fibrinolytic properties, which might alter the peritoneal healing process. Furthermore, different from many nonobstetric types of intraabdominal surgery, most cesarean operations are bacterially contaminated to a varying degree by fluids from the intrauterine cavity. This is especially true when the cesarean occurs while the mother is in labor.

Better-designed prospective studies are needed to confirm or refute the contention that routine peritoneal closure carries benefits for women undergoing cesarean delivery. Available data, although thought provoking, are far from definitive. Changes in current practices should not be made based on these data, unless they are confirmed by subsequent study.

The authors' current approach to peritoneal closure is perhaps as arbitrary as traditional teachings. We have in general abandoned *routine* reapproximation of the *visceral* peritoneum; however, there are circumstances in which closure of the visceral peritoneum is indicated. In the unusual instance of persisting ooze from beneath the vesicouterine fold, which might follow the repair of a laceration or in any case when insertion of a Jackson-Pratt drain is deemed necessary, then close the peritoneum over it. Given the controversy in the literature and the limited data, the authors consider routine *parietal* peritoneal closure optional. We do not routinely reapproximate this layer unless repeated protrusion of the bowel interferes with closing the abdominal wall or the rare Smead-Jones closure is performed. Bowel protrusion is an uncommon problem except when the initial incision was large and midline, or anesthesia is limited or rapidly waning. In those instances in which the peritoneum is closed, we employ small-diameter polyglycolic suture material, minimize tissue handling, and avoid a tight closure to minimize tissue injury.

### **Documentation**

The senior surgeon is required to document the operative procedure in the medical record. A detailed, dictated note about the clinical circum-

stances facing the surgeon at the commencement of the procedure, why the choice for surgery was made, and the pertinent events of the operation are necessary. In this dictation, it is much more important for the surgeon to include a careful description of the patient's anatomy, the type of uterine incision, and any complications than to list the type or size of suture used in the various stages of the operation. If presumed fetal jeopardy or fetal distress was the original indication for the procedure, an umbilical arterial and venous blood gas should be obtained, and the placenta and cord submitted for histologic examination.

### **Complications**

Cesarean delivery has many potential complications. Among the most common are *excessive blood loss* and *postoperative febrile morbidity* from several causes, including endometriosis, urinary tract infection, and pneumonia [185,298–303]. The likelihood for postoperative readmission to the hospital is substantially increased in cesarean deliveries, as opposed to spontaneous vaginal deliveries [304,305]. Immediate postoperative complication rates are greater for clinic patients, for longer procedures, and for operations involving extensive blood loss or those requiring additional surgery. Surgical complications are also related to emergency procedures, when there was labor prior to surgery, when there was a history of prior surgery, adhesions are present, and when VBAC trials are attempted, among other factors [299,306,307]. Extensions of the original myometrial incision laterally into the uterine arteries or downward into the cervix are common misadventures. Other injuries, including damage to bladder, ureter, or bowel, are possible but much less likely [246,306]. Potential long-term complications include adhesion formation, wound disruptions and abnormal placentation in subsequent pregnancies [443].

The components of proper management for a surgical complication have been unchanged for many years. These steps include prompt identification of the injury, control of bleeding, accurate anatomic repair, and subsequent patient notification. Minor incisional extensions, inadvertent laceration of the uterine vessels, and minor bladder injuries are generally easy to repair and present no challenge to the clinician. Prompt and uncomplicated healing can be

confidently anticipated after these events. Extensive injuries to the ureter, bowel, and bladder are another matter. Such injuries, often complicated, should be repaired only by the experienced surgeon.

### Wound Disruption

A fairly common complication of cesarean delivery is disruption of the surgical wound from infection, hematoma, or seroma. The estimated incidence of cesarean delivery wound infection varies from 3% to 15%, with an additional 3% to 14% complicated by seroma or hematoma. The principal management options for a disrupted wound are a delayed wound reclosure or permitting spontaneous closure with secondary healing by intention [308]. When wound disruption is diagnosed, several steps are appropriate. The wound should be carefully probed to ensure the continuity of the fascia. Residual pockets of blood, serum, or pus can be released by finger dissection. The wound is then carefully lavaged using normal saline, or, in a widely exposed wound, normal saline mixed with a dilute solution of hydrogen peroxide. Extensive lavage with concentrated hydrogen peroxide is potentially dangerous and should not be performed. Once the wound is clean, it is loosely packed with sterile gauze and a loose dressing applied to contain seepage. This process is repeated two or more times daily, with removal of the gauze resulting in débridement of the wound to some degree. Once the wound is clean and free from necrotic debris, wound reclosure can be considered after 4 or more days. As the process of lavage and packing proceeds if substantial amounts of necrotic material are noted, surgical débridement is needed, perhaps aided by the use of enzyme preparations. Granulation tissue observed at the wound base heralds the onset of closure. Some surgeons, although advocates of secondary closure, have preferred to wait until the wound appears clean and has developed a good granulating base before attempting resuturing. Although this is the usual technique, there are good data indicating that many wounds can be successfully closed secondarily early in the process, shortening the recovery time markedly. A number of hospitals now have special wound services that can apply special techniques for accelerating closure of contaminated or otherwise problematic wounds. In difficult or atypical cases, this expertise should be sought.

In a recent review, Wechter and coworkers conducted a literature search, and located a series of eight studies of secondary wound closure [308]. Four of these involved 124 women with cesarean or gynecologic surgery wounds. In three of these studies, antibiotics were routinely administered during the healing process. Most secondary closure procedures involved full-thickness suturing of the skin using a monofilament suture material. Alternatively, some wounds were closed with superficial mattress sutures or with a permeable adhesive tape. Overall, healing in surgically reapproximated wounds was significantly faster than those left to heal by secondary intention. The observed difference was more than 6 weeks. Perhaps not surprisingly, successful healing was independent of antibiotic use. Regardless of the technique employed, the successful reclosure rate was above 85%.

From these and other data, it appears that secondary wound closure at an interval of 4 to 6 days after the original disruption has a high success rate with minimal complications. Closure with nonabsorbable monofilament nylon sutures, which are subsequently removed, is the most popular technique and the one that the authors prefer. It is not clear if the administration of antibiotics either speeds the process of wound healing or increases the likelihood of success. Of interest, in the Wechter review, either early wound reclosure at day four after wound disruption or delayed closure eight or more days after the original disruption following the development of granulation tissue was equally successful.

Thus, once the wound is free from necrotic material and is not clinically infected, reclosure after the fourth day should be seriously considered. If additional débridement is required, reclosure can be delayed by several more days. The likelihood of success with reclosure is high and the risk for complications low. The actual method of wound reapproximation does not seem to be an important factor in success.

Rarely, in probing a disrupted wound, or in the presence of a characteristic history, a clinician might suspect dehiscence of the fascia. The usual clinical history provided is that of the sensation of sudden wound or abdominal pressure, sometimes accompanied by the sensation of a "pop." A characteristic copious tan-colored watery discharge follows. Patients with a dehiscence must return to the

**TABLE 18.12 Surgical Techniques to Reduce the Risk of Perioperative Fetal Laceration**


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Initial palpation of the myometrium
Preincision elevation of the uterine wall by surgical clamps
Use of good light, exposure, and active suctioning during myometrial entry
Uterine scoring; use of bandage scissors or a blunt finger dissection for entry
Use of specialized instruments

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operating suite for wound exploration and resuturing. At surgery, devitalized tissues are debrided and closure of the fascia by a modified Smead-Jones approach, a traditional interrupted closure with retention sutures or a running closure with a double, looped suture should be performed. Broad-spectrum antibiotics should also be administered. The skin is usually not closed but left open, packed with sterile gauze, and secondarily closed as described previously in 4 to 6 days. Fortunately, wound dehiscence is now a *rara avis* in obstetric practice. (See Chapter 16, Surgery in Pregnancy.)

### Fetal Injury

Although uncommon, fetal trauma does occur during cesarean delivery [309–313]. Cesareans can reduce the incidence of birth injuries, but the procedure does not prevent all of them and itself has certain fetal risks. Thus, close attention to the well-established mechanics of safe delivery is as important during a cesarean as it is in a vaginal delivery (Table 18.12). Most of these operative injuries are to soft tissue (bruising/ecchymosis) and are of trivial clinical consequence. Much less frequently, other injuries, such as skin lacerations; long-bone fractures, dislocations, spinal, or visceral trauma; and brachial plexus injuries are encountered. In terms of the rare brachial plexus injury occurring in infants delivered by cesareans, some represent true in-utero injuries. The others likely result from traumatic extractions during surgery. Regardless of cause, brachial plexus injuries noted after a cesarean, although certainly possible, remain at best uncommon.

Inadvertent fetal lacerations at cesarean delivery are a persistent problem [309–313]. Although most of these injuries are of minimal clinical consequence, they cause the mother and the family

unnecessary distress and are largely avoidable. In reviews including more than 6,000 cesarean deliveries, the reported incidence varies from 0.55% to 3.12%. Smith reported a large series of cesareans performed for standard obstetric indications when neonatal records could be matched to the mother's chart [310]. A total of 17 lacerations were identified in 896 cesarean deliveries, or 1.9%. Notably, only 1 of the 17 injuries was recorded in the maternal record! In the authors' experience as well, the failure of obstetric surgeons to record fetal injuries is distressingly common. In the Smith article, when the laceration site was recorded for cephalic-presenting infants, two thirds of lacerations were to the fetal head and neck, whereas 10% were on the back. In contrast, when the infant was in a breech or transverse position, the lacerations were principally to the back, buttocks, or lower extremities.

Simple attention to standard surgical techniques avoids most of these injuries. In all instances, care must be taken to operate under direct visualization and incise only the myometrium. Adequate exposure and proper lighting are obvious additional prerequisites. The desire for speed must never trump safety when the uterus is entered. The greatest risk of fetal injuries occurs when the myometrium is very thin, partially replaced by scar tissue, or paradoxically very thick, as in patients operated on before the onset of labor. In both of these instances, it is difficult to detect when the amnion has been reached.

Before the uterine incision is made, the primary surgeon should palpate the myometrium to judge its thickness. In routine cases, the authors employ gentle, recurrent scoring of the myometrium in the midline until the membranes bulge into the wound. If the myometrium is thick, as the incision progresses, the edges are progressively elevated with Allis clamps applied at two adjacent points with progressive bites; the clamps evert the myometrium as the incision is made. If the myometrium is very thin, the authors often employ two Allis clamps applied several centimeters apart. Once the myometrium is grasped, it is elevated away from the fetus similar to the method used to tent and incise the peritoneum. The myometrium is then simply incised between the clamps, thus avoiding the fetus. If the lower segment is tightly drawn over the presenting part, this technique might be technically impossible. Following the entry into the uterine cavity with bulging of the membranes into the wound or membrane

rupture, either a bandage scissors or the surgeon's fingers can be used to widen the wound laterally.

### Alternative Techniques

The continuous effort to reduce morbidity and shorten operative time for cesarean procedures has led to several modifications of the standard Pfannenstiel surgical technique classically taught to obstetric surgeons. Nonetheless, it is fair to say that there are wide differences in standard operative practices between surgeons and institutions [267,439]. As reviewed previously, the standard cesarean technique consists of a low transverse skin incision, followed by sharp separation of subcutaneous tissues, fascia, rectus muscles, and the peritoneum, and sharp entry into the uterus. The surgeon can usually extend the entry wound with bandage scissors. Thereafter, the uterus is closed in two layers, and the partial and visceral peritoneums are reapproximated. The fascia is then closed with interrupted or running sutures. Staples or a subcutaneous stitch are used to close the skin. Closure of the subcutaneous fatty layer, externalization of the uterus for repair, and manual removal of the placenta are considered discretionary.

The principal alternative to the standard cesarean is the *Misgav Ladach (ML) cesarean technique*, itself a derivation of the Joel-Cohen method [314–319]. This surgical approach forgoes sharp tissue layer separation, favoring instead blunt or finger dissection of the fascia, subcutaneous tissues, rectus muscles, uterus, and peritoneum. The abdominal incision is straight transverse and placed slightly higher than that of a classic Pfannenstiel incision. The uterus is closed in a single layer. The fascia is closed by a continuous suture, and in the original procedure, the skin is reapproximated by widely spaced silk sutures, with the intervening areas subjected to 5 minutes of clamping until adherence. Finally, neither the visceral nor the parietal peritoneums are reapproximated. Several clinical trials document that the ML technique reduces operative time and blood loss, consumes less suture material, and could reduce wound morbidity and consumption of analgesics, as well as shortening convalescence [315–319]. However, the surgical techniques as used in varying studies are inconsistent, and not all reports have concurred in finding advantages to the Joel-Cohen type of approach [315].

Elements of the ML technique have been progressively but selectively adopted by many surgeons, but the extent to which the entire surgical program is employed is unknown. The most popular components of the technique have proved to be the blunt dissection of the subcutaneous space, peritoneum, and uterus, and nonclosure of the peritoneum. There is good evidence that blunt expansion of the uterine wound reduces blood loss and does not increase the risk for disruption of the lateral vessels [320,321]. The most controversial elements of the ML technique are the original method for uterine closure and closure/nonclosure of the peritoneum. The ML method of skin closure has not proved popular, at least in the authors' area of practice. Use of a running suture for fascial reapproximation seems to be near universal, however. Among techniques for uterine closure, the double embricating technique is by far the most common, possibly because it seems to control bleeding better than a single-layer closure and hides the original incision. There are controversial data showing that single-layer closures are the equal, of double-layered closures and can be faster, however. Furthermore, the short-term morbidity of one or two layered closures is arguably effectively equal, although it is fair to say that there is no consensus on this issue [255,322–325].

On review, the claimed advantages to the ML technique are of mixed value. The observed reductions in blood loss following ML operations are statistically significant but are also clinically inconsequential. The mean operative time is shortened by 7 to 14 minutes. This, with the apparently smoother convalescence, does have some clinical importance. Whether these apparent benefits are sustained in the prospective trials now underway will be interesting to see. Beyond the potential for postoperative adhesions, whether the peritoneum is closed, the principal outstanding controversy is the possible increased risk of rupture inherent in a single-layer uterine closure [326,327]. As previous noted, these and other questions about technique are being addressed in the large CAESAR study currently underway in England [267] and the international CORONIS trial [439].

### Reduction of Morbidity

The mortal risk from cesarean delivery is now extremely low (1/1,600 procedures or fewer). The



oft-quoted Boston Study reported no maternal mortalities in 10,231 cases of cesarean delivery [328]. Thus, in modern practice, it is the morbidity and not the mortality of cesarean operations that is the issue. Important components of morbidity include febrile morbidity and wound infection. The latter complicates approximately 2% to 15% of cesarean deliveries.

*Prophylactic antibiotics* are always indicated when a cesarean follows labor or with previously ruptured membranes. In recent years, this treatment has been extended to all cases, both elective and nonelective [298,329,330]. A single 1-g to 2-g intravenous dose of a first-generation cephalosporin or amoxicillin/ampicillin administered after cord clamping significantly reduces maternal febrile morbidity. Other antibiotics should be substituted in the setting of known allergy. Andrews and coworkers suggest that the addition of antibiotic coverage for *Ureaplasma urealyticum* (doxycycline and azithromycin) to the usual cephalosporin further reduces postcesarean morbidity, specifically wound infection and time of hospitalization [331]. Single-dose regimens are generally as effective as multidose protocols. Broad-spectrum or second-generation antibiotics are no more effective than first-generation drugs and are not recommended as prophylactic agents. Antibiotics in peritoneal irrigation solutions are no more effective than those given intravenously to the mother at surgery. Prophylactic treatment does not necessarily prevent certain rare, serious infectious complications of cesarean surgery, such as abscess formation. The observed reductions in infection rate are primarily due to decreases in the incidence of endometritis and wound infections. A lesser effect is a reduction in the incidence of urinary tract infections.

In women developing fever during labor, the authors favor prompt treatment with standard therapeutic doses of antibiotics, unless cesarean delivery is to be performed promptly (within approximately 1 hour), in which case the initial dose of the drug is administered after cord clamping. Best practice when the mother becomes febrile during labor, however, is not yet firmly established.

Cesarean delivery includes a series of common perioperative problems, some of which have been previously discussed. Anesthesia issues are discussed in detail in Chapter 9, Obstetric Anesthesia, and interested readers are referred to this source for

additional information. Additional comments on surgical technique are included in Chapter 16, Surgery in Pregnancy.

### Other Comments

Perioperatively, most difficulties that arise are due to maternal obesity, an inadequate initial incision, or the technique of fetal delivery. Problems with surgical exposure are usually due either to maternal obesity or to an inadequate incision. It is in the surgeon's and patient's best interest to make at least a 15-cm skin incision and extend it as required for adequate exposure. The rule for fetal monitoring is simple. Monitoring should be performed with the same level of intensity in the operating suite as in the labor suite. Internal electrode techniques are best, but for low-risk cases, intermittent Doppler heart rate checks are acceptable. In selected cases, if serious complications such as placenta accreta, increta, or percreta are possible, a real-time ultrasound scan can be preferred in the surgical suite immediately before opening the abdomen. This can allow the surgeon to place the uterine incision to avoid the bulk of the placenta. In the case of twins, scanning permits reverification of lie, an important consideration for delivery of either a second twin, or, in cases of higher multiples, the second and subsequent infants.

If there is an anterior wall low placental insertion, the usual myometrial incision often enters the placenta. If this occurs, the best technique is to promptly and bluntly dissect the placenta laterally until the membranes are located and can be entered. An alternative is to continue rapid, sharp dissection directly through the substance of the placenta. The cost of the latter technique is a degree of fetal bleeding, which can be marked unless the delivery is prompt. In the haste to reach the amniotic cavity, the surgeon must take care not to cut the fetus inadvertently during this procedure. Thus, if the placenta is incised, it is prudent to inform the pediatrician, because either neonatal anemia or acute hypovolemia can result.

## CESAREAN HYSTERECTOMY

### Indications

Although some cesarean hysterectomies are planned, most occur as emergencies. The most common obstetric problems leading to hysterectomy

are postdelivery atony and hemorrhage. Hemorrhage can arise from many sources, such as placenta accreta/increta/percreta, uterine rupture, or laceration [220,332–337]. A history of prior cesarean delivery, placenta previa, multiple gestation, fetal macrosomia (>4,500 g), failure of the uterus to respond to an ergot preparation or to oxytocin, prolonged or augmented labor, chorioamnionitis, and high parity are important risk factors for atony and for related postpartum hemorrhage [220,333,338,339]. Infrequently, hysterectomy is indicated for severe infection or for unusual problems such as massive leiomyomata that precludes uterine closure. Rarely, cesarean hysterectomy is performed for sterilization. In view of the enhanced morbidity of the operation, such procedures are discouraged unless other significant pathology or special circumstances are present.

### Procedure

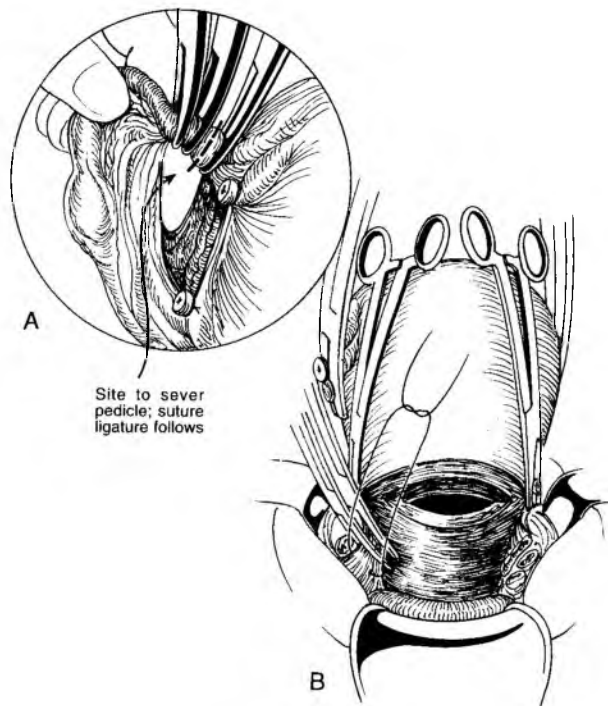
If a hysterectomy has been decided on, or when hysterectomy is deemed likely, it is best to perform a midline vertical skin incision. This incision permits easy extension of the wound if extra space is required. In the more usual case, when the need for hysterectomy occurs after the patient has already been opened by a low transverse skin incision, exposure is usually still adequate if the original incision was generous. If the surgeon requires additional room, the original transverse incision should be immediately extended by laterally incising the “smile” symmetrically on both sides, as necessary. As previously described, the rectus muscles can either be divided in the midline or preferably incised at their pubic insertion and reflected if additional room is needed. Rarely, the original fascial and skin incision are converted to a T. This practice is discouraged except in the most dire of circumstances owing to the inherent weakness of this incision during healing and the poor cosmetic result. Except in unusual circumstances, a T incision is not required if the surgeon simply extends the original incision and has adequate assistance for retraction.

Because cesarean hysterectomies are usually emergencies, time is at a premium. The vascular pedicle cases are large and usually edematous. Important structures, including the bladder and ureters, are all too close to the planes of dissection. Thus, all simple aids for a rapid but safe oper-

ation should be used. *The operator's desire for speed must never trump the safe and methodical approach to the surgery, however.* If the problem forcing the hysterectomy is uterine atony, an unanticipated laceration, or a uterine rupture, prompt control of hemorrhage can be critical. A few moments are all that is required to directly ligate both uterine arteries or apply clamps across these vessels. Such steps immediately restrict the blood loss, permitting time for other procedures and evaluations. Alternatively, a rubber catheter can be passed around the uterus at the level of the endocervix, drawn tight, and temporarily held with a clamp. In case of uterine rupture or a cesarean, the bleeding edges of the tear or wound can be grasped directly with clamps (e.g., Pennington or ring forceps) to staunch localized heavy flow. Too many clamps restrict access and observation; therefore, progressive ligation of bleeding sites or ligation of major feeding vessels, as possible, is always best. Rarely, an isolated arterial vessel will be identified bleeding actively into the operative field. For this, either immediate digital pressure or judicious clamping followed by a prompt ligature is indicated. Obviously, blind clamping and large tissue bites with a suture must be avoided to lessen the risk to adjacent structures, especially the all-too-close ureter.

The hysterectomy is begun at the uterine fundus. The adnexa are identified and elevated by an atraumatic clamp such as a Babcock. The round ligaments and the fallopian tube are then grasped bilaterally with long Kelly's (or similar) clamps at their insertion to the uterus. The clamps are applied with their handles oriented medially. The clamps restrict back bleeding and their application provides a useful handhold for the assistant to apply traction. Active tensioning of the uterus is an important detail in the subsequent surgical operation. With the large uterus in tension and deviated to one side or the other, the various pedicles “fall away” once they are suture ligated/divided, easing the procedure and increasing its safety.

The uteroovarian ligament and the fallopian tube are next doubly clamped with Heaney-Ballentine hysterectomy clamps or similar instruments (Figure 18.16). If necessary, a third clamp can be used to control back bleeding. As the operation continues, double clamping of all major vascular pedicles is strongly suggested. A window is then developed in the broad ligament with blunt and sharp dissection.



**FIGURE 18.16.**

*Technique of cesarean hysterectomy (A). Note the double clamping of major vascular pedicles (B). These pedicles are initially tied, then suture ligated. Deeper tissues are doubly suture ligated. See text for details.*

The adnexa are divided from the uterus with Mayo scissors. The ovarian pedicles are then secured by a tie, followed by a transfixing suture ligature. The securing clamp is not removed until this second suture/ligature has been securely placed, and the surgeon is prepared to set the initial knot. A similar procedure is performed on the other side. This technique isolates the major vascular bundles from the uterus while reliably preventing hemorrhage from the pedicle. The round ligament can either be incorporated into this initial adnexal suture or ligated separately. The authors generally prefer the latter technique. Absorbable large-caliber suture material is best for these ligatures, to prevent cutting through these often-edematous tissues. Classically, No. 1 chromic has been used for this operation, but a polyglycolic acid (or similar maternal) suture of No. 1 or 0 size will also suffice. Again, this is a situation in which the type of suture material is of trivial importance compared with the surgical technique.

The broad ligament is next skeletonized by sharp dissection. The anterior peritoneum of the vesicouterine fold is then incised and dissected from the uterus. The broad ligament is then progressively

separated from the uterus by serial dual clamping followed by double-suture ligation of the isolated pedicles. A clamp to control back bleeding from the uterus is sometimes required. This procedure is progressively followed down both sides of the uterus, taking as many bites as necessary until the uterine arteries have been divided and sutured and the dissection approaches the level of the endocervix.

Once the level of the cardinal ligaments is reached, they are suture ligated. At this point or after control of the uterine arteries, the uterus is best simply excised supracervically and passed from the table. Once the dissection has proceeded to this level, the procedure can be terminated if the bleeding is controlled. In many instances, this is the time to stop, thus avoiding the problem of removal of the cervix and possible injury to its adjacent structures.

Much of the serious morbidity associated with cesarean hysterectomy occurs when the surgeon continues the operation to remove the cervix. Removal of the cervix involves acceptance of a risk of several potentially serious complications. The most problematic of these are cuff hemorrhage and ureteric injury. The ureter is perilously close to the uterine sidewall, and the edematous surrounding tissues mitigate against its easy identification. The cuff is floppy and usually well vascularized. If postoperative bleeding occurs, a site on the vaginal cuff is often the culprit.

If removal of the cervix has been decided, the remaining cervical stump is grasped with tenaculum or similar instrument as a traction aid. Since the major arterial supply has been ligated by this time, back bleeding is usually minimal. Also, once the fundus and bulk of the lower uterine segment are extirpated, the surgical exposure to the remaining cervix is much improved.

If the patient has been in labor, the lower uterine segment is normally both flaccid and dilated. Locating the cervix by external palpation under these circumstances can be difficult. Our technique for identification of the cervix is to grasp the lower uterine segment in the midline anteriorly with one or more Allis clamps and incise it transversely for 2 cm to 3 cm (Figure 18.17). Bleeding is usually minimal. The surgeon then introduces his/her finger into this wound. Usually the cervical edge can then be easily palpated. This technique guides the surgeon in determining the level of the dissection that is required, limiting the possibility of extending the dissection unnecessarily far into the vagina.

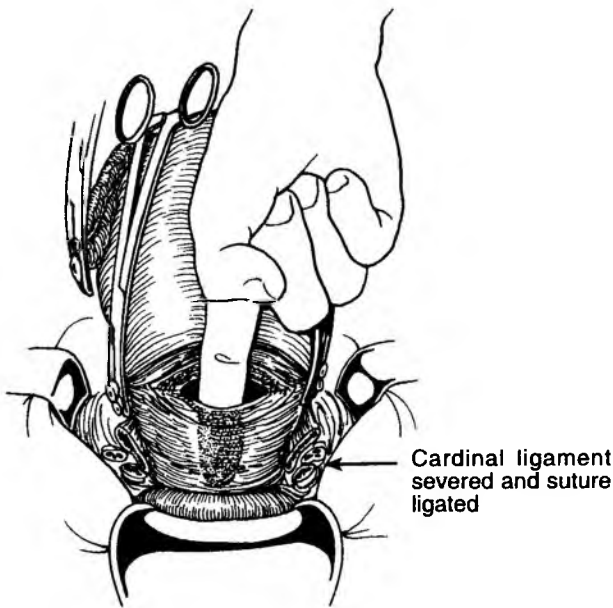


FIGURE 18.17.  
*Cesarean hysterectomy. The surgeon palpates for the cervical edge through an incision in the lower uterine segment; this helps to gauge the extent of dissection.*

When direct vessel ligations are apparently ineffectual in hemorrhage control, remember that another supplying vessel is always present that is likely the cause of the bleeding. In the pelvis, all of the vascular supply for the major structures have a number of supplying vessels of varying caliber. Also, because arteries have no valves, immediate retrograde flow in the arterial system is possible whenever a specific vessel is ligated. Thus, if a hemorrhage occurs from a high cervical or vaginal wall laceration, ligation of the uterine arteries or even hysterectomy might not control the bleeding. A separate vessel arising from the hypogastric artery can be the primary feeding artery. If so, only direct oversewing of this vessel, ligating the hypogastric to control other feeding vessels, or direct embolization will control the bleeding.

To excise the cervical stump, simple or double Kocher clamps should be applied at the lateral edges of the cervix, progressively isolating the cardinal and uterosacral ligaments, which are doubly suture ligated. Once the dissection has reached the level of the exocervix, further advance downward is halted. The cervix is removed by incising the vaginal tissue circumferentially, grasping the cut edge of the vagina with Kocher or similar straight clamps. The authors prefer to run the cervical cuff with a continuous locking suture and leave it open. Electively,

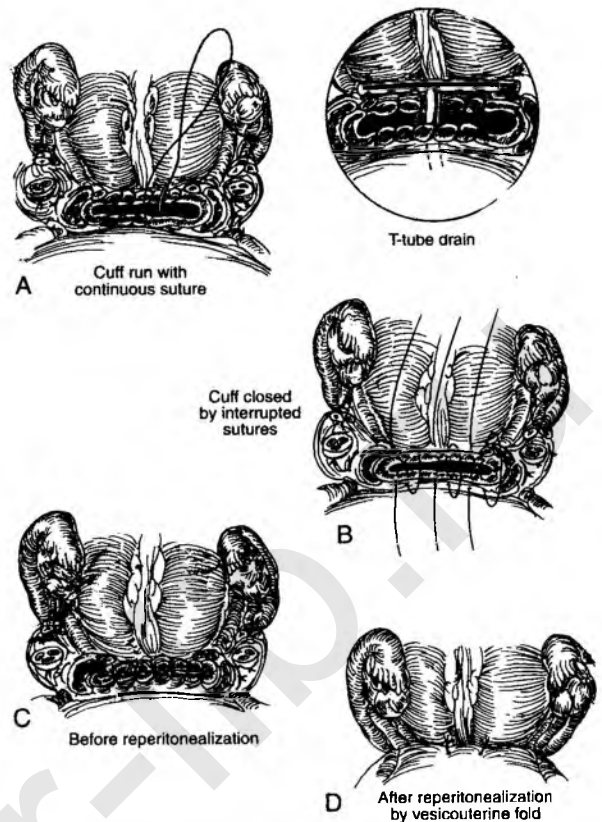


FIGURE 18.18.  
*A-D. Cesarean hysterectomy. Use of a running suture on the vaginal cuff, an alternative technique for complete cuff closure, and a method for reperitonealization are depicted. See text for details.*

the cuff can also be closed (Figure 18.18). Close attention to hemostasis is required, because often bothersome bleeding arises from the cuff or the adjacent pedicles. Simple or figure-of-eight sutures are placed as required to achieve hemostasis. Because of the oft-emergent nature of these procedures and the presence of many large and edematous pedicles, the authors favor routine drainage, inserting a hysterectomy T-tube drain through the cuff and down the vagina. The T portion of the tube is then lightly tacked to the midposterior segment of the cuff with a simple stitch of 3-0 plain suture material to prevent dislodging it during the rest of the procedure.

To complete the closure, the round ligaments are drawn down and sutured to the edges of the vaginal cuff; this part of the technique is elective. To ensure complete hemostasis, lavage of the abdomen and close inspection of all pedicles follow. The vesicouterine fold is then drawn over the cuff and simply

sutured to the peritoneum of the midposterior vaginal wall, avoiding any potential involvement with the laterally situated ureters. This places the open cuff, the major pedicles, and the T-tube drain in the retroperitoneal space. The vaginal drain is usually removed at 48 hours or once full ambulation begins. Prior to closure, reinspection of the operative field confirms hemostasis. The abdomen is then closed in the usual manner. Broad-spectrum antibiotics are administered in full therapeutic doses; this treatment is not required in all cases, however. Minimally, standard single-agent, single-dose antibiotic prophylaxis, which is routine for cesarean delivery, is necessary.

### **Complications**

Cesarean hysterectomy can be a life-saving operation. As noted, the operation is usually performed for hemorrhage, and it can be preceded by a rapid bilateral O'Leary-type uterine artery ligation to restrict blood loss as surgery is begun. The major surgical complications of a cesarean hysterectomy include ureteric or bladder injury, fistula formation, hemorrhage/anemia/transfusion, febrile morbidity, hematoma formation, and wound infection [337,340–342]. Not surprisingly, unscheduled hysterectomy procedures are more likely to have complications, require a longer surgical time, or require blood transfusion [337]. Some of the complications from cesarean hysterectomy are avoidable, especially if they arise from perioperative haste or, paradoxically, from preoperative uncertainty and delay.

During cesarean hysterectomy, a meticulous surgical technique must be balanced with the demand for speed. Especially in emergencies, the surgeon must proceed methodically but relentlessly. Exposure is usually easy and the surgery straightforward unless hematomas or adhesions from prior surgery are present. The greatest difficulty is the decision to proceed to surgery, not in the actual performance of the operation.

When delays occur, most of the lost time occurs before beginning the operation, as clinicians either debate treatment options or vainly hope for improvement while the clinical situation deteriorates. When there is real uncertainty, the accoucheur's best option can be to immediately seek another opinion. The second practitioner, new to the clinical scene, is less influenced by prior events and

can often be more objective. What must be avoided is "paralysis by analysis." Requesting another opinion delays any intervention and is not an excuse for failing to act if the situation is clearly worsening, potentially threatening maternal survival. At one extreme is the cautious practitioner, wary of an unnecessary procedure and wishing for a clear sign to proceed. At the other is the aggressive, surgically oriented physician, pressing for immediate exploration. The former surgeon might permit events to go too far for patient safety. In contrast, the latter physician permits him- or herself to go too far and too fast, risking an unnecessary surgery. Both approaches result in unnecessary morbidity, and best practice lies between these extremes.

In the setting of unanticipated cesarean hysterectomy, communication and explanation to patient and family are critical. Because the pregnant woman might be unable to comprehend explanations in the immediate postoperative state, the family should be closely counseled about the clinical problems, what occurred perioperatively, and the postoperative complications experienced or likely to be encountered. Morbidity is high. In 2003, Baskett reviewed a series of emergency hysterectomies drawn from 110,537 deliveries over the interval 1980 to 2001 reported the need for transfusion in 84.4% and postoperative intensive care in 26.6% [336]. Needless to state, a full discussion with the patient once she is fully able to comprehend is mandatory, as is an extensive note in the medical record.

### **Operations for Hemorrhage**

Whenever severe and acute postpartum blood loss occurs, expert team assistance is required to ensure the best outcome. Initial management for postpartum hemorrhage consists of several virtually simultaneous actions: prompt recruitment of assistants, installation of large-bore intravenous catheters, placement of a Foley catheter, and prompt clinical evaluation for cause and definitive treatment [343,346,352–355,436,437]. For recent reviews of the medical and surgical approaches to hemorrhage, readers are referred to the recent and excellent text by B-Lynch and coworkers [436].

Viewed in isolation, the incidence of hemorrhage at cesarean delivery is approximately 10% [344]. The principal associated risk factors include those

**TABLE 18.13 Immediate Postpartum Hemorrhage: Possible Etiologies**


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Uterine atony
Genital tract lacerations: vaginal, cervical
Secundines
Abnormal placental adherence
Uterine rupture
Uterine inversion
Coagulopathy:
• Hereditary
• Acquired:
• Dead fetus syndrome
• Preclampsia
• Abruptio placentae
• Amniotic fluid embolism

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associated with general anesthesia, chorioamnionitis, preeclampsia, and a prolonged active phase. Postpartum hemorrhage is due principally to atony, secundines, and undiagnosed lacerations. Less frequently the cause is coagulopathy. The incidence of atony can be modified by general obstetric management, specifically active management of the third stage of labor, including routine use of uterotonics, controlled cord traction, and uterine massage postplacental delivery [345]. The principal causes for a coagulopathy are abruptio placentae or amniotic fluid embolism. Additional causes are noted on Table 18.13. These possibilities for postpartum hemorrhage should be rapidly considered and either sustained or rejected, because proper therapy hinges on a prompt diagnosis.

Postpartum hemorrhage is best considered as a clinical sign. The hemorrhage immediately identifies an acute obstetric emergency that has a variety of causes that vary both in pathophysiology and definitive treatment. As the maternal treatment of the hemorrhage commences, so does the search for a cause to arrest the original process [346].

The usual method of immediate maternal resuscitation is the rapid administration of balanced salt solutions and once available, blood products [347]. The situation can prove serious, and surgical exploration for vessel ligation or compression suturing, uterine packing, passage of compressing intrauterine balloons, administration of potent uterotonics, vessel embolization, or hysterectomy might be required for control [348,436].

The use of an antishock garment to compress the lower body and abdomen has also been shown to be helpful in maternal resuscitation. This device is suggested for use especially in developing countries where surgical and blood bank facilities are often not immediately available [349,350].

The principal surgical procedures for controlling obstetric hemorrhage consist of hypogastric or uterine artery ligation, and various techniques for myometrial compression suturing [347,351–353,436]. Other possible procedures include vessel embolization, uterine gauze packing, and the use of an intracavity balloon. The obstetric surgeon must approach the problem of postpartum or perioperative hemorrhage with flexibility, using the techniques best suited to the specifics of the given case. As Baskett [353,354] and others [351,355] point out, knowledge of several methods for hemorrhage control are prudent, because in difficult circumstances, the success of any specific technique is never guaranteed.

#### Uterine Artery Ligation (O'Leary Technique)

*Uterine artery ligation* is a rapid and often successful technique for controlling obstetric hemorrhage from uterine atony or lacerations. The technique was originally described by Waters in 1952 and popularized by O'Leary in the 1960s [356–360]. The operation is potentially less successful for high vaginal or cervical lacerations, because the feeding vessel might not arise from the uterine arteries. In these special circumstances, embolization, direct ligation, or hypogastric ligation can be required for control.

In the O'Leary technique, a suture of No. 1 chromic or polyglycolic acid/polyglactin is passed through 1 cm to 2 cm of myometrium, medial to the uterine artery at the approximate level where a transverse uterine cesarean incision is routinely performed (Figure 18.19). This "low" ligature suture can be placed either slightly higher or lower, depending on the degree of exposure and the surgeon's preference. Usually, placement of the suture is decided after the vesicouterine fold has been reflected, avoiding potential injury to the bladder. The suture is passed through the myometrium anterior to posterior (or the other way around at the surgeon's convenience), and then through a transilluminated clear space in the broad ligament, and firmly tied. Transillumination immediately

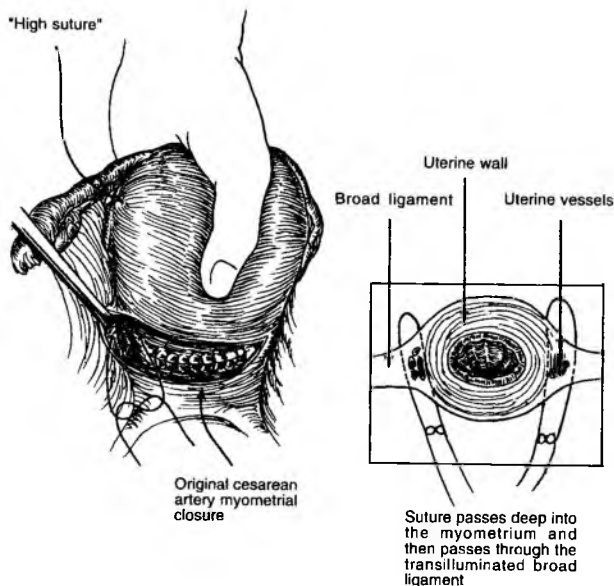


FIGURE 18.19.

**Obstetric hemorrhage: placement of high (left) and low (right). O'Leary uterine artery sutures are depicted. These sutures can be placed bilaterally, if clinical circumstances require.**

demonstrates broad ligament vessels and should be performed as the suture is passed to avoid injury to the large, thin-walled veins traversing this area. Time should not be wasted trying to visualize or specifically identify the uterine artery. In most instances, the vessel is not directly seen but can be palpated by the surgeon. A reasonable (approximately 1 cm–2 cm) bite into the myometrium will include the artery. As the ligature is tied, the vessel is directly compressed. At times, during otherwise routine cesarean operations, one uterine artery is lacerated and retracts into the myometrium. In this situation, unilateral uterine artery ligation, with sutures placed above and below the hysterotomy site as required, usually suffices for control. If bleeding persists following a unilateral ligation, the procedure can be rapidly repeated on the other side. A unilateral or bilateral high ligation of the ascending uterine artery or ligation of the uteroovarian connecting vessels is also possible. Alternatively, the uterine artery can be re-ligated below the original suture and the myometrial wound if bleeding appears to arise from below. Hebisch has also described a technique for transvaginal direct uterine artery ligation, digitally guiding needle placement into the cervix and identifying the artery by palpation [358]. After this somewhat startling technique, success was reported in twelve

or thirteen cases, with no reported complications directly related to the procedure. This approach will probably find few adherents, however.

When bleeding occurs from atony or lacerations, the best approach is a systematic and progressive ligation of feeding vessels until the hemorrhage stops [436]. In uterine bleeding from atony or placenta previa, the surgeon begins by clamping bleeding edges of the cesarean wound, assuming a cesarean was performed. If the bleeding continues, one or both uterine arteries are ligated. This procedure is easily accomplished in only a few minutes and alone can be sufficient for control. If not, a “high” uterine artery or uteroovarian vessel suture to isolate the anastomosis between the uterine and ovarian arteries at the uteroovarian ligament is added [347]. If vessel ligations prove insufficient to control bleeding, several important issues must be considered. If the hemorrhage arises from the uterus and the problem is persisting atony, uterine compression by a modified B-Lynch compression suture or another type of oversewing or localized compression suture is best. If all these procedures fail, hysterectomy is the final option. If the uterus is firmly contracted and the bleeding arises from at or about the cervix or high vaginal area, the feeding vessel might arise directly from the hypogastric artery or there is an occult laceration in the vaginal fornix or cervix. Even removing the uterus in this setting might not arrest the bleeding. This is the rare instance when either a direct or hypogastric ligation or the resort to embolization is the treatment of choice.

### Complications

The principal complication of the direct uterine vessel ligation technique is failure to control the hemorrhage; however, this is not common. In a large series of uterine artery ligations for postcesarean hemorrhage, O'Leary reported only 10 failures in 265 patients with a 1% complication rate [359]. Even with correct bilateral high and low vessel ligation, all blood flow to the uterus is not arrested – only the mean perfusion pressure is reduced. This reduction is usually sufficient to permit other hemostatic mechanisms, including coagulation and myometrial contractions, to act in concert to control the hemorrhage. In O'Leary's reports, complications appeared to be associated with operator inexperience [357,359,360]. Broad ligament hematomas are

possible if the ligature is not passed under direct vision (transillumination). The theoretic risk of ligation of the ureter is avoided by following the protocol for the proper level and technique of suture placement.

### Comments

The advantages of direct uterine ligation are that it is rapid, the exposure is simple, and complications minimal and inconsequential. In addition, the procedure is frequently successful in controlling bleeding. Although this operation is not a substitute for either uterotonics or transfusion, it usually slows and can arrest blood loss. At a minimum, ligations permit time for other steps to be taken. This procedure can also be performed if an inadvertent laceration of the uterine artery occurs during the initial myometrial incision of a cesarean delivery. In this setting, a prompt suture above and below the site of injury after the delivery of the baby usually rapidly controls the problem. Caution must be exercised with high ligatures to avoid trauma to the extrauterine or interstitial portion of the fallopian tube. The bilateral high and low suture technique is also well suited for patients with placenta accreta who wish to preserve fertility. Especially when multiple ligations are performed, the uterus might blanch despite remaining atonic. This is normal. The bleeding is usually controlled, and the uterus retains sufficient blood flow to avoid complications of ischemia. Subsequent menstrual flows as well as future fertility are unaffected. Even successful bipolar laparoscopic uterine vessel coagulation has been reported in delayed postpartum hemorrhage without serious complication, attesting to the robustness of the collateral circulation [361].

There are new techniques for compression of the uterus as a means of hemorrhage control; these involve either the use of intrauterine balloons or methods for suturing the walls of the uterus together with an absorbable suture material. These techniques serve the same physiologic purpose as increasing uterine tone. When the uterus contracts firmly, the interdigitating and overlapping fibers of the myometrium form a multitude of minor "ligatures" that pinch off vessels. Direct manual compression, operative procedures that compress the myometrium, or the intrauterine introduction of a balloon or classic uterine gauze packing all achieve the same result: the direct compression of vessels. As

long as the pressure exerted by sutures, myometrial tissue, balloon, pack, or external pressure exceeds the perfusion pressure, the lumen of the vessels remains occluded, and the hemorrhage is controlled. This permits time for the hemostatic mechanism to act, the surgeons to replace depleted volume, and the uterus to regain its normal tone.

*Bimanual compression*, although an important technique, is at best a temporizing measure. Bimanual compression requires substantial pressure to execute properly; the technique is rapidly fatiguing to the operator, especially when performed from below, and for a conscious parturient, this maneuver is obviously uncomfortable. Furthermore, this technique occupies both of the surgeon's hands. This method is not feasible for long-term hemorrhage control and should be viewed as an immediate stop-gap procedure while other preparations are made and assistants summoned. This said, bimanual compression is all too often not performed when indicated. The advantage of this procedure is that it is both simple and effective. It is folly to permit a woman to exsanguinate under observation when her losses can be markedly reduced by this simple maneuver, which can be performed by a birth assistant other than the primary surgeon.

The classic treatment for atony was *uterine packing* with a continuous gauze strip, with or without presoaking the gauze with vasopressin (Pitressin). A limited role remains for packing in noncesarean cases if an intrauterine balloon is not available and immediate control is necessary while preparations are underway for transfusion, embolization, or laparotomy on transfer.

Packing of the uterus for unresponsive postpartum hemorrhage has fallen from favor in recent decades. Its decline in popularity is mostly due to the introduction of potent uterotonics and the use of intrauterine balloon tamponade. When done properly, gauze packing is both safe and effective, however [362–365,365]. The usual opposition to packing, infection and a risk of concealed hemorrhage, does not figure importantly in modern reports or in the authors' clinical experience. When a pack is placed, potent uterotonics and broad-spectrum antibiotics are administered and the parturient closely monitored. There is no consensus in the literature about when a pack should be removed. The authors usually keep packs in place for 12 hours or less, but 24 to 36 hours is traditional [362].



Packing should be retained in the armamentarium of the obstetric surgeon [347,353]. It might on occasion prove useful, especially in cases of lower segment placental implantation when bleeding persists despite vessel ligation, and when the administration of uterotonics and embolization is not immediately available. (See Chapter 11, The Third Stage.)

The best known of the myometrial compression techniques is the B-Lynch [351,366–369,436]. This procedure is performed at laparotomy. As originally described, the B-Lynch suture required that the uterus be open for its insertion. The authors have found the original technique for placement of the suture to be unnecessarily complex, however. A simple anteroposterior puncture of the lower uterine segment with subsequent tying of a large-caliber chromic suture at the fundus is both simpler and faster and is currently the preferred technique of the authors. The suture is passed directly through the myometrium at the same site as used for O'Leary sutures and then passed externally over the uterine fundus and tied firmly in place, medial to the insertion of the fallopian tubes (Figures 18.20–18.23). When one

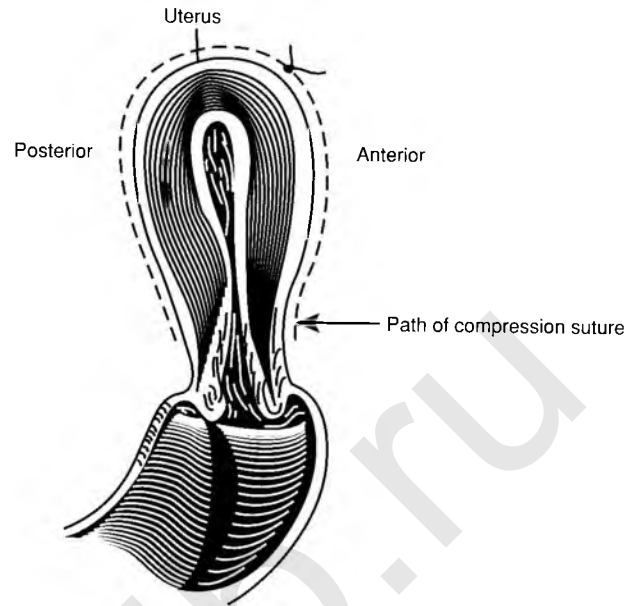


FIGURE 18.21. Anteroposterior view of the postpartum uterus indicates the route for a simple vertical compression suture.

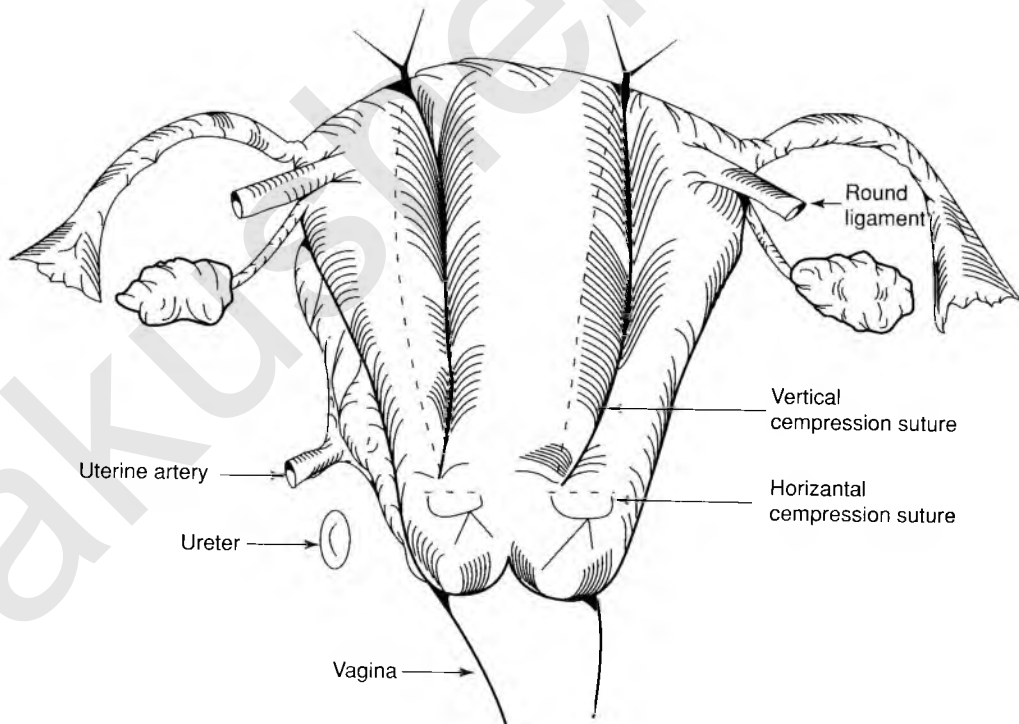
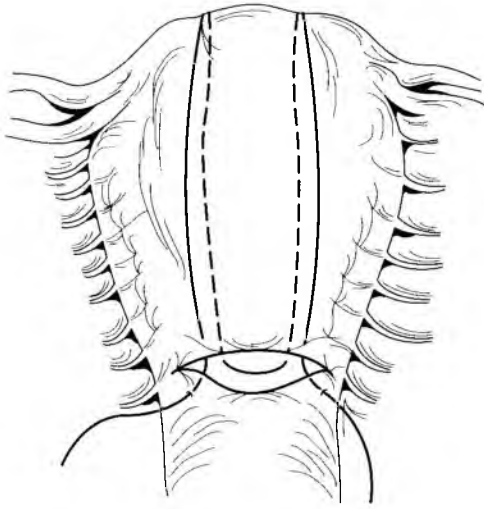
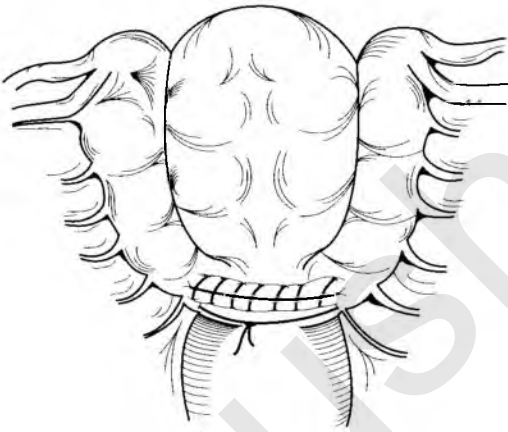


FIGURE 18.20. Modified B-Lynch method: Uterine anatomy depicting placement site for vertical and horizontal compression sutures. See text for details.



**FIGURE 18.22.**  
*Original Technique: B-Lynch compression suture. The initial suture placement is indicated. Note that placement follows cesarean delivery, and that the final tie is positioned below the original myometrial incision. See text for details.*



**FIGURE 18.23.**  
*Appearance of the postpartum uterus after placement and tensioning of a B-Lynch compression suture. See text for details.*

or more sutures is correctly placed on both sides of the uterus and tensioned, the uterus assumes an unusual "M" shape. The authors perform this procedure with heavy chromic suture material (No. 1), both for strength and to avoid cutting into the soft myometrium. There are several variants of the B-Lynch technique that use slightly different methods of suture placement or different suture

materials or sizes. They all employ the same general technique: myometrium-to-myometrium compression by direct tissue approximation, using an absorbable suture [351,367–370].

Several complications of compression sutures have been reported. The most serious include hematometrium, hematocolpos, pyometrium, and myometrial necrosis [371]. These problems presumably result from interference with the drainage of lochia or the uterine blood supply. The most common problem is failure to control the hemorrhage despite the correct suture placement and tensioning. Because this suture is restricted to the body of the uterus, there is no risk of ureteric injury.

Another simple technique involves direct oversewing of bleeding vessels at the placental implantation site [351]. Oversewing is a valuable adjunct when partial control of hemorrhage is achieved and a principal source of the remaining blood loss arises from the placental base. This situation usually occurs when the original placental implantation site was in the lower uterine segment. In the oversewing procedure, discrete areas of bleeding are identified and directly oversewn using figure-of-eight stitches of large-diameter absorbable sutures [355,372]. The operator must carefully avoid overtightening these sutures or the myometrium could be lacerated, potentially increasing the blood loss. A variant of this operation has the sutures placed in a "box" pattern, traversing the full thickness of the myometrium [370]. When through-and-through sutures are placed, care is needed to avoid inadvertently striking a major surface vessel, or involving either the bowel or omentum. The operator must also avoid obstructing or occluding the cervix. If such sutures are placed in the lower uterine segment close to the cervix, passing an instrument such as a ring forceps through the vagina to ensure an open passage is prudent.

Oversewing techniques are best suited to when the number of actively bleeding sites is limited. If the entire placental bed is a sea of upwelling blood from profound atony with many open vessels, oversewing is unlikely to succeed. In this situation, the surgeon's efforts are better directed toward vessel ligations or other types of compressive sutures.

A common but little-discussed problem is how to judge the extent of hemorrhage when the problem is uterine atony and one or more surgical control methods has been performed. This is especially

an issue when a cesarean was not performed or the uterus has already been closed after a cesarean. When the uterus is open at cesarean and atony is diagnosed, the extent of bleeding can be directly observed. Vessels bleeding from the wound edge can be grasped by atraumatic clamps and tensioned. This exposure permits direct observation from the interior of the uterus. Individual bleeding sites from the wound edge can be oversewn to free up space otherwise inconveniently occupied by one or more instruments. The usual technique of methodical, progressive devascularization is then conducted. If bleeding slows appreciably but more control is desirable, oversewing portions of the placental bed can be attempted, as previously discussed.

If the uterus has not yet been opened or has already been closed after a cesarean delivery, direct observation of the extent of bleeding from above is not possible unless the myometrial incision is reopened. In these circumstances, there are several acceptable approaches. The anesthesiologist can be queried about maternal vital signs such as arterial pressure, pulse rate, and urinary output. These parameters are more reflective of total bleeding, however, and only slowly and indirectly suggest how adequately the immediate hemorrhage has been controlled. Because this is not a reasonable technique, the surgeon must act to directly assess the *rate* of blood loss. Bimanual compression of the flaccid uterus is first performed to expel clots and liquid blood. Potent uterotonics should then be administered. The parturient's legs are flexed; her feet placed together and then pressed cephalad, allowing her knees to separate widely. To judge loss, one surgeon goes beneath the drapes and conducts a vaginal examination while the assistant compresses the myometrium from above. With a laparotomy sponge in hand, the lower operator swabs out the upper vagina with one hand while depressing the perineum with the other. At the same time, compression from above evacuates blood and clots into the vagina and the lower uterine segment. This dual technique expels most of the major clots and much of the liquid blood that might otherwise pool in the upper vagina and confuse the situation. Removal of clots from the lower uterine segment also relieves uterine distension, favoring contraction. Intravaginal collections of clots and unclotted blood might be due not to active bleeding but to earlier bleeding. To summarize the technique, when observation for the

rate of bleeding is performed, the surgeon should first remove old liquid and clotted blood and as well extend the observation over a reasonable interval of time to be certain that the rate of loss is accurately estimated.

Once the bleeding abates, the uterus is closed in layers if this has not already been performed. If oozing persists from the myometrial wound, a vacuum drain such as a Jackson-Pratt can electively be inserted behind the vesicouterine fold and the peritoneal reflection closed over the drain, as previously described. The drain is brought out through a separate stab wound in the lower abdomen. Intraperitoneal drainage can also be performed, but the output is usually copious and the drainage difficult to interpret. This technique is not recommended.

The parturient is next closely observed for several hours. Every half hour, for two hours, a vaginal examination is conducted to ensure that the uterus is firm, blood does not pool in the vagina, and to judge overall losses and review the vital signs. If all is stable after two to three hours, hourly evaluations are then performed for the next two to four hours. In the interim, blood and blood products are administered, as required, along with balanced salt solutions to replace losses, maintaining circulating volume and ensuring a good urinary output. Uterotonics are continued and a broad-spectrum first-generation antibiotic is administered. If bleeding has been severe, determination of a platelet count, the fibrinogen level, and other coagulation tests is appropriate. These data and clinical observations are used to judge the adequacy of fluid and blood or blood product replacement and the need for a possible return to the operating suite. If bleeding persists after laparotomy, packing, balloon placement or vessel ligations, embolization, or hysterectomy need to be considered. In clinical settings when immediate control is required, embolization is often not the best initial choice. This technique usually requires at least an hour or more in order to make the necessary arrangements and transport the parturient to a site where this procedure is performed. Thus, although embolization is certainly helpful, it is most practical when the acute bleeding is partially controlled, the patient is hemodynamic stable with her losses successfully replaced by transfusion or the infusion of crystalloids, and the delay to assemble the embolization team is not excessive. Obviously, availability and practicality of emergency embolization services

vary among institutions, and the clinician must take these limitations into account as decisions are made.

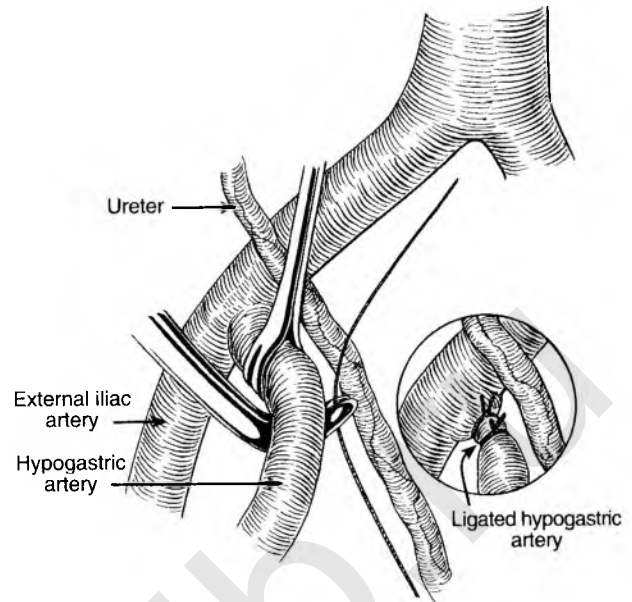
### Hypogastric Vessel Ligation

Ligation of the hypogastric artery was the principal vessel ligation procedure for obstetric hemorrhage before the introduction of techniques for direct uterine artery ligation [373]. In most services, owing to its technical difficulty, complications, and the time required for this procedure, hypogastric ligation is now performed only for limited indications [352,373]. In the usual case of uterine atony, hypogastric ligation has no advantages over direct uterine artery ligation and several serious drawbacks. The principal indication for this operation is in extensive high vaginal or cervical injury, with resulting hemorrhage unresponsive to the usual vessel ligations or the presence of large pelvic hematomas, precluding another approach. Vessel embolization is usually the safer choice for such unusual or difficult cases.

### Procedure

The origin of the hypogastric artery from the common iliac vessel is first located along the pelvic sidewall, and the vessel is dissected free from surrounding tissues (Figure 18.24).

The hypogastric vessels are best exposed by incising the peritoneum at the site where the ureter passes over the pelvic brim. The peritoneal reflection is dissected medially with the attached ureter, using care not to disrupt the periurethral vessels. This reflection will expose the hypogastric vessels. The hypogastric artery has varying branches. *It is best to ligate the vessel distal to the origin of the superficial gluteal artery, if this vessel can be identified.* Proximal ligation interrupts the arterial supply to the superior gluteal vessel, which can result in a partial muscle slough. Once the ligation site is identified, the anterior vessel is carefully dissected free of the connective tissue using a blunt right-angle clamp (e.g., a Mixtner). A space is dissected under the vessel, with close attention to not injuring the thin-walled (and difficult to control if lacerated) underlying hypogastric vein. A double ligature of nonabsorbable suture material – classically heavy silk or umbilical tape – is then drawn under the hypogastric vessel and firmly tied. The vessel is not severed. Surgical errors do occur, and dividing the



**FIGURE 18.24.** *Obstetric hemorrhage. Ligation of the internal iliac (hypogastric) artery is depicted. A double strand of heavy, permanent suture material (e.g., silk or umbilical tape) is used to ligate the vessel. Note that the artery is not severed.*

vessel has no advantage over simple ligation. If by chance the incorrect vessel (or the ureter!) is ligated, it is much easier to remove a suture than to anastomose a major artery or the ureter.

Most obstetric surgeons rarely perform hypogastric ligations. In this operation, it is surprisingly easy to ligate the incorrect vessel (or the ureter). Thus, it is best to attain assistance from another experienced surgeon. If the situation is pressing, the clinician should “talk” their way through the procedure, verbally reviewing the anatomy with the assistant surgeon as each structure is encountered and identified. As noted, meticulous care in the dissection around the artery is mandatory because of the risk(s) of hemorrhage if the thin-walled vein is inadvertently injured. The presence of normal femoral and distal pedal pulses should *always* be confirmed both before and after ligating the chosen vessel.

### Complications

Hypogastric ligation is a more complex and difficult procedure than an O’Leary type direct uterine artery suture [373–375]. Complications from internal iliac ligation can be divided into two categories: those associated with the incorrect

identification of vascular anatomy and those of general technique. Accidental ligation of the external iliac artery leads to leg ischemia, requiring reexploration and a good deal of professional embarrassment and an unpleasant time with the quality assurance reviewers. Careful identification of anatomic landmarks and palpation of distal extremity pulses before and after ligation should avoid this risk. An additional safety check occurs at the time of closure of the retroperitoneum before closing the abdomen. Before the final closure is the time to remind the operator to reevaluate pulses and look for any developing hematomas that might have been missed.

As noted previously, great respect must be given to the thin-walled veins lying under the hypogastric artery, because they are of formidable caliber. Pelvic hematomas from disruption of the hypogastric vein are particularly vexing and most difficult to control. In dissecting the site for the ligature, the tip of the right-angle clamp should be kept snugly against the arterial vessel. The index finger of the other hand can often help to guide the tip around the artery. The ureter is attached to the medial leaf of the peritoneum and can be easily located. Once identified, gentle medial retraction should keep it out of harm's way.

### **Comments**

In selected cases, bilateral internal iliac artery ligation is occasionally performed in the effort to avoid hysterectomy when other methods are ineffective in controlling hemorrhage. As noted before, iliac vessel ligation is not the initial procedure of choice in obstetric hemorrhage, except in the unusual setting of a high vaginal or cervical laceration. This procedure has been largely superseded by direct uterine artery ligation and by embolization.

Although internal iliac artery ligation or other multiple vessel ligation causes profound hemodynamic changes, none of these procedures obliterates all pelvic circulation. Ligation alters the pathways and reverses the direction of blood flow in the valveless arterial vessels but does not totally arrest flow to any site in the pelvis. There is extensive collateral circulation involving a rich network of interconnections between the major named pelvic vessels, including the lumbar-iliolumbar, middle sacral-lateral, sacral, and the superior hemorrhoidal-middle hemorrhoidal arteries. Vessel ligations work

by reducing the pulse pressure in these and other pelvic arterial vessels. This transforms what was originally high-pressure arterial system into a venous-like, low-pressure system, blunting the pressure excursions that normally occur during systole, permitting clots to form, remain, and eventually stem the flow.

For the reasons already discussed, hypogastric artery ligation is not the best initial procedure for obstetric hemorrhage. Although ligations decrease mean arterial pressure by 25%, mean blood flow by 50%, and arterial pulse pressure by 85%, the overall success in controlling hemorrhage and avoiding hysterectomy is only approximately 50% [375]. The exposure can be lengthy, since many obstetricians are unfamiliar with retroperitoneal exploration. Serious surgical complications are possible, albeit relatively uncommon. The ligation also renders a subsequent embolization difficult and at times impossible by precluding access to smaller vessels deep in the pelvis.

Fertility and obstetric performance after either compression sutures or major vessel ligations is of concern. There are small series reporting obstetric performance in women following uterine artery ligation [376] combined uterine and uteroovarian vessel ligation [377], hypogastric artery ligation [378], and placement of B-Lynch compression sutures [379]. The data are best for hypogastric vessel ligation. This procedure does not appear to have an adverse effect on menstrual/ovarian function or fertility. Outcome data for the other ligations/compression sutures are limited and more difficult to interpret. An important variable is the initial reason for the obstetric hemorrhage. As an example, unusual placenta adherence can be a recurrent problem in a subsequent pregnancy and presents a greater threat of complication than a prior history of simple atony. Long-term problems after even multiple vessel ligations are distinctly uncommon.

### **Angiographic Embolization**

When postpartum hemorrhage is sudden and extensive, the parturient must be stabilized and the bleeding stopped by using the most effective and rapid method. Simple atony usually responds to uterotonics. Occasionally, an obvious vessel injury is the cause of the bleeding, and ligation or compression is successful. When standard treatments fail

and surgery is contemplated, selective angiographic embolization of injured vessels needs consideration [380–388,390,391]. Optimally, embolization should be used *before* hypogastric artery ligation or hysterectomy and not as a last resort; however, the question usually is one of practicality. Immediate, severe hemorrhage is usually best handled by proceeding with uterotonics, packing an intrauterine balloon, standard vessel ligations, compression sutures, or hysterectomy, depending on the clinical circumstances. If one or more procedures has been attempted without complete success, or if control is incomplete, or if for any reason a laparotomy is considered inappropriate, embolization has an important role. Embolization techniques are also useful in cases complicated by pelvic hematomas, high vaginal lacerations, or those involving abnormal placental adherence.

Embolization involves cannulation of an artery (usually the femoral vessel) with the area under local anesthesia. Contrast material is then injected by means of a catheter and, under fluoroscopic guidance, the bleeding site is identified. Embolization is performed by first manipulating the catheter to lie within the feeding vessel. Embolic material (e.g., polyvinyl alcohol [PVA], absorbable gelatin sponge [Gelfoam], embosphere particles [starch microspheres]) is then injected to occlude the artery. A postembolization arteriogram confirms occlusion of the vessel. Both PVA and embosphere particles are permanent occlusive agents and are thus less desirable for use in patients in whom retention of fertility is desired. Gelatin sponge is generally recommended for use in postpartum hemorrhage, since vessels embolized with this material will eventually recannulize. Even gelatin sponge embolization has been associated with neurotic injury to the uterus, resulting in amenorrhea, however [389].

Procedure-related complications include local hematomas, pelvic pain, arterial vessel thrombosis, and rarely, partial bladder or vaginal necrosis, small bowel injury, transient paresthesias, and perforation of the iliac or hypogastric artery [381,384,390]. No embolization-related maternal deaths have been reported.

Embolization does not preclude surgical management if bleeding continues; however, the reverse is not always true [352]. The advantages of angiographic embolization include rapid recovery from the procedure and a high success rate. The proce-

cedure is minimally invasive and includes the potential for retained fertility while avoiding the complications of major abdominal surgery [391]. Data in the literature about the long-term effects of vessel embolization are principally case reports or short series [392–395]. Many of the included cases involved procedures originally performed outside of pregnancy for treatment of leiomyomas. It appears, based on very limited data, that pregnancies following embolization for leiomyomas are more fraught with difficulty than those resulting from treatment for obstetric hemorrhage. Additional data are necessary before firm conclusions can be reached, however [396].

The principal disadvantages to embolization include the requirement for a skilled team with easy availability, and a clinical setting that permits delay in achieving definitive hemorrhage control. Early identification of patients at high risk (e.g., abdominal pregnancy, placenta accrete/percreta) occasionally permit the prophylactic passage of catheters in the axillary or femoral arteries, to be used for embolization as proves necessary at delivery [390,397]. Such placement, when possible, potentially alleviates hurried and dangerous surgical maneuvers in high-risk settings. Despite the attractiveness of this idea, however, it has yet to be proved that prophylactic catheter placement actually results in lower blood loss or reductions in maternal morbidity when serious complications such as placenta accreta are the indication for surgery.

Successful *intrauterine or intracervical balloon tamponade* for hemorrhage has been reported in several cases [398]. These cases have involved postpartum hemorrhage from several causes, including atony, post-hysterectomy cervical stump or laceration bleeding, and placental site bleeding. Several different types of balloons have been employed, including the Stenstoken-Blakemore tubes [398–401], urologic hydrostatic balloon catheters [403], modified condom-based balloons [402], Foley catheter balloons [404–407], and commercially produced balloons especially designed for obstetric applications [408].

Condous and coworkers [401] have described what they termed the *tamponade test* to help judge which cases of postpartum hemorrhage require surgery for control. They reported 16 cases of medically intractable obstetric hemorrhage unresponsive to the administration of oxytocin,

ergometrine, or carboprost. Appropriate examinations for secundines and occult lacerations were conducted, and coagulopathy was excluded by standard testing. Thereafter, a Sengstaken-Blakemore tube (tip excised) was passed into the uterus through the cervix and then inflated with 70 ml to 300 ml of warmed saline, sufficient to tamponade the uterus. With this technique, 14 of 16 parturients were (87.5%) successfully controlled by the balloon treatment alone. When the hemorrhage was controlled, the tube was allowed to stay for 12 to 24 hours (longer in some cases), while both broad-spectrum antibiotics and uterotonics were administered. Laparotomy for surgery control was performed in the two failed cases. One of the failures was due to an undiagnosed cervical tear, a complication from a prior cesarean delivery. In this series, the original mode of delivery was cesarean in six cases and vaginal in the remaining ten. Ten of sixteen cases involved atony; four were complicated by retained products (secundines), and one had a cervical laceration. The final case involved an unspecified hematologic abnormality.

Although this observational study is limited by an inconsistent and in some cases possibly inadequate medical protocol for the treatment of hemorrhage/atony, the results are notable. Two points are worthy of comment. First, direct myometrial balloon compression was an effective means of hemorrhage control, and second, routine measures, specifically an initial careful patient examination searching for occult problems (e.g., a clinically unanticipated unsuspected laceration) are mandatory in all cases involving a severe postpartum hemorrhage.

### SYMPHYSIOTOMY

Although unusual in the United States, symphysiotomy is still practiced in the nonindustrialized world as an alternative method of delivery when cesareans are not readily available [429–435]. Although outwardly simple in execution, symphysiotomy has several complexities, and should not be attempted without knowledge of the surgical anatomy of the symphysis and an understanding of the potential complications of the procedure.

#### **Procedure**

The technique is not complex but does require at least two assistants to conduct properly.

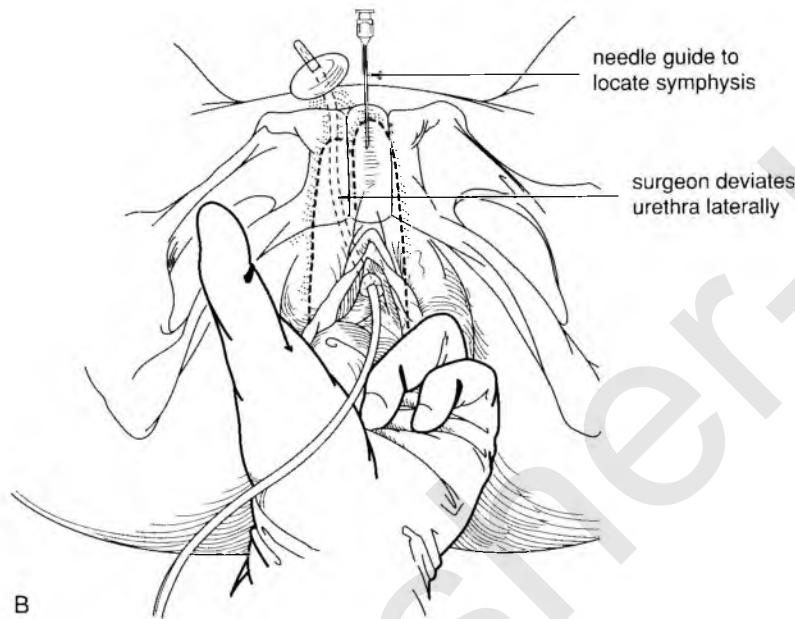
The technique as described is that suggested by Nichols [435]. To commence the procedure, each of the woman's legs is firmly grasped by a birth attendant (Figure 18.25). Their principal assignment is to restrict lateral movement of the parturient's legs to less than a 90° angle, avoiding undue stress on pelvic ligaments once the symphysis has been separated. This procedure can be performed under local anesthesia with simple xylocaine infiltration, or with epidural or spinal anesthesia, which ever proves acceptable in the specific clinical circumstances.

Initially, an indwelling (Foley) catheter is passed into the bladder, and the retention balloon is inflated. If an anesthetic has not already been administered, 1% or 2% xylocaine without epinephrine is first injected superficially into the midportion of the mons pubis and then deep into the gap of the pubic symphysis, which normally is easily palpated. The surgeon then inserts one hand into the vagina and deviates the urethra laterally to avoid injury during the subsequent separation of the symphysis (Figure 18.25B). The catheter tubing makes identification and lateral displacement of the urethra easy. A guide needle is then inserted vertically through the mons pubis and passed between the pubic bones, identifying the midline. Redirection of the needle is sometimes required until the joint space is located. With the needle as a guide, a direct downward puncture wound is made with a scalpel in the skin overlying the mons. The scalpel is introduced with the blade *toward* the operator and directed straight downward until the tip is felt by the vaginal finger (Figure 18.25C1 and 18.25C2). The scalpel is then rocked *upward* (cephalad), cutting through the posterior ligaments of the symphysis. The knife is then removed, rotated 180°, and reinserted and the handle rotated *downward* (caudal) to sever the remaining portion of the connecting ligaments. Sufficient separation of the symphysis has occurred when the surgeon can introduce a vaginal finger into the defect between the pubic bones. *During and after the severance of the ligaments of the symphysis, the attendants holding the mother's legs must rigidly restrict the angle of separation between the knees to less than 90°.*

If delivery is not spontaneous following the symphysiotomy, it can be assisted by vacuum extraction. After delivery, the surgical wound on the mons is closed and a pelvic binder is applied. The catheter is left in for four or more days. Full maternal



A



B

**FIGURE 18.25.**  
*Symphysiotomy. A, The legs are held by assistants, with abduction limited to  $<90^\circ$ . B, With a Foley catheter in place, the urethra is deviated laterally. A needle is inserted to identify the midline pubofibrocartilage and a scalpel is introduced vertically through a stab wound and swept anteriorly as indicated, severing the lower fibers. The vaginal finger limits the depth of the incision. The scalpel is then removed, rotated  $180^\circ$ , inserted into the stab wound, and then swept downward, freeing the remaining symphysis ligaments. See text for details.*

recovery can take 8 to 12 weeks. Potential complications of this procedure include localized bleeding, which normally responds to simple compression, fever, infection, abscess, urethral injury, urinary incontinence, and chronic pelvic instability. According to Nichols [435], long-term orthopedic disability is uncommon. The incidence of the other complications has not been established.

Potential indications for symphysiotomy in Western practice include severe shoulder dystocia when other measures for delivery fail, or cranial entrapment in an unanticipated vaginal breech delivery. Because some of the complications of symphysiotomy can be severe, this procedure should not be conducted without the presence of competent assistants and a surgeon who is aware of the basic technique. Performed by the uninitiated there is

a risk of injury to the bladder, urethra, or to the various pelvic ligaments and associated connective tissues.

## COMMON ADDITIONAL PROCEDURES

### **Surgical Sterilization**

Elective *tubal ligation* is a sterilizing procedure performed either at the time of a cesarean or after some interval postpartum following a vaginal delivery. The infant has been delivered, any vaginal lacerations have been closed, the uterine incision, if performed is sutured, the mother has stable vital signs, and general hemostasis is ensured.

At a cesarean, the fallopian tubes and ovaries are first evaluated for abnormalities and all pelvic organs



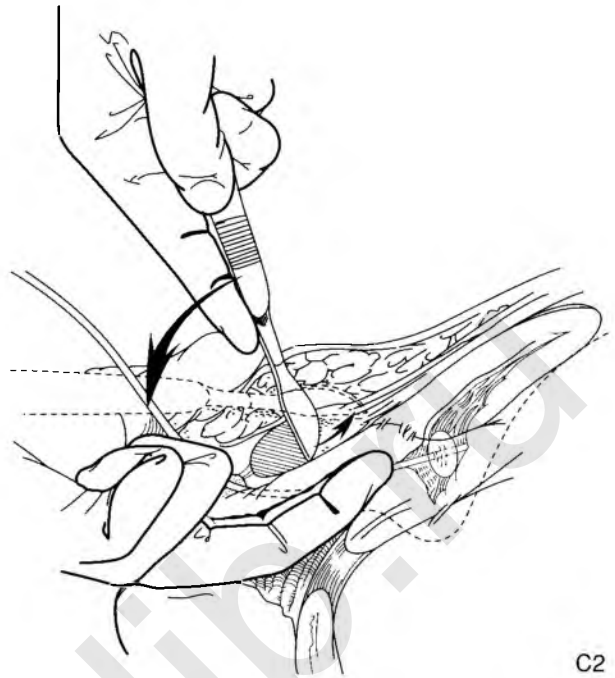
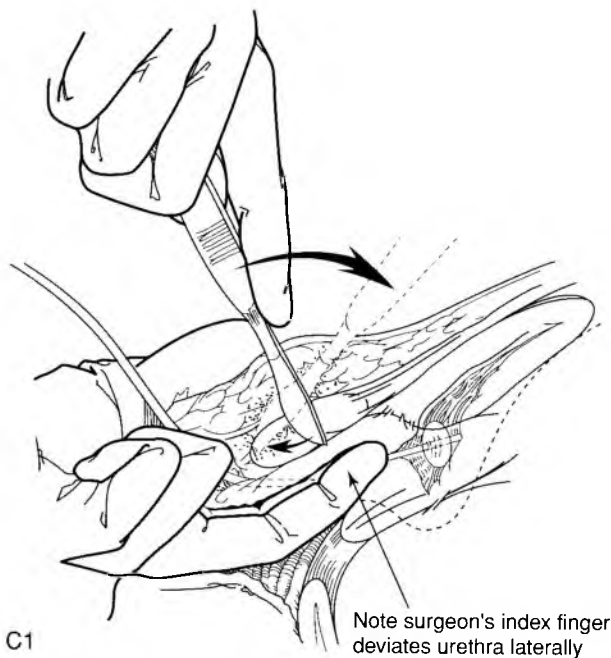


FIGURE 18.25.  
(Continued)

carefully inspected. The surgeon then identifies each fallopian tube by tracing its course from the cornua to the fimbria. The tube is then ligated and severed by one of the procedures described below. After the ligation, the adnexa are closely inspected for hemostasis and adequacy of tubal interruption before the uterus is returned to the anatomic position and the abdomen closed. An excised segment of tube is submitted for subsequent histologic confirmation.

Tubal ligations are also frequently performed after vaginal delivery, usually within 48 hours postpartum. In most cases, postpartum tubal ligations are performed through a subumbilical "minilaparotomy" incision within a few hours of vaginal delivery. In terms of timing, the operation is easiest when performed before puerperal involutions have progressed to the point where access to the adnexa through a paraumbilical incision is difficult.

### Counseling and Consent

Careful preoperative evaluation of women for puerperal tubal sterilization is required. This evaluation includes the assurance of medical factors, such as hemodynamic stability, the woman's acceptabil-

ity for an intraperitoneal procedure, and anesthesia. Equally important is the clinical setting and the consent process. Medical instability of the neonate weighs against proceeding with immediate postpartum tubal sterilization unless this possibility was anticipated and carefully discussed before delivery. Patient uncertainty or extreme emotional lability during the puerperium should also prompt deferring a purely elective sterilization procedure.

Informed consent for any sterilizing operation is central, because the current availability of effective alternatives for contraception makes female sterilization procedures elective. The obstetrician should make certain that the woman is aware of and has considered all of her options for fertility control. Counseling must scrupulously avoid either imposing a choice on the woman or arbitrarily denying her the opportunity for either permanent sterilization or temporary contraception because of age, parity, or social history. A well-thought-out sterilization decision by a young woman of low parity, whether or not she is married or otherwise in a committed relationship, might be entirely reasonable and appropriate for that person. Practitioners who think that they cannot adequately counsel certain patients or who are morally opposed to the patient's decision

should refer these women to another practitioner who is willing to consider performing the requested procedure.

Although consent of the patient's partner is not legally required for a sterilization operation, it is prudent to determine if the partner has been consulted. If not, or more important, if the partner is opposed to the procedure, serious problems with the relationship are likely. If identified, such concerns should be frankly discussed. The woman must understand that the proposed sterilization procedure is intended to be permanent and not reversible. It must also be pointed out that permanent sterility cannot be guaranteed, since failures have been reported with all techniques. Specifically, the possibility of sterilization failure with a resulting pregnancy, especially an ectopic pregnancy, should be discussed. Finally, the risks of surgery and anesthesia must be reviewed if the sterilization does not accompany a cesarean delivery. These discussions should precede the onset of labor. It is best to complete the informed consent and obtain a consent for surgery well prior to the anticipated hospital admission, following the requirements of the specific institution.

After hospital admission for labor and delivery, the woman's birth control plans are reviewed and the desirability of permanent sterilization reconsidered. If the decision remains the same, it is wise to document this in hospital discussion in the medical record and have the patient sign a second consent. The issue of neonatal status should be discussed. Despite all best wishes and intentions, the normality and survival ability of the neonate cannot be absolutely predicted in the delivery room. The patient must understand the distinct limitations of an instant diagnosis that the child is "normal." Because sterilization must be considered final, the decision to undergo this procedure on a preliminary report of the infant's condition is fraught with potential problems and is to be avoided, if possible.

### General Management and Incision

When a postpartum procedure is performed for a woman who has undergone a vaginal delivery, the most common site for the abdominal incision is subumbilical. At this location, the abdominal wall is thinnest because of the absence of intervening muscle, and the peritoneum closely approaches the rec-

tus fascia. For entry, the laxity of the abdominal wall immediately after delivery often makes it possible to proceed through what seems at first to be a very small incision. Either a curved, transverse, or a short vertical midline incision of approximately 2.5 cm to 5.0 cm is made, just sufficient to introduce narrow retractors. If exposure is inadequate, the incision is extended as required. The surgeon should not struggle with an inadequate incision. If exposure is insufficient for safe access to the adnexa, the incision should simply be enlarged. Accurate identification of the fallopian tubes and atraumatic surgery are far more important than the goal of producing a small abdominal scar.

The subcutaneous tissue is next incised down to the fascia. The authors prefer to make a vertical midline incision in the fascia to minimize the potential for trauma to the paramedical subfascial vessels. These vessels are located far enough away from the midline, however, that the alternative, a small transverse subumbilical fascial incision, usually does not place them at risk for injury. After the fascia is incised, the retrofascial tissues are then dissected down to the peritoneum, which is tented and entered as usual. At this point, some surgeons tag the peritoneum with a suture for easy subsequent identification. With retractors holding the wound open, the uterine fundus is identified and the cornua located. Each fallopian tube is grasped in turn with a noncrushing clamp, such as a Babcock, and then traced to its fimbriated end for positive identification. In addition, each tube is closely inspected after the procedure to ensure that a complete transection has occurred, and that adequate hemostasis is present. The fascia and the skin are then closed in separate layers as usual, using absorbable suture material. A subcuticular skin closure is favored. Routine diet and activity may be resumed as soon as recovery from anesthesia is complete.

### Madlener Technique

Introduced early in the 20th century, the *Madlener technique* is now infrequently performed [409]. In this procedure, each fallopian tube is first identified in the usual manner. Then, a knuckle of tube is formed in the isthmic region. The base of the knuckle is next crushed by a heavy clamp. The crush site is subsequently ligated. In the original report, the ligation was performed with silk suture. This

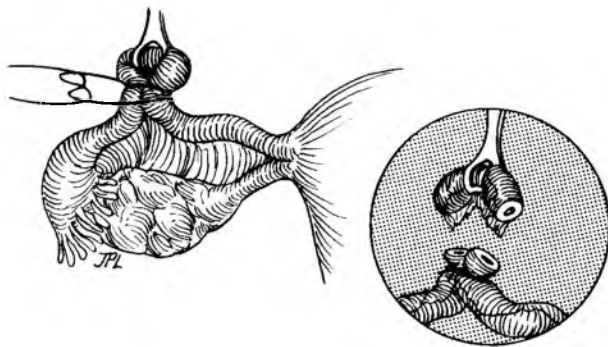


FIGURE 18.26.  
*Pomeroy Technique. See text for details.*

procedure has been supplanted by other techniques because of its unacceptably high failure rate owing to spontaneous tubal recannulation. Pregnancy rates as high as 82 in 1,000 have been reported [410].

### Pomeroy Technique

The *Pomeroy procedure* is the simplest and most popular technique for tubal sterilization. Pomeroy's associates first described this technique in 1930 [411]. In this operation, each fallopian tube is identified and then grasped at a relatively avascular segment of the isthmic region, and drawn up into a knuckle of approximately 1 cm in length for each arm (Figure 18.26). The base of the isolated loop is then doubly ligated. Various suture materials have been proposed for this ligation; the authors favor plain gut, 0 or 00. After placement of the ligature, the entrapped tubal loop is excised. The cut ends of the tube are then inspected to ensure that the proximal and distal lumina have been completely transected, and that there is no bleeding. The ligating suture subsequently dissolves, allowing the cut ends to separate widely during the process of healing.

The objective of the Pomeroy procedure is to keep the cut ends of the tube ligated only long enough to achieve permanent hemostasis. As the tissue heals, the ends become covered by reperi-tonealization, occluding the lumen. Electrosurgical desiccation of the tube at the time of surgery is specifically not advisable, because eschar formation can fuse the ends together, paradoxically increasing the chances for spontaneous recannulation. Similarly, slowly resorbing or permanent suture material should not be used, because this delays the desired separation of the tubal stumps.

An alternative to the Pomeroy technique involves the direct placement of a Hulka [412–414] spring-loaded clip in the fallopian tube. This procedure is feasible at cesarean delivery but has the disadvantage of not providing a histologic specimen to prove tubal transection, and thus has found few adherents.

Multiple studies of the Pomeroy procedure have been published [410]. Failure rates of 2 to 4 in 1,000 procedures are common. Prior concerns regarding the possibility that Pomeroy tubal ligations performed in conjunction with cesarean delivery were associated with a higher failure rate have not been confirmed [415].

### Irving Technique

This technique, first described by Irving in 1924, is moderately popular [416]. The *Irving operation* involves first dividing each tube, then burying the proximal stump into the myometrium, and in the original version, also burying the distal tubal stump in the leaves of the broad ligaments (Figure 18.27). In the procedure, each tube is identified and grasped sufficiently far away from the uterus to permit mobility of the proximal segment. An avascular area of the mesosalpinx is identified and punctured (Figure 18.27A). Ligatures of chromic suture material are placed 2 cm apart around the overlying tubal segment. The proximal ligature is tied in the center of the suture, leaving two long ends, one with the attached needle.

The tubal segment isolated between the ligatures is then excised. The cut ends are examined to ensure that hemostasis is adequate. A stab wound is then made in the uterine fundus with a sharp pointed clamp such as a hemostat, and a tunnel approximately 2 cm long is burrowed directly into the myometrium. A needle is attached to each end of the proximal tubal ligature and passed into the myometrial tunnel and out at its apex, with a 1-cm to 3-cm separation between the suture ends as they exit the uterus. The surgeon then inserts the proximal tubal stump into the myometrial tunnel as the suture is tied, this fixes the tubal stump within the uterine wall (Figure 18.27B). It is often necessary to insert one or more additional sutures at the site where the tube enters the myometrial tunnel to achieve hemostasis and prevent the tube from being dislodged.

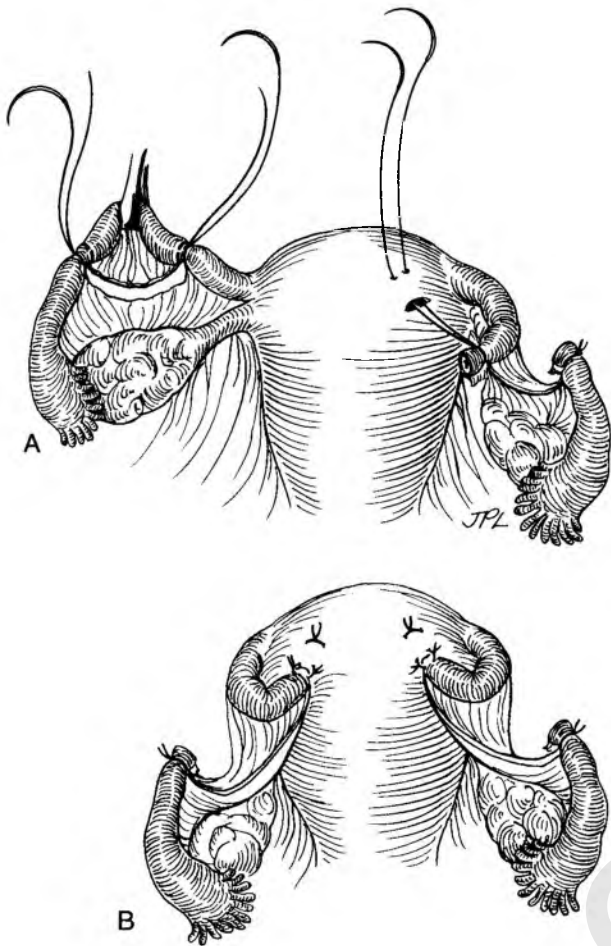


FIGURE 18.27.  
*Irving Technique. See text for details.*

The Irving procedure is highly successful as a sterilization procedure. Failures are rare [417]. Because only a small portion of the tube is excised, the potential for reversibility remains high. This procedure does have its limitations, however. Hemostasis at the site of the uterine perforation is a common problem, and this technique cannot be easily performed through a small paraumbilical incision. For these reasons, the Irving procedure is best suited for postcesarean tubal ligations, when exposure is not an issue.

### Uchida Technique

The *Uchida operation* permanently separates the proximal and distal ends of the divided fallopian tube, exteriorizing one stump into the peritoneal cavity [418]. As with the Irving technique, a knuckle

of fallopian tube is first identified and doubly ligated. After the intervening segment of tube between the ties is excised for histologic confirmation of the tubal separation, the proximal stump is buried within the mesosalpinx. The procedure positions the distal stump within the peritoneal cavity, thus permanently separating the segments. The potential reversibility of this technique depends on how much of the tube is excised as a surgical specimen (Figure 18.28).

In the performance of the Uchida operation, each tube is identified as previously described. To open a potential space, the mesosalpinx overlying the chosen tubal segments is infiltrated with several milliliters of saline, hydrodissecting the peritoneum from the tube (Figure 18.28A).

A linear incision is made in the antimesenteric border of the mesosalpinx overlying the site of infiltration, and a segment of the tube is dissected free of the submucosal tissue (Figure 18.28B). The isolated tubal segment is then ligated with absorbable

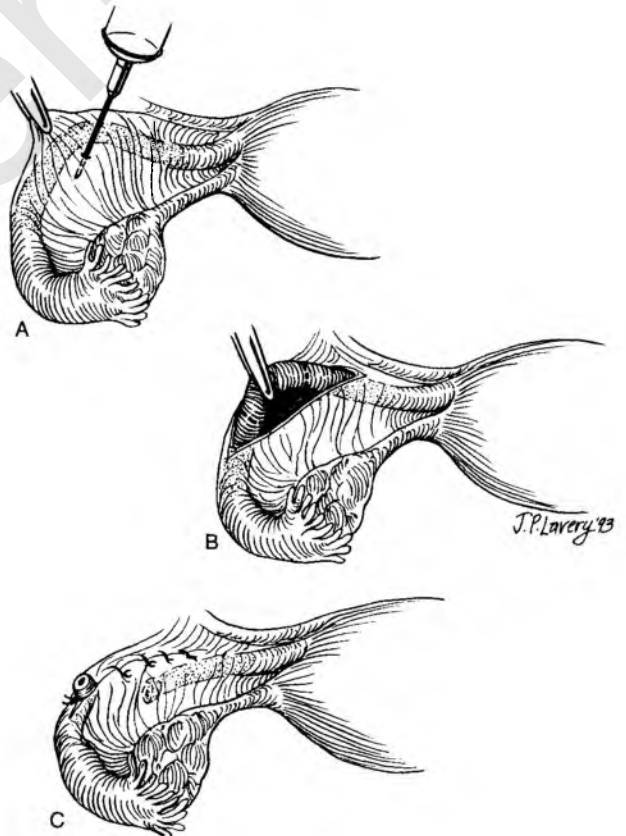


FIGURE 18.28.  
*Uchida Technique. See text for details.*

suture at its proximal and distal ends. The intervening segment is excised and retained for subsequent histologic examination. The proximal end of the tube is allowed to retract into the subserosa of the mesosalpinx. A purse-string suture of absorbable suture isolates the proximal tubal segment in the mesosalpinx, while the distal stump is left fixed in position in the peritoneal cavity (Figure 18.28C).

The Uchida procedure is quite effective. In his personal series, Uchida reported no failures in 20,000 cases [418]. There are no other independent reports of comparable series with which to evaluate this claim, however.

In the original procedure, a 5-cm segment of tube was excised; however, decreasing the length of the excised segment does not significantly jeopardize the long-term results. A shorter excised segment does reduce interference with the tubal blood supply. The greater the length of the residual tube after tubal sterilization, the greater the success rate of subsequent tubal reversal surgery [414]. Because this procedure depends on the success of the isolation of the proximal and distal tubal ends, the length of the excised segment is immaterial to success. As a practical matter, there is no need to excise a longer portion than is necessary for histologic confirmation of complete tubal transaction.

### Fimbriectomy

*Fimbriectomy*, a procedure popularized by Kroener [419], is a rapid and easily performed method of sterilization. Fimbriectomy must be considered a permanent type of sterilization, because it is rarely reversible. If the Kroener operation is chosen, it is extremely important to identify the fimbriated end of each tube definitively and release it from adhesions to surrounding structures. Failures of this technique can occur if the fimbria ovarica, a small strand of fimbria that connects the tube to the ovary, is not included in the pedicle. If the fimbria ovarica is not identified and specifically divided, it can maintain tubal patency and lead to subsequent pregnancy. To perform this sterilization, the portion of the tube distal to the isthmus is simply clamped and then excised. The residual tubal stump is then suture ligated with a delayed absorbable suture, such as chromic (Figure 18.29).

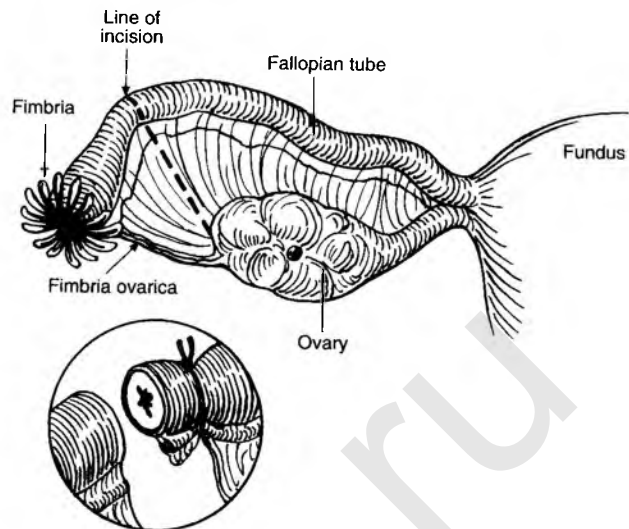


FIGURE 18.29.  
*Kroener Technique.* See text for details.

### COMPLICATIONS

Possible immediate postoperative complications of surgical sterilization include infection, bleeding, intraoperative bowel or bladder injury, thromboembolism, and rarely, death. Serious complications following tubal ligation are at best uncommon. Each procedure also carries the possibility of failure, with subsequent reconnection of the tube and restoration of a functioning lumen. The mortality risk for any of the sterilization procedures listed is very low, approximately 1 to 10 per 100,000 procedures [420,421]. When fatal outcomes occur, most are due to complications from anesthesia or unanticipated medication reactions. Losses due to surgical complications are at best very rare.

Long-term adverse effects of tubal ligation are controversial. Early concern that tubal ligation might adversely affect normal function by altering tubal structure or blood supply have been unsubstantiated. Potential changes in premenstrual symptomatology [422], ovarian function [423], vaginal bleeding patterns [424,425], sexual response [426], and pelvic pain [427] have been investigated. None of the longitudinal studies of women undergoing tubal sterilization has detected any significant differences in these parameters. Women who undergo tubal sterilization before the age of 29 years are at increased risk for hysterectomy compared with the general population, however [428]. This

difference disappears when these women are compared with women of similar age married to vasectomized men. The observed differences apparently result from unidentified factors other than the tubal surgery.

Sterilization failures are often the result of either mistaken identification of some other intraabdominal structure for the fallopian tube, or of incomplete occlusion of the tubal lumina. The potential for these errors is increased in postpartum procedures because of the alterations in the size and appearance of the tubes associated with pregnancy and the small incisions used to enter the abdomen, which restrict observation. Perioperatively, it is essential that each tube is definitely identified, and that care is taken to transect the entire tube.

## REFERENCES

1. National Center for Health Statistics: U.S. cesarean section rate for 2004. Hyattsville, MD: Nat Center Health Statistics: 2004.
2. Katz VL, Wells ST, Kuller JA, Hansen WF, McMahon MJ, Bowes WA Jr, Cesarean delivery: A reconsideration of terminology. *Obstet Gynecol*, 1995. Jul;86(1):152-3.
3. Poma P, Effects of obstetrician characteristics on cesarean delivery rates: A community hospital experience. *Am J Obstet Gynecol*, 1999. Jun;180 (6 Pt 1):1364-72.
4. Burns L, Geller SE, Wholey DR, The effect of physician factors on the cesarean section decision. *Med Care*, 1995. Apr;33(4):365-82.
5. Sachs BP, Kobelin C, Castro MA, Frigoletto F, The risks of lowering the cesarean-delivery rate. *N Engl J Med*, 1999. Jan 7;340(1):54-7.
6. Li T, Rhoads GG, Smulian J, Demissie K, Wartenberg D, Kruse L, Physician cesarean delivery rates and risk-adjusted perinatal outcomes. *Obstet Gynecol*, 2003. Jun;101(6):1204-12.
7. Townner D, Castro MA, Eby-Wilkens E, Effect of mode of delivery in nulliparous women on neonatal intracranial injury. *N Engl J Med*, 1999. Dec 2; 341(23):1709-14.
8. Centers for Disease Control, Differences in maternal mortality among black and white women - United States, 1990. *MMWR*, 1995. 44(1):13-14.
9. Fenton PWC, Reynolds F, Cesarean section in Malawi: Prospective study of early maternal and perinatal mortality. *Br Med J* 2003. Sep 13;327(7415):587.
10. Oladapo OT, Lamina MA, Fakoya TA (2006) Maternal deaths in Sagamu in the new millennium: A facility-based retrospective analysis. *BMC Pregnancy and Childbirth* 10 March, 2006.
11. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin Number 49, December 2003, Dystocia and Augmentation of Labor, 2003.
12. Vintzielos A, Nochimson DJ, Guzman EF, Knuppel RA, Lake M, Schiffrin BS, Intrapartum electronic fetal heart rate monitoring versus intermittent auscultation: A meta-analysis. *Obstet Gynecol*, 1995. 85:149-55.
13. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin: Number 70, December 2005: Intrapartum fetal heart rate monitoring.
14. Miller D, Rabello YA, Paul RH, The modified biophysical profile: Antepartum testing in the 1990s. *Am J Obstet Gynecol*, 1996. 174:812-7.
15. Manning F, Morrison I, Harman CR, Lange IR, Menticoglou S, Fetal assessment based on fetal biophysical profile scoring: Experience in 19,221 referred high-risk pregnancies. II An analysis of false-negative fetal deaths. *Am J Obstet Gynecol*, 1987. 157:880-4.
16. Nageotte M, Towers CV, Asrat T, Freeman RK, Perinatal outcome with the modified biophysical profile. *Am J Obstet Gynecol*, 1994. 170: 1672-6.
17. Hannah M, Hannah WJ, Hewson SA, Hodnett ED, Saigal S, Willan AR, Planned cesarean section versus planned vaginal birth for breech presentation at term: A randomised multicentre trial. *Lancet*, 2000. 356:1375-83.
18. Van Vellen A, Van Cappellen AW, Flu PK, Straub MJ, Wallenburg HC, Effect of external cephalic version in late pregnancy on presentation at delivery: A randomized controlled trial. *Br J Obstet Gynaecol*, 1989. 96:916-21.
19. Mahomed K, Seeras R, Coulson R, External cephalic version at term: A randomized controlled trial using tocolysis. *Br J Obstet Gynaecol*, 1991. 98:8-13.
20. Vezina Y, Bujold E, Varin J, Marquette GP, Boucher M, Cesarean delivery after successful external cephalic version of breech presentation at

- term: A comparative study. *Am J Obstet Gynecol*, 2004. Mar;190(3):763–8.
21. Taffel S, Placek PJ, Liss T, Trends in the United States cesarean section rate for the 1980–1985 rise. *Am J Public Health*, 1985. 77:955–9.
  22. Gould J, Davey B, Stafford RS, Socioeconomic differences in rates of cesarean section. *N Engl J Med*, 1989. Jul 27;321(4):233–9.
  23. Goyert G, Bottoms SF, Treadwell MC, Nehra PC, The physician factor in cesarean birth rates. *N Engl J Med*, 1989. Mar 16;120(11):706–9.
  24. DeMott R, Sandmire HF, The Green Bay cesarean section study. I. The physician factor as a determination of cesarean birth rate. *Am J Obstet Gynecol*, 1990. Jun;162(6):1593–1602.
  25. Bofill JA, Rust OA, Perry KG, Roberts WE, Martin RW, Morrison JC, Operative vaginal delivery: A survey of fellows of ACOG. *Obstet Gynecol*, 1996. Dec;88(6):1007–10.
  26. Demissie K, Rhoads GG, Smulian JC, Balasubramanian BA, Gandhi K, Joseph KS, Kramer M, Operative vaginal delivery and neonatal and infant adverse outcomes: Population-based retrospective analysis. *Br Med J*, 2004. 3 July;329(7456):24–9.
  27. Liu EHC, Sia ATH, Rates of caesarean section and instrumental vaginal delivery in nulliparous women after low-concentration epidural infusions or opioid analgesia: Systematic review. *Br Med J*, 2004. Jun 12; 328(7453):1410.
  28. Marrinan G, Stein M, Placenta previa. *eMedicine*, 2005, DOI: imedicine.com.topic = 559.
  29. Joy S, Lyon D, Placenta previa. *eMedicine*, 2004, DOI: imedicine.com.topic = 3271.
  30. Ko P, Yoon Y, Placenta previa. *eMedicine*, 2005, DOI: imedicine.com/topic = 427.
  31. Faiz A, Ananth CV, Etiology and risk factors for placenta previa: An overview and meta-analysis of observational studies. *J Matern Fetal Neonatal Med*, 2003. Mar;13(3):175–90.
  32. Gilliam M, Rosenberg D, Davis F, The likelihood of placenta previa with greater number of cesarean deliveries and higher parity. *Obstet Gynecol*, 2002. Jun;99(6):976–8.
  33. Ananth C, Smulian JC, Vintzileos AM, The effect of placenta previa on neonatal mortality: A population-based study in the United States 1989 through 1997. *Am J Obstet Gynecol*, 2003. May;188(5):1299–304.
  34. Leerentveld R, Gilberts EC, Arnold MJ, Waldimiroff JW, Accuracy and safety of transvaginal sonographic placental localization. *Obstet Gynecol*, 1990. Nov;76(5 Pt. 1):759–62.
  35. Mustafa SA, Brizot ML, Carvalho HM, Transvaginal ultrasonography in predicting placenta previa at delivery: A longitudinal study. *Ultrasound Obstet Gynecol*, 2002. Oct;20(4): 356–9.
  36. Taipale P, Hillesmaa V, Salonen R, Ylostalo P, Diagnosis of placental previa by transvaginal sonographic screening at 12–16 weeks in a nonselected population. *Obstet Gynecol*, 1997. Mar;89(3):364–7.
  37. Lauria M, Smith RS, Treadwell MC, Comstock CS, Kirk JS, Lee W, Bottoms SF, The use of second-trimester transvaginal sonography to predict placenta previa. *Ultrasound Obstet Gynecol*, 1996. Nov;8(5):337–40.
  38. Chama C, Wanonyi IK, Usman JD, From low-lying implantation to placenta praevia: A longitudinal ultrasonic assessment. *J Obstet Gynaecol*, 2004. Aug;24(5):516–18.
  39. Dashe J, McIntire DD, Ramus RM, Santos-Ramos R, Twickler DM, Persistence of placenta previa according to gestational age at ultrasound detection. *Obstet Gynecol*, 2002. May;99(5 Pt 1):692–7.
  40. Dola C., Garite TJ, Dowling DD, Friend D, Ahdoot D, Asrat T, Placenta previa: Does its type affect pregnancy outcome? *Am J Perinatol*, 2003. Oct;20(7):353–60.
  41. Oppenheimer L, Simpson P, Holmes N, Dabrowski A, Diagnosis of low-lying placenta: Can migration in the third trimester predict outcome? *Ultrasound Obstet Gynecol*, 2001. Aug;18(2):100–2.
  42. Bhide A, Prefumo F, Moore J, Hollis B, Thilaganathan B, Placental edge to internal os distance in the late third trimester and mode of delivery in placenta praevia. *Br J Obstet Gynaecol*, 2003. Sep;110(9):860–64.
  43. Oyelese Y, Smulian JC, Placenta previa, placenta accreta, and vasa previa. *Obstet Gynecol*, 2006. Apr;107(4):927–41.
  44. Lijoi A, Brady J, Vasa previa diagnosis and management. *J Am Board Fam Pract*, 2003. Nov–Dec; 16(6):543–8.
  45. Francois K, Mayer S, Harris C, Perlow JH, Association of vasa previa at delivery with a history of second-trimester placenta previa. *J Reprod Med*, 2003. Oct;48(10):771–4.

46. Catanzarite V, Maida C, Thomas W, Mendoza A, Stanco L, Piacquadio KM, Prenatal sonographic diagnosis of vasa previa: Ultrasound findings and obstetric outcome in ten cases. *Ultrasound Obstet Gynecol*, 2001. Aug;18(2):109–15.
47. Oyelese Y, Catanzarite V, Prefumo F, Lashley S, Schachter M, Tovbin Y, Goldstein V, Smulian JC, Vasa previa: The impact of prenatal diagnosis on outcomes. *Obstet Gynecol*, 2004. May;103(5 Pt. 1):937–42.
48. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin Number 35, May 2002: Diagnosis and Treatment of Cervical Carcinomas, 2002.
49. Donegan W, Cancer and pregnancy. *CA Cancer J Clin* 1983. 33:194–214.
50. Hannigan EV, Cervical cancer in pregnancy. 1990. *Clin Obstet Gynecol* 33:837–45.
51. Nevin J, Soeters R, Dehaeck K, Bloch B, van Wyk L, Cervical carcinoma associated with pregnancy. *Obstet Gynecol Surv*, 1995. Mar;50(3):228–39.
52. Method M, Brost BC, Management of cervical cancer in pregnancy. *Semin Surg Oncol*, 1999. Apr–May;16(3):251–60.
53. Benedet J, Odicino F, Maisonneuve P, Beller U, Creasman WT, Heintz AP, Carcinoma of the cervix uteri. *J Epidemiol Biostat*, 2001. 67(1):7–43.
54. Lee R, Neglia W, Park RC, Cervical carcinoma in pregnancy. *Obstet Gynecol*, 1981. Nov;58:584–89.
55. Weisz B, Schiff E, Lishner M, Cancer in pregnancy: Maternal and fetal implications. *Hum Reprod Update*, 2001. 7(4):384–93.
56. Shivvers S, Miller DS, Preinvasive and invasive breast and cervical cancer prior to or during pregnancy. *Clin Perinatol*, 1997. Jun;24(2):369–89.
57. Sadler L, Saftlas A, Wang W, Exeter M, Whittaker J, McCowan L, Treatment for cervical intraepithelial neoplasia and risk of preterm delivery. *JAMA*, 2004. May 5;291(17):2100–6.
58. Sood A, Sorosky JI, Mayr N, Krogman S, Anderson B, Buller RE, Hussey DH, Radiotherapeutic management of cervical carcinoma that complicates pregnancy. *Cancer*, 1997. Sep 15;80(6):1073–8.
59. Sorosky JI, Squatrito R, Ndubisi BU, Anderson B, Podczaski ES, Mayr N, Buller RE, Stage I squamous cell cervical carcinoma in pregnancy: Planned delay in therapy awaiting fetal maturity. *Gynec Oncol*, 1995. Nov;59(2):207–10.
60. Takushi M, Moromizato H, Sakumoto K, Kanazawa K, Management of invasive carcinoma of the uterine cervix associated with pregnancy: Outcome of intentional delay in treatment. *Gynecol Oncol*, 2002. Nov;87(2):185–9.
61. Van der Vange N, Weverling GJ, Ketting BW, Ankum WM, Samlal R, Lammes FB, The prognosis of cervical cancer associated with pregnancy: A matched cohort study. *Obstet Gynecol*, 1995. Jun; 85(6):1022–6.
62. Sood A, Sorosky JI, Krogman S, Anderson B, Brenda J, Buller RE, Surgical management of cervical cancer complicating pregnancy: A case-control study. *Gynecol Oncol*, 1996. Dec;63(3):294–8.
63. Zemlickis D, Lishner M, Degendorfer P, Maternal and fetal outcome after invasive cervical cancer in pregnancy. *J Clin Oncol*, 1991. Nov;9(11):1956–61.
64. Lishner M, Cancer in pregnancy. *Ann Oncol*, 2003. 14(Supp 3):iii31–6.
65. Sood A, Sorosky JI, Invasive cervical cancer complicating pregnancy: How to manage the dilemma. *Obstet Gynecol Clin North Am*, 1998. Jun;25(2):343–52.
66. McKenna D, Ester JB, Fischer JR, Elective cesarean delivery for women with a previous anal sphincter rupture. *Am J Obstet Gynecol*, 2003. Nov; 189(5):1251–6.
67. Nienaber C, Von Kodolitsch Y, Therapeutic management of patients with Marfan syndrome: Focus on cardiovascular involvement. *Cardiol Rev*, 1999. Nov–Dec;7(6):332–41.
68. Groenink M, Lohuis, TAJ, Tijssen JGP, Naerff MSJ, Hennekam RCM, van der Wall EE, Mulder BJM, Survival and complication-free survival in Marfan's syndrome: Implications of current guidelines. *Heart*, 1999. 82:499–504.
69. Keskin H, Mungan T, Aktepe-Keskin E, Gungor T, Marfan syndrome in pregnancy: A case report. *Ann Saudi Med*, 2002. 22(5–6):356–8.
70. Kim S, Martin N, Hsia EC, Pyeritz RE, Albert DA, Management of aortic disease in Marfan syndrome: a decision analysis. *Arch Intern Med*, 2005. Apr 11;165(7):749–55.
71. Hwa J, Richards JG, Huang H, McKay D, Pressley L, Hughes CF, Jeremy RW, The natural history of aortic dilatation in Marfan syndrome. *Med J Aust*, 1993. Apr 19;158(8):558–62.



72. Pyeritz R, Maternal and fetal complications of pregnancy in the Marfan syndrome. *Am J Med*, 1981. Nov; 71(5):784–90.
73. Rossiter J, Repke JT, Moarales AJ, Murphy EA, Pyeritz RE, A prospective longitudinal evaluation of pregnancy in the Marfan syndrome. *Am J Obstet Gynecol*, 1995. Nov; 173(5):1599–606.
74. Meijboom L, Vos FE, Timmermans J, Boers GH, Zwinderman AH, Mulder MJ, Pregnancy and aortic root growth in the Marfan syndrome: A prospective study. *Eur Heart J*, 2005. May;26(9):914–20.
75. Lind J, Wallenburg HC, The Marfan syndrome and pregnancy: A retrospective study in a Dutch population. *Eur J Obstet Gynecol Reprod Biol*, 2001. Sep;98(1):28–35.
76. Rahman J, Rahman FZ, Rahman W, al-Suleiman SA, Rahman MS, Obstetric and gynecologic complications in women with Marfan's syndrome. *J Reprod Med*, 2003. Sep;48(9):723–8.
77. Pyeritz R, The Marfan syndrome. *Annu Rev Med*, 2000. 51:481–510.
78. Elkayam U, Ostrzega E, Shotan A, Mehra A, Cardiovascular problems in pregnant women with the Marfan syndrome. *Ann Intern Med*, 1995. July 15; 123(3):117–22.
79. Lee M-J, Huang A, Gillen-Goldstein J, Funai EF, Labor and vaginal delivery with maternal aortic aneurysm. *Obstet Gynecol*, 2001. Nov;98(5 Pt 2): 935–8.
80. Sakaguchi M, Kitahara H, Watanabe T, Kono T, Fukui D, Amano J, Successful surgical treatment for acute aortic dissection in pregnancy with Marfan's syndrome. *Jpn J Thorac Cardiovasc Surg*, 2005. Apr;53(4):220–2.
81. Sakala EP, Harding MD, Ehlers-Danlos syndrome Type III and pregnancy – A case report. *J Reprod Med*, 1991. Aug;36(8):622–24.
82. Schalkwijk J, Zweers MC, Steijlen PM, Dean WB, Taylor G, van Vlijmen IM, van Haren B, Miller WL, Bristow J, A recessive form of the Ehlers-Danlos syndrome caused by tenascin-X deficiency. *N Engl J Med*, 2001. Oct 18;345(16):1167–75.
83. Cikrit D, Glover JR, Dalsing MC, Silver D, The Ehlers-Danlos specter revisited. *Vasc Endovascular Surg*, 2002. May–Jun;36(3):213–7.
84. Lurie S, Manor M, Hagay ZJ, The threat of type IV Ehlers-Danlos syndrome on maternal well-being during pregnancy: Early delivery may make the difference. *J Obstet Gynaecol*, 1998. May;18(3):245–8.
85. Rudd N, Nimrod C, Holbrook KA, Byers PH, Pregnancy complications in type IV Ehlers-Danlos syndrome. *Lancet*, 1983. Jan 1;1(8314–5):50–3.
86. Pepin M, Schwarze U, Superti-Furga A, Byers PH, Clinical and genetic features of Ehlers-Danlos syndrome type IV, the vascular type. *N Engl J Med*, 2000. Mar 9;342(10):673–80.
87. Habecker-Green J, Cohn G, Connective tissue disorders. In *Obstetric Syndromes and Conditions*, O'Grady J, Burkman R (eds). New York: Parthenon Publishing, 1998, pp. 283–287.
88. Lind J, Wallenburg HC, Pregnancy and the Ehlers-Danlos syndrome: A retrospective study in a Dutch population. *Acta Obstet Gynecol Scand*, 2002. Apr;81(4):293–300.
89. Weinbaum P, Cassidy SB, Campbell WA, Rickles FR, Vintzileos AM, Nochimson DJ, Tsipouras P, Pregnancy management and successful outcome of Ehlers-Danlos syndrome type IV. *Am J Perinatol*, 1987. Apr;4(2):134–7.
90. Cruikshank D, White CA, Obstetric malpresentations – twenty years' experience. *Am J Obstet Gynecol*, 1973. Aug 15;116(8):1097–1104.
91. Ross M, Devoe LD, Rosen KG, ST-segment analysis of the fetal electrocardiogram improves fetal heart rate tracing interpretation and clinical decision making. *J Matern Fetal Neonatal Med*, 2004. Mar;15(3):181–5.
- 91b. First STAN fetal monitoring centre opens in the USA. Press Release: 22 October, 2007. [www.neovevta.com](http://www.neovevta.com)
92. American College of Obstetricians and Gynecologists and The American Academy of Pediatrics, Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology. Washington, DC: American College of Obstetricians and Gynecologists, 2003.
93. American College of Obstetricians, and Gynecologists, ACOG Practice Bulletin Number 27: Prenatal Diagnosis of Fetal Chromosomal Abnormalities, 2001.
94. Grandjean H, Larroque D, Levi S, Detection of chromosomal abnormalities, an outcome of ultrasound screening: The Eurofetus Team. *Ann N Y Acad Sci*, 1998. Jun 18;847:136–40.
95. Levi S, Routine ultrasound screening of congenital anomalies: An overview of the European experience. *Ann NY Acad Sci*, 1998. Jun 18; 847: 86–98.

- 95b. Breathnach FM, Malone FD, Lambert-Messerlian G, Cuckle HS, Porter TF, Nyberg DA, Comstock CH, Saade GR, Berkowitz RL, Klugman S, Dugoff L, Craigo SD, Timor-Tritsch IE, Carr SR, Wolfe HM, Tripp T, Bianchi DW, D'Alton ME, First and Second Trimester Evaluation of Risk (FASTER) Research Consortium, First- and second-trimester screening: detection of aneuploidies other than Down syndrome. *Obstet Gynecol*, 2007. Sep;110(3):651-7.
96. Lewis D, Tolosa JE, Kaufmann M, Goodman M, Farrell C, Berghella V, Elective cesarean delivery and long-term motor function or ambulation status in infants with meningomyelocele. *Obstet Gynecol*, 2004. Mar;103(3):469-73.
97. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin Number 38: Perinatal Care at the Threshold of Viability, 2002.
98. Baker DA, Risk factors for herpes simplex virus transmission to pregnant women: A couples study. *Am J Obstet Gynecol*, 2005. Dec;193(6):1887-8.
99. Enright A, Prober CG, Neonatal herpes infection: Diagnosis, treatment and prevention. *Semin Neonatol*, 2002. 7(4):283-91.
100. Wald A, Genital herpes. *Clin Evid*, 2002. Dec;(8):1608-19.
101. Wald A, Zeh J, Selke S, Warren T, Ryncarz AJ, Ashley R, Krieger JN, Corey L, Reactivation of genital herpes simplex virus type 2 infection in asymptomatic seropositive persons. *N Engl J Med*, 2000. Mar 23;342(12):844-50.
102. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin: Number 82: Management of Herpes in Pregnancy, 2007.
103. Stone K, Reiff-Eldridge R, White AD, Corder JF, Brown Z, Alexander ER, Andrews EB, Pregnancy outcomes following systemic prenatal acyclovir exposure: Conclusions from the international acyclovir pregnancy registry. *Birth Defects Res A Clin Mol Teratol*, 2004. Apr;70(4):201-7.
104. Dubin J, Howes DS. 2007, HIV infection and AIDS. *eMedicine*, topic = 253.
105. The International Perinatal HIV Group, The mode of delivery and the risk of vertical transmission of human immunodeficiency virus type 1 - a meta-analysis of 15 prospective cohort studies. *N Engl J Med*, 1999. 340(13):977-87.
106. Sullivan J, Prevention of mother-to-child transmission of HIV- what next? *J Acquir Immune Defic Syndr*, 2003. 34(Suppl 1):S67-S72.
107. Brockelhurst P, Interventions for reducing the risk of mother-to-child transmission of HIV infection. *Cochrane Database Syst Rev*, 2002. 1(CD000102).
108. Mandavilli A, The coming epidemic. *Nature*, 2005. 436:496-8.
109. Wu J, Huang DB, Pang KR, Tying SK, Selected sexually transmitted diseases and their relationship to HIV. *Clin Dermatol*, 2004. Nov-Dec;22(6):499-508.
110. McIntyre J, Mothers infected with HIV. *Br Med Bull*, 2003:127-35.
111. Dominguez K, Lindegren ML, D'Almada PJ, Peters VB, Frederick T, Rakusan TA, Ortiz IR, Hsu HW, Melville SR, Fowler MG, Pediatric Spectrum of HIV Disease Consortium, Increasing trend of cesarean deliveries in HIV-infected women in the United States from 1994-2000. *J Acquir Immune Defic Syndr*, 2003. 33(2):232-8.
112. Chou, R, Smits AK, Huffman LH, Fu R, Korhuis PT, U.S. Preventive Services Task Force, Prenatal screening for HIV: A review of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med*, 2005. 143(1):38-54.
113. Semprini A, Fiore S, HIV and pregnancy: Is the outlook for mother and baby transformed? *Curr Opin Obstet Gynecol*, 2004. 16(6):471-5.
114. Kind C, Rudin C, Siegrist CA, Wyler CA, Biedermann K, Lauper U, Irion O, Schupbach J, Nadal D, Prevention of vertical HIV transmission: Additive protective effect of elective Cesarean section and zidovudine prophylaxis. *Swiss Neonatal HIV Study Group. AIDS*, 1998. 12(2):205-10.
115. Tuomala R, Watts DH, LI D, Vajaranant M, Pitt J, Hammill H, Landesman S, Zorrilla C, Thompson B, and Women and Infants Transmission Study, Improved obstetric outcomes and few maternal toxicities are associated with antiretroviral therapy, including highly active antiretroviral therapy during pregnancy. *J Acquir Immune Defic Syndr*, 2005. 38(4):449-73.
116. International Perinatal HIV Group, Duration of ruptured membranes and vertical transmission of HIV-1: A meta-analysis from 15 prospective cohort studies. *AIDS*, 2001. 15(3):357-68.
117. Watts D, Covington DL, Beckerman K, Garcia P, Scheuerle A, Dominguez K, Ross B, Sacks S, Chavers S, Tilson H, Assessing the risk of birth defects associated with antiretroviral exposure

- during pregnancy. *Am J Obstet Gynecol*, 2004. 191(3):985-92.
118. Watts D, Lambert JS, Stiehm ER, Bethel J, Whitehouse J, Fowler MG, Read J, Complications according to mode of delivery among human immunodeficiency virus-infected women with CD4 lymphocyte counts of  $\leq 500/\mu\text{L}$ . *Am J Obstet Gynecol*, 2000. 183(1):100-7.
  119. European Mode of Delivery Collaboration, Elective caesarean-section versus vaginal delivery in prevention of vertical HIV-1 transmission: A randomised clinical trial. The European Mode of Delivery Collaboration. *Lancet*, 1999. 353(9158): 1035-9.
  120. Marcollet A, Goffinet F, Firtion G, Pannier E, Le Bret T, Brival ML, Mandelbrot L, Differences in postpartum morbidity in women who are infected with the human immunodeficiency virus after elective cesarean delivery, emergency cesarean delivery, or vaginal delivery. *Am J Obstet Gynecol*, 2002. 186(4):784-9.
  121. Mohlala B, Tucker TJ, Besser MJ, Williamson C, Yeats J, Smit L, Anthony J, Puren A, Investigation of HIV in amniotic fluid from HIV-infected pregnant women at full term. *J Infect Dis*, 2005. 192(3):488-91.
  122. Perinatal HIV Guidelines Working Group, Recommendations for Use of Antiretroviral Drugs in Pregnant HIV-1-Infected Women for Maternal Health and Interventions to Reduce Perinatal HIV-1 Transmission in the United States. Washington, DC: Public Health Resources and Services Administration, 2005.
  123. Capparelli E, Rakhmanina N, Mirochnick M, Pharmacotherapy of perinatal HIV. *Semin Fetal Neonatal Med*, 2005. Apr;10(2):161-75.
  124. Delke I, Special considerations for the HIV-infected patient with preterm labor. *Clin Perinatol*, 2003. 30(4):855-74.
  125. Centers for Disease Control, Recommendations of the US Public Health Service Task Force on the use of zidovudine to reduce perinatal transmission of human immunodeficiency virus. *MMWR*, 1994. 43: No. RR-11.
  126. Mrus J, Tsevat J, Cost-effectiveness of interventions to reduce vertical HIV transmission from pregnant women who have not received prenatal care. *Med Decis Making*, 2004. 24(1):30-9.
  127. Halpern M, Read JS, Ganoczy DA, Harris DR, Cost-effectiveness of cesarean section delivery to prevent mother-to-child transmission of HIV-1. *AIDS* 2000. 14(6):691-700.
  128. Conte D, Fraquelli M, Prati D, Prevalence and clinical course of chronic hepatitis C virus infection and rate of HCV vertical transmission in a cohort of 15,250 pregnant women. *Hepatology*, 2000. Mar;31(3):751-5.
  129. Dinsmoor M, Hepatitis in pregnancy. *Curr Womens Health Rep*, 2001. Aug;1(1):27-30.
  130. Resti M, Azzari C, Mannelli F: Tuscany Study Group on Hepatitis C Virus Infection, Mother to child transmission of hepatitis C virus: Prospective study of risk factors and timing of infection in children born to women seronegative for HIV-1. *Br Med J*, 1998. Aug 15;317(7156): 437-40.
  131. Marine-Barjoani E, Bongain A, Berrebi A, Is delivery mode related to perinatal transmission of hepatitis C virus (HCV)? 40th Annual Meeting of the European Association for the Study of the Liver. Paris, France, April 13-17, 2005.
  132. Vandelli C, Roman L, Tisminetzky S, Stroffolini T, Ventura E, Zanetti A, Lack of evidence of sexual transmission of hepatitis C among monogamous couples: Results of a 10-year prospective follow-up study. *Am J Gastroenterol*, 2004. May;99(5):855-9.
  133. Chung JW, Acute hepatitis C virus infection. *Clin Infect Dis*, 2005. July 1;41(Suppl):S14-S17.
  134. Strader D, Wright T, Thomas DL, Seeff LB, Diagnosis, Management, and Treatment of Hepatitis C. *Hepatology*, 2004. 39(4):1147-71.
  135. Mok J, Pembrey L, Tovo PA, Nowell ML, European Paediatric Hepatitis C Virus Network, When does mother to child transmission of hepatitis C occur? *Arch Dis Child Fetal Neonatal Ed* 2005. Mar;90(2):F156-60.
  136. Crowther C, Caesarean delivery for the second twin (Cochrane Review). *Cochrane Database Syst Rev* 1996, 1. DOI: 10.1002/14651858. CD000047.
  137. Persad V, Baskett TF, O'Connell CM, Scott HM, Combined vaginal-cesarean delivery of twin pregnancies. *Obstet Gynecol*, 2001. Dec;98(6):1032-7.
  138. Dafour P VD, Vanderstichele S, Ducloy AS, Depret S, Monnier JC, Intravenous nitroglycerine for internal podalic version of the second twin in transverse lie. *Obstet Gynecol*, 1998. Sep;92(3):416-19.

139. Simhayoff N, Sheiner E, Levy A, Mahhel RD, Mazor M, Hallak M, To induce or not to induce labor: A macrosomic dilemma. *Gynecol Obstet Invest*, 2004. 58(3):121-5.
140. Menticoglou S, Manning FA, Morrison, Harmon CR, Must macrosomic fetuses be delivered by a caesarean section? A review of the outcome for 786 babies greater than or equal to 4,500 g. *Aust N Z J Obstet Gynaecol*, 1992. May;32(2):100-103.
141. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin. Number 22, November 2000. Fetal Macrosomia.
- 141b. Nahum GG. Estimation of fetal weight. *eMedicine* ([www.emedicine.com](http://www.emedicine.com)), topic = 3281.
142. Fischer R, Hageboutros A, Thrombocytopenia in pregnancy. *eMedicine*, 2004. DOI: [emedicine.com/med/topic3480](http://emedicine.com/med/topic3480)
143. Levy JA, Murphy LD, Thrombocytopenia in pregnancy. *J Am Board Fam Pract*, 2002. Jul-Aug;15(4):290-7.
144. Boehlen F, Hohlfeld P, Extermann P, de Moerloose P, Maternal antiplatelet antibodies in predicting risk of neonatal thrombocytopenia. *Obstet Gynecol*, 1999. Feb; 93(2):169-73.
145. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin: Number 6, September 1999: Thrombocytopenia in Pregnancy.
146. Poggi SH, Hematologic disease and pregnancy. *eMedicine*, 2002.
147. Lescale K, Eddleman KA, Cines DB, Antiplatelet antibody testing in thrombocytopenic pregnant women. *Am J Obstet Gynecol*, 1996. Mar;174(3):1014-8.
148. Burrows R, Kelton JG, Fetal thrombocytopenia and its relation to maternal thrombocytopenia. *N Engl J Med*, 1993. Nov 11;329(20):1463-6.
149. Samuels P, Bussel JB, Braitman LE, Estimation of the risk of thrombocytopenia in the offspring of pregnant women with presumed immune thrombocytopenic purpura. *N Engl J Med*, 1990. Jul 26;323(4):229-35.
150. Sharif B, Kuban K, Prenatal intracranial hemorrhage and neurologic complications in alloimmune thrombocytopenia. *J Child Neurol*, 2001. Nov;16(11):838-42.
151. Johnson J, Samuels P, Review of autoimmune thrombocytopenia: Pathogenesis, diagnosis, and management in pregnancy. *Clin Obstet Gynecol*, 1999. Jun;42(2):317-26.
152. Kaplan C, Immune thrombocytopenia in the foetus and the newborn: Diagnosis and therapy. *Transfus Clin Biol*, 2001. Jun;8(3):311-4.
153. Bussel J, Alloimmune thrombocytopenia in the fetus and newborn. *Semin Thromb Hemost*, 2001. Jun;27(3):245-52.
154. Overton T, Duncan KR, Jolly M, Letsky E, Fisk NM, Serial aggressive platelet transfusion for fetal alloimmune thrombocytopenia: platelet dynamics and perinatal outcome. *Am J Obstet Gynecol*, 2002. Apr;186(4):826-31.
155. Tiblad E, Olsson I, Petersson K, Shanwell A, Winiarski J, Wolff K, Westgren M, Experiences with fetomaternal alloimmune thrombocytopenia at a Swedish hospital over a 10-year period. *Acta Obstet Gynecol Scand*, 2003. Sep;82(9):803-6.
156. Radder C, Brand A, Kanhai HH, A less invasive treatment strategy to prevent intracranial hemorrhage in fetal and neonatal alloimmune thrombocytopenia. *Am J Obstet Gynecol*, 2001. Sep;185(3):683-8.
157. Rosen M, Dickinson JC, Westhoff CL, Vaginal birth after cesarean: A meta-analysis of morbidity and mortality. *Obstet Gynecol*, 1991. Mar;77(3):465-70.
158. Molloy B, Shirl O, Duignan NM, Delivery after cesarean section: Review of 2176 consecutive cases. *Br Med J (Clin Res Ed)*, 1987. Jun 29;294(6588):1645-7.
159. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin Number 54, July 2004: Vaginal Birth after Previous Cesarean Delivery, 2004.
160. Bayer-Zwirello L, O'Grady JP, Patel SS, ACOG's 1999 VBAC guidelines: A survey of western Massachusetts obstetric services. *Obstet Gynecol*, 2000. 95(4 Suppl):S 73.
161. Smith G, Pell JP, Pasupathy D, Dobbie R, Factors predisposing to perinatal death related to uterine rupture during attempted vaginal birth after caesarean section: Retrospective cohort study. *Br Med J*, 2004. Aug;329(7462):375.
162. Phelan JP, Clark SI, Diaz F, Vaginal birth after cesarean. *Am J Obstet Gynecol*, 1987. 157:1510-15.
163. Hender I, Bujold E, Effect of prior vaginal delivery or prior vaginal birth after cesarean delivery on obstetric outcomes in women undergoing trial of labor. *Obstet Gynecol*, 2004. Aug;104(2):273-7.

164. Phelan J, Uterine rupture. *Clin Obstet Gynecol*, 1990. 33:432-7.
165. Chauhan S, Martin JN Jr, Henrich CE, Morrison JC, Magann EF, Maternal and perinatal complications with uterine rupture in 142,075 patients who attempted vaginal birth after cesarean delivery: A review of the literature. *Am J Obstet Gynecol*, 2003. 189:408-17.
166. Guise JM, McDonagh MS, Osterweil P, Nygren P, Chan BKS, Helfand M, Systematic review of the incidence and consequences of uterine rupture in women with previous cesarean section. *Br Med J*, 2004. Jul 3;329(7456):19-23.
167. Flamm BL, Vaginal birth after cesarean (VBAC). *Best Pract Res Clin Obstet Gynaecol*, 2001. 15:81-92.
168. Flamm BL, Newman LA, Thomas SJ, Fallon D, Yoshida MM, Vaginal birth after cesarean delivery: results of a 5-year multicenter collaborative study. *Obstet Gynecol*, 1990. Nov;76(5 Pt 1):750-4.
169. Miller D, Diaz G, Paul RH, Vaginal birth after cesarean: A 10-year experience. *Obstet Gynecol*, 1994. Aug;84(2):255-8.
170. McMahon ML, Luther ER, Bowes WA Jr, Olshan AF, Comparison of a trial of labor with an elective second cesarean section. *N Engl J Med*, 1996. Sep 5;335(10):689-95.
171. Chauchan S, Magann EF, Wiggs CD, Barrilleaux PS, Martin JN Jr, Pregnancy after classic cesarean delivery. *Obstet Gynecol*, 2002. Nov;100(5 Pt 1):946-50.
172. Rageth JC, Juzi C, Grossenbacher H, Delivery after previous cesarean: A risk evaluation: Swiss Working Group of Obstetric and Gynecologic Institutions. *Obstet Gynecol*, 1999. Mar;93(3):332-7.
173. Lyndon-Rochelle M, Holt VI, Martin DP, Easterling TR, Association between method of delivery and maternal rehospitalization. *JAMA*, 2000. May;283(18):2411-6.
174. Guise JM, Vaginal delivery after cesarean section: determining thresholds for risks requires more than uterine rupture rates. *Br Med J*, 2004. Aug 14;329:359-60.
175. Caughey A, Shipp TD, Repke JT, Zelop C, Coehn A, Lieberman E, Trial of labor after cesarean delivery: The effect of previous vaginal delivery. *Am J Obstet Gynecol*, 1998. Oct;179(4):938-41.
176. Zelop CM, Shipp TD, Repke JT, Cohen A, Lieberman E, Effect of previous vaginal delivery on the risk of uterine rupture during a subsequent trial of labor. *Am J Obstet Gynecol*, 2000. Nov;183(5):1184-6.
177. Cowan R, Kinch RA, Ellis GB, Andersen R, Trial of labor following cesarean delivery. *Obstet Gynecol*, 1994. 83:933-6.
178. Paul R, Phelan JP, Yeh Sy, Trial of labor in the patient with a prior cesarean birth. *Am J Obstet Gynecol*, 1985. Feb 1;151(3):297-304.
179. Flamm B, Goings FR, Fuelberth NJ, Fischermann E, Jones C, Hersh E, Oxytocin during labor after previous cesarean section: Results of a multicenter study. *Obstet Gynecol*, 1987. Nov;70(5):709-12.
180. Blanchette H, Blanchette M, McCabe J, Vincent S, Is vaginal birth after cesarean safe? Experience at a community hospital. *Am J Obstet Gynecol*, 2001. Jun;184(7):1478-87.
181. Stovall T, Shaver DC, Solomon SK, Anderson GD, Trial of labor in previous cesarean section patients, excluding classical cesarean sections. *Obstet Gynecol*, 1987. Nov;70(5):713-7.
182. Lydon-Rochelle M, Holt VL, Easterling TR, Martin DP, Risk of uterine rupture during labor among women with a prior cesarean delivery. *N Engl J Med*, 2001. Jul;345(1):3-8.
183. Mozurkewich EL, Hutton EK, Elective repeat cesarean delivery versus trial of labor: A meta-analysis of the literature from 1989 to 1999. *Am J Obstet Gynecol*, 2000. Nov;183(5):1187-97.
184. Deutchman M, Roberts RG, VBAC: Protecting patients, defending doctors. *Am Fam Phys*, 2003. Mar 1;67(5):931-2; 935-6.
185. Allen V, O'Connell CM, Liston RM, Baskett TF, Maternal morbidity associated with cesarean delivery without labor compared with spontaneous onset of labor at term. *Obstet Gynecol*, 2001. Sep;102(3):477-82.
186. Phelan J, Clark SL (eds), *Cesarean Delivery*. New York: Elsevier, 1988.
187. Ophir E, Oetlinger M, Yagoda A, Markovits Y, Rojansky N, Shapiro H, Breech presentation after cesarean section: Always a section? *Am J Obstet Gynecol*, 1989. Jul;161(1):25-8.
188. Flamm B, Fried MW, Lonky NM, Guiles WS, External cephalic version after previous cesarean section. *Am J Obstet Gynecol*, 1991. Aug; 165(2):370-2.
189. Cragin EB, Conservatism in obstetrics. *NY Med J*, 1916. 104:1-3.

190. Shimonovitz S, Botosneano A, Hochner-Celnikier D, Successful first vaginal birth after cesarean section: A predictor of reduced risk for uterine rupture in subsequent deliveries. *Isr Med Assoc J*, 2000. Jul;2(7):526–8.
191. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin: Number 10, November, 1999: Induction of Labor. 1999.
192. Golde S, Tahilramaney MP, Platt LD, Use of ultrasound to predict fetal lung maturity in 247 consecutive elective cesarean deliveries. *J Reprod Med*, 1984. Jan;29(1):9–11.
193. Tahilramaney M, Platt LD, Golde SH, The use of femur length measured by ultrasound to predict fetal maturity. *J Perinatol*, 1991. Jun;11(2):157–60.
194. Alvarez J, Richardson DK, Ludmir J, Prediction of respiratory distress syndrome by the novel dipalmitoyl phosphatidylcholine test. *Obstet Gynecol*, 1996. Mar;87(3):429–33.
195. Parvin C, Kaplan LA, Chapman JF, McManamon TG, Gronowski AM, Predicting respiratory distress syndrome using gestational age and fetal lung maturity by fluorescent polarization. *Am J Obstet Gynecol*, 2005. Jan;192(1):199–207.
196. Winn-McMillan T, Karon BS, Comparison of the TDx-FLM II and lecithin-to-sphingomyelin ratio assays in predicting fetal lung maturity. *Am J Obstet Gynecol*, 2005. Sep;193(3 Pt 1):778–82.
197. Karcher R, Sykes E, Batton D, Uddin Z, Ross G, Hockman E, Shade GH Jr, Gestational age-specific predicted risk of neonatal respiratory distress syndrome using lamellar body count and surfactant-to-albumin ratio in amniotic fluid. *Am J Obstet Gynecol*, 2005. Nov;193(5):1680–4.
198. Dubin S, Assessment of fetal lung maturity: Practice parameter. *Am J Clin Path*, 1998. Dec;110(6):723–32.
199. Minkoff H, Chervenak FA, Elective primary cesarean delivery. *N Engl J Med*, 2003. Mar 6;348(10):946–50.
200. Minkoff H, Powderly KR, Chervenak F, McCullough LB, Ethical dimensions of elective primary cesarean delivery. *Obstet Gynecol*, 2004. Feb;103(2):387–92.
201. American College of Obstetricians and Gynecologists, ACOG Committee Opinion Number 289: Surgery and Patient Choice: The Ethics of Decision Making. *Obstet Gynecol*, 2003. 102: 1101–6.
202. Hannah M, Planned elective cesarean section: A reasonable choice for some women? *Can Med Assoc J*, 2004. Mar 2;170(5):813–4.
203. National Institutes of Health: Department of Health and Human Services. In *Cesarean Delivery on Maternal Request*: March 27–29, 2006. 2006. Bethesda, Maryland.
204. Anderson GM, Making sense of rising caesarean section rates [editorial]. *Br Med J*, 2004. Sep 25; 329(7468):696–7.
205. Sharma G, Is patient-choice primary cesarean rational? *OBG Management*, 2006. May; 18(5).
206. Hildingsson I, Waldenstrom U, Radestad I, Few women wish to be delivered by caesarean section. *Br J Obstet Gynaecol*, 2002. Jun;109(6):618–23.
207. Wax J, Cartin A, Pinette MG, Blackstone J, Patient choice cesarean: An evidence-based review. *Obstet Gynecol Surv*, 2004. Aug;59(8):601–16.
208. Wu J, Hundley AF, Visco AG, Elective primary cesarean delivery: Attitudes of urogynecology and maternal-fetal medicine specialists. *Obstet Gynecol*, 2005. Feb;105(2):301–6.
209. Bergholt T, Ostberg B, Legarth J, Weber T, Danish obstetricians' personal preference and general attitude to elective cesarean section on maternal request: A nation-wide postal survey. *Acta Obstet Gynecol Scand*, 2004. Mar;83(3):262–6.
210. Cotzias C, Paterson-Brown S, Fisk NM, Obstetricians say yes to maternal request for elective caesarean section: A survey of current opinion. *Eur J Obstet Gynecol Reprod Biol*, 2001. Jul;97(1):15–6.
211. Faas-Fehervary P, Schwarz K, Bauer L, Melchert F, Caesarean section on demand: Influence of personal birth experience and working environment on attitude of German gynaecologists. *Eur J Obstet Gynecol Reprod Biol*, 2005. Oct 1;122(2): 162–6.
212. Ghetti C, Chan BK, Guise JM, Physicians' responses to patient-requested Cesarean delivery. *Birth*, 2004. Dec;31(4):280–4.
213. Society of Obstetricians and Gynaecologists of Canada, SOGC Advisory: March 10, 2004: C-Sections on Demand – SOGC's Position.
214. Simpson K, Thorman KE, Obstetric "conveniences": Elective induction of labor, cesarean birth on demand, and other potentially unnecessary interventions. *J Perinat Neonatal Nurs*, 2005. Jun; 19(2):134–44.

215. Cardosi RJ, Porter KB, Cesarean delivery of twins during maternal cardiopulmonary arrest. *Obstet Gynecol*, 1998. Oct;92(4 Pt 2):695-7.
216. O'Connor R, Sevarino FB, Cardiopulmonary arrest in the pregnant patient: A report of a successful resuscitation. *J Clin Anesth*, 1994. Jan-Feb;6(1):66-8.
217. Lurie S, Mamet Y, Cesarean delivery during maternal cardiopulmonary resuscitation for status asthmaticus. *Emerg Med J*, 2003. May;20(3):296-7.
218. DePace N, Betesh JS, Kolter MN, 'Postmortem' cesarean section with recovery of both mother and offspring. *JAMA*, 1982. Aug 27;248(8):971-3.
219. Lopez-Zeno J, Carlo WA, O'Grady JP, Fanaroff AA, Infant survival following delayed postmortem cesarean delivery. *Obstet Gynecol*, 1990. 76:991-2.
220. Yamani Zamzami TY, Indication of emergency peripartum hysterectomy: review of 17 cases. *Arch Gynecol Obstet*, 2003. Aug;268(3):131-5. Epub 2003 May 20. PMID: 12756583.
221. Awwad J, Azar GB, Aouad AT, Raad J, Karam KS, Postmortem cesarean section following maternal blast injury [case report]. *J Trauma*, 1994. Feb;36(2):260-1.
222. Katz MV, Dotters DJ, Droegemueller W, Perimortem cesarean delivery. *Obstet Gynecol*, 1986. Oct;68(4):571-6.
223. Whitten M, Irvine LM, Postmortem and perimortem cesarean section. *J R Soc Med*, 2000. Jan;93(1):6-9.
224. Thomas R, Sotheran W, Postmortem and perimortem cesarean section. *J Royal Soc Med*, 2000. Apr;93(4):215-6.
225. Page-Rodriguez A, Gonzalez-Sanchez JA, Perimortem cesarean section of twin pregnancy: Case report and review of the literature. *Acad Emerg Med*, 1999. Oct;6(10):1072-4.
226. Onwuhafua P, Dying undelivered. *J Obstet Gynaecol*, 2002. Mar; 22(2):155-8.
227. Esposito M, DeLony R, Goldstein PJ, Postmortem cesarean section with infant survival: A case report of an HIV-infected patient. *Md Med J*, 1997. Oct;46(9):467-70.
228. Bloom S, Spong CY, Weiner SJ, Landon MB, Rouse DJ, Varner MW, Moawad AF, Caritis SN, Harper M, Wapner RJ, Sorokin Y, Miodovnik M, O'Sullivan M J, Sibai B, Langer O, Gabbe SG, for the National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network, Complications of anesthesia for cesarean delivery. *Obstet Gynecol*, 2005. Aug;106(2):281-7.
229. McVay P, Hoag RW, Hoag MS, Toy PT, Safety and use of autogenous blood donation during the third trimester of pregnancy. *Am J Obstet Gynecol*, 1989. Jun;160(6):1479-88.
230. Rebarber A, Lonser R, Jackson S, Copel JA, Sipes S, The safety of intraoperative autologous blood collection and autotransfusion during cesarean section. *Am J Obstet Gynecol*, 1998. Sep;179(3 Pt 1):715-20.
231. Wilkinson C, Enkin MW, (1996) Lateral tilt for caesarean section (Cochrane Review). *Cochrane Database Syst Rev*. DOI: 10.1002/14651858.CD000120.
232. Eckstein K, Marx GF, Aorto-caval compression and uterine displacement. *Anesthesiology*, 1974. Jan;40(1):92-6.
233. Abuhamad A, O'Sullivan MJ, Operative techniques for cesarean section. In Plauche SC, Morrison JC, O'Sullivan MJ (eds): *Surgical Obstetrics*. Philadelphia: WB Saunders, 1992, pp. 417-29.
234. Alexander J, Fischer JE, Boyajian M, Palmquist J, Morris MU, The influence of hair-removal methods on wound infections. *Arch Surg*, 1983. Mar;118(3):347-52.
235. Hendrix S, Schimp V, Martin J, Singh A, Kruger M, McNelley SG, The legendary superior strength of the Pfannenstiel incision: A myth? *Am J Obstet Gynecol*, 2000. Jun;182(6):1446-51.
236. Wall P, Deucy EE, Glantz JC, Pressman EK, Vertical skin incisions and wound complications in the obese parturient. *Obstet Gynecol*, 2003. Nov;102(5 Pt 1):952-6.
237. Chauhan, SP, Henrichs CE (2002) Choosing the best incision for cesarean delivery. *OBG Management*, 2002. Nov;14(11):1-8.
238. Houston M, Raynor BD, Postoperative morbidity in the morbidly obese parturient woman: Supraumbilical and low transverse approaches. *Am J Obstet Gynecol*, 2000. May;182(5):1033-5.
239. Finan M, Mastrogiannis DS, Spellacy WN, The Allis test for easy cesarean delivery. *Am J Obstet Gynecol*, 1991. Mar;164(3):772-5.
240. Ayers J, Morley GW, Surgical incision for cesarean section. *Obstet Gynecol*, 1987. Nov;70(5):706-8.

241. Boyle J, Gabbe SG, T and J vertical extensions in low transverse cesarean births. *Obstet Gynecol*, 1996. Feb;87(2):238-43.
242. Patterson L, O'Connell CM, Baskett TF, Maternal and perinatal morbidity associated with classic and inverted T cesarean incisions. *Obstet Gynecol*, 2002. Oct;100(4):633-7.
243. Shipp T, Zelop CM, Repke JT, Cohen A, Caughey AB, Lieberman E, Intrapartum uterine rupture and dehiscence in patients with prior lower uterine segment vertical and transverse incisions. *Obstet Gynecol*, 1999. Nov;94(5 Pt 1):735-40.
244. Naef R, Ray MA, Chauhan SP, Roach H, Blake PG, Martin JN. Trial of labor after cesarean delivery with a lower-segment, vertical uterine incision: Is it safe? *Am J Obstet Gynecol*, 1995. Jun;172(6):1666-73.
245. Halperin M, Moore DC, Hannah WJ, Classical versus low-segment transverse incision for preterm caesarean section: Maternal complications and outcome of subsequent pregnancies. *Br J Obstet Gynaecol*, 1988. Oct;95(10):990-6.
246. Yasin S, Walton DL, O'Sullivan MJ, Problems encountered during cesarean delivery. In Plauche WC, Morrison JC, O'Sullivan MJ (eds): *Surgical Obstetrics*. Philadelphia: WB Saunders, 1992.
247. Landesman R, Graber ED, Abdominovaginal delivery: Modification of the cesarean section operation to facilitate delivery of the impacted head. *Am J Obstet Gynecol*, 1984. Mar 15;148(6):707-10.
248. Murless B, Lower segment cesarean section: A new head extractor? *Br Med J*, 1945. 1234.
249. Blickstein I, Difficult delivery of the impacted fetal head during cesarean section: Intraoperative disengagement dystocia. *J Perinat Med*, 2004. 32(4): 465-9.
250. Wilkinson C, Enkin MW, Manual removal of placenta at caesarean section (Cochrane Review). *Cochrane Database Syst Rev*, 1996. DOI: 10.1002/14651858.CD000130.
251. Magann E, Dodson MK, Allbert JR, McCurdy CM Jr, Martin RW, Morrison JC, Blood loss at time of cesarean section by method of placental removal and exteriorization versus in situ repair of the uterine incision. *Surg Gynecol Obstet*, 1993. 177(4):389-92.
252. Hidar S, Jennane TM, Bouguizane S, Lassoued L, Bibi M, Khairi H, The effect of placental removal method at cesarean delivery on perioperative hemorrhage: A randomized clinical trial. *Eur J Obstet Gynecol Reprod Biol*, 2004. Dec 1;117(2):179-82.
253. Jacobs-Jokhan DHG, Extra-abdominal versus intra-abdominal repair of the uterine incision at caesarean section (Cochrane Review). *Cochrane Database Syst Rev*, 2004.
254. Hershey D, Quilligan EJ, Extraabdominal uterine exteriorization and cesarean delivery. *Obstet Gynecol*, 1978. Aug;52(2):189-92.
255. Enkin M, Wilkinson C, Single- versus two-layer suturing for closing the uterine incision at caesarean section (Cochrane Review). *Cochrane Database Syst Rev*, 1996.
256. Chatterjee S, Scar endometriosis: A clinicopathologic study of 17 cases *Obstet Gynecol*, 1980. Jul;56(1):81-4.
257. Hoskins I, Ordorica SA, Frieden FJ, Young BK, Performance of cesarean section using absorbable staples. *Surg Gynecol Obstet*, 1991. Feb;172(2): 108-12.
258. Martens M, Reduction of infectious morbidity with uterine stapling device. *Adv Ther*, 1990. Mar-Apr;7(2):105-6.
259. Burkett G, Jensen LP, Lai A, O'Sullivan MJ, Yasin S, Beydoun S, McLeod AG, Evaluation of surgical staples in cesarean section. *Am J Obstet Gynecol*, 1989. Sep;161(3):540-5.
260. van Dongen P, Nijhuis JG, Jongsma HW, Reduced blood loss during caesarean section due to a controlled stapling technique. *Eur J Obstet Gynecol Reprod Biol*, 1989. Aug;32(2):95-102.
261. Bond S, Harrison MR, Slotnick RN, Anderson J, Flake AW, Adnizk NS, Cesarean delivery and hysterectomy using an absorbable stapling device. *Obstet Gynecol*, 1989. Jul;74(1):25-8.
262. Wilkinson CE, MW, Absorbable staples for uterine incision at caesarean section. *The Cochrane Database of Systematic Reviews* 1996, 2006(2).
263. Villeneuve M, Khalife S, Marcoux S, Blanchet P, Surgical staples in cesarean section: A randomized controlled trial. *Am J Obstet Gynecol*, 1990. Nov;163(5 Pt 1):1641-6.
264. Gilson GJ, Kephart WH, Izquierdo LA, Joffe GM, Qualls CR, Curet LB, Comparison of absorbable uterine staples and traditional hysterotomy during cesarean delivery. *Obstet Gynecol*, 1996. Mar; 87(3):384-8.



265. Harrigill F, Miller HG, Haynes DE, The effect of intraabdominal irrigation at cesarean delivery on maternal morbidity: A randomized trial. *Obstet Gynecol*, 2003. Jan;101(1):80-5.
266. Anderson E, Gates S, Techniques and materials for closure of the abdominal wall in caesarean section. *Cochrane Database Syst Rev*, 2004, (4).
267. Perinatal Trials Service: National Perinatal Epidemiology Unit Institute of Health Sciences, CESAR Study 2005; [www.npeu.ox.ac.uk/caesar/](http://www.npeu.ox.ac.uk/caesar/).
268. Ramsey P, White AM, Guinn DA, Lu GC, Ramin SM, Davies JK, Neely CL, Newby C, Fonseca L, Case AS, Kaslow RA, Rouse DJ, Hauth JC, Subcutaneous tissue reapproximation, alone or in combination with drain, in obese women undergoing cesarean delivery. *Obstet Gynecol*, 2005. May;105(5):967-73.
269. Magann E, Chauhan SP, Rodts-Palenik S, Bufkin S, Martin JN Jr, Morrison JC, Subcutaneous stitch closure versus subcutaneous drain to prevent wound disruption after cesarean delivery: a randomized clinical trial. *Am J Obstet Gynecol*, 2002. Jun;186(6):1119-23.
270. Gates S, Anderson ER, Wound drainage for caesarean section (Cochrane Review) Issue 1. *Cochrane Database Syst Rev*. 2005.
271. Frishman G, Schwartz T, Hogan JW, Closure of Pfannenstiel skin incisions: Staples vs. subcuticular suture. *J Reprod Med*, 1997. Oct;42(10):627-30.
272. Alderdice F, McKenna D, Dornan J, Techniques and materials for skin closure in caesarean section. *Cochrane Database Syst Rev*. 2003.
273. Chelmow D, Rodriguez EJ, Sabatini MM, Suture closure of subcutaneous fat and wound disruption after cesarean delivery: A meta-analysis. *Obstet Gynecol*, 2004. May;103(3 Pt 1):974-80.
274. Myers S, Bennett TL, Incidence of significant adhesions at repeat cesarean section and the relationship to method of prior peritoneal closure. *J Reprod Med*, 2005. Sep;50(9):659-62.
275. Lyell D, Caughey AB, Hu E, Daniels K, Peritoneal closure at primary cesarean delivery and adhesions. *Obstet Gynecol*, 2005. Aug;106(2):275-80.
276. Bamigboye A, Hofmeyr GJ, Non-closure of peritoneal surfaces at caesarean section - A systematic review. *S Afr Med J*, 2005. Feb;95(2):123-6.
277. Tulandi T, Al-Jaroudi D, Nonclosure of peritoneum: A reappraisal. *Am J Obstet Gynecol*, 2003. Aug;189(2):609-12.
278. Irion O, Luzuy F, Beguin F, Nonclosure of the visceral and parietal peritoneum at caesarean section: A randomised controlled trial. *Br J Obstet Gynaecol*, 1996. Jul;103(7):690-4.
279. Nagele F, Karas H, Spitzer D, Staudach A, Karasegh S, Beck A, Husslein P, Closure or nonclosure of the visceral peritoneum at cesarean delivery. *Am J Obstet Gynecol*, 1996. Apr;174(4):1366-70.
280. Pietrantoni M, Parsons MT, O'Brien WR, Collins E, Knuppel RA, Spellacy WN, Peritoneal closure or non-closure at cesarean. *Obstet Gynecol*, 1991. Feb;77(2):293-6.
281. Cheong Y, Bajekal N, Li TC, Peritoneal closure - to close or not to close. *Hum Reprod*, 2001. Aug;16(8):1548-51.
282. Duffy D, di Zerega GS, Is peritoneal closure necessary? *Obstet Gynecol Surv*, 1994. Dec;49(12):817-22.
283. Cheong Y, Laird SM, Li TC, Shelton JB, Ledger WL, Cooke ID, Peritoneal healing and adhesion formation/reformation. *Hum Reprod Update*, 2001. Nov-Dec;7(6):556-66.
284. Weerawetwat W, Buranawanich S, Kanawong M, Closure vs. non-closure of the visceral and parietal peritoneum at cesarean delivery: 16-year study. *J Med Assoc Thai*, 2004. Sep;87(9):1007-11.
285. Elkins T, Stovall TG, Warren J, Ling FW, Meyer NL, A histologic evaluation of peritoneal injury and repair: Implications for adhesion formation. *Obstet Gynecol*, 1987. Aug;70(2):225-8.
286. Ellis H, Heddle R, Does the peritoneum need to be closed at laparotomy? *Br J Surg*, 1977. Oct;64(10):733-6.
287. Menzies D, Postoperative adhesions: Their treatment and relevance in clinical practice. *Ann Royal Coll Surg Engl*, 1993. 75:147-53.
288. El-Mowafi DM, Diamond MP, Gynecologic surgery and subsequent bowel obstruction. *Geneva Found Med Ed Res*. DOI: [www.gfmer.ch](http://www.gfmer.ch).
289. Morales K, Gordon M, Bates GW, Post cesarean adhesions may compromise infant well-being [abstr. no. 18]. *The American College of Obstetricians and Gynecologists, Armed Forces District Meeting*, Nov 2, 2005.
290. Cheong Y, Laird SM, Shelton JB, Ledger WL, Li TC, Cooke ID, The correlation of adhesions and peritoneal fluid cytokine concentrations: A pilot study. *Hum Reprod*, 2002. 17(4):1039-45.

291. Haney, A, Doty E, Expanded polytetrafluoroethylene (Gore-Tex Surgical Membrane) is superior to oxidized regenerated cellulose (Interceed TC7+) in preventing adhesions. *Fertil Steril*, 1995. May;63(5):1021-8.
292. Diamond M, Reduction of adhesions after uterine myomectomy by Seprafilm membrane (HAL-F): A blinded, prospective, randomized, multicenter clinical study. The Seprafilm Adhesion Study Group. *Fertil Steril*, 1996. Dec;66(6):906-10.
293. Farquhar C, Vandekerckhove P, Watson A, Vail A, Wiseman D, Barrier agents for preventing adhesions after surgery for subfertility (Cochrane Review). *Cochrane Database Syst Rev*, 1999.
294. Interceed (TC7 +) Adhesion Barrier Study Group, Prevention of postsurgical adhesions by Interceed (TC7+), an absorbable adhesion barrier: A prospective randomized multicenter clinical study. *Fertil Steril*, 1989. 51:933-9.
295. Demirel Y, GURSOY S, Duran B, Erden O, Cetin M, Balta O, Cetin A, Closure or nonclosure of the peritoneum at gynecological operations: Effect on postoperative pain. *Saudi Med J*, 2005. Jun;26(6):964-8.
296. Chanrachakul B, Hamontri S, Herabutya Y, A randomized comparison of postcesarean pain between closure and nonclosure of peritoneum. *Eur J Obstet Gynecol Reprod Biol*, 2002. Feb 10;101(1):31-5.
297. Roset E, Boulvain M, Irion O, Nonclosure of the peritoneum during caesarean section: Long-term follow-up of a randomised controlled trial. *Eur J Obstet Gynecol Reprod Biol*, 2003. May 1;108(1):40-4.
298. Killian C, Graffunder EM, Vinciguerra TJ, Venezia RA, Risk factors for surgical-site infections following cesarean section. *Infect Control Hosp Epidemiol*, 2001. Oct;22(10):613-7.
299. Wen S, Rusen ID, Walker M, Liston R, Kramer MS, Baskett T, for the Maternal Health Study Group, Canadian Perinatal Surveillance System, Comparison of maternal mortality and morbidity between trial of labor and elective cesarean section among women with previous cesarean delivery. *Am J Obstet Gynecol*, 2004. Oct;191(4):1263-9.
300. Nielsen T, Hokegard KH, Postoperative cesarean section morbidity: A prospective study. *Am J Obstet Gynecol*, 1983. Aug 15;146(8):911-6.
301. Couto R, Pedrosa TMG, Nogueira JM, Gomes DL, Neto MF, Rezende NA, Post-discharge surveillance and infection rates in obstetric patients. *Int J Gynaecol Obstet*, 1998. Jun;61(3):227-31.
302. Burrows LJ, Meyn LA, Weber AM, Maternal morbidity associated with vaginal versus cesarean delivery. *Obstet Gynecol*, 2004. May;103(5 Pt 1):907-12.
303. Martens M, Kolrud BL, Faro S, Maccato M, Hamill H, Development of wound infection or separation after cesarean delivery: Prospective evaluation of 2,431 cases. *J Reprod Med*, 1995. Mar;40(3):171-5.
304. Liu S, Heaman M, Joseph KS, Liston RM, Huang L, Sauve R, Kramer MS, for the Maternal Health Study Group of the Canadian Perinatal Surveillance System, Risk of maternal postpartum readmission associated with mode of delivery. *Obstet Gynecol*, 2005. Apr;105(4):836-42.
305. Lydon-Rochelle M, Holt VL, Martin DP, Easterling TR, Association between method of delivery and maternal rehospitalization. *JAMA*, 2000. May 10;283(18):2411-6.
306. Phipps M, Watabe B, Clemons JL, Weitzen S, Myers DL, Risk factors for bladder injury during cesarean delivery. *Obstet Gynecol*, 2005. Jan;105(1):156-60.
307. Nielsen T, Hokegard KH, Cesarean section and intraoperative surgical complications. *Acta Obstet Gynecol Scand*, 1984. 63(2):103-8.
308. Wechter M, Pearlman MD, Hartmann KE, Reclosure of the disrupted laparotomy wound: A systemic review. *Obstet Gynecol*, 2005. 106(2):376-83.
309. Gerber A, Accidental incision of the fetus during cesarean section delivery. *Int J Gynaecol Obstet*, 1974. 12:46-8.
310. Smith J, Hernandez C, Wax JR, Fetal laceration injury at cesarean delivery. *Obstet Gynecol*, 1997. Sep;90(3):344-6.
311. Dessole S, Cosmi E, Balata A, Uras L, Caserta D, Capobianco G, Ambrosini G, Accidental fetal lacerations during cesarean delivery: Experience in an Italian level III university hospital. *Am J Obstet Gynecol*, 2004. Nov;191(5):1673-7.
312. Okaro J, Anya SE, Accidental incision of the fetus at cesarean section. *Niger J Med*, 2004. Apr-Jun;13(2):211.

313. Haas D, Ayers AW, Laceration injury at cesarean section. *J Matern Fetal Neonatal Med*, 2002. Mar; 11(3):196–8.
314. Holmgren G, Sjöholm L, Stark M, The Misgav Ladach method for cesarean section: Method description. *Acta Obstet Gynecol Scand*, 1999. Aug; 78(7):615–21.
315. Franchi M, Ghezzi F, Balestreri D, Beretta P, Maymon E, Miglierina M, Bolis PF, A randomized clinical trial of two surgical techniques for cesarean section. *Am J Perinatol*, 1998. 15(10): 589–94.
316. Bjorklund K, Kimaro M, Urassa E, Lindmark G, Introduction of the Misgav Ladach caesarean section at an African tertiary centre: A randomised controlled trial. *Br J Obstet Gynaecol*, 2000. Feb; 10(2):209–16.
317. Darj E, Nordstrom ML, The Misgav Ladach method for cesarean section compared to the Pfannenstiel method. *Acta Obstet Gynecol Scand*, 1999. Jan;78(1):37–41.
318. Fatusic Z, Kurjak A, Jasarevic E, Hafner T, The Misgav Ladach method – a step forward in operative technique in obstetrics. *J Perinat Med*, 2003. 31(5):395–8.
319. Ansaloni L, Brundisini R, Morion G, Kiura A, Prospective, randomized, comparative study of Misgav Ladach versus traditional cesarean section at Nazareth Hospital, Kenya. *World J Surg*, 2001. Sep;25(9):1164–72.
320. Magann E, Chauhan SP, Bufkin L, Field K, Roberts WE, Martin JN Jr, Intra-operative haemorrhage by blunt versus sharp expansion of the uterine incision at caesarean delivery: A randomised clinical trial. *Br J Obstet Gynaecol*, 2002. Apr;109(4):448–52.
321. Rodriguez A, Porter KB, O'Brien WF, Blunt versus sharp expansion of the uterine incision in low-segment transverse cesarean section. *Am J Obstet Gynecol*, 1994. Oct;171(4):1022–5.
322. Hauth J, Owen J, Davis RO, Transverse uterine incision closure: One versus two layers. *Am J Obstet Gynecol*, 1992. Oct;167(4 Pt 1):1108–11.
323. Jelsema R, Wittingen JA, Vander-Kolk KJ, Continuous, nonlocking, single-layer repair of the low transverse uterine incision. *J Reprod Med*, 1993. May;38(5):393–6.
324. Hohlagschwandtner M, Chalubinski K, Nather A, Husslein P, Joura EA, Continuous vs. interrupted sutures for single-layer closure of uterine incision at cesarean section. *Arch Gynecol Obstet*, 2003. Apr;268(1):26–8.
325. Chapman SJ, Owen J, Hauth JC, One- versus two-layer closure of a low transverse cesarean: The next pregnancy. *Obstet Gynecol*, 1997. Jan;89(1):16–18.
326. Bujold E., Bujold C, Hamilton EF, Harel F, Gauthier RJ, The impact of a single-layer or double-layer closure on uterine rupture. *Am J Obstet Gynecol*, 2002. Jun;186(6):1326–30.
327. Durnwald C, Mercer B, Uterine rupture, perioperative and perinatal morbidity after single-layer and double-layer closure at cesarean delivery. *Am J Obstet Gynecol*, 2003. Oct;189(4): 925–9.
328. Frigoletto F, Ryan KJ, Phillippe M, Maternal mortality rate associated with cesarean delivery: An appraisal. *Am J Obstet Gynecol*, 1980. Apr 1;126 (7):969–70.
329. Smaill F, Hofmeyr GJ, Antibiotic prophylaxis for cesarean section (Cochrane Review). *Cochrane Database Syst Rev*, 2002.
330. Chelmow D, Ruehli MS, Huang E, Prophylactic use of antibiotics for nonlaboring patients undergoing cesarean delivery with intact membranes: A meta-analysis. *Am J Obstet Gynecol*, 2001. Mar; 184(4):656–61.
331. Andrews W, Hauth JC, Cliver SP, Savage K, Goldenberg RL, Randomized clinical trial of extended spectrum antibiotic prophylaxis with coverage for ureaplasma urealyticum to reduce post-cesarean delivery endometritis. *Obstet Gynecol*, 2003. 101(6):1183–9.
332. Stanco L, Schrimmer DB, Paul RH, Mishall DR Jr, Emergency peripartum hysterectomy and associated risk factors. *Am J Obstet Gynecol*, 1993. Mar;168(3 Pt 1):879–83.
333. Clark S, Yeh SY, Phelan JP, Bruce S, Paul RH, Emergency hysterectomy for the control of obstetric hemorrhage. *Obstet Gynecol*, 1984. Sep;64(3): 376–80.
334. Kastner E, Rigueroa R, Garry D, Maulik D, Emergency peripartum hysterectomy: Experience at a community teaching hospital. *Obstet Gynecol*, 2002. 99(6):971–5.
335. Miller D, Chollet JA, Goodwin TM, Clinical risk factors for placenta previa-placenta accreta. *Am J Obstet Gynecol*, 1997 Jul; 177. (1):210–4.
336. Baskett TF, Emergency obstetric hysterectomy. *J Obstet Gynaecol* 2003. Jul;23(4):353–5.

337. Plauche W, Gruich FG, Bourgeois MO, Hysterec-tomy at the time of cesarean section: Analysis of 108 cases. *Obstet Gynecol*, 1981. Oct;58(4):459–64.
338. Oleen MA, Marino JP, Controlling refractory atonic postpartum hemorrhage with Hemabate sterile solution. *Am J Obstet Gynecol*, 1990. Jan; 162(1):205–8.
339. Rouse D, Leindecker S, Landon M, Bloom SL, Varner MW, Moawad AH, Spong CY, Caritis SN, Harper M, Wapner RJ, Miodovnik M, O'Sullivan MJ, Sibai BM, Langer O, National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network, The MFMU Cesarean Registry: Uterine atony after primary cesarean delivery. *Am J Obstet Gynecol*, 2005. Sept;193(3 Pt 2):1056–60.
340. Suchartwatnachai C, Linasuitta V, Chaturachinda K, Obstetric hysterectomy: Ramathibodi's experience 1969–1987. *Int J Gynaecol Obstet*, 1991. Nov;36(3):183–6.
341. Yancey M, Harless FE, Benson W, Brady K, The perioperative morbidity of scheduled cesarean hysterectomy. *Obstet Gynecol*, 1993. Feb;81(2): 205–10.
342. Gonsoulin W, Kennedy RT, Guidry KH, Elective versus emergency cesarean hysterectomy cases in a residency program setting: A review of 129 cases from 1984–1988. *Am J Obstet Gynecol*, 1991. Jul; 165(1):91–4.
343. American College of Obstetricians, and Gynecol-ogists, ACOG Educational Bulletin: Number 76, October, 2006. Postpartum Hemorrhage.
344. Combs A, Murphey EL, Laros RK Jr, Factors associated with hemorrhage in cesarean deliveries. *Obstet Gynecol*, 1991. Jan;77(1):77–82.
345. International Confederation of Midwives (ICM) and International Federation of Gynecology and Obstetrics (FIGO), Management of the third stage of labour to prevent postpartum hemorrhage. *J Obstet Gynaecol Can*, 2003. Nov;136(11):952–3.
346. Clark S, Managing postpartum hemorrhage: Estab-lish a cause. *OBG Management*, 2002. Nov;14 (11):1–12.
347. Bonnar J, Massive obstetric haemorrhage. *Bal-lier's Clin Obstet Gynaecol*, 2000. 14(1):1–18.
348. Bouwmeester F, Bolte AC, van Geijn HP, Pharma-cological and surgical therapy for primary postpar-tum hemorrhage. *Curr Pharm Des*, 2005. 11(6): 759–73.
349. Miller S, Lester F, Hensleigh PA, Prevention and treatment of postpartum hemorrhage: New advances for low-resource settings. *J Midwifery Womens Health*, 2004. 49(4):283–92.
350. Hensleigh P, Anti-shock garment provides resusci-tation and haemostasis for obstetric haemorrhage. *Br J Obstet Gynaecol*, 2002. Dec;109:1377–84.
351. Tamizian O, Arulkumaran S, The surgical manage-ment of post-partum haemorrhage. *Best Pract Res Clin Obstet Gynaecol*, 2002. 16(1):81–98.
352. Shevell T, Malone FD, Management of obstet-ric hemorrhage. *Semin Perinatol*, 2003. 27(1):86–104.
353. Baskett TF, *Essential Management of Obstetric Emergencies*, ed 3. Redland, Bristol, England: Clinical Press Limited. 1999.
354. Baskett TF, Surgical management of severe obstet-ric hemorrhage: Experience with an obstetric hem-orrhage equipment tray. *J Obstet Gynaecol Can*, 2004. 26(9):805–8.
355. Li Y, Yin CS, Chen FM, Chao TC, A use-ful technique for the control of severe cesarean hemorrhage: report of three cases. *Chang Gung Med J*, 2002. 25(8):548–52.
356. Waters E, Surgical management of postpartum hemorrhage with particular reference to ligation of uterine arteries. *Am J Obstet Gynecol*, 1952. (5):1143–8.
357. O'Leary J, O'Leary JA, Uterine artery ligation in control of intractable postpartum hemorrhage. *Am J Obstet Gynecol*, 1966. Apr 1;94(7):920–4.
358. Hebisch G, Huch A, Vaginal uterine artery liga-tion avoids high blood loss and puerperal hysterec-tomy in postpartum hemorrhage. *Obstet Gynecol*, 2002. 100(3):574–8.
359. O'Leary J, Uterine artery ligation in the control of postcesarean hemorrhage. *J Reprod Med*, 1995. 40(3):189–93.
360. O'Leary J, Stop OB hemorrhage with uterine artery ligation. *Contemp Ob/Gyn*, 1986. 28:13–6.
361. Chou Y, Wang PH, Yuan CC, Yen YK, Liu WM, Laparoscopic bipolar coagulation of uterine ves-sels to manage delayed postpartum hemorrhage. *J Am Assoc Gynecol Laparosc*, 2002. Nov;9(4): 541–4.
362. Haier R, Control of postpartum hemorrhage with uterine packing. *Am J Obstet Gynecol*, 1993. 169(2):317–23.

363. Hsu S, Rodgers B, Lele A, Yeh J, Use of packing in obstetric hemorrhage of uterine origin. *J Reprod Med*, 2003. 48(2):69–71.
364. Druzin M, Packing of lower uterine segment for control of postcesarean bleeding in instances of placenta previa. *Surg Gynecol Obstet*, 1989. 169:543–5.
365. Bagga R, Jain V, Kalra J, Chopra S, Gopalan S, Uterovaginal packing with rolled gauze in postpartum hemorrhage. *Med Gen Med*, 2004. Feb 13;6(1):50.
366. B-Lynch C, Coker A, Lawal AH, Abu J, Cowen MJ, The B-Lynch surgical technique for the control of massive postpartum haemorrhage: An alternative to hysterectomy? Five cases reported. *Br J Obstet Gynaecol*, 1997. 104(3):372–5.
367. Hayman R, Arulkumaran S, Steer PJ, Uterine compression sutures: Surgical management of postpartum hemorrhage. *Obstet Gynecol*, 2002. 99(3):502–6.
368. Ferguson JN, Bourgeois FJ, Underwood PB Jr, B-Lynch suture for postpartum hemorrhage. *Obstet Gynecol*, 2000. Jun;95(6 Pt 2):1020–2.
369. Smith KL, Baskett TF, Uterine compression sutures as an alternative to hysterectomy for severe postpartum hemorrhage. *J Obstet Gynaecol Can*, 2003. Mar;25(3):197–200.
370. Cho J, Jun HS, Lee CN, Hemostatic suturing technique for uterine bleeding during cesarean delivery. *Obstet Gynecol*, 2000. 96(1):129–31.
371. Ochoa M, Allaire AD, Stitely ML, Pyometria after hemostatic square suture technique. *Obstet Gynecol*, 2002. 99(3):506–9.
372. Abu-Musa A, Seoud M, Suidan F, A new technique for control of placental site bleeding. *Int J Gynaecol Obstet*, 1998. 60:169–70.
373. Clark S, Phelan JP, Yeh SY, Bruce SR, Paul HR, Hypogastric artery ligations for obstetric hemorrhage. *Obstet Gynecol*, 1985. Sep;66(3):353–6.
374. Evans S, McShane P, The efficacy of internal iliac artery ligation in obstetric hemorrhage. *Surg Gynecol Obstet*, 1985. Mar;160(3):250–3.
375. Burchell C, Physiology of internal iliac artery ligation. *J Obstet Gynaecol Br Commonw*, 1968. 75:642–51.
376. O'Leary J, Pregnancy following uterine artery ligation. *Obstet Gynecol*, 1980. Jan;55(1):112–3.
377. Verspyck E, Resch B, Sergent F, Marpeau L, Surgical uterine devascularization for placenta accreta: Immediate and long-term follow-up. *Acta Obstet Gynecol Scand*, 2005. May;84(5):444–7.
378. Nizard J, Barrinque L, Frydman R, Fernandez H, Fertility and pregnancy outcomes following hypogastric artery ligation for severe post-partum haemorrhage. *Hum Reprod*, 2003. Apr;18(4):844–8.
379. Api M, Api O, Yala M, Fertility after B-Lynch suture and hypogastric artery ligation. *Fertil Steril*, 2005. Aug;84(2):509.
380. Hansch E, Chitkara U, McAlpine J, El-Sayed Y, Dake MD, Razavi MK, Pelvic arterial embolization for control of obstetric hemorrhage: A five-year experience. *Am J Obstet Gynecol*, 1999. 180(6 Pt 1):1454–60.
381. Deux J, Bazot M, LeBlanche AF, Tassart M, Khalil A, Berkane N, Uzan S, Boudghene F, Is selective embolization of uterine arteries a safe alternative to hysterectomy in patients with postpartum hemorrhage. *Am J Roentgenol*, 2001. 177(1):145–9.
382. Pelage JP, Repiquet D, Herbreteau D, Le Dref O, Houdart E, Jacob D, Kardache M, Schurando P, Truc JB, Rymer R, Secondary postpartum hemorrhage: Treatment with selective arterial embolization. *Radiology* 1999. Aug;212(2):385–9.
383. Pelage J, LeDref O, Jacob D, Soyer P, Herbreteau D, Rymer R, Selective arterial embolization of the uterine arteries in the management of intractable post-partum hemorrhage. *Acta Obstet Gynecol Scand*, 1999. 78(8):698–703.
384. Chung JW, Jeong HJ, Joh JH, Park JS, Jun JK, Kim SH, Percutaneous transcatheter angiographic embolization in the management of obstetric hemorrhage. *J Reprod Med*, 2003. Apr;48(4):268–76.
385. Yamashita Y, Harada M, Yamamoto H, Miyazaki T, Takahashi M, Miyazaki K, Okamura H, Transcatheter arterial embolization of obstetric and gynaecological bleeding: Efficacy and clinical outcome. *Br J Radiol*, 1994. 67(798):530–4.
386. Velling T, Brennan FJ, Hall LD, Watabe JT, Role of the interventional radiologist in treating obstetric-gynecologic pathology. *Am J Roentgenol*, 2000. Nov;175(5):1273–8.
387. Turner J, Embolization, Hemorrhage. *eMedicine*, 2005.topic = 760.
388. Vedantham S, Goodwin SC, McLucas B, Mohr G, Uterine artery embolization: An underused

- method of controlling pelvic hemorrhage. *Am J Obstet Gynecol*, 1997. 176(4):938-48.
389. Chitrit Y, Zafy S, Pelang JP, Ledref O, Khoury R, Caubel P, Amenorrhea due to partial uterine necrosis after uterine artery embolization for control of refractory postpartum hemorrhage. *Eur J Obstet Gynecol Reprod Biol*, DOI: 16024157.
  390. Ojala K, Perala J, Kariniemi J, Ranta P, Raudaskoski T, Tekay A, Arterial embolization and prophylactic catheterization for the treatment for severe obstetric hemorrhage. *Acta Obstet Gynecol Scand*, 2005. Nov;84(11):1075-80.
  391. Boulleret C, Chahid T, Gallot D, Mofid R, Tran Hai D, Ravel A, Garcier JM, Lemery D, Boyer L, Hypogastric arterial selective and superselective embolization for severe postpartum hemorrhage: A retrospective review of 36 cases. *Cardiovasc Intervent Radiol*, 2004. Jul-Aug;27(4):344-8.
  392. Descargues G, Mauger-Tinlot F, Douvrin F, Clavier E, Lemoine JP, Marpeau L, Menses, fertility and pregnancy after arterial embolization for the control of postpartum haemorrhage. *Hum Reprod*, 2004. Feb;19(2):339-43.
  393. Goldberg J, Pereira L, Berghella V, Pregnancy after uterine artery embolization. *Obstet Gynecol*, 2002. Nov;100(5 Pt 1):869-72.
  394. Wang H, Garmel S, Successful term pregnancy after bilateral uterine artery embolization for postpartum hemorrhage. *Obstet Gynecol*, 2003. Sep;102(3):603-4.
  395. Carpenter T, Walker WJ, Pregnancy following uterine artery embolisation for symptomatic fibroids: A series of 26 completed pregnancies. *Br J Obstet Gynaecol*, 2005. Mar;112(3):321-5.
  396. American College of Obstetricians, and Gynecologists, ACOG Committee Opinion: Number 293. Uterine Artery Embolization, 2004.
  397. Cardosi RJ, Nackley AC, Londono J, Hoffman MS, Embolization for advanced abdominal pregnancy with a retained placenta: A case report. *J Reprod Med*, 2002. Oct;47(10):861-3.
  398. Fahy U, Sved A, Burke G, Successful balloon tamponade of postcesarean hysterectomy pelvic bleeding: A case report. *Acta Obstet Gynecol Scand*, 2003. 82:97-8.
  399. Seror J, Allouche C, Elhaik S, Use of Sengstaken-Blakemore tube in massive postpartum hemorrhage: A series of 17 cases. *Acta Obstet Gynecol Scand*, 2005. Jul;84(7):660-4.
  400. Japaraj R, Raman S, Sengstaken-Blakemore tube to control massive postpartum haemorrhage. *Med J Malaysia*, 2003. Oct;58(4):604-7.
  401. Condous G, Arulkumaran S, Symonds I, Chapman R, Sinha A, Razi K, The "tamponade test" in the management of massive postpartum hemorrhage. *Obstet Gynecol*, 2003. Apr;101(4):767-2.
  402. Akhter S, Begum MR, Kabir ZK, Rashid M, Laila TR, Zabeen F, Use of a condom to control massive postpartum hemorrhage. *Med Gen Med*, 2003. Sep 11;5(3):1-11.
  403. Johanson R, Kumar M, Obhrai M, Young P, Management of massive postpartum haemorrhage: Use of a hydrostatic balloon catheter to avoid laparotomy. *Br J Obstet Gynaecol*, 2001. Apr;108(4):420-2.
  404. De Loor JA, van Dam PA, Foley catheters for uncontrollable obstetric or gynecologic hemorrhage. *Obstet Gynecol*, 1996. Oct;88(4 Pt 2):737.
  405. Marcovici I, Scoccia B, Postpartum hemorrhage and intrauterine balloon tamponade: A report of three cases. *J Reprod Med*, 1999. Feb;44(2):122-6.
  406. Bowen LW, Beeson JH, Use of a large Foley catheter balloon to control postpartum hemorrhage resulting from a low placental implantation: A report of two cases. *J Reprod Med*, 1985. Aug;30(8):623-5.
  407. Danso D, Reginald P, Combined B-lynch suture with intrauterine balloon catheter triumphs over massive postpartum haemorrhage. *Br J Obstet Gynaecol*, 2002. Aug;109(8):963.
  408. Bakri Y, Amri A, Abdul Jabbar F, Tamponade-balloon for obstetrical bleeding. *Int J Gynaecol Obstet*, 2001. Aug;74(2):139-42.
  409. Madlener M, Ubersterilisierende Operationen an den Tuben. *Zentralb fur Gynak*, 1919. 43:380-4.
  410. Garb A, A review of tubal ligation failures. *Obstet Gynecol Surv*, 1957. 12:291-305.
  411. Bishop ENW, A simple method of sterilization. *NY State Med J*, 1930. 30:214-6.
  412. Lee S, Jones JS, Postpartum tubal sterilization: A comparative study of Hulka clip application and the modified Pomeroy technique. *J Reprod Med*, 1991. 36:703.
  413. Hulka J, Mercer JP, Fishburne JI, et al, Spring clip sterilization: 1-year follow-up of 1079 cases. *Am J Obstet Gynecol*, 1976. 125:1039.

414. Nichols D, Clarke-Pearson DL, *Gynecologic, Obstetric, and Related Surgery*, 2nd ed. St. Louis: Mosby, Inc., 2000; p. 1248.
415. Husbands M, Pritchard JA, Pritchard SA, Failure of tubal sterilization accompanying cesarean section. *Am J Obstet Gynecol*, 1970. 107:966–7.
416. Irving F, A new method of insuring sterility following cesarean section. *Am J Obstet Gynecol*, 1924. 8:335–7.
417. Lopez-Zeno J, Muallem NS, Anderson JB, The Irving sterilization technique: A report of a failure. *Int J Fertil*, 1990. 35:23–5.
418. Uchida H, Uchida tubal sterilization. *Am J Obstet Gynecol*, 1975. 121:153.
419. Kroener WF, Surgical sterilization by fimbriectomy. *Am J Obstet Gynecol*, 1969. 104:247–54.
420. Escobedo LG, Grubb GS, et al, Case-fatality rates for tubal sterilization in U.S. hospitals, 1979–1980. *Am J Obstet Gynecol*, 1989. 160:147–50.
421. Chick PHFM, Paterson PJ, A comprehensive review of female sterilization – tubal occlusion methods. *Clin Reprod Fertil*, 1985. 3:81–97.
422. Rojansky NHU, Prevalence and severity of premenstrual changes after tubal sterilization. *J Reprod Med*, 1991. 36:551–5.
423. Alvarez FFA, Brache V, Tejada AS, Segal S, Prospective study of the pituitary-ovarian function after tubal sterilization by the Pomeroy or Uchida techniques. *Fertil Steril*, 1989. 51:604–8.
424. Shy KK, Stergachis A, Grothaus LG, Wagner EH, Hecht J, Anderson G, Tubal sterilization and risk of subsequent hospital admission for menstrual disorders. *Am J Obstet Gynecol*, 1992. Jun;166(6 Pt 1):1698–705; discussion 1705–6.
425. Wilcox LS, Martinez-Schnell B, Peterson HB, Ware JH, Hughes JM, Menstrual function after tubal sterilization. *Am J Epidemiol*, 1992. Jun 15;135(12):1368–81.
426. Shain RN, Miller WB, Holden AE, Rosenthal M. Impact of tubal sterilization and vasectomy on female marital sexuality: Results of a controlled longitudinal study. *Am J Obstet Gynecol*, 1991. Mar;164(3):763–71.
427. Rulin MC, Davidson AR, Philliber SG, Graves WL, Cushman LF. Long-term effect of tubal sterilization on menstrual indices and pelvic pain. *Obstet Gynecol*, 1993. Jul;82(1):118–21.
428. Stergachis A, Shy KK, Grothaus LC, Wagner EH, Hecht JA, Anderson G, Normand EH, Raboud J, Tubal sterilization and the long-term risk of hysterectomy. *JAMA*, 1990. Dec 12;264(22):2893–8.
429. Vurkuyl, DA. Think globally act locally: the case for symphysiotomy. *PLOS Med*, 2007 Mar 27;4(3):e71.
430. Bjorklund K. Minimally invasive surgery for obstructed labour: A review of symphysiotomy during the twentieth century (including 5000 cases). *BJOG*, 2002. Mar;109(3):236–48.
431. Mola GD. Symphysiotomy: Technique, problems and pitfalls, and how to avoid them. *P N G Med J*, 1995. Sep;38(3):231–8.
432. Menticoglou SM, Symphysiotomy for the trapped aftercoming parts of the breech: A review of the literature and a plea for its use. *Aust N Z J Obstet Gynaecol*, 1990. Feb;30(1):1–9.
433. Mola GD, Symphysiotomy or caesarean section after failed trial of assisted delivery. *P N G Med J*, 1995. Sept;38(3):172–7.
434. van Roosmalen J, Safe motherhood: cesarean section or symphysiotomy? *Am J Obstet Gynecol*, 1990. Jul;163(1 Pt 1):1–4.
435. Nichols DH, Episiotomy, repair of fresh obstetric lacerations, and symphysiotomy. in Nichols DH, Clarke-Pearson DL (eds) *Gynecologic, Obstetric, and Related Surgery*, Second Edition. St. Louis, Mosby, 2000, pp 1066–80.
436. B-Lynch C, Keith LG, Lalonde AB, et al., *A Textbook of Postpartum Hemorrhage*. Duncow, UK, Sapiens Publishing, 2006.
437. Stitley ML, Bherman RB, Postpartum hemorrhage: Solutions to 2 intractable cases. *OBG Management*, 2007. 19;(4):64–76.
438. Chao A, Safe delivery of the fetal head during cesarean section. *OBG Management*, 2003. Jan 15;(1):
439. The CORONIS Trial Collaborative Group: The CORONIS Trial. International study of caesarean section surgical techniques: A randomized factorial trial. *BMC Pregnancy and Childbirth* 2007, <http://www.biomedcentral.com/1471-2393/7/24>.
440. Warshak CR, Eskander R, Hull A, et al, Accuracy of ultrasonography and magnetic resonance imaging in the diagnosis of placenta accreta. *Obstet Gynecol*, 2006 108;(3 pt 1):573–81.
441. Comstock CH, Antenatal diagnosis of placenta accreta: a review. *Ultrasound Obstet Gynecol*, 2005. Jul;26(1):89–96.

442. Gielchinsky Y, Rojansky N, Fasouliotis SJ, Placenta accreta – summary of 10 years: A survey of 310 cases. *Placenta*, 2002. Feb–Mar;23(2–3):210–4.
443. Wu S, Kocherginsky M, Hibbard FU. Abnormal placentation: Twenty – year analysis. *Am J Obstet Gynecol*, 2005. 192:1458–61.
444. Tully L, Gates S, Brocklehurst P, et al, Surgical techniques used in caesarean section operations in the UK: A survey of current practice. *Eur J Obstet Gynecol Reprod Biol*, 2002. 102:120–6.
445. Heidenreich W, Bruggenjurgen K, Modified Sarafoff suture for single-layer closure of uterotomy in cesarean section. A prospective study. *Zentralbl Gynakol*, 1995. 117:40–4.
446. Sood AK, Single versus double layer of low transverse uterine incision at cesarean section. *J Obstet Gynecol India*, 2005. 55:231–6.
447. Chapman SJ, Owen J, Hauth JC, One-versus two-layer closure of a low transverse cesarean: The next pregnancy. *Obstet Gynecol*, 1997. 89:16–18.
448. Bujold E, Bujold C, Hamilton EF, et al, The impact of a single-layer or double-layer closure on uterine rupture. *Am J Obstet Gynecol*, 2002. 186:1326–30.
449. Dodd JM, Anderson ER, Gates S, Surgical techniques involving the uterus at the time of caesarean section. (Protocol) *Cochrane Database of Systematic Reviews* 2004, 2.
450. Belizan J, Althabe F, Barros F, et al, Rates and implication of cesarean sections in Latin America: Ecological study. *BMJ*. 1999. 319:1397–1402.
451. Sreevidya S, Sathiyasekaran BW, High caesarean rates in Madras (India): A population-based cross sectional study. *Br J Obstet Gynaecol*, 2003. 110:106–11.
452. Siddiqui M, Goldszmidt E, Fallah S, et al, Complications of exteriorized compared with in situ uterine repair at cesarean delivery under spinal anesthesia: A randomized controlled trial. *Obstet Gynecol*, 2007. 110(3):570–5.
453. Bamigboye AA, Hofmeyr GJ, Closure versus non-closure of the peritoneum at caesarean section. *The Cochrane Database of Systematic Reviews* 2003, 3.
454. Quintero RA, Kontopoulos EV, Bornick PW, Allen MH, In utero laser treatment of type II vasa previa. *J Matern Fetal Neonatal Med*, 2007. Dec;20(12):847–51.
455. Stoodley MA, Macdonald RL, Weir BK, Pregnancy and intracranial aneurysms. *Neurosurg Clin North Am*, 1998. Jul;9(3):549–56.
456. Kriplani A, Relan S, Misra NK, Mehta VS, Takkar D, Ruptured intracranial aneurysm complicating pregnancy. *Int J Gynaecol Obstet*, 1995. Feb;48(2):201–6.
457. Piotin M, de Souza Filho CB, Kothimbakam R, Moret J, Endovascular treatment of acutely ruptured intracranial aneurysms in pregnancy. *Am J Obstet Gynecol*, 2001. Nov;185(5):1261–2.
458. Riviello C, Ammannati R, Bordi L, Lamassa M, Mennonna P, Parretti E, Tondi F, Mello G, Pregnancy and subarachnoid hemorrhage: A case report. *J Matern Fetal Neonatal Med*, 2004. Oct;16(4):245–6.
459. Roman H, Descargues G, Lopes M, Emery E, Clavier E, Diguët A, Freger P, Marpeau L, Proust F, Subarachnoid hemorrhage due to cerebral aneurysmal rupture during pregnancy. *Acta Obstet Gynecol Scand*, 2004. Apr;83(4):330–4.
460. D’Haese J, Christiaens F, D’Haens J, Camu F, Combined cesarean section and clipping of a ruptured cerebral aneurysm: A case report. *J Neurosurg Anesthesiol*, 1997. Oct;9(4):341–5.
461. Brisman JL, Soliman E, 2007. Cerebral aneurysm. *eMedicine*, topic = 3468.



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*For this relief much thanks*

William Shakespeare (1564–1616)

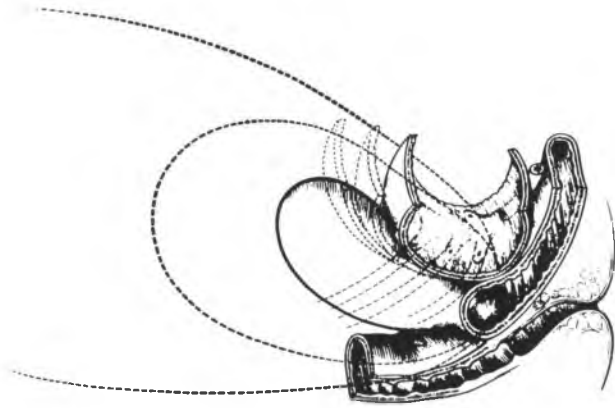
*The Tragedy of Hamlet, Prince of Denmark, I,i*

Urologic disorders during pregnancy merit special consideration, because pregnancy induces extensive anatomic and physiologic changes in both the genital and urinary tracts. These alterations increase the susceptibility of the urinary tract to certain diseases and injuries. Labor and delivery also can result in urinary tract damage and possibly other pelvic floor disorders. Additionally, the treatment of many conditions during pregnancy must be tempered by considerations of fetal risk, including possible premature delivery and the potential induction of fetal anomalies.

This chapter reviews conditions occurring in the urinary tract during pregnancy that place the mother, and often the fetus, at risk. This jeopardy is either a result of these diseases or is due to the clinician's attempts at treatment. Conditions discussed in this chapter that might require surgery during pregnancy include urolithiasis, urinary tract obstruction, accidental and iatrogenic lower urinary tract injury, urethral diverticula, genitourinary fistulas, complications of previous urologic surgery, and urinary tract carcinoma. This chapter critically appraises the current literature and discusses contemporary concepts of diagnosis and management of these conditions specific to pregnancy.

### ANATOMIC CHANGES

In normal pregnancy, each kidney elongates by approximately 1 cm because of the increase in vascular volume and interstitial space. As the uterus enlarges, the bladder rises out of the pelvis and into the abdomen, thus altering its relationship and that of the urethra and ureters to the cervix and uterus. By the third trimester, the bladder becomes more of an abdominal than a pelvic organ. Normally, the ureter passes beneath the uterine artery 1.5 cm to 2 cm lateral to the cervical isthmus as it courses medially to enter the bladder trigone. During pregnancy, the ureter is displaced by the enlarging lower uterine segment and lies closer to the uterus and cervix. The expanding uterus also elevates the bladder trigone, making this normally concave structure convex, displacing the ureteral orifices laterally (Figure 19.1).



**FIGURE 19.1.**  
*Changing relationships of bladder base and ureters to cervix and lower uterine segment during the three trimesters of pregnancy, showing elevation of the trigone and lateral displacement of ureteral orifices. (From Mattingly RF, Borkowf HI: Lower urinary tract injuries in pregnancy. In: Barber HRK, Graber EA (eds): Surgical Disease in Pregnancy. Philadelphia: WB Saunders, 1974; with permission.)*

The left ureter, moreover, is drawn anteriorly as a result of uterine dextrorotation. These changes, with the new abdominal position of the bladder, render the ureter, bladder, and the urethra much more susceptible to accidental and surgical trauma [1–3].

Whereas changes in lower urinary tract anatomy associated with pregnancy predispose to injury, alterations in physiology explain the propensity for urinary tract obstruction [4]. Hydronephrosis and hydroureter, ureteral elongation, dilation, and tortuosity commonly accompany pregnancy. In the late third trimester, some degree of bilateral hydroureteronephrosis is present in 90% of pregnant women [2,3]. This physiologic hydroureteronephrosis of pregnancy can be seen as early as 6 weeks of gestation (Figure 19.2). The kidney and ureters revert to their normal prepregnancy status by 6 weeks postpartum.

Theories proposed to explain these changes include both mechanical obstruction from the gravid uterus or adjacent vessels, and the physiologic effects of progesterone and prostaglandins, causing decreased ureteral peristalsis and bladder atony. The mechanical theory is supported by the fact that, when present, hydroureter and hydronephrosis occur on the right side much more frequently than on the left. The propensity for right ureteral dilation results from a combination of uterine dextro-



**FIGURE 19.2.**  
*Intravenous urogram in sixth week of pregnancy demonstrates early dilation of upper ureters bilaterally. Note that lower ureter (arrow) retains its normal caliber. (From Freed SZ, Herzig N (eds): Urology in Pregnancy. Baltimore, Williams & Wilkins, 1982; with permission.)*

rotation, right ureteral compression by the engorged right ovarian vessels, and cushioning of the left ureter by the sigmoid colon and iliac arteries. A mechanical cause for upper urinary tract dilation is also favored by sharp termination of ureteral dilation at the pelvic brim, as documented by intravenous pyelography (IVP) [5]. In addition, ureteral dilation is most evident after 20 weeks of gestation, presumably as a consequence of the gravid uterus compressing the ureter against the pelvic brim. Finally, hydronephrosis and hydroureter are not common in women with renal transplants or pelvic kidneys, lending further support to the mechanical obstruction theory [5].

Elevated progesterone levels were thought to be primarily responsible for smooth muscle relaxation, hypotonicity, and dilation of the renal pelvis and ureter in pregnancy; however, ureteral dilation does not occur even when large doses of synthetic progesterone are used for chemotherapy [6]. Additionally, investigations of the amplitude and frequency of ureteral contractions reveal no changes during pregnancy, and ureteral tone is actually increased [7]. For these reasons, progesterone and other pregnancy-related hormones probably play only a limited role in ureteral dilation during pregnancy. Mechanical effects are thought to predominate.

Bladder capacity increases progressively throughout the second and third trimesters of pregnancy. This change is thought to be a result of bladder atony because of the effect of progesterone, combined with partial urethral obstruction by the fetal head. Bladder capacity during pregnancy increases to 450 ml to 600 ml, compared with 400 ml in nonpregnant controls [3]. Bladder volume in excess of 1,000 ml, which is not unusual during labor or the puerperium, predisposes the bladder to rupture from external trauma or neglected labor [4,8]. Pregnancy can also affect the urethra. A urodynamic study on 14 healthy and continent primigravida women performed during the first trimester, late third trimester, and again 5 to 7 days postpartum showed an increase in total and functional urethral length as well as in urethral closure pressure [9]. Besides these anatomic changes in the lower urinary tract, it is evident that pregnancy and vaginal delivery can also affect the pelvic floor and predispose to stress urinary incontinence and pelvic organ prolapse [10–12].

### PHYSIOLOGIC CHANGES

Many systemic changes are normal during pregnancy, and many homeostatic mechanisms are altered. The kidney plays an important role in maintenance of water and electrolyte control, and its function is affected by pregnancy-related alterations in the cardiovascular and respiratory systems. Plasma volume increases nearly 50%, whereas red cell volume increases to a lesser extent, leading to physiologic anemia of pregnancy. Cardiac output increases early in the first trimester by 1 l/min to 2 l/min, an increase of 20% to 40% over normal values [13]. During pregnancy, effective renal plasma

flow increases by 60% to 80% in the first and second trimesters, and then decreases in the third trimester. The glomerular filtration rate (GFR) increases 30% to 50% early in pregnancy and remains elevated until term [13].

There is cumulative retention of total body water throughout pregnancy, with a mean gain of 7 liters to 8.5 liters over the 40 weeks of gestation. Throughout pregnancy, total osmolality is 10 mmol less than normal nonpregnant levels, and the plasma sodium concentration is decreased. Despite this change, 500 mEq to 900 mEq of sodium is retained throughout pregnancy [13]. The 50% increase in GFR results in a massive increase in the filtered sodium load. This increased filtration is accompanied by a concomitant increase in the amount of sodium reabsorbed.

With increased glomerular clearance, there is an increase in the excretion of protein and glucose, which can exceed the resorption capacity of the tubules. For this reason, proteinuria up to 300 mg/day is considered normal in pregnancy. Two or more episodes of glucosuria are seen in 10% of pregnant women, because the renal threshold for glucose decreases by 10% to 15%, to approximately 140 mg/dl [14]. Because of the increase in GFR and the filtration fraction in the presence of unchanged production of creatinine and urea, serum creatinine and blood urea nitrogen levels decrease to 0.7 mg/dl and 10 mg/dl, respectively [15].

### COMMON URINARY COMPLAINTS

#### **Urinary Frequency and Nocturia**

The normal homeostatic changes involving the urinary tract are frequently responsible for the gravid woman's urinary complaints. Other pathologic changes, as a consequence of tissue damage either from pregnancy or labor, can accentuate these physiologic changes and result in persistent symptoms, however.

The most common urinary complaint in early pregnancy is urinary frequency. The onset of frequency can be as early as the first trimester. This symptom can be explained by pressure on the bladder from an enlarging, anteflexed uterus, and by increased urine production resulting from changes in GFR exceeding tubular reabsorption. Late in the third trimester, when the presenting part of the fetus enters the pelvis, urinary frequency can recur

or worsen. Using a definition of frequency as more than seven daytime voids and more than one nighttime void, Francis [16] compared 400 healthy pregnant women with normal nonpregnant women and found that urinary frequency was reported by 59% in early pregnancy, 61% in midpregnancy, and 81% in late pregnancy. Parboosingh and Doig reported that 66% of pregnant women experienced nocturia by the third trimester, using a definition of nocturia as at least three nighttime voids [17]. Another study on women in their first trimester of pregnancy found that 40% complained of frequency and 23% of nocturia [18]. The symptoms of urinary frequency are unrelated to the effect of posture or bladder capacity but are caused by polydypsia and polyuria of pregnancy [16]. During the first trimester, both fluid intake and urine output are increased and remain constant until the third trimester, causing frequency. During the third trimester, a decrease in sodium output leads to a decrease in urine output; however, the large uterus exerts more pressure on the bladder, and frequency persists. Sodium excretion and the mobilization of dependent edema at night were the major reasons for nocturia [17]. The antenatal symptoms of diurnal and nocturnal frequency might also be related to increased fluid intake combined with increased time spent in recumbency.

### Voiding Difficulties

During the first and second trimesters, urinary hesitancy occurs in up to 27% of pregnant women [19]. Urinary retention can occur early during pregnancy in women with a retroverted uterus, or those having an enlarging fibroid or a pelvic mass. The retention usually resolved by 16 weeks of gestation as the uterus grows out of the pelvis. Either bladder drainage or self-catheterization is sometimes needed to manage this problem. Alternatively, the obstruction on the bladder neck can be relieved by manual reduction of the retroverted, incarcerated uterus or placement of a Hodge pessary [20].

Postpartum urinary retention is more common, with an incidence of 1.7% to 17.9% [1]. For women who received epidural anesthesia during labor and delivery, the bladder can take up to 8 hours after the last dose to regain sensation. Other risk factors for postpartum retention include instrumental delivery, a first labor, and a long duration of labor (>13 hours). Prolonged postpartum bladder disten-

sion can lead to permanent detrusor dysfunction and altered bladder sensation [1]. Hence, women with risk factors for postpartum urinary retention should be monitored carefully after delivery to ensure adequate voiding. If necessary, catheterization should be employed to avoid bladder overdistension.

### Urinary Urgency and Urge Incontinence

Urinary urgency and urge incontinence are common in pregnancy. In one study, 62% of pregnant women reported the symptoms of urgency, and 18% complained of urge incontinence [21]. In another study of a large number of nulliparous women (549 subjects), only 22.9% reported urgency and 8% urge incontinence during pregnancy [22]. When compared with their prepregnancy state, complaints of urgency and urge incontinence in pregnancy in this same group of women were 10 and 16 times higher, respectively. Urinary urgency and urge incontinence can also occur postpartum, with 7.8% of parturients reporting urgency and 2.2% reporting urge incontinence at as much as 12 weeks after delivery [22]. As suggested by some published data [21,23], the etiology of urgency and urge incontinence in pregnancy is explained not by the development of detrusor instability alone, but rather the combination of detrusor instability, low bladder compliance, and urethral instability.

### Stress Urinary Incontinence

Urinary symptoms of stress urinary incontinence are also quite common in pregnancy. In some series, the symptoms of stress incontinence have been reported in up to 85% of pregnant women [16,24]. There is a poor correlation between symptoms and urodynamic findings for incontinence, however [25,26]. Although the symptoms of stress (32%) and urge incontinence (26%) were frequently reported by pregnant patients in one series, urodynamic studies demonstrated genuine stress incontinence in only 7%, and detrusor instability in 36% of these symptomatic women [25]. No significant differences in symptoms of frequency, nocturia, urgency, and urge incontinence were found in patients with or without detrusor instability, indicating that these symptoms often occur in patients without urodynamic changes. Stanton and coworkers [19] have demonstrated no relationship between stress or urge

incontinence with descent or engagement of the presenting part. Nonetheless, when present, symptoms generally increased to term.

Symptoms of stress incontinence usually disappear in the puerperium. At 3 months postpartum, however, 10% of patients who developed stress incontinence during pregnancy and 29% who developed stress incontinence after delivery still complained of this symptom [27]. At 1 year postpartum, these numbers decreased to 3.5% and 25%, respectively [24]. These findings suggest that stress incontinence developing after delivery carries more serious risk than that developing during pregnancy [27]. This supposition is corroborated by electrophysiologic data showing pudendal and perineal nerve damage as results of labor and vaginal delivery [28]. Stress incontinence that appears during pregnancy seems to be caused by a different pathologic process from that developing after vaginal delivery. Although urodynamic stress incontinence (previously "genuine" stress incontinence) from partial denervation of the pudendal nerve appears to be responsible for most postpartum difficulties, detrusor instability is likely the primary cause for the symptom of stress incontinence during pregnancy [29]. There is a significant correlation between the length of the second stage of labor and the development of stress postpartum incontinence [10–12]. Viktrup and coworkers [24] reported that 7% of primigravida women developed *de novo* stress incontinence postpartum.

The exact cause of stress incontinence in the postpartum period is unclear; however, the etiology is most likely multifactorial, related to functional and structural changes, nerve damage to the lower urinary tract and pelvic floor, and collagen abnormalities. The mechanism for maintaining continence during pregnancy, despite an increase in intravesical pressure, is thought to be related to the increases in both urethral length and maximal urethral closure pressure. Iosif and Ulmsten reported that during pregnancy, the absolute urethral length increased by a mean of 6.7 mm and the functional urethral length increased by a mean of 4.8 mm [30]. The maximal urethral closure pressure increased to 93 cm H<sub>2</sub>O at 38 weeks and then decreased to the prepregnancy value of 69 cm H<sub>2</sub>O after delivery. These changes were not apparent in pregnant women who complained of stress incontinence. The

increased blood volume during pregnancy could increase the urethral sphincter volume and amplitude of vascular pulsations in the urethral wall, and subsequently increase the urethral closure pressure. The opposite effect has been demonstrated in pregnant women with genuine stress incontinence, who had lower amplitude of vascular pulsations in the urethral wall than did their continent counterparts [1,30].

Some researchers have suggested that vaginal delivery, rather than pregnancy itself, predisposes women to stress incontinence. Wilson and coworkers reported that the risk of stress incontinence was significantly less in those who had undergone cesarean birth, whether elective or in labor, especially primigravidas [31]. The prevalence of stress incontinence in women who had three or more cesarean deliveries was similar to that of those who delivered vaginally, however (38.9% and 37.7%, respectively). Although the issue is still under debate, some authors propose that incontinence in this situation might be a result of nerve damage from bladder distension during cesarean delivery [10–12,31]. After delivery, the urethral length and closure pressure were significantly higher in women who had cesareans than in those who delivered vaginally [32]. These data demonstrate that cesarean delivery might prevent or minimize the development of stress incontinence in the postpartum period.

Damage to the pudendal nerve and its branches has been demonstrated after childbirth [10–12,33]. Since the perineal branch of the pudendal nerve innervates the striated muscles of the urethra and pelvic floor, damage to this nerve in childbirth can lead to stress incontinence. Patients with genuine stress incontinence have been shown to have abnormal conduction in the perineal branch of the pudendal nerve [10–12]. Snooks and coworkers reported that, at 2 to 3 days postpartum, prolongation of pudendal nerve terminal motor latencies was found in 42% of those delivered vaginally, but none in those delivered by cesarean [33]. The pudendal nerve latency returned to baseline at 2 months postpartum in 60% of the women with evidence of nerve damage. Allen and coworkers found evidence of denervation injury in 80% of women after vaginal delivery [28]. The degree of pudendal nerve damage correlated positively with the use of

forceps, longer second stage of labor, larger baby, and multiparity.

Vaginal delivery can also lead to permanent damage to the urethral sphincter mechanism and subsequently predispose to stress incontinence [10–12]. Sphincter weakness was caused not only by the loss of motor units but also by asynchronous activity in those remaining. Trauma from vaginal delivery might also cause urethral detachment, lowered bladder neck position, and increasing bladder neck mobility. Using nulligravid controls, Peschers and coworkers reported that bladder neck position was significantly lower and bladder neck mobility was significantly greater after vaginal delivery than after cesarean birth [34]. Significant lowering of bladder neck after forceps delivery was also reported [35]. Alterations in urethral and bladder neck support and increases in bladder neck mobility that occur after vaginal or instrumental deliveries could lead to stress incontinence. (For additional discussion, see Chapter 23, Birth Injuries.)

### Urinary Tract Infection

A major cause of urinary symptoms in pregnancy is acute cystitis, which complicates up to 15% of pregnancies [1–3,36]. The prevalence of asymptomatic bacteriuria in pregnant women is similar to that in a nonpregnant population, but there is a three- to fourfold higher progression rate to bladder and, most notably, kidney infection [1,2]. The decrease in bladder tone and increase in ureteral volume, with the facilitatory effect of estrogen, particularly in the growth of *Escherichia coli*, contribute to the risk of acute cystitis and pyelonephritis in pregnancy. Pregnant patients with urinary tract infections, like their nonpregnant counterparts, complain of frequency, urgency, dysuria, suprapubic discomfort, and occasional gastrointestinal distress. The standard colony count for urinary tract infection is  $\geq 10^5$  colony-forming units (CFU)/ml of urine; however, counts as low as  $10^2$  CFU/ml might represent active infection in pregnancy [36]. Two thirds of pregnant women with acute cystitis have a negative initial screening culture, underscoring the necessity for repeat periodic screening for bacteriuria in pregnancy [15,36]. If found positive for bacteriuria, pregnant women should be treated even if they are asymptomatic. Antibiotic treatment for asymptomatic bacteriuria

can include amoxicillin, nitrofurantoin, or one of the cephalosporins. Trimethoprim should not be used in early pregnancy because it can affect neural tube development. Sulfonamides can also be administered, but not in late pregnancy when they can increase the risk of kernicterus. Tetracyclines should be avoided because they cause bone and teeth dysplasia and discoloration. If a woman treated with appropriate antibiotics for cystitis or pyelonephritis fails to respond or has recurrent infection, the presence of a urinary tract anomaly or urolithiasis must be considered.

Acute pyelonephritis complicates 1% to 2% of pregnancies and is associated with maternal and fetal morbidity and mortality [2,3,37]. The patients typically complain of fever, costovertebral angle tenderness, and cystitis symptoms. Sepsis and respiratory distress can occur in severe cases. Treatment should be aggressive, with hydration, analgesics, and intravenous antibiotics. Second- or third-generation cephalosporins and a short course of an aminoglycoside are usually employed.

## URINARY CALCULI

### Epidemiology

Urolithiasis is an uncommon event, reported in 0.03% to 0.53% of pregnancies, an incidence similar to that of nonpregnant women [38]. Despite its low incidence, this problem is significant, because renal colic is the most common nonobstetric cause of pain necessitating hospitalization in pregnancy. Urolithiasis can cause urinary tract infection, leading to compromised renal function, permanent renal damage, or promote preterm labor. Drago and Rohner report that two thirds of women with renal colic presented with preterm labor secondary to complications of urolithiasis [39]. Multiparous women are three times more likely to be affected by urolithiasis than are nulliparas. This increased incidence probably is reflective of the increased predisposition to stone formation in older women [40]. Urolithiasis occurs with equal frequency in all three trimesters, although this issue was formerly a question of considerable debate [41]. The diagnosis of urolithiasis is more likely to be made in the second or third trimester, probably because ureteral dilation in late pregnancy leads to passage of renal and upper

ureteral stones into the distal ureters, with resultant symptoms [40].

### Etiology

The physiologic hydroureteronephrosis of pregnancy was previously thought to be a major etiologic factor in the development of upper urinary tract stones. If this were true, however, one would expect to see a right-sided predominance, which is not the case. Many metabolic alterations occur in pregnancy, some of which predispose to stone formation. Gestational hypercalcinuria is normal, with a magnitude up to two- to threefold greater than in nonpregnant women. Hypercalcinuria has been attributed to enhanced placental formation of 1,25-dihydroxycholecalciferol with suppressed parathormone levels [42]. The hypercalcinuria of pregnancy is attributed to the 50% increase in GFR or to the increase in clearance of uric acid. Urine in pregnancy is also supersaturated with calcium oxalate and calcium carbonate, increasing the likelihood of precipitation probably resulting from the relatively alkaline urine that characterizes pregnancy [41].

Despite these factors, there is no increase in the incidence of stone formation during pregnancy. Furthermore, pregnancy does not increase the risk of urolithiasis in women who are known to be "stone formers," probably because of increased excretion of citrate, magnesium, and other crystalline inhibitors [41]. In addition, calcium oxalate crystal growth is inhibited by acidic glycoproteins, which are excreted in increased amounts during pregnancy [43]. Although the relatively alkaline urine of pregnancy predisposes to calcium carbonate lithiasis, it also results in a protective effect on uric acid stone formation. This combination of multiple protective metabolic alterations apparently offsets the factors predisposing to urolithiasis. In pregnant stone formers, there is an increased incidence of struvite or "infection stones" (13%); however, the composition of stones in pregnant patients and in their nonpregnant counterparts is similar [2]. The composition of urinary calculi is calcium, 90%; uric acid, 5% to 10%; and cystine, 3% [44].

There is a recognized but unexplained association between calcium urolithiasis and habitual abortion, which imparts even greater concern for the stone-forming patient who is pregnant or desiring pregnancy [45]. Because of this association, other

complications of urolithiasis, and the many ramifications of management associated with pregnancy, some experts recommend that gravid patients be treated for asymptomatic calculi.

Underlying systemic disease leading to urolithiasis must be considered in the pregnant as in the nonpregnant patient. Recurrent calcium stone formation can result from primary hyperparathyroidism, destructive bone disease, renal tubular acidosis, or sarcoidosis. For uric acid stones, hyperuricemia can be caused by gout, polycythemia vera, myeloid metaplasia, lymphoma, or leukemia. Cystine stones are seen with inborn errors of metabolism. If present, these primary systemic conditions, rather than urolithiasis alone, carry a higher risk for the pregnancy. Although the possibility of primary conditions in calcium stone formers should be eliminated by a metabolic evaluation in the postpartum period, most patients are found to have idiopathic hypercalcinuria without underlying systemic disease.

### Diagnosis

With a few exceptions, symptoms of urolithiasis in pregnant women are the same as those in nonpregnant women. Nausea, vomiting, and severe flank pain are the most common complaints, mimicking acute pyelonephritis, but usually without fever. Because of pregnancy-induced ureteral dilation, renal colic is less common in the second and third trimesters. For the same reason, gross hematuria is less common, although microscopic hematuria occurs in 60% to 90% of cases [3]. Hematuria is not a specific indicator for calculi in pregnancy because it can result from vascular dilation from both collecting system enlargement and local hormonal changes [46]. Other common symptoms and signs of urolithiasis include frequency, dysuria, groin pain or tenderness, and pyuria. Occasionally, the patient with a renal stone, or even with a ureteral calculus, after initial colic, may be completely asymptomatic.

Because of the obscurity of the presentation in pregnancy, diagnosis of urolithiasis is frequently delayed. Alternatively, urolithiasis can present solely as an unresponsive or recurrent urinary tract infection. In these instances, a high index of suspicion must be maintained to arrive at the correct diagnosis. An evaluation for urolithiasis is warranted in

any patient who, after being treated for urinary tract infection with appropriate parenteral antibiotics, remains febrile after 48 hours. Symptomatic women with previous episodes of urolithiasis, those with a family history of urolithiasis, or those who have undergone prior urologic surgery are at increased risk for stone formation.

Because the typical signs and symptoms of urolithiasis are often obscured by pregnancy, the physician is frequently faced with a diagnostic challenge. The diagnosis is suspected on physical examination when tenderness is elicited over the costovertebral angle, flank, lower abdomen, or groin, although the clinical picture is frequently confused with that of pyelonephritis. Hematuria, pyuria, bacilluria, and a moderate leukocytosis without fever can all be signs of urolithiasis.

Current opinion on the optimal diagnostic technique for urolithiasis has undergone evolution over the past few years and is not uniform. Traditionally, a limited or "one-shot" IVP or modified excretory urography has been accepted as the preferred test and is still recommended by many authorities [3]. A limited IVP in pregnancy typically consists of a scout film, a 20-minute film and, if delayed excretion is present, a 60-minute film (Figure 19.3). These films result in less than 0.6 mGy of radiation exposure to the fetus and maternal ovaries. This dosage is well below the 5-mGy to 15-mGy dose, which is associated with a 1% to 3% risk of congenital anomalies in the first trimester. The risk of carcinogenesis is less than 1% with x-ray exposures under 1 mGy. Fluoroscopy, with rare exception, should be avoided during pregnancy.

An IVP is warranted if renal colic and gross hematuria exist; if there is persistent nausea, vomiting, or fever; if there is a positive culture after administering parenteral antibiotics for 48 hours; or if the blood urea nitrogen (BUN) and creatinine levels increase, suggesting complete obstruction. A single abdominal radiograph, carrying a fetal exposure of 0.2 mGy, is recommended prior to IVP. Whether or not a radiopaque calculus is seen, a limited excretory urogram is indicated to evaluate function and to eliminate the possibility of obstruction [41].

A review by Stothers and Lee of the management of 80 pregnant patients with renal colic demonstrated that delayed films were of no benefit when the IVP did not show the location of the calculus after 20 minutes [47]. Impaired visualization



**FIGURE 19.3.** Ureteral calculus (arrow) demonstrated in right distal ureter on IVP. (From MacNeily AE, Goldenberg SL, Allen GJ, et al: *Sonographic visualization of the ureter in pregnancy*. *J Urol* 1991; 146: 299; with permission.)

of the calculus was attributed to overlying fetal bony structures, overlying maternal pelvic bones, and poor bowel preparation. In cases of nonvisualization, the diagnosis was made by retrograde pyelography, which the authors recommended as the next step in the evaluation process, in lieu of delayed IVP films, which they found only added additional radiation exposure to the fetus.



Because of the inherent risk of radiation exposure to the fetus, many physicians view roentgenographic studies, however limited in dosage, as being relatively contraindicated in pregnancy. Whereas some authorities think that ultrasonography is of virtually no value in the detection of ureteral calculi, others think of it as the initial diagnostic method of choice [2,38,48]. In Hendricks and colleagues' series, in 10 of 15 pregnant patients, the diagnosis of urinary tract calculi was established by ultrasonic scan [48]. Eight of these ten patients were in the third trimester of pregnancy, attesting to the value of ultrasonography in the diagnosis of urolithiasis, even in advanced pregnancy. In addition to lacking radiation exposure, ultrasonography has the advantage of being an accurate noninvasive means of detecting hydronephrosis.

Ultrasound scan is nonspecific because of its inability to distinguish pregnancy-induced hydronephrosis from hydronephrosis secondary to ureteral calculi. MacNeily and coworkers describe a technique to differentiate physiologic from pathologic dilation of the renal collecting system in pregnant patients by combining routine ultrasound scan with color flow Doppler. In physiologic hydronephrosis, the distal ureter extends down to the level of the common iliac artery, leaving the pelvic ureters unaffected unless there is also pathologic obstruction. Using ultrasound scan with color flow Doppler, the iliac artery can be easily differentiated from the dilated pelvic ureter, establishing the correct diagnosis (Figure 19.4).

In summary, a review of the literature demonstrates ample support for the use of ultrasonography, transabdominally and transvaginally, as an initial diagnostic technique for the detection of urinary stones. If radiographic studies are performed, an initial IVP film and one at 20 minutes after dye injection are best. X-ray evaluations (i.e., the limited intravenous or retrograde pyelogram) are reserved for women in whom ultrasound diagnosis is not definitive. Renal function assessment and urine microscopic studies should also be undertaken.

### Management

In complicated cases, expectant management of urolithiasis in pregnant patients, just as in nonpregnant subjects, is the best initial treatment [50]. Only 50% of pregnant women, however, compared with

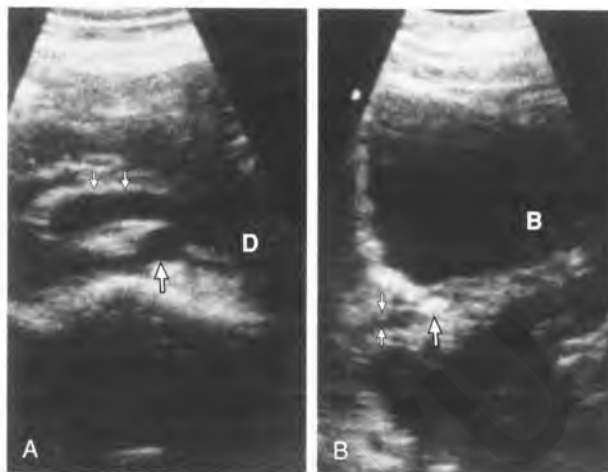


FIGURE 19.4.

*A, On sagittal view, real-time ultrasound scan demonstrates dilated ureter (small arrows) over and beyond iliac vessels (large arrow). The distal ureter (D) courses toward the right side of scan. B, A sagittal view of the bladder (B) demonstrates a stone (large arrow) at the ureterovesical junction (ureter, small arrows). (From MacNeily AE, Goldenberg L, Allen GJ, et al: Sonographic visualization of the ureter in pregnancy. J Urol 1991; 146: 300; with permission.)*

80% of nonpregnant women, are likely to undergo spontaneous passage of ureteral calculi with a combination of hydration, analgesia, and, with infected stones, antibiotic therapy [41,48,50]. If hydration and analgesia fail to result in passage of calculi, segmental epidural anesthesia can be attempted. In the literature over the past three decades there have been occasional reports of the successful use of epidural anesthesia to facilitate the passage of impacted ureteral stones, presumably by decreasing ureteral spasm [48].

Surgical management of renal lithiasis is indicated for the following conditions: intractable renal colic unresponsive to conservative management, concomitant premature labor refractory to tocolysis, persistent massive hydronephrosis with impaired renal function, calculus pyelonephritis, urosepsis, or obstruction of a solitary kidney. The operative management of pregnant and nonpregnant patients with urolithiasis has changed radically since the mid-1980s, with the advent of new techniques for endoscopic and extracorporeal manipulation of calculi. The use of extracorporeal shock wave lithotripsy (ESWL) has revolutionized the treatment of ureteral calculi in nonpregnant patients.

The difficulty in modulating the energy dispersed from the lithotripsy to the enlarged uterus and fetus, compounded by the potential difficulty of localizing calculi, which necessitates prolonged ionizing radiation exposure, makes pregnancy an absolute contraindication to the use of ESWL.

Because of the close anatomic relationship between the distal ureter and the uterus and ovaries, concern has been raised regarding the possible mutagenic effect or a decrease in female fertility from lithotripsy [51]. As a result, many centers regard ESWL of lower ureteral calculi as contraindicated in all female patients in the reproductive years. A first-trimester spontaneous abortion was also reported in a woman after lithotripsy of a distal ureteral calculus [51]. In a retrospective review, Vogel concluded that lithotripsy did not affect female fertility and did not lead to increased teratogenic risk [52]. This finding is not unexpected, considering the energy levels involved in this procedure. The exposure throughout the urinary tract from ESWL varies from 100 mSv to 300 mSv, with the gonadal dose being estimated at 30% of these figures. According to published guidelines, a gonadal dose of less than 100 mSv is insignificant, whereas a dose of greater than 200 mSv is considered a possible indication for termination of pregnancy [52].

The treatment of choice for distal ureteral calculi in pregnant patients who require intervention is cystoscopic examination, followed by either passage of a ureteral stent to relieve obstruction (Figure 19.5), or ureteroscopy with calculus manipulation [48,53–55]. Stents can displace calculi proximally, allowing definitive therapy to be postponed until after delivery. Rittenberg and Bagley recommend stent placement for any pregnant woman at the time a ureteral calculus is diagnosed [54]. Internal stents usually can be placed under radiographic guidance with or without local anesthesia. The disadvantage of internal stents, aside from the possible risk of radiation exposure, is encrustation, which necessitates stent changes every 4 to 8 weeks, according to some authorities [56]. Goldfarb and coworkers theorize that the increased tendency for stent encrustation is related to the physiologic hypercalciuria and hyperuricosuria associated with pregnancy [57]. For this reason, some researchers recommend restriction of dietary calcium in pregnancy for patients with ureteral stents or known urolithiasis [56].



**FIGURE 19.5.** Arrows indicate ureteral calculi, the cause of obstruction bypassed with bilateral ureteral stents in a gravid patient. Although the upper ureters are dilated, the lower ureters are of normal caliber. (From Freed SZ, Herzig N (eds): *Urology in Pregnancy*. Baltimore: Williams & Wilkins, 1982; with permission.)

Wolf and colleagues described a new technique for ureteral stent placement during pregnancy using *endoluminal ultrasound* [58]. This technique uses an interventional ultrasound system (IVUS), consisting of a 20-MHz transducer attached to a flexible 6.2-Fr catheter, which is passed to the renal pelvis without need for ureteral dilation. Once the IVUS catheter is in place, an indwelling stent is passed beside it, and the IVUS catheter is removed. This technique has the advantage of traditional stents but does not require the use of a roentgenogram to ensure correct placement.

In practice, however, most stones are manipulated retrograde using a cystoscope or ureteroscope [59]. After the ureteral orifice has been dilated

with a balloon dilator catheter, a ureteroscope is passed into the distal ureter. This technique is easier for stones in the lowermost ureter, especially in advanced pregnancy, because the fetal head usually compresses the bladder, limiting access to the upper ureter [59]. The potential risks of this procedure include ureteral perforation (17%), ureteral stricture formation (5%), sepsis, and mucosal laceration with extravasation of contrast material [60]. Ureteroscopy with basket extraction is relatively contraindicated in patients with active urinary tract infections, sepsis, ureteral strictures, calculi larger than 1 cm in diameter, multiple calculi, and solitary kidneys [59].

Both retrograde ureteral catheterization and ureteroscopy can be difficult or impossible owing to distortion of the distal ureter by the enlarged uterus. If efforts to decompress from below are unsuccessful because of advanced pregnancy, proximal drainage with percutaneous nephrostomy becomes the procedure of choice [61]. This procedure can be performed with the patient under local anesthesia using ultrasound guidance. Problems with this technique include difficulties resulting from positioning the gravid patient into a prone position, and the risks of premature delivery [61] and external drainage [55]. External drainage has the disadvantages of patient discomfort, bacterial colonization, potential tube dislodgement, erosion, and bleeding [55]. To avert some of these complications, patients undergoing initial nephrostomy drainage can sometimes be converted to a stent later in pregnancy. Percutaneous tubes do have advantages over internal stents, in that bladder discomfort from indwelling stents is avoided, and incrustation of tubes is thought to be less likely owing to their accessibility for periodic irrigation. Kavoussi and coworkers report tube occlusion with debris in five of six patients with percutaneous drains, however, necessitating changing drainage catheters every 6 weeks [62].

Rarely, the aforementioned methods are unsuccessful, and more invasive surgical procedures are needed. These operative techniques, which carry greater fetal and maternal morbidity, include ureterolithotomy, pyelolithotomy/pyelostomy, and partial nephrectomy [3]. The optimal time for surgery is usually considered to be the second trimester, but it often is not possible to delay urologic intervention, endoscopic or open. Ideally, if one of the temporizing measures mentioned

previously is feasible, further intervention is best delayed until the postpartum period. Asymptomatic renal calculi that are present before pregnancy can become symptomatic during pregnancy, as a result of dislodgement brought about by physiologic hydronephrosis. To avoid this complication and its ramifications for treatment options during pregnancy, and for the well-being of the pregnancy, some authorities have suggested prophylactic ESWL of asymptomatic caliceal stones in women of child-bearing age who are planning pregnancies [55].

The xanthine oxidase inhibitor allopurinol has been used in nonpregnant patients with increased serum or urine levels of uric acid, who have either uric acid or calcium oxalate stones. Allopurinol is a class C drug in pregnancy. Animal studies have shown adverse effects, but no human studies have been performed. D-Penicillamine, used to treat cystinuria, is known to be teratogenic in rats and is rated a class D (unsafe) drug in pregnancy. Some authorities emphasize that both drugs have been used successfully and safely in pregnancy with little evidence of fetal harm, however [2]. Others think that their unknown effects on the human fetus contraindicate their use [41]. Because the safety of these drugs is not fully validated, they are best avoided. Thiazide diuretics have been used in pregnancy to reduce urinary calcium excretion in patients with recurrent calcium oxalate stone formation. Considered a Class B drug (presumed safe based on animal studies), hydrochlorothiazide has been used in pregnancy but carries some risk of fetal thrombocytopenia, hypoglycemia, and hyponatremia [63,64]. If thiazide diuretics are administered to these high-risk patients, close monitoring is mandatory.

## LOWER URINARY TRACT OBSTRUCTION

### **Ureteral Compression**

Acute ureteral obstruction is uncommon in pregnancy, and when seen is usually not due to intrinsic blockage (e.g., by calculi, sloughed renal papillae or other conditions) but to direct compression by the gravid uterus as the ureters cross the pelvic brim [65]. Extrinsic ureteral compression, caused by pelvic masses, uterine prolapse, or leiomyomas, has been reported. Pelvic neoplasms are thought to cause retention by interfering with bladder neck

relaxation. Extensive ureteral compression can also be caused by an overdistended uterus from multiple gestation or hydramnios. Ureteral obstruction from intrinsic or extrinsic sources can result in severe pain from acute hydronephrosis, renal infection, sepsis, or preterm labor.

The treatment for extrinsic ureteral obstruction in pregnancy depends on its etiology. Hydramnios, for example, can be managed by repeated drainage of amniotic fluid or by induction and delivery after fetal pulmonary maturity has occurred. The obstructed ureter can sometimes be decompressed simply by placing the patient on bedrest in the lateral recumbent position. If conservative management fails, a double-pigtail ureteral stent is placed, with removal 4 to 6 weeks postpartum [65]. If stent placement is technically impossible, a temporary percutaneous nephrostomy is warranted if obstruction is severe or complete.

### Impacted or Incarcerated Uterus

The gravid uterus infrequently causes urinary tract obstruction when it is in a retroverted position by incarceration in the true pelvis during the second trimester [20,66,67]. In this situation, the cervix presses against the trigone, obstructing the urethra and causing frequency, overflow incontinence, or urinary retention, depending on the degree of compression. Although unusual, this condition must be recognized and treated promptly. Rarely, death has been attributed to a retroverted, impacted, gravid uterus [67]. Spontaneous miscarriage is also a possible complication. Treatment consists of emptying the bladder, followed by manual replacement of the uterus, with the patient in either the dorsal lithotomy or knee-chest position, and the application of slow, steady transvaginal or transrectal pressure. Anesthesia is rarely needed to permit these manipulations. Subsequent intermittent or continuous bladder drainage by catheterization and placement of a pessary to retain normal positioning is also sometimes necessary until the uterus enlarges sufficiently, rising out of the pelvis, thereby avoiding reincarceration.

### Urinary Retention in Labor

Urinary retention occurs in 10% to 15% of postpartum women, usually as a result of minor trauma to

the bladder or its nerve supply; however, retention is actually more common antenatally [68]. A factor that contributes to urinary retention in pregnancy is the physiologic yet significant drop in intravesical pressure during voiding [68]. In labor, compression of the bladder neck by the engaged fetal head is a possible mechanism. During the second stage of labor, compression of the bladder and urethra between the fetal head and the pubic symphysis can cause edema and irritation to the bladder nerves and detrusor muscle, leading to temporary retention.

Postpartum, trauma and pain from injuries to the perineum can cause reflex contraction of the voluntary (striated) muscle around the lower urethra [16]. This condition, added to the effects of epidural anesthesia, can lead to impaired voiding [69]. The mechanism of urinary retention after cesarean delivery is thought to be a disturbance of the autonomic nerve fibers during mobilization of the bladder, which results in inertia of the detrusor muscle [8].

Recognition of bladder distension during labor is important in avoiding obstructed labor, postpartum retention, bladder rupture, and tissue necrosis leading to fistula formation. Gentle, firm suprapubic pressure (Credé maneuver) usually facilitates bladder emptying, but if this is unsuccessful, catheterization is warranted. Because indwelling catheterization can produce local irritation, interfere with subsequent voiding, and initiate a cycle of retention, further catheterization, and infection [68,69], indwelling catheters are best avoided unless absolutely necessary. If relief is required, and the Credé maneuver does not empty the bladder, intermittent catheterization using meticulous technique is best.

A pelvic kidney is a rare cause of obstructed labor. The incidence of patients with pelvic kidney in labor over a 21-year period at Johns Hopkins Hospital was 1 in 4,886 deliveries [70]. The coincidence of a solitary pelvic kidney and pregnancy is much less common [71]. In these cases, injury during delivery would be disastrous, because this kidney is the sole renal excretory organ. In these women, cesarean delivery is recommended. Because preterm infants are less likely to disrupt the kidney, a trial of labor in carefully selected cases is permissible [68]. Antepartum ultrasonography is helpful in establishing the correct diagnosis.

## **LOWER URINARY TRACT INJURIES AND TRAUMA**

### **Accidental Trauma**

Accidental trauma complicates as many as 5% to 10% of pregnancies [72]. Although most of these accidents are inconsequential, trauma is responsible for more maternal deaths than any other pregnancy-related disease. The most common serious injury to the pregnant woman is blunt trauma from motor vehicle accidents, often resulting in multiple injuries, and responsible for 80% of pelvic fractures [73].

A pelvic fracture is the most common cause of urethral injury during pregnancy [4]. When the patient presents to the hospital, she is often seen by emergency department physicians who might be unfamiliar with obstetric management or, alternatively, by obstetricians unacquainted with management of her injuries. Delays in diagnosis and management in this setting are not uncommon. In a series of 2,000 reported cases of pelvic fracture, Orkin reported the combined incidence of bladder and urethral rupture of 15% [74]. Despite this substantial incidence, urethral and bladder injuries in trauma patients with pelvic fractures are frequently not suspected and often overlooked. Perry and Husmann, in their review of urethral injuries in female subjects after pelvic fracture reported a missed diagnosis rate of 50%, resulting in significant morbidity [75]. Although 80% of patients with known pelvic fracture had blood at the vaginal introitus, only 50% underwent vaginal examination! When the diagnosis of bladder injury is missed, significant morbidity can result from extravasated, infected urine, which can lead to necrotizing fasciitis, septic shock, or cardiovascular collapse.

Urethral injury should be suspected if there is difficulty with urethral catheterization, or if the patient is anuric after a Foley catheter is removed. Additionally, if the gravida experiences difficulty voiding or has vulvar edema (resulting from direct trauma or extravasation of urine) after catheter removal, one should suspect urethral trauma.

Evaluation of suspected urethral injury includes cystourethroscopy and radiographic evaluation with retrograde urethrography. Pregnant trauma patients at 20 or more weeks of gestation are best brought directly to the labor and delivery suite for obstetric evaluation. After fetal evaluation by electronic

monitoring and evaluation of the uterus and its contents by ultrasonography, a careful vaginal examination should be performed if pelvic fracture is suspected or documented. Obviously, specialized consultation and treatment are required in these cases. Surgical management of pelvic fracture and urethral injuries is beyond the scope of this chapter. Obstetric co-management is the rule.

As pregnancy advances, the bladder progressively becomes more of an abdominal organ. Its capacity steadily increases in the last two trimesters to a volume greater than 1 liter [4]. These changes, in addition to partial urethral obstruction from a deeply engaged presenting part, predispose to bladder overdistension and possible rupture. When bladder rupture occurs, it is usually extraperitoneal and can result in blood loss and shock. Trauma to the bladder and urethra can result from accidental injury, labor, obstetric manipulations, and surgery (e.g., cesarean delivery, cesarean hysterectomy, and termination of pregnancy).

### **INTRAPARTUM INJURIES**

In modern practice, prolonged labor, usually involving some degree of disproportion, occasionally results in compression necrosis of the posterior urethra between the fetal head and pubic symphysis. This condition is common in remote areas of developing countries, particularly where trained specialists and hospitals are not immediately unavailable, and obstructed labor continues for several days. In this situation, a urethrovaginal or vesicovaginal fistula develops. With the low incidence of prolonged labor and a concomitant increase in the cesarean birth rate, obstetric injuries to the bladder and urethra have become rare in the Western world. In the past, severe, prolonged pelvic compression occasionally resulted in partial or complete destruction of the urethra, accounting for 5% of the obstetric fistulas requiring repair. Postoperative sequelae of corrected fistulas included urethral stricture in 12% and persistent stress urinary incontinence in 16% [4].

Difficult forceps applications and forceps rotations, with their inherent risk of injury to the urethra as it is compressed behind the unyielding pubic symphysis, can cause injury, including complete urethral transection. Forceps lacerations and, less commonly, the effects of prolonged labor with

unremitting pressure of the presenting part on the bladder can also result in vesicovaginal fistulas. Such heroic deliveries, however, have essentially disappeared in modern practice, and such complications remain at best rare. Infrequently, precipitate labor results in an anterior vaginal laceration that includes the urethra, bladder, or distal ureter. When laceration of the anterior vaginal wall occurs, repair can inadvertently incorporate the bladder floor in the vaginally placed sutures, resulting in a vesicovaginal fistula or ureteral ligation.

Advances in obstetric care have minimized some factors predisposing to bladder injury, while increasing others. Until recently, spontaneous rupture of the bladder associated with uterine rupture during labor was described frequently, with bladder injury accompanying 22% of uterine ruptures [76]. Today, most reports of uterine rupture come from developing countries. In the developing world, the incidence of uterine rupture is 1 in 92 hospital admissions, in contrast to the incidence in the United States of 1 in 1,000 to 1 in 1,500 deliveries [77,78]. In modern practice, most ruptures occur in scarred uteri during spontaneous or oxytocin-induced labor in patients with a prior cesarean delivery scar (i.e., vaginal birth after cesarean [VBAC]).

Spontaneous lacerations will probably be on the rise in the future as a cause for injury to bladder and ureter. The increase in the number of patients with cesarean uterine scars, the performance of VBACs, widespread use of oxytocin augmentation and induction, and prostaglandin cervical ripening – all predisposing to scar separation – will likely increase the frequency of uterine rupture. In addition, transverse cesarean incisions, although associated with one fifth the incidence of dehiscence compared with classic incisions (1%–2% vs. 5%), are more likely to be associated with extension into the bladder if catastrophic rupture occurs [79].

Although obstructed labor is likely to result from dystocia, including malpresentation and cephalopelvic disproportion, an overdistended bladder rarely can be a cause as well, potentially leading to bladder necrosis and fistula formation. As previously discussed, a laboring woman who cannot void is best managed by the Credé maneuver, with the application of gentle suprapubic pressure. If the parturient is still unable to void after these maneuvers, intermittent catheterization is best. Indwelling bladder catheters are not routinely used because of the

risk of infection and the possibility of damage to the bladder base and urethra with descent of and compression by the presenting part [4].

Occasionally obstetric lower urinary tract injury results as a complication of placenta percreta. In placenta percreta, penetration of chorionic villi through the myometrium can result in uterine rupture, bladder invasion and rupture, or massive hemorrhage. Placenta percreta involving the bladder is almost always associated with placenta previa. The likelihood of previa is strongly related to prior obstetric events, including previous cesarean birth, prior dilatation and curettage, and grand multiparity [79]. The placenta accreta/increta/percreta syndrome should be suspected in a pregnancy at risk when on ultrasonic scan, there is loss of the usual subplacental sonolucent area, consistent with absence of the decidua basalis; and when multiple echofree “lakes” are observed within the placenta, among other findings. (See Chapter 18, Cesarean Delivery, and Chapter 11, The Third Stage.)

Case reports in the literature describe management of placenta percreta by cesarean delivery, hysterectomy, and segmental resection of the bladder [80]. At delivery, hemostasis can be aided by ligation or embolization of the anterior branches of the hypogastric artery, with concomitant transfusion of blood and blood products as required [80,81]. Placenta accreta diagnosed antepartum has also been managed by uterine artery embolization with hydroxycellulose (Gelfoam). This treatment has resulted in limited blood loss at cesarean delivery with uterine conservation in a nulliparous woman [81].

### Surgical Injuries

In the last 30 years, the percentage of cesarean deliveries has increased from below 10% to 30% or more, with a concomitant decrease in the number of urethral and trigonal injuries from prolonged labor and difficult forceps procedures. The increasing number of cesarean deliveries has been accompanied by a greater number of intraoperative injuries to the bladder dome and ureters, however. Injury to the bladder is the most common surgical injury of the lower urinary tract, complicating 0.5% to 1.0% of abdominal procedures [82]. The incidence of ureteral injuries is about 0.1% [83]. Whereas the diagnosis of bladder injuries can be

made immediately, recognition of ureteral injuries is often delayed, and these injuries should be suspected if the patient has persistent flank tenderness and unilateral hydronephrosis postpartum.

## **BLADDER INJURY AND MANAGEMENT**

### **Bladder Injuries**

During labor and delivery, the bladder is vulnerable to trauma and injury. Mucosal congestion, submucosal hemorrhage, and capillary oozing around the trigone have been observed cystoscopically after delivery [1]. Physician haste, failure to catheterize the bladder prior to cesarean delivery, and adhesions from prior surgery are common causes of bladder laceration on entering the abdomen. Routine preoperative catheterization reduces this risk. In addition to decompressing the bladder, catheterization allows for easy identification of the bladder base by palpation of the catheter bulb. The currently popular low transverse uterine incision is more likely to be associated with injury to the bladder and ureters compared with vertical incisions, because of anatomic proximity of these structures to the lower uterine segment. If the lower uterine segment is not well developed, such as in patients who undergo cesarean deliveries before the onset of labor, the bladder also can be less well demarcated and thus more vulnerable to injury.

One of the most common associations with bladder injury is prior cesarean delivery. Previous surgery often results in dense adhesions between the bladder and lower uterine segment (the vesicouterine fold), with superior advancement of the bladder over the uterus. The normal anatomic planes can be obscured by such adhesions. The increased vascularity of the bladder and lower uterine segment in pregnancy adds to the risk of inadvertent cystotomy. Blunt dissection in the vesicouterine fold at the bladder base in attempts to mobilize the bladder flap with vigorous use of a sponge stick or finger can cause inadvertent injury. Newton reports a threefold likelihood of bladder injury with repeat cesarean deliveries compared with primary operations (0.6% vs. 0.19%), with most of these occurring during attempts to dissect the adhesions between the bladder and lower uterine segment [3]. The risk of bladder injury is increased to 1.5% after four or more previous uterine incisions. To avoid injury, careful sharp dissec-

tion is recommended. Additionally, repeat cesarean delivery is also associated with a greater incidence of vesicovaginal fistula compared with a primary operation, 0.6% versus 0.31% [2]. This increased risk for vesicovaginal fistula is related to probably inadvertent bladder injury.

Another cause of bladder injury during cesarean delivery is incision into the vagina rather than the lower uterine segment. A markedly effaced and dilated cervix can be difficult to distinguish from the vagina, which is then unintentionally incised. Although the bladder can be easily dissected from the lower uterine segment, it is not as readily separated from the vagina and can be entered inadvertently [84].

Plauché and coworkers reported inadvertent operative cystotomy in 3 of 100 cesarean hysterectomies by [85]. In Plauché's review of 5,185 cases of cesarean hysterectomy, he reported a 0.46% incidence of vesicovaginal fistula [86]. Bladder injury during cesarean hysterectomy can be avoided in most cases by careful separation of the bladder from the cervix and upper vagina, avoidance of lateral dissection into the base of the broad ligament, and slow sharp dissection. After cesarean hysterectomy, if there is any uncertainty concerning a cystotomy, the authors recommend testing the bladder to ensure its integrity. This test is performed by distending the bladder with 300 ml to 400 ml of saline containing indigo carmine or methylene blue dye. If an injury is detected, it should be promptly closed in layers and the catheter left in place for a minimum of 7 days.

Less common fistulas include the vesicocervical, vesicouterine, and urethrovaginal. Vesicocervical fistulas usually occur during cesarean birth from insufficient separation of the bladder from the uterus. Like the vesicovaginal fistula, this defect is demonstrated by leakage of blue-stained dye after an indigo carmine solution is instilled retrograde into the bladder with a tampon or gauze placed in the vagina. In another unusual entity, vesicouterine fistula, urinary leakage might not occur at all. Patients with vesicouterine fistulas can present with amenorrhea and cyclic hematuria [87].

### **Bladder Repair**

Inadvertent cystotomy recognized during surgery is rarely a cause of patient morbidity and is easy to

repair. The dome of the bladder is the site most frequently injured. If the injury is limited to the dome of the bladder, the defect is simply closed in two layers with 2-0 or 3-0 running absorbable suture on a small needle. The first stitch can incorporate all layers including bladder mucosa, although many surgeons attempt to omit the bladder mucosa and include only the submucosa and muscularis layers [82,88]. The second imbricating layer may be either a parallel Lembert or a perpendicular Connell stitch. Filling the bladder with sterile milk, methylene blue, or indigo carmine dye solution can reveal other bladder defects. Although the bladder repair need not be watertight, a reasonable effort to isolate major defects and produce a layered closure is required [88]. Bladder drainage by transurethral or preferably suprapubic catheter for 7 to 10 days is recommended. If the injury is not clearly in the bladder dome but leakage has been documented, vertical extension of the cystotomy should be implemented, with the injection of indigo carmine dye intravenously to ensure ureteral integrity. Injuries extending into the trigone or ureteric orifices might require ureteric implantation or stenting.

### Vesicovaginal Fistula Repair

Vesicovaginal fistulas are rarely seen in the United States, but they are more common in developing countries. If the labor is obstructed, necrosis can develop in the anterior vaginal wall and the bladder, predisposing to fistula formation. For the treatment of vesicovaginal fistula, current therapy is either an immediate repair or, alternatively, a delayed repair after inflammation has resolved [89-91]. A vesicovaginal fistula is repaired immediately if the diagnosis is made, within 2 to 3 days of the precipitating injury – usually gynecologic surgery or obstetric trauma. If the fistula is detected more than 3 days after injury, a 1- to 3-month waiting period with continuous drainage is necessary before attempting a repair. Spontaneous closure can be expected in 20% to 30% of cases during this period of observation. A vesicovaginal fistula can be repaired either vaginally or abdominally [90,91]. The preferred route is transvaginal, but an abdominal route is sometimes necessary, depending on the site and extent of the fistula. The location of the fistula in relation to the ureteral orifices is especially important. There should be sufficient exposure to

excise scar tissue and place two rows of suture without encroaching on the ureteral orifices or ureters. Ureteral catheterization can be used during surgery to mark the ureters and to avoid their inadvertent ligation or injury. If there is doubt about the use of a vaginal approach to provide satisfactory access for suturing, an abdominal transvesical approach is preferred.

Definitive vesicovaginal fistula repair by the transvaginal approach involves excision of the fistulous tract and scar tissue, with mobilization of the tissue planes beyond the scar tissue, followed by a layered closure. Healing of the repaired fistula depends on good vascularization of the edge of the dissection, and the absence of tension at the repair site. The vaginal epithelium should be separated from the bladder at least 2 cm circumferentially around the fistula. The bladder is then closed in two layers, taking care to avoid stress at the site of reapproximation (Figure 19.6).

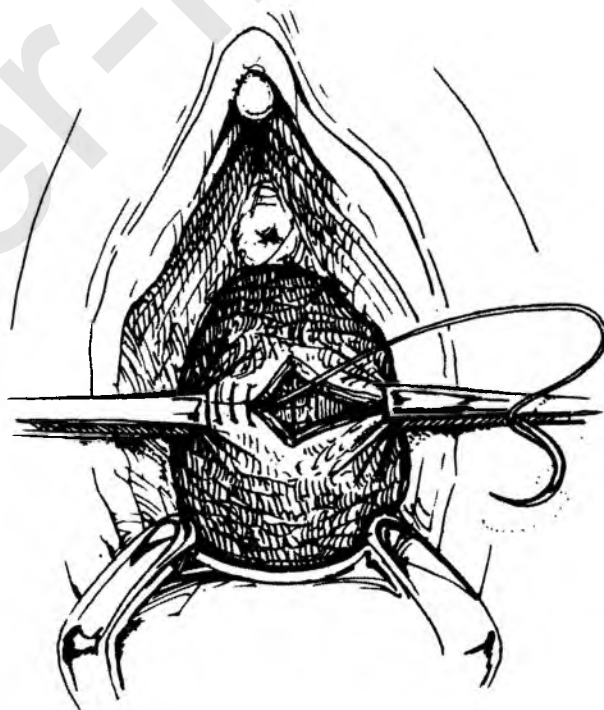


FIGURE 19.6.

*Vaginal closure of vesicovaginal fistula. After excision of fistula tract, the bladder is closed in two vertical layers. The figure illustrates the vaginal epithelium being closed in a horizontal plane at right angles to the bladder closure. (From Plauché WC, Morrison JC, O'Sullivan MJ (eds): Surgical Obstetrics. Philadelphia: WB Saunders, 1992; with permission.)*





FIGURE 19.7.

*Martius bulbocavernosus fat pad graft for fistula from vagina to urethra or bladder. A, Lateral labia majora opened vertically and fat pad next to muscle mobilized, preserving vascular pedal inferiorly. B and C, Fat pad tunneled beneath labia minora and vaginal mucosa and sutured to fascia of urethra and bladder. D, Vaginal mucosa and vulvar incision closed separately without tension. (From Thompson JD, Rock JA: *TeLinde's Operative Gynecology*. Philadelphia: JB Lippincott, 1992: p. 815; with permission.)*

If the blood supply is suboptimal or supportive tissues are weak, a vascular pedicle such as a Martius flap of bulbocavernosus muscle and fat can be interposed between the bladder and vaginal epithelium (Figure 19.7). The bulbocavernosus interposition is performed by tunneling under the dissected vaginal epithelium deep to the labium majorum. The distal bulbocavernosus muscle, transected to create a vascular flap, is rotated through the tunnel and sutured over the repair. The resultant pedicle graft, with its proximal blood supply intact, improves vascularization and healing. The fistula repair is completed by closing the vaginal epithelium horizontally, preferably at right angles to the line of bladder closure. Although suprapubic drainage is preferable for avoiding infection, Foley transurethral catheter drainage is reasonable, provided that the bulb does

not rest on the repair site. Loose vaginal packing for 1 day and bladder drainage for 7 to 10 days are recommended. Although this technique is suitable for closure of simple vesicovaginal fistula, more complicated fistulas require different urologic techniques, which are beyond the scope of this chapter.

### Repair of Other Vesical Fistulas

Surgical repair of vesicocervical and vesicouterine fistulas includes separation of the bladder from the uterus or cervix, and careful identification and removal of the fistulous tract. The repair is performed in a meticulous, dry, layered-closure method through an abdominal incision [90,91].

## URETHRAL INJURY AND MANAGEMENT

### Urethral Injuries

Compression injuries to the urethra often go undetected when they occur, and patients then present with urethrovaginal fistula formation, usually 5 to 7 days after the original trauma. If the urethrovesical junction is involved, incontinence can result. In either case, cystourethroscopy and cystourethrography are advisable to define the extent of the injury or fistula and to exclude the presence of other fistulous tracts.

### Urethral Repair

Urethral injury should be repaired at the time of diagnosis by reapproximation over a urethral catheter or stent. A two-layer repair, closing mucosa and muscularis separately with interrupted 3–0 absorbable sutures transversely, prevents urethral constriction during healing. Once suturing is completed, the urethral catheter can be replaced by a suprapubic bladder catheter to prevent continued trauma and pressure on the suture line, with possible resultant pressure necrosis and fistula formation. Voiding trials can be begun 3 to 7 days after repair, depending on the severity of the injury.

### Urethrovaginal Fistula Repair

Repair of a urethrovaginal fistula should be performed only after resolution of edema and inflammation, usually 6 weeks to 6 months after the date of the injury. If the fistula is small, it might resolve with

decompression and suprapubic catheter drainage while under observation. Repair of urethrovaginal fistulas is generally performed in the same manner as that for urethral injuries [90,91].

## URETERAL INJURY AND MANAGEMENT

### General Considerations

Ureteral injury resulting from vaginal birth or operative delivery is unusual, and the consequences are potentially serious. Unilateral ureteral injury, especially if unrecognized, can lead to kidney loss. Bilateral damage or ureteral damage with a solitary kidney can be life threatening. Trauma is a major cause of injury to the lower urinary tract. When trauma results in injury to the ureter, it frequently occurs where the ureter crosses the pelvic brim. Ureteral injury, however, is much more likely to happen during gynecologic or obstetric surgery [74].

Rarely, ureteral injury occurs during vaginal delivery as a result of precipitate delivery or a difficult forceps application. More frequently, however, attempts to repair extensive lacerations of the cervix or vagina can ligate, lacerate, or transect the lower ureter. This type of injury is usually not recognized immediately postpartum, and ureterovaginal fistula formation can appear 4 to 6 weeks later. If the lower ureter was injured by forceps, in most cases the lateral margin of the uterus and the adjacent uterine vasculature were also damaged. In this instance, an emergency hysterectomy might be necessary to control the resulting hemorrhage [4]. The ureteral injury might go unrecognized during surgical attempts to control hemorrhage, because attention is focused on maintaining hemostasis. In these instances, the ureter therefore should be visually inspected and checked, if necessary, for patency perioperatively by the passage of a stent.

With the increase in the number of low transverse cesarean incisions compared with classic incisions, there has also been an increase in the occurrence of ureteral trauma. Lateral extension of the cesarean incision into the broad ligament with concomitant bleeding from uterine vessels is the usual scenario. During hurried attempts at controlling blood loss, the ureter is cross-clamped or inadvertently incorporated into the uterine artery pedicle in a mass ligature. The key to avoidance of ureteral injury is careful identification of the uterine and hypogastric arteries. Eighty to ninety percent of ureteral injuries

involve the distal ureter from its location beneath the uterine vessels to its entry into the trigone [82]. Applying pressure with a sponge stick proximally to stop blood flow until an accurate identification of these structures is made is a useful maneuver when suturing near the pelvic sidewall is required to control hemorrhage. The lateral parietal peritoneum is opened to expose the hypogastric artery. The hypogastric artery is then ligated distal to the superior gluteal artery. Alternatively and preferably, the uterine artery is directly ligated by the O'Leary method, avoiding the problem entirely. (See Chapter 18, Cesarean Delivery and Surgical Sterilization.) The ureter is usually easy to identify positively at the pelvic brim. Its course can then be directly followed into the pelvis. If there is a problem, this step should not be omitted.

Cesarean hysterectomy is associated with a 0.44% incidence of ureteral injury and a 0.09% incidence of ureterovaginal fistula [88]. Conditions present at cesarean hysterectomy predisposing to ureteral injury include distortion of pelvic anatomy from the enlarged uterus, the presence of edematous tissues in the lower pelvis, and adhesions from prior cesarean births or laparotomies [89]. Increased vascularity also increases the risk for brisk bleeding, especially from the uterine arteries, which are close to the ureters. In addition, distortion (caused by hematomas or pelvic tumors) and cervical dilation further increase the risk of ureteral injury. Aside from the risk of ureteral injury at the level of the uterine vessels, the ureter is also vulnerable to injury at the juxtavesical region during vaginal angle closure. The vaginal cuff is large, edematous, friable, and well supplied with vessels. Recurrent bleeding from the cuff or the cardinal ligament is common, and repeated suturing is usually required for control. Ureteric injury can be avoided by meticulous technique, with close attention to avoiding blind clamping or suturing. If there is any uncertainty, the ureters should be simply traced directly into the pelvis as previously described. Stents can be placed if ureteral patency is uncertain. A better surgical technique is to avoid cervical removal during an emergency postpartum hysterectomy. (See Chapter 18, Cesarean Delivery and Surgical Sterilization, for additional discussion.)

Injury to the ureter either with a vacuum aspirator or a sharp curette occurs rarely during surgical interruption of pregnancy. The literature contains at least one report of uterine perforation

during sharp curettage, with the segment of ureter discovered by the pathologist in the specimen along with products of conception [92]! Complete avulsion of the ureter after termination of pregnancy with a vacuum aspirator, with delayed appearance in vaginal discharge, has also been reported [93]. Most uterine perforations are caused by efforts at cervical dilation. Because there has been an increase in two-step termination procedures with cervical dilation by Laminaria prior to curettage, a concomitant decrease in uterine perforations and their sequelae is expected. (See Chapter 6, Pregnancy Termination, for a discussion of recommended techniques.)

When a urologic injury or abnormality is suspected, an IVP is indicated and a retrograde ureterogram is sometimes required [93–96]. Cystoscopy is performed after one ampule of indigo carmine dye has been given intravenously. Passing a ureteral stent should also be attempted but might be unsuccessful. If a ureterovaginal fistula is suspected, giving indigo carmine intravenously should result in staining of a tampon in the vagina, whereas bladder instillation of the dye does not cause tampon staining. If an attempt at retrograde stent placement is successful, the stent is left in place for 6 weeks to allow for the possibility of spontaneous closure. In ureterovaginal fistulas, Dowling and colleagues, however, report successful ureteral catheterization in only 5% of cases [97]. If a trial of stent passage from below fails, an attempt to pass a stent antegrade through a percutaneous nephrostomy should be made. The stent is removed 6 weeks later, and serial IVPs every 3 months for 1 year are obtained to ensure patency of the ureter. The timing of ureterovaginal fistula repair is controversial. Although some physicians suggest a waiting period of 4 to 8 weeks, most recommend early repair [89,95].

Several types of ureteral injuries occur and are discussed separately below: ligation injuries, with or without urine leakage; a needlestick or incision into the ureter; and complete transection of the ureter. Management of injuries is optimal at the time of surgery, or in general, as soon as the injury is diagnosed.

### **Crush Injuries, Suture Ligation, Incision, and Needle Puncture**

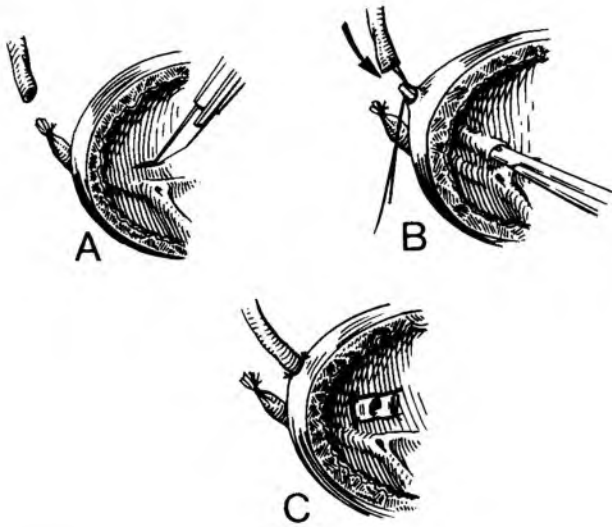
If the ureter is inadvertently included in a clamp or ligation, release of the ureter should be followed by

several minutes of observation for ureteral peristalsis and return of normal color. A ureteral stent can be placed for 5 to 7 days if observation is not reassuring. Some authorities recommend stent placement only if there is a question of tissue viability. In the authors' opinion, if the ureter appears normal, management need include only suction drainage adjacent to the site of ligation to prevent formation of a urinoma [95]. Many experts favor a simple nonsuction Penrose-type drain, theoretically to avoid suction drainage of the ureter with resultant trauma. If further surgery is required, ureteroneocystostomy is preferred, with ureteroureterostomy being reserved for damage to the midureter. A small incision or needle puncture to the ureter can be repaired with interrupted 5–0 absorbable sutures, and a suction or nonsuction drain of operator's preference left proximal to the injury for a few days.

If ureteral obstruction is diagnosed postoperatively, cystoscopically guided ureteral catheterization is recommended. Ureteral fistulas can be managed similarly, possibly obviating the need for further surgery. If a ureteral catheter cannot be passed to the renal pelvis, the catheter should be left in place to facilitate identification of the level of obstruction during subsequent surgery or nephrostomy. If tissue edema and inflammation are extensive, renal decompression by percutaneous nephrostomy is sometimes a necessary temporizing measure, followed by delayed reconstruction. If complete ureteral obstruction is diagnosed within 48 to 72 hours of the original injury, immediate repair is preferable if technically possible; however, some surgeons prefer delaying the repair for 6 to 8 weeks. Many patients, if given a choice, prefer immediate repair, and their preferences should also be considered in surgical decision making.

### **Ureteral Transections**

For complete severance of the ureter within 8 cm of the bladder, the repair of choice is ureteroneocystostomy. For injuries above this area, ureteroureterostomy is sometimes necessary. Ureteroneocystostomy involves transecting the ureter just above the site of distal ligation and spatulating the proximal ureter distally with 4–0 or 5–0 absorbable pilot sutures placed at the site of spatulation (Figure 19.8). A cystotomy is then performed. A small incision is then made in the posterior bladder, just large



**FIGURE 19.8.** Submucosal tunnel technique of ureteroneocystostomy. A, An incision is made in bladder mucosa near original ureteral orifice. B, A 2-cm submucosal tunnel is created with curved clamp; the cut end of the ureter is tagged and pulled into bladder lumen. C, After the distal ureter is spatulated and everted, it is sutured in four corners to the bladder mucosa. The ureter is also sutured to the bladder adventitia. (From Walters MD, Karram MM: *Clinical Urogynecology*. St. Louis: Mosby-Year Book, 1993; with permission.)

enough to admit the spatulated ureter and its pilot sutures. The ureteral sutures are sewn to the bladder wall and tied with the knots outside the bladder mucosa. The periureteral tissue is reapproximated to the bladder muscularis. The anterior cystostomy is closed with 2-0 chromic gut sutures, and the prevesical space is drained.

Another type of ureteroneocystostomy, designed to be an intentional antireflux technique, involves creating a 2- to 3-cm submucosal tunnel in the bladder wall (Figure 19.8). Only an experienced surgeon should perform this procedure. Although a nonrefluxing tunnel of bladder muscularis is preferred, this might not be technically possible. Most patients with the anastomosis, without bladder tunneling described previously, have experienced no reflux or other significant problems [96].

If the ureter is transected above the pelvic junction, it is best repaired by end-to-end anastomosis. An alternative procedure, performed when >10 cm of distal ureter has been damaged, is transureteroureterostomy [96]. This technique is performed by anastomosing the short transected ureter to the nor-



**FIGURE 19.9.** Psoas hitch technique for relief of tension on suture site in ureteroneocystostomy. The bladder is drawn cephalad if tension exists near the distal ureteral repair. (From Walters MD, Karram MM: *Clinical Urogynecology*. St. Louis: CV Mosby, 1999; with permission.)

mal contralateral ureter, end to side. Despite a very low complication rate, many surgeons are reluctant to adopt this procedure, for fear of compromising the healthy ureter [96].

A tension-free anastomosis is essential to the success of ureteral implantation. Tension between the ureter and bladder can be relieved by incising the parietal peritoneum on the involved side and mobilizing the bladder. The superior lateral aspect of the bladder wall is then fixed to the psoas fascia [98]. This *psoas hitch* is illustrated in Figure 19.9. Another technique, which can bridge a gap of up to 10 cm, is the *Boari flap procedure*. This procedure uses a wide-based bladder flap to create a tube into which the

ureter is anastomosed [99,100]. Other more radical techniques for bridging a long ureteral gap include the interposition of an isolated segment of bowel and autotransplantation of the kidney with preservation of its blood supply and collecting system into the pelvis [100,101]. These more complex operations should be reserved for the highly experienced urologic or urogynecologic surgeon and should never be attempted by less-experienced surgeons without immediate, expert assistance readily available.

## URETHRAL DIVERTICULUM

### **Etiology**

A suburethral diverticulum in pregnancy, although an uncommon finding, can complicate gestation. Its asymptomatic development almost always antedates the pregnancy. The etiology of suburethral diverticulum is either congenital or acquired as a result of infection and microabscess formation in the urethral glands. In pregnancy, recurrent suburethral diverticulitis is aggravated by edema and thickening of the urethral wall. Retrograde contamination of the bladder can result in recurrent episodes of acute cystitis or ascending pyelonephritis.

### **Diagnosis**

In four cases reported by Moran and coworkers, the clinical presentation of urethral diverticulum in pregnancy includes the palpation of a paraurethral mass, irritative urethral symptoms, urinary incontinence, urinary tract infection, voiding difficulty, or urethral pain and discharge. The diagnosis of urethral diverticulum is always suspected when the discharge of purulent exudate from the urethral meatus is observed during urethral massage. Palpation of a tender suburethral mass and visualization of the diverticular orifice on urethroscopy suggest this diagnosis as well. Transvaginal ultrasonography can also be successful in patient evaluation. Definitive radiographic diagnosis by double-balloon urethrography using a Tratner or Davis catheter should be delayed until after delivery.

### **Management**

Because of the inherent risk of repair breakdown and fistula formation, a suburethral diverticulum

is best managed conservatively during pregnancy. The diverticulum is not considered an indication for cesarean delivery, although Allen and coworkers have reported obstructed labor from a urethral diverticulum [103]. More than 50% of urethral diverticula regress postpartum. Repeated urethral massage, diverticular aspiration, and broad-spectrum antibiotic therapy can aid regression, however. Incision and drainage have also been employed [102]. Surgical treatment is best postponed until 3 to 6 months postpartum because of the high failure rate during pregnancy that is associated with surgery on the edematous, inflamed, friable, and highly vascular urethral mucosa and paraurethral tissues. The diverticulum is repaired transvaginally by opening the mucosa, partial removal of the diverticular sack, and layered closure. Partial removal of the sack is advocated to leave sufficient urethral mucosa in situ for a closure without undue mucosal tension or stenosis. There is no contraindication for vaginal delivery in these women; however, diverticular aspiration might be needed during the second stage of labor to aid the delivery and to prevent urethral damage or rupture of the diverticular sac [102].

## PREVIOUS UROLOGIC SURGERY

Advances in surgical management of many urinary tract abnormalities, congenital and acquired, have led to an increased number of pregnant women who have had previous surgery on the urinary tract. These operations include augmentation cystoplasty (for intractable detrusor instability or myelodysplasia), urinary diversion procedures (ileal conduit or ureterosigmoidostomy), ureteral reimplantation (usually for vesicoureteral reflux), and incontinence procedures (retropubic urethropexy, suburethral sling, tension-free vaginal tape, transobturator tape, or artificial urinary sphincters).

In the past, patients with abnormal urinary tracts were managed primarily by cutaneous diversion. These patients often sustained a resultant decline in physical appearance and self-image, making reproduction a less desirable option. The replacement of cutaneous diversion by continent internal diversion procedures, in combination with intermittent catheterization, has no doubt made pregnancy a more realistic possibility for these women.

## General Recommendations

For all pregnant patients with a history of surgically altered urinary tracts, close monitoring for infection by monthly culture and suppressive antibiotic therapy (e.g., macrocrystalline nitrofurantoin, 100 mg nightly) are advisable. Symptomatic infections are treated with an appropriate antibiotic for at least 10 days; thereafter, suppressive treatment is resumed. Management includes meticulous patient instruction, frequent clinical visits, bedrest, and pelvic examinations as clinically indicated. Patients should be informed of the risks of urinary tract infection, as well as of preterm labor, and carefully advised of warning signs. Monthly BUN and serum creatinine determinations should be performed, with close monitoring for other symptoms and signs of ureteral obstruction. Rixe recommends measurement of 24-hour creatinine clearance and urine protein levels instead of simple measurement of BUN and serum creatinine, since the latter provide only crude indices of renal function [104]. More than 50% of renal function must be lost before either BUN or creatinine levels reflect an abnormality or before any symptoms of renal insufficiency develop.

Most patients with prior urinary tract surgery usually can deliver vaginally. Exceptions include patients with urinary diversion to the sigmoid colon, or those who have undergone vesical neck elevation or surgery for sphincter repair. If a patient has had extensive urologic surgery, such as enterocystoplasty, an experienced urologic surgeon should be immediately available if cesarean delivery is required or elected.

## Enterocystoplasty

Infection, obstruction, and trauma at cesarean delivery are potential complications for all pregnant patients because of the changes in pelvic anatomy caused by the enlarging uterus. The altered urinary tract anatomy in patients who have undergone ileocecal cystoplasty bladder augmentation presents additional antepartum and intrapartum risks. The enlarging uterus has the potential to compromise the mesenteric blood supply, leading to ischemia or hemorrhage [105]. In the event of amniocentesis or cesarean delivery, special care is necessary to recognize and not disturb the mesenteric blood supply of the augmented bladder. Clinicians should recall that marked adhesions from previous exten-

sive abdominopelvic surgery are frequently present in these patients.

Pregnancies in women who have had prior augmentation cystoplasty are often complicated by urinary tract infections or pyelonephritis (60%) and premature labor (26%) [106]. Hill and Kramer's review of 15 pregnancies in 15 women after augmentation cystoplasty reported 10 vaginal births and 5 cesarean deliveries [106]. Two of these operations were performed for obstetric indications, and three were performed electively to preserve vesical neck or artificial sphincter construction. One patient who was continent before delivery became incontinent after vaginal delivery. In a retrospective study of 19 pregnancies in 18 women who have undergone clam enterocystoplasty, vaginal delivery was found to be a safe option, even in those who had had an anti-incontinence procedure in addition to enterocystoplasty [107]. Women who have undergone enterocystoplasty are not at increased risk for incontinence and should undergo vaginal delivery if possible to avoid the risk of surgical injury to the augmented bladder. If cesarean delivery is necessary for these women, the authors recommend that a urologic surgeon experienced in augmentation cystoplasty be present at the time of the laparotomy.

## Artificial Urinary Sphincters

Some researchers have recommended that patients who have undergone reconstruction of the vesical neck or implantation of an artificial genitourinary sphincter undergo cesarean delivery to avoid disruption of the continence mechanism. Fishman and Scott have suggested that the obstetrician who best understands the clinical case should individualize the mode of delivery for patients with an artificial urinary sphincter [108]. They report seven patients with artificial urinary sphincters who underwent nine deliveries: five vaginal and four cesarean. All patients who underwent vaginal delivery remained dry, whereas one patient who experienced slight leakage during pregnancy underwent a cesarean and continued to leak after delivery. Hill and Kramer think that vaginal delivery is contraindicated in patients who have undergone artificial sphincter placement and augmentation cystoplasty and that elective cesarean delivery is the best management for such patients [106]. For pregnant women with artificial urinary sphincters, it is

recommended that broad-spectrum antibiotics be administered in the immediate perinatal period and that the urethral cuff of the implant be deflated frequently in the third trimester and during labor and delivery [108,109]. In addition, if cesarean delivery is necessary, abdominal ultrasonography should be used to locate the components of the device to avoid perioperative injury. Electrocautery should be avoided or restricted to the tissues superficial to the uterus to avoid injury to the silicone tubing of the implant.

Long-term efficacy of artificial urinary sphincter and the impact of pregnancy on three young female patients were reported recently [110]. These three patients had a good long-term outcome with the artificial urinary sphincter, including one patient with two pregnancies that ended in a normal vaginal delivery. An artificial urinary sphincter also does not appear to affect the pregnancy and can be considered as a favorable option in young women who have intrinsic sphincter deficiency and who are planning future childbearing.

### Anti-incontinence Procedures

Bladder neck reconstruction, when performed for incontinence, is usually deferred until after childbearing is completed to avoid potential damage to the repair during childbirth. When pregnancy occurs after incontinence surgery, the question arises about the optimal mode of delivery. Cutner and coworkers [111] advocate urodynamic investigation to guide this decision. Vaginal delivery is often considered the best choice if the patient develops severe genuine stress incontinence antepartum, because the urethral sphincter mechanism might already be damaged. Reliance on symptomatology alone is inadequate, however, because incontinence in pregnancy can be due to detrusor instability [25,26]. Therefore, optimal management of patients with previous anti-incontinence procedures is controversial. Most clinicians recommend routine cesarean delivery, although others permit a trial of vaginal birth. In a questionnaire survey, 40% of the 149 surveyed clinicians reported that they would always perform cesarean delivery, whereas 28% thought that a trial of labor and vaginal delivery was indicated in patients who have undergone previous anti-incontinence procedures [112]. In this survey, postpartum continence was preserved in 73% of the women with vaginal delivery as opposed to 95%

of those having undergone cesareans. Because no prospective or validated data are available for either choice, a legitimate case can be made for either mode of management. Patient counseling regarding potential risks of each type of delivery is essential for proper informed consent. A detailed note in the medical record outlining the issues and the reasons for the approach taken is prudent. The patient's preferences should also be honored.

In recent years, *tension-free vaginal tape* (TVT) and *transobturator tape* (TOT) have become popular suburethral sling procedures [113,114]. In one case report, the patient became pregnant after the TVT procedure, and ultrasound assessment during pregnancy revealed an unchanged location and topography of the polypropylene (Prolene) tape [115]. Cesarean delivery was performed at term, and the position of the tape continued to be unchanged in the postpartum period. Most clinicians recommend cesarean deliveries for women who have had previous TVT procedures; however, spontaneous vaginal delivery at term was reported on a patient who had had a TVT procedure before pregnancy [116]. At 5 months postpartum, this patient was continent, and urodynamic evaluation showed normal urethral pressure profiles and sufficient maximal urethral closure pressure. The position of polypropylene (Prolene) tape was unchanged as shown by introital ultrasound. This case suggests that vaginal delivery could be an option for women who had tension-free suburethral slings such as TVT or TOT; however, no definitive recommendation can be made owing to the lack of validated data.

### Urinary Diversion

Potential problems associated with pregnancy for patients after urinary diversion include premature delivery (20%–50%), pyelonephritis (15%), urinary obstruction (13%), and intestinal obstruction (10%) [105]. In patients who have had previous ureterosigmoidostomy, cesarean delivery is indicated to maintain continence of the sphincter. In a series of four patients with ureterosigmoidostomy, reversible dilation of the upper urinary tract was observed, and with antibiotic prophylaxis, each of these patients did have one episode of urinary tract infection during pregnancy [117]. One patient developed preeclampsia, three patients underwent cesarean birth, and one patient underwent vaginal delivery. During pregnancy, urologic examinations were

performed every 4 weeks by renal ultrasound scan, calculation of the resistive index, blood gas analysis, and blood tests for electrolytes, BUN, and creatinine levels. No postpartum or neonatal complications were reported. In another series, severe upper urinary tract infections during pregnancy were reported in two patients with existing ileal conduits who had discontinued antibiotic prophylaxis [118]. These cases suggest that antibiotic prophylaxis is mandatory throughout the duration of the pregnancy.

Many women who have undergone urinary diversion procedures have done so because of congenital bladder exstrophy. This abnormality, which is due to a defect in structures derived from infraumbilical mesenchyme, is accompanied by a wide separation of the pubic rami. This weakens the pelvic floor, leading to uterine prolapse. Because vaginal delivery can put the patient at greater risk for prolapse, Freed recommends cesarean delivery in this unique clinical setting to prevent pelvic relaxation [8]. In the authors' experience, the liberal use of serial ultrasound examinations during pregnancy, and judicious timing and use of cesarean delivery is a reasonable method of management. Co-management by and consultation with urologic and perinatal colleagues is advisable.

Another obstetric consideration in patients with corrected bladder exstrophy is fetal malpresentation. The separation of the pubic rami divert the presenting part away from the vaginal canal. In 25% of these women, the lie is transverse or the presentation is breech. If pregnant patients with corrected bladder exstrophy are candidates for vaginal delivery, perineorrhaphy is often necessary because of the usual presence of a high posterior vaginal wall. In many of these patients, the vaginal introitus is immediately below the urethra. In some cases, accompanying vaginal stenosis has made cesarean delivery necessary.

### Ureteral Reimplantation

Ureteral reimplantation has been performed routinely for primary vesicoureteral reflux for more than 50 years. Austenfeld and Snow retrospectively reviewed complications occurring in pregnancies established at least 15 years after the original procedure [119]. In 30 women having 64 pregnancies, urinary tract infection was present in 48% after reimplantation, and in 57% during pregnancy.

Pyelonephritis occurred in 17% during pregnancy, compared with 4% in the nonpregnant state. The authors attributed the high rate of pyelonephritis not to recurrent reflux during pregnancy but to occult bacteriuria missed on prenatal screening. Spontaneous abortions occurred in 8 of 64 pregnancies (13%) between 9 and 21 weeks of gestation. Urinary tract infection was present in six of these eight cases. In a larger series, Mansfield and coworkers retrospectively reviewed 141 pregnancies in 62 women who had childhood ureteral reimplantation and found that 40% of these pregnancies were complicated by urinary tract infections and 15% resulted in spontaneous abortion [120]. Recently, in another series of 47 pregnant women with childhood ureteral reimplantation, urinary tract infection was present in 28%, preeclampsia in 7%, and transient gestational ureteric obstruction in 0.05% (2 patients) [121]. Another two cases of late gestational ureteral obstruction were reported in patients who had had a successful Politano-Leadbetter ureteral reimplantation 17 to 22 years earlier [122]. The obstructions required urinary drainage by percutaneous nephrostomies during pregnancy and gradually subsided postpartum. These data demonstrate the high risk of urinary tract infection and the utility of antibiotic prophylaxis during pregnancy. In these women, the risk of spontaneous abortion is not significantly different from that of the general population. Ureteral obstruction, although rare, should be recognized and promptly treated.

The authors of these studies did not provide recommendations for mode of delivery or descriptions of any difficulty during vaginal or cesarean delivery. It was suggested that after reimplantation, occult urinary infection might be a precursor to spontaneous abortion and ascending infection. For these high-risk patients, more aggressive urinary screening and prompt treatment of infection during pregnancy, in addition to antibiotic prophylaxis, are recommended.

## GENITOURINARY MALIGNANCY

### Epidemiology

Genitourinary malignancies reported during pregnancy include cancers of the bladder and kidney [123,124]. Such tumors presenting during pregnancy are rare, because most patients with these



diseases are more than 50 years old and male (male-to-female ratio of 3:1). Fewer than 50 cases of kidney tumors [125] and 27 cases of bladder cancer [126] have been reported in pregnant patients. When seen in Western practice, bladder cancer is transitional cell in origin in over 90% of cases [127]. Cancer of the bladder is more common in the Far East because of chronic schistosomal bladder infection. Such bilharzia-related carcinomas are usually squamous cell in origin [127].

### Etiology

The bladder is sensitive to induction by carcinogens, including tobacco smoke, dyes, and organic chemicals. Other agents capable of tumor initiation include phenacetin, which can cause interstitial nephropathy, and foreign bodies or infections, which can cause chronic bladder irritation. Long-term indwelling catheters and bladder schistosomiasis are associated with chronic inflammation and can predispose to squamous cell carcinoma. Laurie and coworkers have reported adenocarcinoma in urachal remnants in the bladder dome. Renal malignancy, a relatively uncommon cancer, represents about 3% of all carcinomas in adults. In contrast to bladder cancer, no known relationship exists between renal cancer and industrial or occupational carcinogens. It has been postulated that renal cancer presenting during pregnancy might be linked to the relative state of systemic immunosuppression in pregnancy. Impaired immune surveillance in pregnancy could allow malignant cells to proliferate [129]. An explanation for the rarity of renal cancer is not satisfied by this theory, however, and the etiology of this disorder remains unknown.

### Diagnosis

Hematuria, the most common clinical symptom of bladder cancer in pregnancy, can be mistaken for cystitis, urolithiasis, or vaginal bleeding. Hematuria was present almost universally in one report but absent in two of three patients according to another [123,124]. Hematuria during pregnancy should be evaluated by a catheterized urine specimen; this sample should be examined microscopically and by culture. The most common cause of hematuria in pregnancy is cystitis. Thus, if the patient's signs and symptoms are consistent with a urinary tract infec-

tion, initial conservative management of hematuria with antibiotics is best. Failure of the hematuria to resolve after appropriate antibiotic therapy requires cystoscopic evaluation, regardless of the period of gestation. When unresolved hematuria appears in the third trimester, the differential diagnosis of placenta percreta should be entertained, particularly in patients who had undergone previous cesarean birth. If the source of bleeding is not identified at cystoscopy, a renal ultrasound is warranted, followed in sequence by urinary cytology, magnetic resonance imaging (MRI), a restricted-exposure IVP, and cystography [125,127].

Cystoscopy with topical anesthesia is tolerated well in pregnancy and without any fetal adverse effects. Cystoscopy also provides the added advantage of tissue biopsy for subsequent analysis. Ultrasonography is a valuable tool because it is noninvasive. In the case of upper urinary tract pathology, renal ultrasound scan might delineate a renal mass or hydronephrosis. Ultrasound scan can also detect bladder tumors with a 95% detection rate for lesions greater than 2 cm; however, for tumors less than 0.5 cm in diameter, the rate of detection falls to less than 33%. MRI has the advantage of not exposing the fetus to ionizing radiation. The technique has proved useful in establishing the extent of the malignancy and the nature of the tumor.

### Management

Bladder carcinoma can be treated at any time in pregnancy by transurethral resection if the lesion is superficial, papillary, and low grade in appearance [127,130]. Although the number of case reports is limited owing to the rarity of the disease, transurethral resection has been reported to be safe in pregnancy. This procedure also provides tissue samples for histologic analysis and could be a potential cure for a superficial tumor. For invasive bladder cancer, the management is based on gestational age and the patient's wishes. In the first and second trimesters, pregnancy can be terminated and the cancer managed per standard protocol. If the patient is in the third trimester or in second trimester and desires to proceed with the pregnancy, a modified metastatic work-up with MRI, chest radiography, and bone scan can be performed first to guide the decision [127]. Muscle-invasive bladder cancer without evidence of metastasis is managed surgically

by cystectomy and urinary diversion, in combination with a hysterectomy or a cesarean hysterectomy [123]. Metastatic bladder cancer carries a poor prognosis and, although systemic chemotherapy has produced some encouraging results, the fetal effects of these regimens are unknown. Chemotherapy should be delayed until the immediate postpartum period [127,129].

Radical nephrectomy is the treatment for renal carcinoma regardless of pregnancy status. Although nephrectomy should be performed soon after diagnosis, a 2- to 3-week delay while awaiting fetal maturity in the third trimester is acceptable. With current medical advances, more than 90% of infants delivered after 27–28 weeks survive and have a good prognosis. Recently, laparoscopic radical nephrectomy for renal cell carcinoma has been used effectively and safely in the first or second trimester, and the patients delivered vaginally at term [131,132]. Adjunctive radiation therapy any time during pregnancy is contraindicated, and there is no effective chemotherapy.

## CONCLUSION

Although urologic and urogynecologic complications can present unique opportunities for problem solving and challenging diagnostic dilemmas for the practicing obstetrician, most conditions can be managed with care and precision when principles of systematic rational decision making are followed. Liberal use of consultants and evidence-based management plans can be expected to produce optimal outcomes. In conditions where compelling evidence basis is unavailable, particularly in deciding the mode of delivery, the individual patient's preferences should be strongly considered and honored. All discussions with the patient and all management decisions should be amply documented. Any changes in the patient's condition or proposed changes in management should likewise be carefully discussed and documented. Adherence to these guidelines should ensure the safest best practice.

## REFERENCES

1. Chaliha C, Stanton SL: Urological problems in pregnancy. *BJU International* 2002;89:469–476.
2. Goldberg RP, Lobel RW, Sand PK: The urinary tract in pregnancy. In: Bent AE, Ostergard DR, Cun-

- diff GW, Swift SE (eds): *Urogynecology and Pelvic Floor Dysfunction*. Baltimore: Williams & Wilkins, 2003: pp. 225–243.
3. Newton ER: The urinary tract in pregnancy. In: Walters MD, Karram MM (eds): *Urogynecology and Reconstructive Pelvic Surgery*. St. Louis: CV Mosby, 1999: pp. 399–417.
4. Mattingly RF, Borkowf HI: Surgical disease in pregnancy. In: Barber HRK, Graber EA (eds): *Lower Urinary Tract Injuries in Pregnancy*. Philadelphia: WB Saunders, 1974: pp. 440–464.
5. Meyers DR, Lee RV, Munschauer RW: Dilatation and nontraumatic rupture of the urinary tract during pregnancy: A review. *Obstet Gynecol* 1985;66: 809–815.
6. Voigt R, Voigt P, Klinger G, Schneider HJ: Urographic investigations on the effect of high-dose progestagen therapy. *Rofö* 1976;125:458–460.
7. Marchant DJ: Effects of pregnancy and progestational agents on the urinary tract. *Am J Obstet Gynecol* 1972;112: 487–501.
8. Freed SZ: The bladder and urethra in pregnancy. In: Freed SZ, Herzig N (eds): *Urology in Pregnancy*. Baltimore: Williams & Wilkins, 1982: pp. 22–31.
9. Iosif S, Ingermarsson I, Ulmsten U: Urodynamic studies in normal pregnancy and the puerperium. *Am J Obstet Gynecol* 1980;137: 696–700.
10. Goldberg RP, Sand PK: Obstetric issues and the female pelvis. In: Vasavada SP, Appell RA, Sand PK, Raz S (eds): *Female Urology, Urogynecology, and Voiding Dysfunction*. New York: Marcel Dekker, 2005: pp. 95–116.
11. Baessler K, Schuessler B: Pregnancy, childbirth, and pelvic floor damage. In: Bourcier AP, McGuire EJ, Abrams P (eds): *Pelvic Floor Disorders*. Philadelphia: Elsevier Saunders, 2004: pp. 33–42.
12. Toozs-Hobson P, Cutner A: Pregnancy and childbirth. In: Cardozo L, Staskin D (eds): *Textbook of Female Urology and Urogynaecology*. London: Isis Medical Media Ltd, 2001: pp. 977–992.
13. Robertson EG: Alterations in renal function during pregnancy. In: Buchsbaum HJ, Schmidt JD (eds): *Gynecologic and Obstetric Urology*. Philadelphia: WB Saunders, 1993: pp. 607–620.
14. Newton ER: The acute abdomen in pregnancy. In: Sciarra JJ, Speroff L, Simpson JL (eds): *Gynecology and Obstetrics*. Philadelphia: JB Lippincott, 1991: pp. 1–17.
15. Klein EA: Urologic problems of pregnancy. *Obstet Gynecol Survey* 1984;39:605–615.

16. Francis WJ: Disturbances of bladder function in relation to pregnancy. *J Obstet Gynaecol Br Emp* 1960;67:353-366.
17. Parboosingh J, Doig A: Studies of nocturia in normal pregnancy. *J Obstet Gynaecol Br Commonw* 1973;80:888-895.
18. Cutner A, Carey A, Cardozo LD: Lower urinary tract symptoms in early pregnancy. *Br J Obstet Gynaecol* 1992;12:75-78.
19. Stanton SL, Kerr-Wilson R, Harris VG: The incidence of urological symptoms in normal pregnancy. *Br J Obstet Gynaecol* 1980;87:897-900.
20. Myers DL, Scotti RJ: Acute urinary retention and the incarcerated, retroverted, gravid uterus. *J Reprod Med* 1995;40:487-490.
21. Cutner A, Cardozo LD, Benness CJ: Assessment of urinary symptoms in early pregnancy. *Br J Obstet Gynaecol* 1991;98:1283-1286.
22. Chaliha C, Kalia V, Stanton SL, Monga A, Sultan AH: Antenatal prediction of postpartum urinary and fecal incontinence. *Obstet Gynecol* 1999;94:689-694.
23. Chaliha C, Kalia V, Monga A, Sultan AH, Stanton SL: Pregnancy, childbirth and delivery: A urodynamic viewpoint. *Br J Obstet Gynaecol* 2000;107:1354-1359.
24. Viktrup L, Lose G, Rolff M, Barfoed K: The symptom of stress incontinence caused by pregnancy or delivery in primiparas. *Obstet Gynecol* 1992;79:945-949.
25. Cutner A, Cardozo LD, Benness CJ: Assessment of urinary symptoms in the second half of pregnancy. *Int Urogynecol J* 1992;3:30-32.
26. Cutner A, Cardozo LD, Benness CJ, Carey A, Cooper D: Detrusor instability in early pregnancy. *Neurourol Urodyn* 1990;9:328-329.
27. Viktrup L, Lose G, Rolff M, Barfoed K: The frequency of urinary symptoms during pregnancy and puerperium in the primipara. *Int Urogynecol J* 1993;4:27-30.
28. Allen RE, Hosker GL, Smith ARB, Warrell DW: Pelvic floor damage and childbirth: A neurophysiological study. *Br J Obstet Gynaecol* 1990;97:770-779.
29. Cutner A, Cardozo LD: The lower urinary tract in pregnancy and the puerperium. *Int Urogynecol J* 1992;3:317-323.
30. Iosif S, Ulmsten U: Comparative urodynamic studies of continent and stress-incontinent women in pregnancy and the puerperium. *Am J Obstet Gynecol* 1981;140:645-650.
31. Wilson PD, Herbison RM, Herbison GP: Obstetric practice and the prevalence of urinary incontinence. *Br J Obstet Gynaecol* 1996;103:154-161.
32. Van Geelen JM, Lemmens WA, Eskes TK, Martin CB Jr: The urethral pressure profile in pregnancy and after delivery in healthy nulliparous women. *Am J Obstet Gynecol*. 1982 Nov;144(6):636-649.
33. Snooks SJ, Swash M, Setchell M, Henry MM: Injury to the innervation of pelvic floor sphincter musculature in childbirth. *Lancet* 1984;2:546-550.
34. Peschers U, Schaer G, Anthuber C, Delancey JOL, Schuessler B: Changes in vesical neck mobility following vaginal delivery. *Obstet Gynecol* 1996;88:1001-1006.
35. Meyer S, Schreyer A, De Grandi P, Hohlfeld P: The effects of birth on urinary continence mechanisms and other pelvic floor characteristics. *Obstet Gynecol* 1998;92:613-618.
36. Mittal P, Wing DA: Urinary tract infections in pregnancy. *Clin Perinatol* 2005;32:749-764.
37. Hill JB, Sheffield JS, McIntire DD, Wendel GD Jr: Acute pyelonephritis in pregnancy. *Obstet Gynecol* 2005;105:18-23.
38. Horowitz E, Schmidt JD: Renal calculi in pregnancy. *Clin Obstet Gynecol* 1985;28:324-338.
39. Drago JR, Rohner TJ Jr: Management of urinary calculi in pregnancy. *Urology* 1982;20:578-581.
40. Lattanzi DR, Cook WA: Urinary calculi in pregnancy. *Obstet Gynecol* 1980;56:462-466.
41. Demby AM, Schmidt JD: Urinary calculi in pregnancy. In: Buchsbaum HJ, Schmidt JD (eds): *Gynecologic and Obstetric Urology*. Philadelphia: WB Saunders, 1992: pp. 691-703.
42. Gertner JM, Coustan DR, Kliger AS, Mallette LE, Ravin N, Broadus AE: Pregnancy as a state of physiologic absorptive hypercalciuria. *Am J Med* 1986;81(3):451-456.
43. Nakagawa Y, Abram V, Parks JH, Lau HS, Kawooya JK, Coe FL: Urine glycoprotein crystal growth inhibitors: Evidence for a molecular abnormality in calcium oxalate nephrolithiasis. *J Clin Invest* 1985;76:1455-1462.
44. Harris RE, Dunnihou DR: The incidence and significance of urinary calculi in pregnancy. *Am J Obstet Gynecol* 1967;99:237-241.
45. Honore LE: The increased incidence of renal stones in women with spontaneous abortion: A

- retrospective study. *Am J Obstet Gynecol* 1980;137:145–146.
46. Waltzer WC: The urinary tract in pregnancy. *J Urol* 1981;125:271–276.
  47. Stothers L, Lee LM: Renal colic in pregnancy. *J Urol* 1992;148:1383–1387.
  48. Hendricks SK, Ross SO, Krieger JN: An algorithm for diagnosis and therapy of management and complications of urolithiasis during pregnancy. *Obstet Gynecol* 1991;172:49–54.
  49. MacNeily AE, Goldenberg SL, Allen GJ, Ajzen SA, Cooperberg PL: Sonographic visualization of the ureter in pregnancy. *J Urol* 1991;146:298–301.
  50. McAleer SJ, Loughlin KR: Nephrolithiasis and pregnancy. *Curr Opin Urol* 2004;14:123–127.
  51. Vieweg J, Weber HM, Miller K, Hautmann R: Female fertility following extracorporeal shock wave lithotripsy of distal ureteral calculi. *J Urol* 1992;148:1007–1010.
  52. Vogel H: *Risiken der Röntgendiagnostik*. Munich: Urban & Schwarzenberg, 1986: pp. 193–196.
  53. Loughlin KR, Bailey BBJ: Internal ureteral stents for conservative management of ureteral calculi during pregnancy. *N Engl J Med* 1986;315:1647–1649.
  54. Rittenberg MH, Bagley DH: Ureteroscopic diagnosis and treatment of urinary calculi during pregnancy. *Urology* 1988;32:427–428.
  55. Denstedt JD, Raxui H: Management of urinary calculi during pregnancy. *J Urol* 1992;148:1072–1075.
  56. Weber HN, Dillon RW, Sotolongo JR: Urologic complications in pregnancy. In: Cherry SH, Merkatz IR, Wayne RC (eds): *Cherry and Merkatz's Complications of Pregnancy*. Philadelphia: Williams & Wilkins, 2000: pp. 536–559.
  57. Goldfarb RA, Neerhut GJ, Lederer E: Management of acute hydronephrosis of pregnancy by ureteral stenting: Risk of stone formation. *J Urol* 1989;141:921–922.
  58. Wolf MC, Hollander JB, Salisz JA, Kearney DJ: A new technique for ureteral stent placement during pregnancy using endoluminal ultrasound. *Surg Gynecol Obstet* 1992;175:575–576.
  59. Vest JM, Warden SS: Ureteroscopic stone manipulation during pregnancy. *Urology* 1990;35:250–252.
  60. Kramolowsky EV: Ureteral perforation during ureteroscopy: Treatment and management. *J Urol* 1987;138:36–38.
  61. Khoo L, Anson K, Patel U: Success and short-term complication rates of percutaneous nephrostomy during pregnancy. *J Vasc Interv Radiol* 2004;15:1469–1473.
  62. Kavoussi LR, Albala DM, Basler JW, Apte S, Clayman RV: Percutaneous management of urolithiasis during pregnancy. *J Urol* 1992;148:1069–1071.
  63. Rodriguez SU, Leikin SL, Hiller MC: Neonatal thrombocytopenia associated with antepartum administration of thiazide drugs. *N Engl J Med* 1964;270:881–884.
  64. Gray MJ: Use and abuse of thiazides in pregnancy. *Clin Obstet Gynecol* 1968;11:568–578.
  65. Eckford SD, Gingell JC: Ureteric obstruction in pregnancy – diagnosis and management. *Br J Obstet Gynaecol* 1991;98:1137–1140.
  66. Hess LW, Nolan TE, Martin RW, Martin JN Jr, Wiser WL, Morrison JC: Incarceration of the retroverted gravid uterus: Report of four patients managed with uterine reduction. *South Med J* 1989;82:310–312.
  67. Silva PD, Berberich W: Retroverted impacted gravid uterus with acute urinary retention: Report of two cases and a review of the literature. *Obstet Gynecol* 1986;68:121–123.
  68. Herzig N: Urologic problems of labor and delivery. In: Freed SZ, Herzig N (eds): *Urology in Pregnancy*. Baltimore: Williams & Wilkins, 1982: pp. 208–215.
  69. Hée P, Lose G, Beier-Holgersen R, Engdahl E, Falkenløve P: Postpartum voiding in the primiparous after vaginal delivery. *Int Urogynecol J* 1992;3:95–99.
  70. Anderson GW, Rice GG, Harris BA Jr: Pregnancy and labor complicated by pelvic ectopic kidney. *J Urol* 1951;65:760–776.
  71. Anderson GW, Rice GG, Harris BA Jr: Pregnancy and labor complicated by pelvic ectopic kidney anomalies. *Obstet Gynecol Surv* 1949;4:737–773.
  72. Newton M, Newton ER: Surgical problems in pregnancy. In: Sciarra JJ, Speroff L, Simpson JY (eds): *Gynecology and Obstetrics*. Philadelphia: JB Lippincott, 1991; 4: pp. 1–11.
  73. Buchsbaum HJ: Accidental injury complicating pregnancy. *Am J Obstet Gynecol* 1968;102:752–769.
  74. Orkin LA: Trauma to the bladder, ureter, and kidney. In: Sciarra JJ, Speroff L, Simpson JL (eds): *Gynecology and Obstetrics*. Philadelphia: JB Lippincott, 1991; 1: pp. 1–8.

75. Perry MO, Husmann DA: Urethral injuries in female subjects following pelvic fracture. *J Urol* 1992;147:139-143.
76. Hassim AM: Rupture of a diverticulum of the uterus. *Am J Obstet Gynecol* 1968;101:1132-1134.
77. Rendle-Short CW: Rupture of gravid uterus in Uganda. *Am J Obstet Gynecol* 1960;79:1114-1120.
78. Raghavaiah NV, Devi AI: Bladder injury associated with rupture of the uterus. *Obstet Gynecol* 1975; 46:573-576.
79. Cunningham G, Gant NF, Leveno KJ, Gilstrap L C, Hauth J C, Wenstrom KD: *Williams Obstetrics*, ed 22. New York: McGraw-Hill, 2005: pp. 423-563.
80. Klotz PG, Khalaff HM: Placenta percreta invading the bladder: Report of two cases. *J Urol* 1989;141: 938-939.
81. Taefi P, Kaiser TF, Sheffer JB, Courney NG, Hodson JM: Placenta percreta with bladder invasion and massive hemorrhage: Report of a case. *Obstet Gynecol* 1970;36:686-687.
82. Mattingly RF, Borkowf HI: Acute operative injury to the lower urinary tract. *Clin Obstet Gynecol* 1978;5:123-147.
83. Loughlin KR: Management of urologic problems during pregnancy. *Urology* 1994;44:159-169.
84. Faricy PO, Augspurger RR, Kaufman JM: Bladder injuries associated with cesarean sections. *J Urol* 1978; 120:762-763.
85. Plauché WC, Wycheck JG, Iannessa MJ, Roussett KM, Mickal A: Cesarean hysterectomy at Louisiana State University, 1975 through 1981. *South Med J* 1983;76:1261-1263.
86. Plauché WC: Peripartal hysterectomy. In: Plauché WC, Morrison JC, O'Sullivan MJ (eds): *Surgical Obstetrics*. Philadelphia: WB Saunders, 1992: pp. 447-465.
87. Youssef AF: "Menuria" following lower segment cesarean section. *Am J Obstet Gynecol* 1957;73: 759-767.
88. Plauché WC: Obstetric genital trauma. In: Plauché WC, Morrison JC, O'Sullivan MJ (eds): *Surgical Obstetrics*. Philadelphia: WB Saunders, 1992: pp. 383-404.
89. Gerber GS, Schoenberg HW: Female urinary tract fistulas. *J Urol* 1993;149:229-236.
90. Hilton P: Obstetric fistulae. In: Cardozo L, Staskin D (eds): *Textbook of Female Urology and Urogynaecology*. London: Isis Medical Media Ltd, 2001: pp. 711-719.
91. Hilton P: Surgical fistulae. In: Cardozo L, Staskin D (eds): *Textbook of Female Urology and Urogynaecology*. London: Isis Medical Media Ltd, 2001: pp. 691-709.
92. Gangai MD: An unusual surgical injury to the ureter. *J Urol* 1973;109:32.
93. Dimopoulos C, Giannopoulos A, Pantasopoulos D, Vlahos L: Avulsion of the ureter from both ends as a complication of interruption of pregnancy with vacuum aspirator. *J Urol* 1977;118: 108.
94. Yasin SY, Walton, DL, O'Sullivan MJ: Problems encountered during cesarean delivery. In: Plauché WC, Morrison JC, O'Sullivan MJ (eds): *Surgical Obstetrics*. Philadelphia: WB Saunders, 1992: pp. 431-445.
95. Landau SY: Ureteroneocystostomy: A review of 72 cases with a comparison of two techniques. *J Urol* 1962;87:343-349.
96. Hodges CV, Moore RJ, Lehman TH: Clinical experiences with transureteroureterostomy. *J Urol* 1963;90:552-562.
97. Matthews R, Marshall FF: Versatility of the adult psoas hitch ureteral reimplantation. *J Urol* 1997;158:2078-2082.
98. Konigsberg H, Blunt KJ, Muecke EC: Use of Boari flap in lower ureteral injuries. *Urology* 1975;5:751-755.
99. Benson MC, Ring KS, Olsson CA: Ureteral reconstruction and bypass: Experience with ileal interposition, the Boari flap-psoas hitch and renal autotransplantation. *J Urol* 1990;143:20-23.
100. Dowling RA, Corriere JN Jr, Sandler CM: Iatrogenic ureteral injury. *J Urol* 1981;135:912-915.
101. Hardy JD: High ureteral injuries: Management by autotransplantation of the kidney. *JAMA* 1963; 184:97-101.
102. Moran PA, Carey MP, Dwyer PL: Urethral diverticula in pregnancy. *Aust N Z J Obstet Gynaecol* 1998;38:102-106.
103. Allen LE, Mount J, Cline D, Mertz JH: Pelvic dystocia secondary to urethral diverticulum and urinary retention. *J Urol* 1969;102:451-453.
104. Roxe DM: Renal disease in pregnancy. In: Sciarra JJ, Speroff L, Simpson JL (eds): *Gynecology and Obstetrics*. Philadelphia: JB Lippincott, 1991; 3: pp. 1-16.

105. Doyle BA, Smith SP, Stempel LE: Urinary undiversion and pregnancy. *Am J Obstet Gynecol* 1988; 158:1131–1132.
106. Hill DE, Kramer SA: Management of pregnancy after augmentation cystoplasty. *J Urol* 1990;144:457–459.
107. Fenn N, Barrington JW, Stephenson TP: Clam enterocystoplasty and pregnancy. *Br J Urol.* 1995;75: 5–6.
108. Fishman JJ, Scott FB: Pregnancy in patients with the artificial urinary sphincter. *J Urol* 1993;150:340–341.
109. Petrou SP, Elliott DS, Barrett DM: Artificial urethral sphincter for incontinence. *Urology* 2000; 56:353–359.
110. Toh K, Diokno AC: Management of intrinsic sphincter deficiency in adolescent females with normal bladder emptying function. *J Urol* 2002;168: 1150–1153.
111. Cutner A, Cardozo LD, Wise BG: The effects of pregnancy on previous incontinence surgery: Case report. *Br J Obstet Gynaecol* 1991;98:1181–1183.
112. Dainer M, Hall CD, Choe J, Bhatia N: Pregnancy following incontinence surgery. *Int Urogynecol J Pelvic Floor Dysfunct* 1998;9:385–90.
113. Atherton MJ, Stanton SL: The tension-free vaginal tape reviewed: An evidence-based review from inception to current status. *Br J Obstet Gynaecol* 2005;112:534–546.
114. Shindel AW, Klutke CG: Urethral slings placed by the transobturator approach: Evolution in the technique and review of the literature. *Curr Urol Rep* 2005;6:385–392.
115. Gauruder-Burmester A, Tunn R: Pregnancy and labor after TVT-plasty. *Acta Obstet Gynecol Scand* 2000;80:283–284.
116. Seeger D, Truong ST, Kimmig R: Spontaneous delivery following tension-free vaginal tape procedure. *Int Urogynecol J Pelvic Floor Dysfunct* 2006;17:676–678.
117. Volkmer BG, Seidl EM, Gschwend JE, de Petriconi R, Bach D, Kleinschmidt K: Pregnancy in women with ureterosigmoidostomy. *Urol.* 2002;60:979–982.
118. Gontero P, Masood S, Sogni F, Fontana F, Mufti G, Frea B: Upper urinary tract complications in pregnant women with an ileal conduit: Lessons learned from two cases. *Scand J Urol Nephrol* 2004;38:523–524.
119. Austenfeld MS, Snow BW: Complications of pregnancy in women after reimplantation for vesicoureteral reflux. *J Urol* 1988;140:1103–1106.
120. Mansfield JT, Snow BW, Cartwright PC, Wadsworth K: Complications of pregnancy in women after childhood reimplantation for vesicoureteral reflux: An update with 25 years of followup. *J Urol* 1995;154:787–790.
121. Mor Y, Leibovitch I, Zalts R, Lotan D, Jonas P, Ramon J: Analysis of the long-term outcome of surgically corrected vesico-ureteric reflux. *BJU International* 2003;92:97–100.
122. Mor Y, Leibovitch I, Fridmans A, Farkas A, Jonas P, Ramon J: Late post-reimplantation ureteral obstruction during pregnancy: A transient phenomenon? *J Urol* 2003;170:845–848.
123. Derus JA, Begun FP, Jacobs SC: Genitourinary malignancies in pregnancy. In: Buchsbaum HJ, Schmidt JD (eds): *Gynecologic and Obstetric Urology*. Philadelphia: WB Saunders, 1993: pp. 705–713.
124. Gonzalez-Blanco S, Mador DR, Vickar DB, McPhee MS: Primary bladder carcinoma presenting during pregnancy in 3 cases. *J Urol* 1989;141:613–614.
125. Malamud SC, Holland JP: Neoplasia and pregnancy. In: Cherry SH, Merkatz IR, Wayne RC (eds): *Cherry and Merkatz's Complications of Pregnancy*. Philadelphia: Williams & Wilkins, 2000: pp. 638–661.
126. Spahn M, Bader P, Westermann D, Echtele D, Frohneberg D: Bladder carcinoma during pregnancy. *Urol Int* 2005;74:153–159.
127. Wai CY, Miller DS: Urinary bladder cancer. *Clin Obstet Gynecol* 2002;45:844–854.
128. Lurie A, Eisenkraft SH, Shotland Y, Lurie M: Mucin-producing adenocarcinoma of the bladder of urachal origin. *Urol Int* 1983;38:12–15.
129. Williams SF, Bitran JD: Cancer and pregnancy. *Clin Perinatol* 1985;12:609–623.
130. Fehrenbaker LG, Rhodes JC, Derby DR: Transitional cell carcinoma of bladder during pregnancy: Case report. *J Urol* 1972;108:419–420.
131. O'Connor JP, Biyani CS, Taylor J, Agarwal V, Curley PJ, Browning AJ: Laparoscopic nephrectomy for renal-cell carcinoma during pregnancy. *J Endourol* 2004;18:871–874.
132. Sainsbury DC, Dorkin TJ, MacPhail S, Soomro NA: Laparoscopic radical nephrectomy in first-trimester pregnancy. *Urology* 2004;64:1231.e7–e8.

Shaun M. Kunisaki

Russell W. Jennings

*... material is not lacking – particularly in this vast field of Medicine – in which to prove one's ability, that is, by perfecting things which have been left incomplete and untouched by the ancients or others and by making new contributions to knowledge...*

Gaspere Tagliacozzi (1547–1599)

*De curtorum chirurgia per insitionem* (c 1597)

A. Read (trans.)

London: Jones, 1867.

Most disorders identified in the fetus are best managed in the early postnatal period. Over the last 25 years, however, fetal surgery has emerged from the realm of medical curiosity into an exciting, multidisciplinary specialty now capable of improving patient outcomes for a wide variety of diseases. Recent technologic progress now allows clinicians to both diagnosis and treat many fetal anomalies accurately while maintaining a high level of maternal safety. As expectant parents become increasingly educated about the potential benefits of fetal surgery, obstetricians must become familiar with some of the more recent, state-of-the-art advances that are currently shaping the field.

The purpose of this chapter is to provide an overview of the principles of modern operative fetal intervention. To this end, the authors outline the basic ethical and diagnostic issues pertinent to the practice of fetal surgery and describe the major operative approaches to gain access to the fetus. The discussion then turns to some of the established as well as experimental prenatal therapies that are currently being employed for several lethal and non-lethal anomalies (Table 20.1).

### Fetal Ethics and Informed Consent

Because of the unique physical relationship between mother and fetus, prenatal surgical therapies inherently place two patients at risk for potential complications. Only one patient (the fetus) can derive any direct benefit from the surgical intervention, however. Thus, before considering any fetal surgical procedure, clinicians involved in the care of these patients should be cognizant of potential maternal-fetal conflicts and prepared to discuss these ethical issues openly with expectant mothers during prenatal counseling sessions.

According to many groups, including the American College of Obstetrics and Gynecology (ACOG), the moral imperative of any fetal intervention is to respect the mother in her decision regarding the treatment of her fetus [1,2]. Although every reasonable effort should be made to protect the fetus, they argue that the previable fetus has no independent

TABLE 20.1 Fetal Interventions

Procedure	Approach	Disorder
Ablation of placenta vessels	Fetoscopic	TTTS <sup>a</sup>
Umbilical cord occlusion/division	Fetoscopic	TTTS TRAP sequence* Discordant twins
Ex-utero intrapartum treatment (EXIT)	Open	Massive airway obstruction
Thoracoamniotic shunt	Percutaneous	Primary hydrothorax Cystic adenomatoid malformation
Lobectomy	Open	Cystic adenomatoid malformation
Teratoma resection	Open	Sacroccygeal teratoma Pericardial teratoma
Vesicoamniotic shunt	Percutaneous	Bladder outlet obstruction
Posterior urethral valve ablation	Fetoscopic	Bladder outlet obstruction
Tracheal occlusion	Fetoscopic	Diaphragmatic hernia
EXIT-to-ECMO	Open	Diaphragmatic hernia
Myelomeningocele closure	Open	Myelomeningocele
Amniotic band release	Fetoscopic	Amniotic band syndrome
Aortic valvuloplasty	Percutaneous	Aortic stenosis
Atrial septoplasty	Percutaneous	Aortic stenosis with restrictive septum
Pacemaker insertion	Open	Complete heart block

TTTS, twin-twin transfusion syndrome; TRAP, twin reversed arterial perfusion; EXIT, ex-utero intrapartum treatment; ECMO, extracorporeal membrane oxygenation.

\*See text for details.

moral status. Expectant mothers therefore should be under no obligation to undergo fetal therapy, even if the treatment is deemed to have a favorable risk-benefit ratio. In contrast, groups such as the American Academy of Pediatrics (AAP) have vouched for an ethical framework that holds a stronger consideration for the welfare of the fetus [3]. Proponents of this view have articulated that the moral imperative is for the pregnant woman to take some responsibility and willingness to undergo some degree of physical harm for the sake of fetal well-being.

In practice, maternal safety has remained the highest priority in fetal surgery. Some have suggested that pregnant women are a particularly vulnerable group of patients who might have a low threshold to consent to highly invasive fetal therapies, even if the benefits to their unborn children could be small [4]. In light of this, the establishment of government-sponsored clinical trials has played an important role in fostering new fetal therapies while preserving maternal well-being [5,6]. For example, many experimental fetal procedures are available in the United States only through controlled trials performed at experienced centers

that have demonstrated sufficient resources, commitment, and expertise to execute these fetal therapies properly and safely. (See Chapter 26, Ethical Issues.)

A key component to ethical fetal care is a detailed explanation of surgical risks and benefits through the informed consent process. This process should be performed by a multidisciplinary care team that has a good, evidence-based understanding of the effectiveness of a given prenatal intervention when compared with expectant management. There are many instances in which the risk-benefit ratio seems clearly unfavorable, such as in cases when fetal demise is imminent (e.g., placentomegaly, severe hydrops), leading to what is known as the maternal mirror syndrome (Ballantyne syndrome). In these situations, early clinical experience has shown that fetal intervention is never appropriate and that mothers should be counseled accordingly [7]. For cases in which the fetus already has relatively mature lungs, thereby giving the patient a reasonable chance of remaining viable outside of the womb, preterm delivery with postnatal surgical intervention is usually the best management option.



Of course, risk-benefit quantification is not always an easy task given the experimental nature of many fetal therapies. In these circumstances, a nondirective counseling approach remains vital to the informed consent process. For cases in which fetal surgical intervention is contemplated, all maternal risks should be discussed openly and candidly. These risks include preterm membrane rupture, preterm labor, wound infections, chorioamnionitis, uterine hemorrhage, loss of uterus, and damage to adjacent organs. For some procedures, a maternal blood transfusion is necessary. Preterm labor can subject the mother to prolonged periods of bedrest and expose her to the risks of aggressive tocolysis, including pulmonary edema [8]. Open fetal procedures generally require a nonclassic hysterotomy, which mandates that a cesarean be performed for all subsequent deliveries to minimize the theoretical risk of uterine rupture during active labor. Moreover, mothers must be aware that no prenatal intervention is universally successful in terms of improving fetal well-being. Finally, the morbidities associated with the delivery of a premature baby cannot be overestimated.

Fortunately, fetal surgical interventions have a good overall track record in terms of minimizing maternal risk [9]. To the authors' knowledge, there have been only 3 maternal deaths in over 400 fetal procedures performed over the last 25 years, yielding an overall maternal mortality risk of less than 0.9%. In a large series of women who underwent ex-utero intrapartum treatment (EXIT) to salvage fetuses with airway compromise, short-term maternal complications, including blood loss, were comparable to those observed after standard cesarean delivery [10]. The uterine rupture rate after open fetal surgery has been found to be similar to rates seen after a conventional lower uterine segment hysterotomy [11]. Finally, women can be informed that there is currently no evidence that fetal interventions, whether they are performed by open hysterotomy or by minimally invasive techniques, have an adverse effect on future fertility [12].

### Preoperative Diagnosis

In 1963, the first successful fetal surgical procedure was performed when Liley, a New Zealand perinatologist, transfused blood into the peritoneum of a hydropic fetus afflicted with severe Rh disease



**FIGURE 20.1.** *T2-weighted fetal MRI of a left-sided diaphragmatic hernia. This coronal view showed the presence of abdominal viscera into the chest cavity with mediastinal shift.*

[13]. Liley made the diagnosis without imaging by analyzing the amniotic fluid with spectrophotometry. From these early beginnings, fetal therapy has witnessed numerous major advances in noninvasive diagnostic technologies. Current imaging modalities, particularly high-resolution ultrasonography with Doppler interrogation and ultrafast magnetic resonance imaging (MRI), can now characterize many fetal anomalies with a high degree of accuracy and in exquisite detail (Figure 20.1) [14,15].

It is the opinion of the authors that fully trained pediatric radiologists play a vital and essential role in the evaluation of all referred cases. Because patients are often misdiagnosed or incompletely diagnosed by the referring institution, pediatric radiologists must review all diagnostic imaging and suggest additional studies if they can be deemed helpful in facilitating an accurate diagnosis and prenatal care plan. Furthermore, the expertise of our radiology colleagues is indispensable during fetal procedures, since ultrasonography is an important perioperative tool used in all percutaneous, fetoscopic, and open surgical procedures [16].

A listing of common inclusion and exclusion criteria used for considering a patient for fetal therapy is shown in Table 20.2. No preoperative workup for a fetal intervention is complete without a

TABLE 20.2 Fetal Surgery Criteria

**Inclusion Criteria**

Fetal anomaly in which prenatal intervention offers a favorable risk–benefit ratio

Normal fetal karyotype

Maternal age  $\geq 18$  years

**Exclusion Criteria**

Gestational age suggestive of likely viability ex utero

Associated severe fetal anomalies

Multifetal pregnancy (unless TTTS, TRAPS, imminent death in a discordant twin)

Placenta previa or history of placental abruption

Cervical insufficiency

Significant medical comorbidities

Inadequate support networks

Inability to comply with travel and medical follow-up

TTTS, twin-twin transfusion syndrome; TRAP, twin reversed arterial perfusion.

formal evaluation looking for other anomalies. Because many fetal disorders are associated with an increased risk for karyotype abnormalities, a diagnostic amniocentesis remains essential to eliminate the possibility of chromosomal defects that would be a contraindication to fetal intervention. Additionally, fetal echocardiography might be indicated for some disorders, such as in diaphragmatic hernia, because concomitant cardiac disease portends a much worse prognosis [17,18].

**General Operative Approaches**

The operative approaches in fetal surgery are uniquely challenging because of the small working environment and the fact that the fetus is encased within multiple layers, including the maternal abdominal wall, uterus, and chorioamniotic membranes. Three basic operative approaches, namely *open procedures*, *fetoscopic procedures*, and *percutaneous interventions*, have evolved and are all currently used for the surgical management of fetal disorders.

**OPEN FETAL SURGERY**

The principles of modern open fetal procedures were pioneered in the late 1970s and 1980s by Harrison, a pediatric general surgeon at the University of California, San Francisco [19–23]. Open surgery requires a cadre of specialists, each with a defined

role within the operating suite. A pediatric surgeon, assisted by the patient's perinatologist, should perform these types of interventions.

Preoperative preparation typically begins several hours before the procedure, with the administration of indomethacin 50 mg per rectum to minimize perioperative preterm labor. All mothers should receive a lumbar epidural catheter to minimize postoperative pain and uterine irritability. Deep general anesthesia (approximately 2 minimum alveolar concentration [MAC]) with isoflurane or desflurane is used to decrease uterine tone, thereby preserving maternal-fetal gas exchange at the placental interface. A roll should be placed under the woman's right side to partially relieve inferior vena cava compression by the uterus. Common perioperative maternal monitoring includes an invasive arterial line, continuous echocardiography and pulse oximetry, and an end-tidal carbon dioxide monitor. The placement of two large-bore intravenous lines is standard. Aggressive fluid resuscitation with crystalloid is generally avoided because of the risk of postoperative pulmonary edema associated with tocolytic agents, particularly magnesium sulfate.

After the mother is prepped and draped from midthorax to midhigh, a low transverse abdominal incision is made. In the setting of a posterior placenta, a vertical midline fascial incision is extended from the umbilicus to the pubic symphysis. In cases of an anterior placenta, the rectus muscles and underlying fascia must be divided transversely so that the uterus can be rotated out of the abdomen to enable a posterior hysterotomy. A large abdominal ring retractor is placed to facilitate better exposure. The position of the placenta is mapped by ultrasound scan and marked on the surface of the uterus.

At this point, the inhalational anesthetic is adjusted to ensure complete relaxation of the gravid uterus. Ephedrine is given to maintain adequate maternal blood pressure. A hysterostomy site is then identified by locating an area along the upper uterine segment that is at least 5 cm away from the placenta. Two large monofilament stay sutures are then placed in parallel to the proposed hysterotomy site to help facilitate a bloodless entry into the uterus. A 2-cm hysterotomy is made with electrocautery to allow for positioning of a specially designed uterine stapler device containing lactomer staples (U.S. Surgical Corp.). The stapler helps to create an 8- to 10-cm

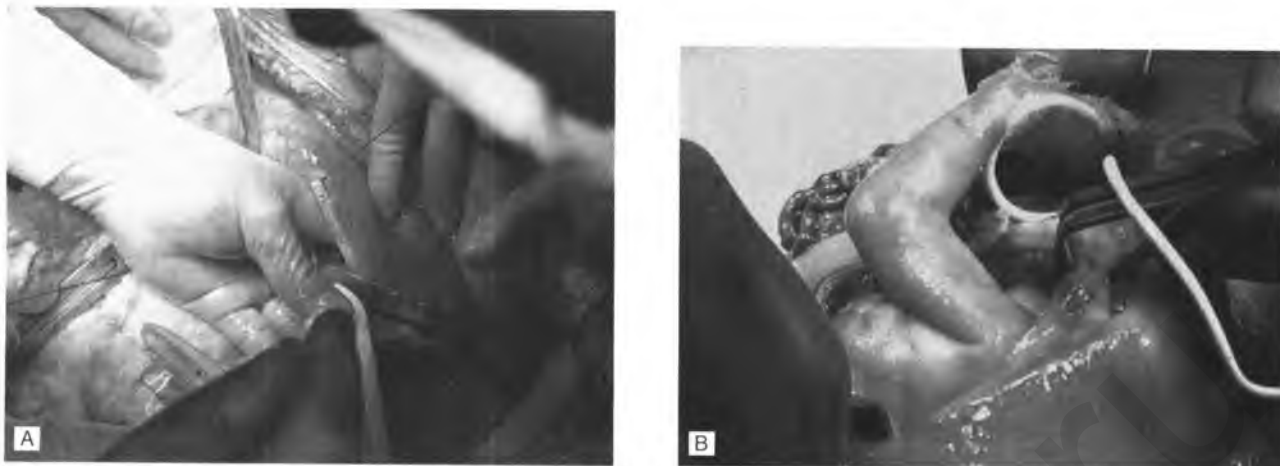


FIGURE 20.2.

*A, In all open fetal surgery cases, a maternal hysterotomy is performed using a specially designed uterine stapler device containing lactomer staples. B, Proper exposure of the fetal thorax in preparation for a left fetal thoractomy and lobectomy. A transcutaneous pulse oximeter is a useful adjunct for perioperative fetal monitoring. For color reproduction, see Color Plate 4.*

bloodless hysterotomy and maintains the integrity of the fragile chorion and amnion in a setting of complete uterine relaxation (Figure 20.2A). Back-biting uterine clamps can further facilitate adequate hemostasis.

Once the uterus is opened, medications can be given for fetal analgesia (e.g., fentanyl .10 mg/kg–.20 mg/kg IM) and neuromuscular blockade (e.g., vecuronium 200 mg/kg IM). Only the necessary fetal anatomy required for surgical manipulation is removed from the uterus. To maintain uterine distension, a red rubber catheter connected to a level I-type rapid infuser is placed deep into the amniotic cavity for continuous infusion of warm lactated Ringer's solution at 400 ml/min. A sterile transcutaneous pulse oximeter that is typically used for micropremature infants is placed around the palm and protected from light interference using aluminum foil (Figure 20.2B). In more extensive operative procedures (e.g., teratoma resection), fetal intravenous access should be attained to enable appropriate resuscitation if required [24]. During the fetal portion of the operation, normal fetal oxygen saturation readings should be maintained between 60% and 75%. Any oxygen saturation level of less than 50% suggests fetal hypoperfusion secondary to low cardiac output or kinking of the umbilical cord. At the conclusion of the proce-

cedure, antibiotics (e.g., nafcillin 500 mg) are infused into the amniotic cavity. A meticulous closure of the hysterotomy is essential because the presence of amniotic fluid between the membranes and the myometrium is a powerful stimulus to preterm labor. The uterus is typically closed in two layers, using full-thickness interrupted 0 polydioxanone (PDS) and a second layer of running 2–0 PDS. Some institutions have used fibrin glue on the hysterotomy incision to help to ensure adequate hemostasis. Parenteral tocolytic therapy begins at the time of uterine closure, using a 6-g bolus of magnesium sulfate followed by a continuous infusion at 2 g/hr. The maternal abdomen is closed in layers in the standard fashion.

Postoperatively, fetal well-being and uterine activity are recorded externally with a tocodynamometer. Magnesium and indomethacin are continued for at least 2 days. Few data currently support the improved efficacy of nitroglycerin as a long-term tocolytic agent [25,26]. Daily fetal echocardiograms are performed to assess for possible tricuspid regurgitation and premature closure of the ductus arteriosus because of the indomethacin. Mothers are maintained on strict bed rest and transitioned to a subcutaneous terbutaline pump or oral nifedipine until delivery. Because preterm labor occurs in 100% of all open fetal procedures, the

median interval between the fetal operation and delivery is only about 5 weeks.

### Fetoscopic Surgery

Transabdominal fetoscopy was developed in the early 1970s for the purposes of diagnosing certain genetic disorders, including hemoglobinopathies, myelomeningocele, and Duchenne's muscular dystrophy [27]. As one could imagine, these early fetoscopes were quite cumbersome and offered poor optical resolution. By the early 1980s, interest in fetoscopy waned as ultrasonography became a routine part of obstetric practice. Moreover, there were initially some concerns that the bright lights emitted from the endoscope might be harmful to the developing visual pathways, but this has never been substantiated [28].

Over the last decade, fetoscopy has enjoyed a resurgence among pediatric surgeons and perinatalogists alike. Mostly because of incremental advances in surgical technique and instrumentation, the current endoscopes are lightweight, offer higher-resolution capabilities, and have an expanded therapeutic repertoire (Figure 20.3) [29,30]. Although the tasks performed during fetoscopic surgery are usually not as complex as those performed during open fetal surgery, fetoscopy still demands a high level of operator expertise. In all fetoscopic cases, intraoperative ultrasound scan provides additional real-time guidance. Excellent communication between the surgeon and sonologist remains essential.



FIGURE 20.3.  
*Fetoscopy after a maternal laparotomy. For color reproduction, see Color Plate 5.*

Preoperative preparation for fetoscopic surgery is done in a fashion similar to that used in open fetal surgery. Placement of an epidural catheter and intensive blood pressure monitoring are not routinely indicated, however [31]. Fetoscopy can be performed under regional or standard general anesthesia, based on patient and institutional preferences. Deep general anesthesia is not required because profound uterine relaxation is not mandatory in these cases. The mother is typically positioned in a modified lithotomy position, with the knees low enough to allow the operator to work between the abducted legs. As in open fetal surgery, tilting the patient to the left can help to minimize obstruction of the inferior vena cava.

If the placenta is located anteriorly, a minilaparotomy can be a useful adjunct to allow for forward uterine displacement and a transfundal trocar puncture. A lateral approach is discouraged in this setting because of the course of the uterine vessels. For a posteriorly located placenta, a fully percutaneous approach is often feasible after a 1- to 2-mm incision is made in the maternal abdominal wall. The uterus is usually entered using a diamond-cut radially expanding trocar rather than a Veress needle, because a diamond-cut trocar minimizes tenting and separation of the uterine membranes. Multiple ports are sometimes required for saline irrigation and for performing more complex surgical tasks. Because the fetus is free floating in amniotic fluid, transabdominal or transuterine fixation of the fetus can be of help during the procedure.

Unlike many other areas in medicine that use surgical endoscopy, fetoscopy is performed within a fluid-filled cavity. Carbon dioxide is not a commonly used distension medium because it has been associated experimentally, even at low pressures, with air embolism, fetal acidosis, and placental insufficiency [32,33]. In addition, a liquid medium is ideal for transmitting clear sonographic images. One drawback to operating in a liquid medium is that visualization can often be difficult through turbid amniotic fluid, which is common with advancing gestational age. Visualization can be improved with the placement of an extra port for the infusion of an isotonic, optically neutral solution [34]. Excessive amnioinfusion should be avoided, however, because it can place undue stress on the gestational membranes, thereby increasing the risk of premature

membrane rupture. Formal closure of smaller trocar sites is generally not required, although larger trocar sites should probably be closed using absorbable figure-of-eight sutures. Tocolytic therapy should be initiated at the conclusion of the procedure.

Fetoscopy offers several distinct advantages when compared with open fetal surgery. First and foremost, endoscopes enable the operator to induce much less trauma to the maternal abdominal wall and uterus, thereby reducing uterine irritability, tocolytic requirements, and preterm labor. The current instruments measure between 1.2 mm and 5 mm in diameter and are typically about 18 cm in length. A theoretical benefit of fetoscopy is better preservation of fetal homeostasis [35]. Finally, the fetoscopic approach does not preclude the opportunity for a subsequent vaginal delivery.

Despite these advantages relative to open fetal surgery, the endoscopic approach can have significant drawbacks, depending on the clinical scenario. For example, there can be considerable difficulty performing an operation in a tiny working space. Moreover, performing some of the more complex tasks can still be next to impossible with the currently available instruments. The emerging field of robotics has yet to be applied clinically in fetal surgery but could prove to be a valuable tool for selected fetal cases in the future [36].

### Percutaneous Approaches

Percutaneous fetal therapies began in the early 1980s, when Frigoletto and others began placing ventriculoamniotic shunts for the treatment of fetal hydrocephalus [37]. These procedures proved to be technically feasible and enjoyed a brief period of enthusiasm at multiple centers worldwide. Minimally invasive neurologic interventions were soon abandoned, however, because they were associated with a high procedure-related death rate and were never able to demonstrate improved neurologic outcomes [38].

Currently, percutaneous interventions play an important role in draining other space-occupying, fluid-filled structures, such as the pleural space and bladder (Figure 20.4) [39,40].

These procedures are generally performed in an outpatient setting under continuous ultrasound guidance and require minimal maternal sedation



FIGURE 20.4.  
*Percutaneous procedure under ultrasound guidance. For color reproduction, see Color Plate 6.*

(e.g., diazepam, morphine). In selected cases, fetal muscle blockade is required by intramuscular injection or through the umbilical vein.

Needle aspirations can often be performed using a 20- or 22-gauge spinal needle. Although these procedures usually do not achieve a long-term therapeutic result because of fluid reaccumulation within 48 to 72 hours, they can be helpful for diagnostic purposes and can be useful just prior to birth for facilitating an easier delivery and resuscitation postnatally.

For long-term drainage, percutaneously placed catheters provide superior long-term decompression. In such cases, a 2.5-mm trocar assembly is placed into the cavity of interest. The sharp trocar is then withdrawn into the introducer sleeve and replaced with the shunt catheter. Finally, the introducer sleeve is withdrawn, leaving the proximal end of the catheter in the amniotic space. The most commonly used shunts are 2.1-mm Silastic catheters with pigtails that end at 90 degrees to each other, such as the KCH fetal bladder catheter (Rocket Medical, Washington, UK) or the Harrison fetal bladder stent (Cook Urological, Bloomington, IN). The double-pigtail design helps to minimize shunt dislodgement.

Outpatient tocolytics are commonly used in the early postoperative period. Weekly ultrasound scans are recommended to assess for shunt function and to determine general fetal well-being. Overall, there is a 10% to 15% rate of obstruction and a 20% to 30% rate of migration. Although percutaneous

approaches are the least invasive of all operative interventions, premature rupture of the membranes remains a risk, although to a lesser degree than in other approaches. Most fetuses are born in the early-to-middle third trimester.

## ESTABLISHED APPLICATIONS

### **Twin–Twin Transfusion Syndrome**

Anomalous vascular connections are common in monochorionic twins. In about 15% of these gestations, these vascular connections can lead to a significant imbalance of blood flow between the twins, a condition known as *twin–twin transfusion syndrome* (TTTS). Chronic TTTS can be seen sonographically by a discordance in weight and amniotic fluid volume, resulting from a relative hypovolemia in the donor twin and a relative hypervolemia in the recipient twin [41]. The release of vasoactive mediators, including endothelin, might also be involved in the pathophysiology of TTTS [42].

In untreated TTTS cases diagnosed before 26 weeks' gestation, mortality rates exceed 80% to 90%, with most deaths occurring in the recipient twin secondary to high-output cardiac dysfunction and hydrops [43]. In addition, neurologic sequelae, including cerebral palsy, hemiparesis, and spastic quadriplegia, frequently occur in the surviving twin [44]. The mechanism for the neurologic impairment is not completely understood but is thought to be secondary to either a release of tissue thromboplastin or a sudden drop in vascular resistance when the co-twin dies [45,46].

Until recently, serial amnioreduction was the mainstay of therapy in virtually all severe cases of TTTS. Amnioreduction is helpful in relieving the hydramnios that can precipitate preterm labor and can improve uteroplacental blood flow by decreasing pressure on the chorionic plate [43]. Unfortunately, this approach has had limited impact on minimizing neurologic morbidity.

Fetoscopic laser photocoagulation of the aberrant placental vessels has emerged as the preferred therapy in many cases of TTTS. Although the procedure was originally described through a maternal laparotomy, laser ablation can now be accomplished using a completely percutaneous approach [47]. Most approaches employ a 2-mm fetoscope

and a neodymium:YAG (Nd:YAG) laser, which is used to photocoagulate nonpaired placental vessels adjacent to placental cotyledons on the chorionic plate. Curved endoscopes and instruments can be used in cases with an anterior placenta [48]. The effectiveness of selective versus nonselective ablation of aberrant vessels has been debated [49].

The first-line effectiveness of fetoscopic laser ablation when compared with serial amnioreduction before 26 weeks' gestation was recently supported by a randomized trial based in Europe [50]. In this study of over 140 patients, short-term survival of at least one twin after fetoscopic ablation and therapeutic amnioreduction was 76% and 56%, respectively. In addition, neurologic morbidity at 6 months occurred in only 31% of patients who underwent ablation, compared with 52% in those who had serial amnioreduction. Data from another prospective trial, sponsored by the National Institutes of Health had more equivocal results [50a].

There are currently insufficient data to determine whether fetoscopic laser ablation is a preferred therapy for TTTS beyond 26 weeks' gestation. It is also unclear whether treatment should be customized (i.e., amnioreduction versus laser ablation) according to the stage of TTTS, as was articulated by Quintero [51,52]. In practice, perinatologists often perform an amnioreduction as an initial therapy to check response, particularly in cases detected later in gestation. Amnioreduction has the advantage of being extremely safe, cheap, and readily available. In contrast, expertise in fetoscopic ablation surgery is currently available in only a few centers, which continues to limit enthusiasm for its use.

Other alternative interventions for the management of TTTS can be considered in certain situations. Endoscopic cord occlusion is indicated when death is imminent for one twin (e.g., severe hydrops), to minimize neurologic morbidity in the surviving twin [53]. Needle septotomy of the inter-twin amniotic membrane has also been advocated as an alternative approach to repeat amnioreductions [54]. Theoretically, septotomy allows for equilibration of the amniotic fluid volumes and might minimize the need for multiple procedures. A recently published randomized trial has shown no improvement in terms of perinatal survival after septotomy when compared with serial amnioreduction, however [55]. Furthermore, critics of

septotomy have expressed concern about the theoretical risk of cord entanglement that might occur following the procedure [56].

### Twin Reversed Arterial Perfusion Sequence

The twin reversed arterial perfusion (TRAP) sequence is a rare complication of monochorionic twin gestations, occurring in approximately 1% of all cases. In this condition, a nonviable (usually acardiac and acephalic) twin receives its blood flow by reversed perfusion by the umbilical artery directly from the normally functioning twin (also called the *pump twin*) [57]. The natural history of the TRAP sequence varies, with some pump twins surviving to delivery under conservative management or with pharmacologic agents such as digoxin and indomethacin [58]. The added perfusion burden placed on the structurally normal twin leads to high-output cardiac failure and fetal demise in approximately 50% to 70% of cases, however. When the weight discordance between the twins is greater than 75%, the mortality rate approaches 90% [57].

Currently, the favored therapeutic approach in most cases of TRAP sequence is selective feticide of the anomalous twin by fetoscopic umbilical cord occlusion [59]. Since the first successful case reports [60,61], a variety of fetoscopic methods for successful cord occlusion have been described, including suture ligation, monopolar diathermy, bipolar diathermy, YAG laser, and radiofrequency ablation [62]. In the setting of a monoamniotic pregnancy, the cord of the anomalous twin should also be cut to minimize the risk of cord entanglement. Survival rates after fetoscopic cord occlusion are now greater than 80% in most reported series [59,63].

Several other methods for selective termination in the TRAP sequence have been described. One needle-based approach that appears to be a reasonable option is intrafetal ablation of pulsatile tissue, a procedure that has been advocated at some centers because of its technical simplicity [64]. Open hysterotomy with removal of the anomalous twin (*sectio parva*) has also been performed but is largely now of historical interest because of high maternal morbidity, including abruptio placenta, preterm labor, and pulmonary edema associated with tocolysis [65]. Direct injection of toxic substances into the cord of the anomalous twin is not performed

because of the risk of inadvertent transmigration of the infusate into the pump twin.

### Airway Obstruction

Fetal airway obstruction can be caused by intrinsic defects within the larynx or trachea, or more commonly, by extrinsic compression from a massive oropharyngeal teratoma or cervicomedial lymphatic malformation. Current, state-of-the-art imaging by ultrasound scan and MRI can be used to estimate the degree of obstruction and the probable difficulty of managing the airway with conventional orotracheal intubation at delivery (Figure 20.5) [15,66–68].

In severe cases of airway obstruction and hydrops in the previable fetus, fetal tracheostomy has been performed [69,70]. In most instances, however, the fetus with airway obstruction can survive until late in gestation, at which time open fetal surgery with control of the airway can be done prior to umbilical cord clamping. This procedure is now commonly termed EXIT.

The management of the fetal airway immediately prior to delivery was first described over 15 years ago [71,72]. In these early reports there was no attempt to prevent uterine contraction and placental separation, however, resulting in likely cessation of uteroplacental gas exchange. Over time, the techniques employed in the EXIT procedure have become standardized, enabling surgeons to perform procedures under controlled conditions while the fetus remains on uteroplacental bypass [73,74]. In cases of airway obstruction, the EXIT procedure has proved to be very useful because it avoids the hypoxia, brain injury, and death that can be associated with a neonatal airway crisis (Figure 20.6) [15,75,76].

The EXIT procedure begins with the administration of deep maternal general anesthesia. A maternal laparotomy and low transverse uterine segment hysterotomy are performed using the standard techniques of open fetal surgery. The fetal head and thorax are then exposed, while keeping the fetus connected to the umbilical cord. Uterine distension is maintained with a continuous instillation of normal saline into the amniotic cavity. Endotracheal intubation is usually attempted using a Miller 0 or 1 blade, or with a 2.5-mm rigid bronchoscope (Figure 20.7). Once the airway is secured, the umbilical cord is then cut, and the fetus is delivered to the awaiting



FIGURE 20.5. T2-weighted fetal MRI of an airway obstruction secondary to a massive oropharyngeal teratoma.

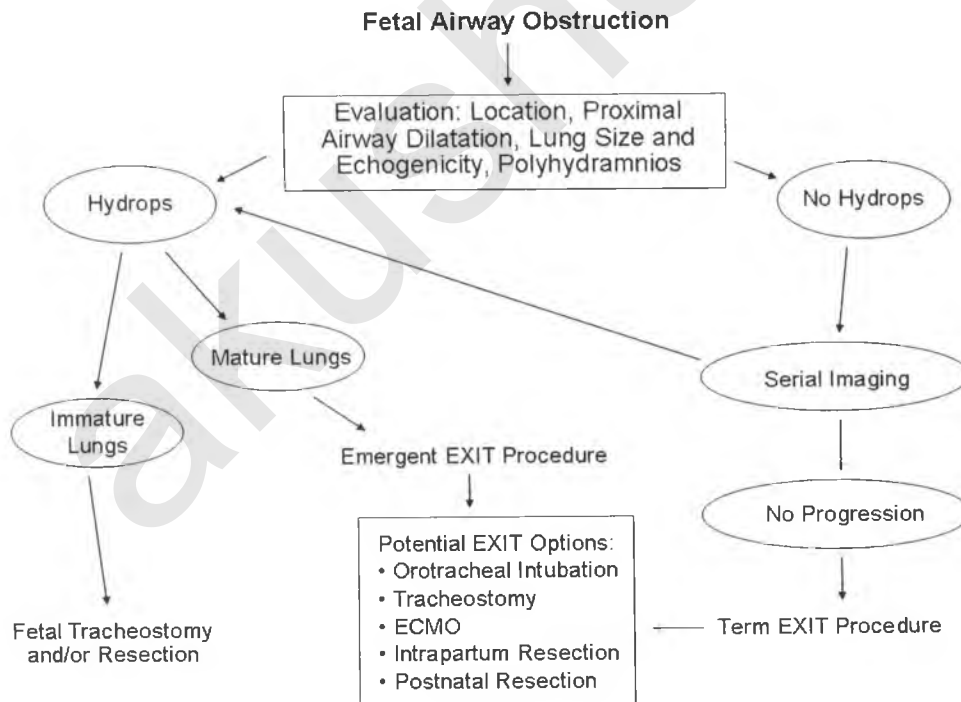


FIGURE 20.6. Algorithm for the management of fetal airway obstruction.



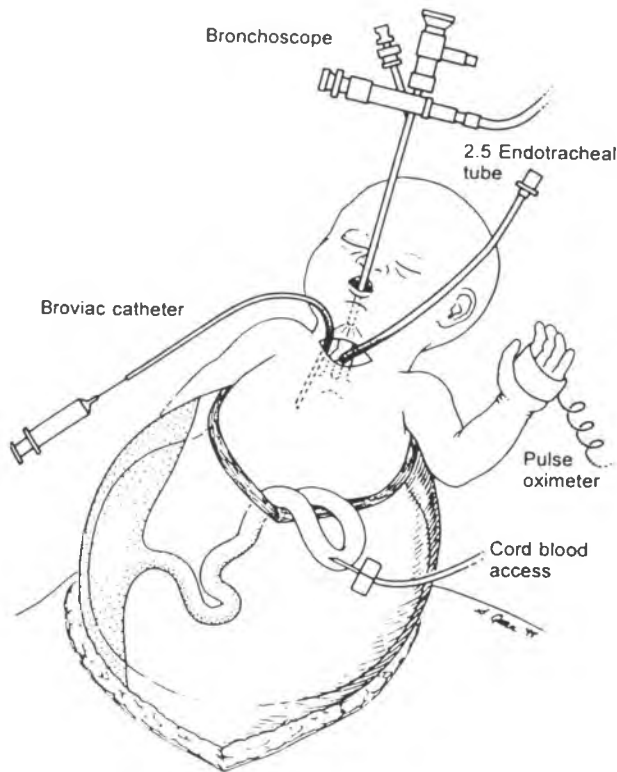


FIGURE 20.7.  
Ex-utero intrapartum treatment (EXIT).

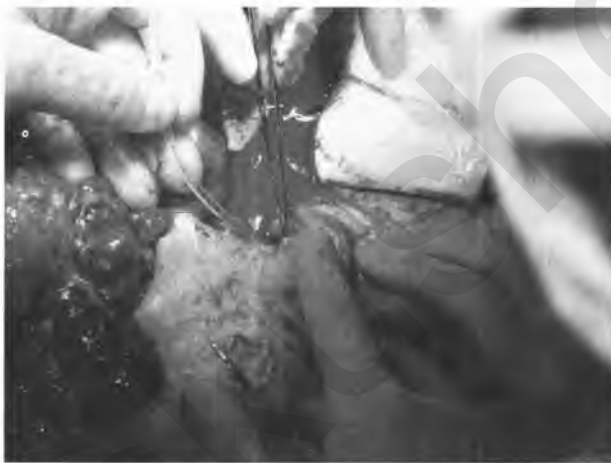


FIGURE 20.8.  
A tracheostomy during the EXIT procedure, performed for massive airway obstruction. For color reproduction, see Color Plate 7.

neonatology team. When endotracheal intubation is impossible, the surgeon still has abundant time to perform a tracheostomy, because most fetuses can be maintained on placental bypass for 45 to 90 minutes (Figure 20.8). In the rare case in which gas

exchange might still be impossible, another viable option is to proceed directly to placement on extra-corporeal membrane oxygenation (ECMO) prior to delivery [68].

Currently, the EXIT procedure for airway obstruction is the most common intervention using the techniques of open fetal surgery. The maternal morbidity of this procedure has been evaluated, showing comparable maternal outcomes relative to standard cesarean section [10]. The only exception is the wound infection rate, which is slightly higher after the EXIT procedure.

### Thoracic Anomalies

*Primary hydrothoraces* are uncommon, with an estimated incidence of 1 in 12,000 pregnancies. Based on some of the larger clinical series, the overall survival is approximately 50% for untreated fetuses [77–79]. Fetal surgical intervention for this condition was first reported in 1988 [80]. Although there have been no large, prospective studies comparing fetal intervention with expectant management for this disorder, prenatal intervention is advised in cases of early hydrops in fetuses less than 32 weeks' gestation [78]. Some centers also advocate fetal intervention in all hydrothoraces identified before 24 weeks, because of the increased risk for significant pulmonary hypoplasia. For all effusions that reaccumulate within 48 to 72 hours after an initial thoracentesis performed under ultrasound guidance, a thoracoamniotic shunt should be inserted. Survival after shunt placement is estimated to be about 70% [78,79].

*Congenital cystic adenomatoid malformations* (CCAMs) are benign hamartomatous masses that might have bronchial atresia as part of their underlying pathophysiology [81]. Adzick has classified CCAMs based on gross anatomic and sonographic features. Macrocystic lesions contain cysts greater than 5 mm in diameter, whereas microcystic lesions are predominantly solid with cysts less than 5 mm [82]. Although some studies have suggested that the natural history of CCAMs remains relatively undefined [7,83,84], experience from the authors' center has shown that most macrocystic and microcystic CCAMs tend to decrease in size relative to thoracic cavity volume after about 25 weeks' gestation. Therefore, most prenatal CCAMs have good outcomes under expectant management [85,86].

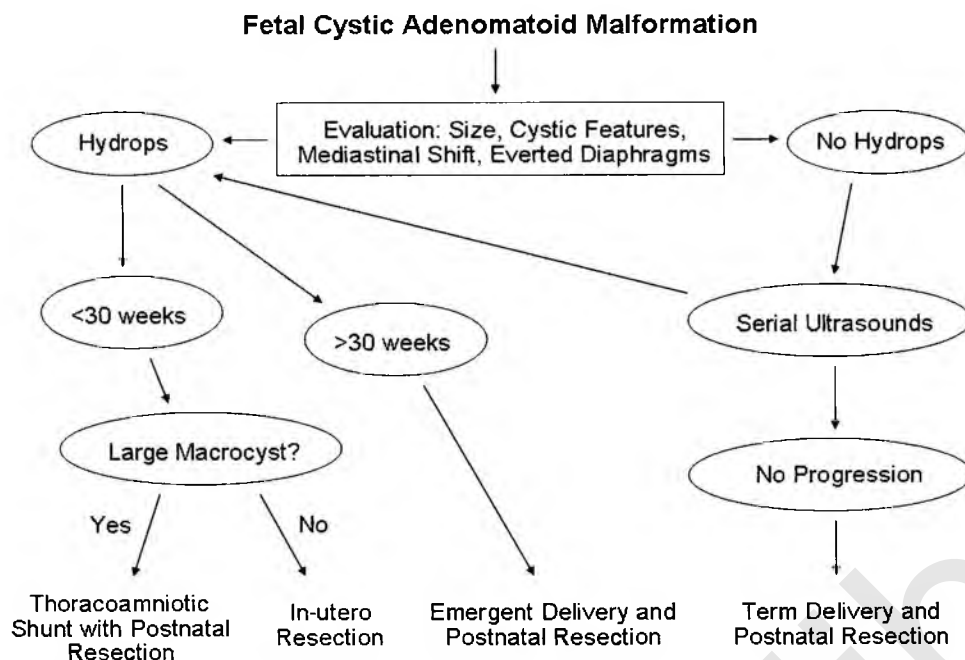


FIGURE 20.9.

*Algorithm for the management of fetal cystic adenomatoid malformations.*

Unfortunately, there remains a small subset of fetuses with CCAMs who develop profound pulmonary compression secondary to mass effect on the developing lung [85]. Furthermore, these space-occupying lesions can obstruct the esophagus, resulting in polyhydramnios. More rarely, large CCAMs can compress the inferior vena cava and heart, leading to hydrops and in-utero demise.

Currently, there is no consensus on the optimal antenatal management for large macrocystic CCAMs. A common management algorithm is shown in Figure 20.9. Some institutions have used the *cystic adenomatoid volume ratio* (CVR), defined as the volume of the CCAM divided by the head circumference, to identify ideal candidates for fetal shunting [87]. The CVR has yet to be widely embraced as a reliable prognosticator of outcome. At the authors' institution, fetuses less than 30 weeks' gestation, with enlarging macrocystic lesions associated with significant mediastinal shift or everted hemidiaphragms, are considered good candidates for a thoracoamniotic shunt. Again, well-designed prospective studies are lacking, but survival after thoracoamniotic shunt placement is approximately 70% based on some of the larger reported case series [78,88].

Rapidly enlarging solid lung lesions, including bronchopulmonary sequestrations and microcystic CCAMs, are not amenable to catheter drainage. In such cases, various ablation devices, including lasers and coagulators, have been used [86]. Unfortunately, these devices have not been shown to be effective because they all have a tendency to induce significant local edema in the immediate postprocedural period [89]. Thus, for the fetus less than 30 weeks' gestation in early hydrops, fetal lobectomy is the only reliable mass reduction therapy.

This procedure was first reported by Harrison in 1990 and requires an open hysterotomy and fetal thoracotomy through the fifth intercostal space (Figure 20.10) [90]. The pulmonary hilar structures are individually ligated or transected using a TA-30 vascular stapling device (U.S. Surgical Corp.). The perioperative morbidity associated with this procedure can be high, but overall survival rates have been reported to be approximately 50% to 60% [82,91].

For the fetus presenting with a massive CCAM beyond 34 weeks' gestation, the ability to oxygenate and ventilate the patient after delivery can be severely compromised. At the authors' institution, one viable option can be EXIT-to-ECMO, followed by immediate postnatal lung resection once the patient is stabilized. Another approach, fetal



**FIGURE 20.10.**  
*Fetal lobectomy for a cystic adenomatoid malformation.*  
For color reproduction, see Color Plate 8.

lobectomy during the EXIT procedure, has been described by the Children's Hospital of Philadelphia group and appears to have low rates of maternal-fetal morbidity and mortality [92].

### Sacrococcygeal Teratoma

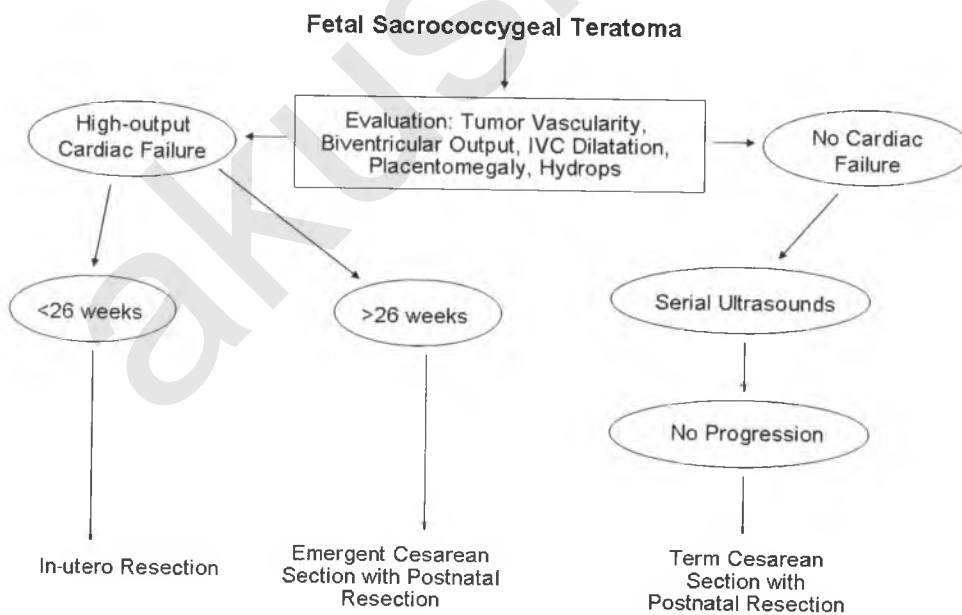
*Sacrococcygeal teratomas* (SCT) are rare tumors with an incidence of 1 in 40,000 births. Although malignancy rates for prenatal SCT are low, these lesions can be associated with significant arteriovenous shunting in a subset of patients. Noncystic SCTs

have a tendency to have a rich blood supply, and therefore some fetuses are at significant risk for high-output cardiac failure and hydrops. In-utero disruption of the aberrant vascular physiology remains the only option for salvage of the fetus [93].

Fetuses are followed closely by serial ultrasound scans and echocardiograms looking for evidence of high-output cardiac failure (Figure 20.11). Many fetuses with SCTs never develop high-output cardiac failure on serial imaging and are delivered close to term by a cesarean.

Prenatal surgical intervention is entertained only in cases of early hydrops before 26 weeks' gestation. Experience has shown that these tumors are best approached by open fetal resection (Figure 20.12) [24,94]. An umbilical tape tourniquet or vascular stapling device can be used to minimize blood loss. In addition, a red rubber catheter is typically placed into the rectum to facilitate the dissection. Other less invasive approaches, particularly radiofrequency ablation, have been tried and have yet to achieve consistent results to date because of problems associated with iatrogenic thermal injury [95,96]. In theory, these ablative approaches might also be associated with significant risk for the tumor lysis syndrome [97].

For the preterm fetus after 26 weeks' gestation, betamethasone administration followed by cesarean delivery allows an alternative to fetal surgery. At the authors' institution, these SCT patients are allowed



**FIGURE 20.11.**  
*Algorithm for the management of fetal sacrococcygeal teratoma.*



FIGURE 20.12.  
*Sacrococcygeal teratoma prior to fetal resection. For color reproduction, see Color Plate 9.*

to grow and develop in the neonatal intensive care unit until they develop early signs of infection, at which time the SCT is resected.

### Bladder Outlet Obstruction

*Bladder outlet obstruction* is currently the only genitourologic condition that is amenable to fetal sur-

gical intervention. The obstruction is usually secondary to posterior urethral valves, although urethral atresia and prune-belly syndrome are also well-described entities that can cause this condition [98]. Important diagnostic signs of a high-grade, lower urinary tract obstruction are an enlarged bladder, thickening of the bladder wall, and hydroureter in association with a dilated upper urethra. The sonographic appearance of the kidneys should be noted but might not be predictive of renal function in this setting. Obstructive uropathy in the presence of oligohydramnios is particularly worrisome because it is associated with high perinatal mortality secondary to severe pulmonary hypoplasia and renal dysplasia.

Patients with bladder outlet obstruction are considered good candidates for a prenatal intervention if they have a male karyotype, have documented oligohydramnios, and show relatively preserved renal function as demonstrated on serial vesicocenteses (Figure 20.13) [98]. There is currently no role for a fetal procedure in the setting of a normal amniotic fluid volume. If serial fetal urine sampling every 48 to 72 hours reveals *hypotonic* urine (typically defined as sodium <100 mEq/l; chloride <90 mmol/l; osm <200 mmol/l; and microglobulin <6 mg/l), the fetus is likely to have adequate renal

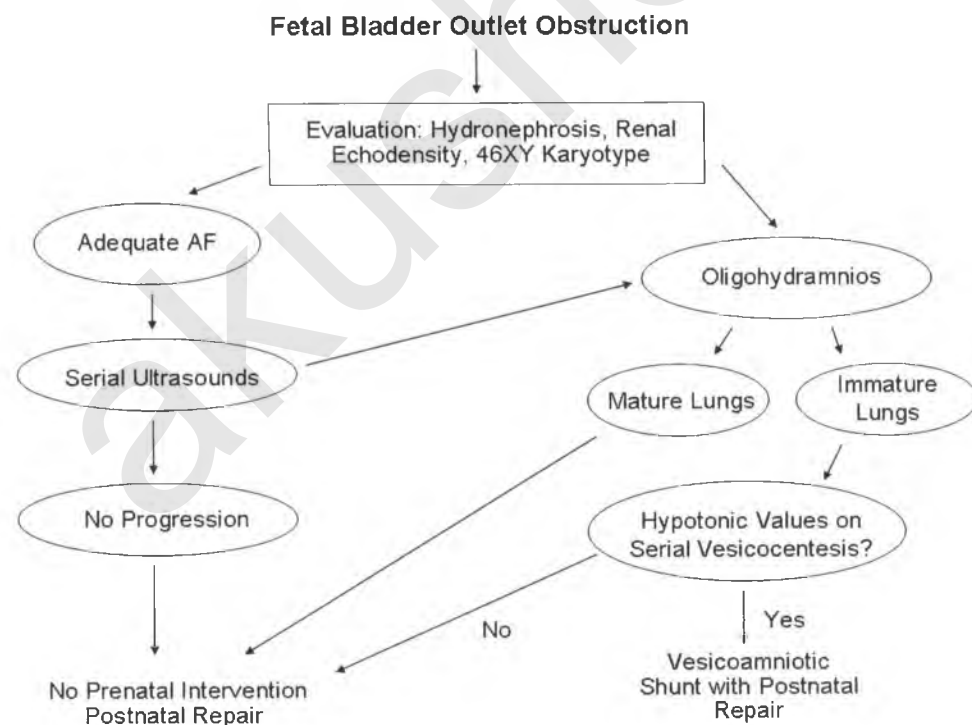


FIGURE 20.13.  
*Algorithm for the management of fetal bladder outlet obstruction.*

function and is a suitable prenatal surgical candidate [99].

The first fetal procedures for the treatment of bladder outlet obstruction were performed using open fetal surgical techniques [100]. Given the morbidities associated with an open vesicostomy, however, vesicoamniotic shunting using a double-pigtail catheter has now become the treatment of choice for appropriately identified patients. The ideal location for shunt placement is below the umbilical cord insertion and slightly lateral to the midline. Decompression of the urinary tract and improvement in the lung hypoplasia can be achieved in most cases. Unfortunately, periprocedural morbidity remains high [40]. Although there is now reasonable long-term postnatal data demonstrating good urologic function after fetal urinary tract decompression, up to one third of all children develop renal failure and require dialysis or renal transplantation [101,102].

Several other approaches can be appropriate in selected cases of bladder outlet obstruction. Simple aspiration of the bladder contents is an option that can be particularly useful as an initial therapy for fetuses less than 20 weeks' gestation. One interesting experimental approach is fetal cystoscopy with ablation of the posterior urethral valves through a 1.3-mm endoscope [103,104]. This approach offers several theoretical advantages when compared to vesicoamniotic shunting, but the results to date have been mixed, mostly because of technical difficulties using currently available instrumentation. Open vesicostomy remains largely of historical interest but can be considered if vesicoamniotic shunting fails to resolve the oligohydramnios.

## EXPERIMENTAL APPLICATIONS

### **Diaphragmatic Hernia**

*Congenital diaphragmatic hernia* (CDH) occurs in approximately 1 in 2,500 live births and has long been a disease of significant interest for fetal intervention because of its association with severe pulmonary hypoplasia [105,106]. In several large clinical series, postnatal survival rates for prenatally diagnosed cases of severe CDH patients remain below 50%, despite the use of state-of-the-art technologies, including permissive hypercapnea, nitric oxide, and ECMO [107–110]. In addition, the pul-

monary morbidity among many survivors born with CDH continues to be significant [111,112].

The basis for fetal surgery in CDH was originally established through careful studies in animal models by Harrison and colleagues [113]. Subsequent findings led to the first human attempts at open fetal CDH repair, which were initiated in 1989. These operations required an open maternal hysterotomy, fetal thoracotomy, and a subcostal laparotomy to reduce the abdominal contents and repair the diaphragmatic defect [114]. Although this approach proved to be technically feasible, open diaphragmatic repair was ultimately abandoned because of a failure to demonstrate improved postnatal outcomes when compared with standard postnatal care [115]. In addition, open repair could not be safely performed in the most severely affected fetuses, namely those with liver herniation, because of kinking of the umbilical vein when the liver was reduced back into its normal anatomic position [116]. Other methods to reduce the abdominal viscera, such as the creation of a gastroschisis in utero, have been described but are associated with substantial and unnecessary morbidity [117].

Currently, the most promising approach for the prenatal treatment of CDH remains *temporary tracheal occlusion*, a procedure that induces lung hyperplasia by preventing the egress of lung liquid secreted by fetal pulmonary epithelium. Wilson and colleagues at Children's Hospital, Boston, first introduced the concept of tracheal occlusion as a means of inducing fetal CDH lung growth [118]. In a series of subsequent experiments, they demonstrated that suture ligation of the trachea prevented lung hypoplasia in fetal lambs with surgically created diaphragmatic hernias [119,120]. Work by Harrison's group [121] made significant advances on this principle, which led to human application using a titanium clip [122], and more recently, the deployment of a detachable intratracheal balloon placed endoscopically [101,123]. The balloon was later removed during an EXIT procedure [76], and the actual diaphragmatic defect was repaired postnatally.

Thus far, clinical outcomes of fetal tracheal occlusion for the treatment of CDH have been mixed [124–126]. The results of a single-center U.S. randomized controlled trial sponsored by National Institutes of Health (NIH) were recently published [126]. In this study, successful endoscopic

deployment of the tracheal balloon was achieved after a maternal laparotomy was performed at 25 weeks' gestation. This resulted in an impressive survival rate of 73% in a cohort of patients with liver herniation and a lung-to-head ratio of less than 1.4. This survival rate did not prove to be significantly different when compared with those who were treated with aggressive postnatal management (survival rate = 77%).

There are several important points worth mentioning about the NIH trial. First, the success of the control group emphasizes that ongoing progress in the postnatal management of diseases like CDH will continue to raise the bar against which fetal therapies should be judged. An alternative interpretation of the observed survival rates in this study raises the question of whether the sonographic lung-to-head ratio is a reliable prenatal predictor for identifying patients with high mortality under conventional management [127]. Finally, the trial highlighted the continuing problem of preterm labor and the subsequent long-term patient morbidity that is common in all survivors [128].

Although enthusiasm for tracheal occlusion has tempered after the results of the NIH study became known, the fetal tracheal occlusion approach continues to be explored worldwide. The currently ongoing Eurofoetus trial in Europe, led by Deprest and colleagues, has yielded some very promising results in a cohort of CDH patients [129]. In this study, fetuses with liver herniation and a lung-to-head ratio of less than 1.0 undergo tracheal occlusion at a median gestational age of 26 weeks using a single, percutaneous 3.3-mm port. Interim results have demonstrated a 50% survival rate in a cohort of patients considered to have a less than 10% survival under conventional postnatal management [130].

Unfortunately, the Eurofoetus trial has several flaws, including the lack of a randomized design. There is also no standardization of postnatal care. Moreover, a comparison with a group of patients treated with state-of-the-art, postnatal therapy as practiced at the major American referral centers is probably necessary before direct translation of the European approach could be justified in the United States. Nevertheless, the lessons learned from the European experience, with ongoing research in postoperative tocolysis, surgical technique, and control of fetal lung growth through the

use of intrapulmonary adjuncts, might eventually produce clinical results that could justify tracheal occlusion in the most severe cases [131–134].

Finally, a more recent perinatal intervention, EXIT-to-ECMO, is currently being employed in at least three U.S. centers as an alternative approach in the resuscitation of patients with severe CDH identified in utero. During the EXIT procedure, fetuses are placed on ECMO if they fail a trial of ventilation. The rationale for this approach is that by transitioning the patient directly from placental support to ECMO support, the hypoxia, acidosis, hypotension, and barotrauma associated with vigorous neonatal resuscitation can be avoided. Although the early data from the authors' center are encouraging, whether this approach is superior to conventional postnatal therapy in terms of mortality and morbidity remains to be determined [134a].

### Myelomeningocele

A *myelomeningocele* (MMC) is a relatively common and nonlethal neural tube defect, with estimated incidence of 1 in 1,500 births. Depending on the level of the MMC, patients face lifelong disabilities secondary to paraplegia, urinary and fecal incontinence, sexual dysfunction, and skeletal malformations. In addition, most of these children require multiple operations to treat the hydrocephalus that occurs in association with the Chiari II malformation [135]. The current standard of care for MMC remains elective cesarean at term followed by early postnatal closure of the spinal cord defect, ventriculoperitoneal (VP) shunting if necessary, and aggressive rehabilitation. An alternative approach, fetal closure of the MMC defect, has been under intense clinical investigation in recent years, however.

The rationale for in-utero MMC repair is that closure of the defect as early as possible could arrest ongoing neural destruction, thereby allowing improved neurologic function according to the "two-hit hypothesis," originally described by Hefez and colleagues [136]. This theory suggests that the spinal cord damage is caused by 1) a primary defect in neurulation at the fourth week of gestation, and 2) secondary injury from chronic exposure to urea and other toxic components contained within the amniotic fluid. Detailed pathologic evaluations of human MMC cord specimens, as well as sonographic observations that some MMC fetuses

have decreased lower extremity activity as gestation progresses, both lend support to this hypothesis [137,138]. Another rationale for in-utero closure has been to minimize cerebrospinal fluid leakage, thereby preventing further brain maldevelopment, including hindbrain herniation and ventriculomegaly [137].

Initial animal work on closure of surgically created fetal spinal cord defects proved to be very promising, demonstrating improvements in both distal spinal cord function as well as brain development [139,140]. Based on these results, the first human fetal MMC repairs were initiated [141]. Early approaches attempted in-utero repair by maternal laparotomy, with fetoscopic coverage of the defect using a maternal split-thickness skin graft [142]. The minimally invasive approach has since been abandoned, however, because it proved to be too cumbersome and difficult to perform. In addition, fetal mortality and morbidity in these early cases were high [143]. Since 1997, an open hysterotomy approach, with closure of the fetal spinal cord defect in multiple layers under an operating microscope, has been employed [144].

The current results of fetal MMC repair are based on observational data from 3 U.S. centers – Vanderbilt University Medical Center, Children’s Hospital of Philadelphia, and the University of California at San Francisco Medical Center. Their combined experience has demonstrated a consistent resolution in hindbrain herniation as well as a decrease in the incidence of shunt-dependent hydrocephalus among infants when compared with historical controls [145–149]. The results of this procedure have not been as favorable as the animal studies have shown, however. There does not appear to be a significant benefit of fetal repair on distal neurologic function [150]. Furthermore, in-utero repair poses an increased risk for prematurity, and several fetal deaths have been reported [145–148].

In an effort to ensure patient safety and to assess better whether the observed reduction in shunt-dependent hydrocephalus might simply be a reflection of confounding factors such as physician bias, the effectiveness of fetal MMC repair is currently being evaluated in an NIH-sponsored trial, known as the Management of Myelomeningocele Study (MOMS). In this trial, fetuses are randomized to undergo either fetal closure or standard postnatal therapy. Other U.S. institutions have agreed

not to perform any fetal MMC repairs pending these results. To be eligible, all fetuses must present before 25 weeks’ gestation and have a MMC defect between T1 and S1. Hindbrain herniation should be demonstrated on fetal MRI. Accrual rates have been slow, but over 100 subjects have now entered the trial.

Regardless of the findings of the MOMS trial, many questions regarding in-utero MMC repair are likely to remain unanswered. What are the long-term neurologic outcomes associated with in-utero repair? What is the best way to close the defect? Does a modest decrease in the need for a ventriculoperitoneal shunt justify the maternal-fetal risks of open fetal surgery for a nonlethal condition? Would a fetal intervention earlier in gestation lead to greater improvements in distal neurologic function? All of these questions, as well as the impact of robotics on enabling a less invasive fetoscopic repair, must be carefully evaluated to determine the ultimate utility of fetal repair for the large number of MMC fetuses identified each year [151].

### Aortic Stenosis

The natural history of critical aortic stenosis follows a predictable pathophysiology in the developing fetal heart [152]. Left ventricular outflow tract obstruction results in ventricular overload, which then progresses to chronic myocardial wall ischemia, and eventually to myocardial fibrosis. Within weeks, the left ventricle becomes unable to support systemic circulation, a condition referred to as the *hypoplastic left heart syndrome* (HLHS).

Given the irreversible damage incurred by the fetal heart, prenatally diagnosed aortic stenosis has been associated with significant mortality and morbidity at birth [153]. Currently, most HLHS children undergo a series of staged surgical reconstructions (known as the Norwood procedure), but up to 25% of these infants die during or soon after their first operation [154]. Additionally, these procedures do not alleviate the central problem of a single ventricle circulation. As a result, many affected patients face a lifetime of cardiac and neurologic morbidity. Cardiac transplantation has the advantage of restoring two-ventricle physiology; however, available donor organs are scarce, and transplantation itself is associated with substantial long-term morbidities because of chronic immunosuppression.

Although increasingly rare, compassionate care still remains an option at a few centers.

In-utero aortic valvuloplasty is a relatively new and emerging therapy that aims to prevent, reverse, or minimize the degree of left ventricular hypoplasia associated with fetal aortic stenosis. Although the procedure was first reported in 1991, there were few cases performed initially, in part because early results proved to be disappointing [155,156]. In a review of these cases published in 2000, postprocedural and long-term mortality were 58% and 92%, respectively [157]. Only recently has a large clinical experience accumulated, particularly at Children's Hospital, Boston, where over 60 such aortic valve procedures have been performed to date [158].

Patients are selected based on various echocardiographic parameters, including flow across the aortic valve, as well as left ventricular size and contractility. Under continuous ultrasound guidance, the fetus is paralyzed with an intramuscular agent. A trocar is passed directly into the left ventricle, and a wire is inserted and passed across the stenotic aortic valve (Figure 20.14). A 3-mm balloon catheter is then passed over the wire, and the balloon is inflated. The procedure can be performed through a maternal laparotomy or as a fully percutaneous intervention, if fetal positioning allows. Thus far, early results have shown that a technically successful aortic valvuloplasty can be accomplished in most patients before 29 weeks' gestation [158,159]. Ongoing left heart

growth can be seen following aortic valvuloplasty, and a two-ventricle physiology develops in about one third of all live-born infants. Most patients who do not develop a fully functional left ventricle after aortic valvuloplasty remain candidates for standard postnatal operative repair.

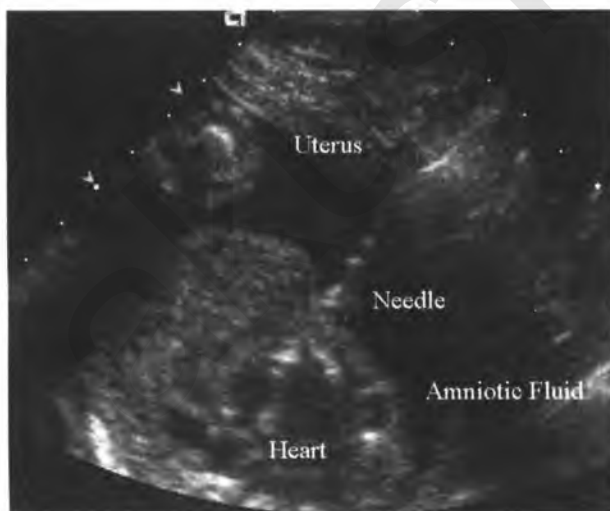
Fetal cardiac interventions could also prove to be useful for a distinct subset of aortic stenosis fetuses presenting with premature closure of the atrial septum, a condition referred to as a *restrictive atrial septum*. These fetuses are at a much higher risk for subsequent mortality and morbidity [160]. Theoretically, decompression of the left ventricle might allow for improved pulmonary venous return to the right heart. In addition, left atrial decompression might allow time for pulmonary remodeling. On this basis, fetal atrial septoplasty has been performed and has been shown to be technically feasible [161]. The heart is entered directly through the right atrial wall, and a wire is passed through the septum and into the left atrium. Either a 3-mm balloon or a YAG laser can be used to create channels in the atrial septum [162].

Despite some of these promising early results in management of fetal aortic stenosis, the effectiveness of these procedures relative to conventional postnatal therapies must be better defined. Issues of patient selection, perioperative fetal imaging, timing of intervention, and operative technique are all undoubtedly important in determining the precise role of these therapies in the near future [159].

## CONCLUSION

Although most prenatally diagnosed anomalies are best managed after birth, several disorders have predictable, irreversible, and devastating consequences under expectant prenatal management. In some of these situations, fetal surgical therapies now have something to offer. The recent increase in the number of fetal care centers being established worldwide is a testament to the growing acceptance of fetal interventions among both health professionals and the lay public alike.

As rapid advances in prenatal imaging and minimally invasive instrumentation continue, improvements in existing and emerging applications seem inevitable. Specialists across the maternal-fetal spectrum should remain hopeful of the future but also must understand the enormous ethical, diagnostic,



**FIGURE 20.14.**  
*Intraoperative ultrasound demonstrating successful needle entry into the fetal heart in preparation for the aortic valve balloon dilatation.*



and therapeutic challenges involved in the fetal therapeutic enterprise. Physicians must also remain cautious about the precipitous introduction of prenatal interventions of unproven value. Maternal safety should always remain paramount, and therapies that do not prove to be effective in appropriately controlled clinical studies must be abandoned in a timely manner.

## REFERENCES

1. Patient choice and the maternal-fetal relationship, No. 214, April, 1999. Committee on Ethics. *Int J Gynaecol Obstet* 1999;65:213-5.
2. Chervenak FA, McCullough LB. The fetus as a patient: An essential concept for the ethics of perinatal medicine. *Am J Perinatol* 2003;20:399-404.
3. American Academy of Pediatrics. Committee on Bioethics. Fetal therapy – ethical considerations. *Pediatrics* 1999;103:1061-3.
4. Levine C, Faden R, Grady C, Hammerschmidt D, Eckenwiler L, Sugarman J. "Special scrutiny": A targeted form of research protocol review. *Ann Intern Med* 2004;140:220-3.
5. Chescheir NC, Socol M. The National Institutes of Health Workshop on Fetal Treatment: Needs assessment and future directions. *Obstet Gynecol* 2005;106:828-33.
6. Lyerly AD, Gates EA, Cefalo RC, Sugarman J. Toward the ethical evaluation and use of maternal-fetal surgery. *Obstet Gynecol* 2001;98:689-97.
7. Adzick NS. Management of fetal lung lesions. *Clin Perinatol* 2003;30:481-92.
8. DiFederico EM, Harrison M, Matthay MA. Pulmonary edema in a woman following fetal surgery. *Chest* 1996;109:1114-7.
9. Longaker MT, Golbus MS, Filly RA, Rosen MA, Chang SW, Harrison MR. Maternal outcome after open fetal surgery: A review of the first 17 human cases. *JAMA* 1991;265:737-41.
10. Noah MM, Norton ME, Sandberg P, Esakoff T, Farrell J, Albanese CT. Short-term maternal outcomes that are associated with the EXIT procedure, as compared with cesarean delivery. *Am J Obstet Gynecol* 2002;186:773-7.
11. Wilson RD, Johnson MP, Flake AW, et al. Reproductive outcomes after pregnancy complicated by maternal-fetal surgery. *Am J Obstet Gynecol* 2004;191:1430-6.
12. Farrell JA, Albanese CT, Jennings RW, Kilpatrick SJ, Bratton BJ, Harrison MR. Maternal fertility is not affected by fetal surgery. *Fetal Diagn Ther* 1999;14:190-2.
13. Liley AW. Intrauterine transfusion of foetus in haemolytic disease. *Br Med J* 1963;5365:1107-9.
14. Quinn TM, Hubbard AM, Adzick NS. Prenatal magnetic resonance imaging enhances fetal diagnosis. *J Pediatr Surg* 1998;33:553-8.
15. Rahbar R, Vogel A, Myers LB, et al. Fetal surgery in otolaryngology: A new era in the diagnosis and management of fetal airway obstruction because of advances in prenatal imaging. *Arch Otolaryngol Head Neck Surg* 2005;131:393-8.
16. Myers LB, Bulich LA, Mizrahi A, et al. Ultrasonographic guidance for location of the trachea during the EXIT procedure for cervical teratoma. *J Pediatr Surg* 2003;38:E12.
17. Cohen MS, Rychik J, Bush DM, et al. Influence of congenital heart disease on survival in children with congenital diaphragmatic hernia. *J Pediatr* 2002;141:25-30.
18. Graziano JN. Cardiac anomalies in patients with congenital diaphragmatic hernia and their prognosis: A report from the Congenital Diaphragmatic Hernia Study Group. *J Pediatr Surg* 2005;40:1045-9; discussion 9-50.
19. Nakayama DK, Harrison MR, Seron-Ferre M, Villa RL. Fetal surgery in the primate II: Uterine electromyographic response to operative procedures and pharmacologic agents. *J Pediatr Surg* 1984;19:333-9.
20. Adzick NS, Harrison MR, Glick PL, et al. Fetal surgery in the primate III: Maternal outcome after fetal surgery. *J Pediatr Surg* 1986;21:477-80.
21. Harrison MR, Adzick NS. Fetal surgical techniques. *Semin Pediatr Surg* 1993;2:136-42.
22. Harrison MR, Anderson J, Rosen MA, Ross NA, Hendrickx AG. Fetal surgery in the primate I: Anesthetic, surgical, and tocolytic management to maximize fetal-neonatal survival. *J Pediatr Surg* 1982;17:115-22.
23. Jennings RW, Adzick NS, Longaker MT, Lorenz HP, Harrison MR. Radiotelemetric fetal monitoring during and after open fetal operation. *Surg Gynecol Obstet* 1993;176:59-64.
24. Graf JL, Paek BW, Albanese CT, et al. Successful resuscitation during fetal surgery. *J Pediatr Surg* 2000;35:1388-9.

25. Bisits A, Madsen G, Knox M, et al. The Randomized Nitric Oxide Tocolysis Trial (RNOTT) for the treatment of preterm labor. *Am J Obstet Gynecol* 2004;191:683–90.
26. Duckitt K, Thornton S. Nitric oxide donors for the treatment of preterm labour. *Cochrane Database Syst Rev* 2002.
27. Quintero RA, Puder KS, Cotton DB. Embryoscopy and fetoscopy. *Obstet Gynecol Clin North Am* 1993;20:563–81.
28. Luks FI, Deprest JA, Peers KH, Desimpelaere L, Vandenberghe K. Infrared fetoscopy in the sheep. *Fetal Diagn Ther* 1994;9:327–30.
29. Skarsgard ED, Bealer JF, Meuli M, Adzick NS, Harrison MR. Fetal endoscopic ('Fetendo') surgery: The relationship between insufflating pressure and the fetoplacental circulation. *J Pediatr Surg* 1995;30:1165–8.
30. Deprest JA, Gratacos E. Obstetrical endoscopy. *Curr Opin Obstet Gynecol* 1999;11:195–203.
31. Myers LB, Cohen D, Galinkin J, Gaiser R, Kurth CD. Anaesthesia for fetal surgery. *Paediatr Anaesth* 2002;12:569–78.
32. Pelletier GJ, Srinathan SK, Langer JC. Effects of intraamniotic helium, carbon dioxide, and water on fetal lambs. *J Pediatr Surg* 1995;30:1155–8.
33. Fauza DO, Rawn J, Fishman SJ. Fetal response to neutral gas and liquid media for intraamniotic distension. *J Pediatr Surg* 1999;34:591–4.
34. Evrard VA, Verbeke K, Peers KH, et al. Amnioinfusion with Hartmann's solution: A safe distention medium for endoscopic fetal surgery in the ovine model. *Fetal Diagn Ther* 1997;12:188–92.
35. Smith RP, Gitau R, Glover V, Fisk NM. Pain and stress in the human fetus. *Eur J Obstet Gynecol Reprod Biol* 2000;92:161–5.
36. Knight CG, Lorincz A, Johnson A, et al. Robot-enhanced fetoscopic surgery. *J Pediatr Surg* 2004;39:1463–5.
37. Frigoletto FD, Jr., Birnholz JC, Greene MF. Antenatal treatment of hydrocephalus by ventriculoamniotic shunting. *JAMA* 1982;248:2496–7.
38. Manning FA, Harrison MR, Rodeck C. Catheter shunts for fetal hydronephrosis and hydrocephalus: Report of the International Fetal Surgery Registry. *N Engl J Med* 1986;315:336–40.
39. Nicolaidis KH, Blott M, Greenough A. Chronic drainage of fetal pulmonary cyst. *Lancet* 1987;1:618.
40. Wilson RD, Johnson MP. Prenatal ultrasound-guided percutaneous shunts for obstructive uropathy and thoracic disease. *Semin Pediatr Surg* 2003;12:182–9.
41. Duncan KR, Denbow ML, Fisk NM. The aetiology and management of twin-twin transfusion syndrome. *Prenat Diagn* 1997;17:1227–36.
42. Bajoria R, Sullivan M, Fisk NM. Endothelin concentrations in monochorionic twins with severe twin-twin transfusion syndrome. *Hum Reprod* 1999;14:1614–8.
43. Fox C, Kilby MD, Khan KS. Contemporary treatments for twin-twin transfusion syndrome. *Obstet Gynecol* 2005;105:1469–77.
44. Lopriore E, Nagel HT, Vandebussche FP, Walther FJ. Long-term neurodevelopmental outcome in twin-to-twin transfusion syndrome. *Am J Obstet Gynecol* 2003;189:1314–9.
45. Bejar R, Vigliocco G, Gramajo H, et al. Antenatal origin of neurologic damage in newborn infants. II. Multiple gestations. *Am J Obstet Gynecol* 1990;162:1230–6.
46. Fusi L, McParland P, Fisk N, Nicolini U, Wigglesworth J. Acute twin-twin transfusion: A possible mechanism for brain-damaged survivors after intrauterine death of a monochorionic twin. *Obstet Gynecol* 1991;78 (Pt 2):517–20.
47. De Lia JE, Cruikshank DP, Keye WR, Jr. Fetoscopic neodymium:YAG laser occlusion of placental vessels in severe twin-twin transfusion syndrome. *Obstet Gynecol* 1990;75:1046–53.
48. Deprest JA, Van Schoubroeck D, Evrard VA, Flageole H, Van Ballaer P, Vandenberghe K. Fetoscopic Nd:YAG laser coagulation for twin-twin transfusion syndrome in cases of anterior placenta. *J Am Assoc Gynecol Laparosc* 1996;3(4, Suppl):S9.
49. Quintero RA, Comas C, Bornick PW, Allen MH, Kruger M. Selective versus non-selective laser photocoagulation of placental vessels in twin-to-twin transfusion syndrome. *Ultrasound Obstet Gynecol* 2000;16:230–6.
50. Senat MV, Deprest J, Boulvain M, Paupe A, Winer N, Ville Y. Endoscopic laser surgery versus serial amnioreduction for severe twin-to-twin transfusion syndrome. *N Engl J Med* 2004;351:136–44.
- 50a. Crombleholme TR, Shera D, Lee H, et al. A prospective, randomized, multicenter trial of amnioreduction vs selective fetoscopic laser photocoagulation for the treatment of severe twin-twin

- transfusion syndrome. *Am J Obstet Gynecol* 2007; 197:396e1-9.
51. Quintero RA, Dickinson JE, Morales WJ, et al. Stage-based treatment of twin-twin transfusion syndrome. *Am J Obstet Gynecol* 2003;188:1333-40.
  52. Taylor MJ, Govender L, Jolly M, Wee L, Fisk NM. Validation of the Quintero staging system for twin-twin transfusion syndrome. *Obstet Gynecol* 2002;100:1257-65.
  53. Taylor MJ, Shalev E, Tanawattanacharoen S, et al. Ultrasound-guided umbilical cord occlusion using bipolar diathermy for Stage III/IV twin-twin transfusion syndrome. *Prenat Diagn* 2002;22:70-6.
  54. Saade GR, Belfort MA, Berry DL, et al. Amniotic septostomy for the treatment of twin oligohydramnios-polyhydramnios sequence. *Fetal Diagn Ther* 1998;13:86-93.
  55. Moise KJ, Jr., Dorman K, Lamvu G, et al. A randomized trial of amnioreduction versus septostomy in the treatment of twin-twin transfusion syndrome. *Am J Obstet Gynecol* 2005;193(Pt 1):701-7.
  56. Feldman DM, Odibo A, Campbell WA, Rodis JF. Iatrogenic monoamniotic twins as a complication of therapeutic amniocentesis. *Obstet Gynecol* 1998;91(Pt 2):815-6.
  57. Van Allen MI, Smith DW, Shepard TH. Twin reversed arterial perfusion (TRAP) sequence: A study of 14 twin pregnancies with acardius. *Semin Perinatol* 1983;7:285-93.
  58. Hanafy A, Peterson CM. Twin-reversed arterial perfusion (TRAP) sequence: Case reports and review of literature. *Aust N Z J Obstet Gynaecol* 1997;37:187-91.
  59. Tan TY, Sepulveda W. Acardiac twin: A systematic review of minimally invasive treatment modalities. *Ultrasound Obstet Gynecol* 2003;22:409-19.
  60. McCurdy CM, Jr., Childers JM, Seeds JW. Ligation of the umbilical cord of an acardiac-acephalus twin with an endoscopic intrauterine technique. *Obstet Gynecol* 1993;82(4 Pt 2 Suppl):708-11.
  61. Quintero RA, Reich H, Puder KS, et al. Brief report: Umbilical-cord ligation of an acardiac twin by fetoscopy at 19 weeks of gestation. *N Engl J Med* 1994;330:469-71.
  62. Tsao K, Feldstein VA, Albanese CT, et al. Selective reduction of acardiac twin by radiofrequency ablation. *Am J Obstet Gynecol* 2002;187(3):635-40.
  63. Arias F, Sunderji S, Gimpelson R, Colton E. Treatment of acardiac twinning. *Obstet Gynecol* 1998;91(5 Pt 2):818-21.
  64. Sepulveda W, Corral E, Aiello H, et al. Intrafetal alcohol chemosclerosis of acardiac twins: A multi-center experience. *Fetal Diagn Ther* 2004;19:448-52.
  65. Bruner JP, Wheeler TC, Bliton MJ. Sectio parva for fetal preservation. *Fetal Diagn Ther* 1999;14:254-6.
  66. Hubbard AM, Crombleholme TM, Adzick NS. Prenatal MRI evaluation of giant neck masses in preparation for the fetal EXIT procedure. *Am J Perinatol* 1998;15:253-7.
  67. Morof D, Levine D, Grable I, et al. Oropharyngeal teratoma: Prenatal diagnosis and assessment using sonography, MRI, and CT with management by ex utero intrapartum treatment procedure. *AJR Am J Roentgenol* 2004;183:493-6.
  68. Levine D, Jennings R, Barnewolt C, Mehta T, Wilson J, Wong G. Progressive fetal bronchial obstruction caused by a bronchogenic cyst diagnosed using prenatal MR imaging. *AJR Am J Roentgenol* 2001;176:49-52.
  69. Paek BW, Callen PW, Kitterman J, et al. Successful fetal intervention for congenital high airway obstruction syndrome. *Fetal Diagn Ther* 2002;17:272-6.
  70. Kohl T, Hering R, Bauriedel G, et al. Fetoscopic and ultrasound-guided decompression of the fetal trachea in a human fetus with Fraser syndrome and congenital high airway obstruction syndrome (CHAOS) from laryngeal atresia. *Ultrasound Obstet Gynecol* 2006;27:84-8.
  71. Zerella JT, Finberg FJ. Obstruction of the neonatal airway from teratomas. *Surg Gynecol Obstet* 1990;170:126-31.
  72. Langer JC, Tabb T, Thompson P, Paes BA, Caco CC. Management of prenatally diagnosed tracheal obstruction: Access to the airway in utero prior to delivery. *Fetal Diagn Ther* 1992;7:12-6.
  73. Skarsgard ED, Chitkara U, Krane EJ, Riley ET, Halamek LP, Dedo HH. The OOPS procedure (operation on placental support): In utero airway management of the fetus with prenatally diagnosed tracheal obstruction. *J Pediatr Surg* 1996;31:826-8.
  74. Mychaliska GB, Bealer JF, Graf JL, Rosen MA, Adzick NS, Harrison MR. Operating on placental support: The ex utero intrapartum treatment

- procedure. *J Pediatr Surg* 1997;32:227–30; discussion 30–1.
75. Bouchard S, Johnson MP, Flake AW, et al. The EXIT procedure: Experience and outcome in 31 cases. *J Pediatr Surg* 2002;37:418–26.
  76. Hirose S, Harrison MR. The ex utero intrapartum treatment (EXIT) procedure. *Semin Neonatol* 2003;8:207–14.
  77. Aubard Y, Derouineau I, Aubard V, Chalifour V, Preux PM. Primary fetal hydrothorax: A literature review and proposed antenatal clinical strategy. *Fetal Diagn Ther* 1998;13:325–33.
  78. Wilson RD, Baxter JK, Johnson MP, et al. Thoracoamniotic shunts: Fetal treatment of pleural effusions and congenital cystic adenomatoid malformations. *Fetal Diagn Ther* 2004;19:413–20.
  79. Smith RP, Illanes S, Denbow ML, Soothill PW. Outcome of fetal pleural effusions treated by thoracoamniotic shunting. *Ultrasound Obstet Gynecol* 2005;26(1):63–6.
  80. Rodeck CH, Fisk NM, Fraser DI, Nicolini U. Long-term in utero drainage of fetal hydrothorax. *N Engl J Med* 1988;319:1135–8.
  81. Kunisaki SM, Fauza DO, Nemes LP, et al. Bronchial atresia: The hidden pathology within a spectrum of prenatally diagnosed lung masses. *J Pediatr Surg* 2006;41:61–5.
  82. Adzick NS, Harrison MR, Crombleholme TM, Flake AW, Howell LJ. Fetal lung lesions: Management and outcome. *Am J Obstet Gynecol* 1998;179(4):884–9.
  83. Miller JA, Corteville JE, Langer JC. Congenital cystic adenomatoid malformation in the fetus: natural history and predictors of outcome. *J Pediatr Surg* 1996;31:805–8.
  84. Roggin KK, Breuer CK, Carr SR, et al. The unpredictable character of congenital cystic lung lesions. *J Pediatr Surg* 2000;35:801–5.
  85. Laberge JM, Flageole H, Pugash D, et al. Outcome of the prenatally diagnosed congenital cystic adenomatoid lung malformation: A Canadian experience. *Fetal Diagn Ther* 2001;16:178–86.
  86. Davenport M, Warne SA, Cacciaguerra S, Patel S, Greenough A, Nicolaidis K. Current outcome of antenally diagnosed cystic lung disease. *J Pediatr Surg* 2004;39:549–56.
  87. Crombleholme TM, Coleman B, Hedrick H, et al. Cystic adenomatoid malformation volume ratio predicts outcome in prenatally diagnosed cystic adenomatoid malformation of the lung. *J Pediatr Surg* 2002;37:331–8.
  88. Dommergues M, Louis-Sylvestre C, Mandelbrot L, et al. Congenital adenomatoid malformation of the lung: When is active fetal therapy indicated? *Am J Obstet Gynecol* 1997;177:953–8.
  89. Bruner JP, Jarnagin BK, Reinisch L. Percutaneous laser ablation of fetal congenital cystic adenomatoid malformation: Too little, too late? *Fetal Diagn Ther* 2000;15:359–63.
  90. Harrison MR, Adzick NS, Jennings RW, et al. Antenatal intervention for congenital cystic adenomatoid malformation. *Lancet* 1990;336:965–7.
  91. Kuller JA, Yankowitz J, Goldberg JD, et al. Outcome of antenatally diagnosed cystic adenomatoid malformations. *Am J Obstet Gynecol* 1992;167(4 Pt 1):1038–41.
  92. Hedrick HL, Flake AW, Crombleholme TM, et al. The ex utero intrapartum therapy procedure for high-risk fetal lung lesions. *J Pediatr Surg* 2005;40:1038–43; discussion 44.
  93. Graf JL, Albanese CT, Jennings RW, Farrell JA, Harrison MR. Successful fetal sacrococcygeal teratoma resection in a hydropic fetus. *J Pediatr Surg* 2000;35:1489–91.
  94. Hedrick HL, Flake AW, Crombleholme TM, et al. Sacrococcygeal teratoma: Prenatal assessment, fetal intervention, and outcome. *J Pediatr Surg* 2004;39:430–8; discussion 8.
  95. Paek BW, Jennings RW, Harrison MR, et al. Radiofrequency ablation of human fetal sacrococcygeal teratoma. *Am J Obstet Gynecol* 2001;184:503–7.
  96. Lam YH, Tang MH, Shek TW. Thermocoagulation of fetal sacrococcygeal teratoma. *Prenat Diagn* 2002;22:99–101.
  97. Jona JZ. Progressive tumor necrosis and lethal hyperkalemia in a neonate with sacrococcygeal teratoma (SCT). *J Perinatol* 1999;19:538–40.
  98. Freedman AL, Johnson MP, Gonzalez R. Fetal therapy for obstructive uropathy: Past, present, future? *Pediatr Nephrol* 2000;14:167–76.
  99. Freedman AL, Bukowski TP, Smith CA, et al. Use of urinary beta-2-microglobulin to predict severe renal damage in fetal obstructive uropathy. *Fetal Diagn Ther* 1997;12:1–6.
  100. Harrison MR, Golbus MS, Filly RA, et al. Fetal surgery for congenital hydronephrosis. *N Engl J Med* 1982;306:591–3.

101. Holmes N, Harrison MR, Baskin LS. Fetal surgery for posterior urethral valves: Long-term postnatal outcomes. *Pediatrics* 2001;108:E7.
102. Biard JM, Johnson MP, Carr MC, et al. Long-term outcomes in children treated by prenatal vesicoamniotic shunting for lower urinary tract obstruction. *Obstet Gynecol* 2005;106:503–8.
103. Quintero RA, Hume R, Smith C, et al. Percutaneous fetal cystoscopy and endoscopic fulguration of posterior urethral valves. *Am J Obstet Gynecol* 1995;172(1 Pt 1):206–9.
104. Welsh A, Agarwal S, Kumar S, Smith RP, Fisk NM. Fetal cystoscopy in the management of fetal obstructive uropathy: Experience in a single European centre. *Prenat Diagn* 2003;23:1033–41.
105. Harrison MR, Adzick NS, Nakayama DK, de Lorimier AA. Fetal diaphragmatic hernia: Fatal but fixable. *Semin Perinatol* 1985;9:103–12.
106. Adzick NS, Vacanti JP, Lillehei CW, O'Rourke PP, Crone RK, Wilson JM. Fetal diaphragmatic hernia: Ultrasound diagnosis and clinical outcome in 38 cases. *J Pediatr Surg* 1989;24:654–7; discussion 7–8.
107. Harrison MR, Adzick NS, Estes JM, Howell LJ. A prospective study of the outcome for fetuses with diaphragmatic hernia. *Jama* 1994;271:382–4.
108. Dillon E, Renwick M, Wright C. Congenital diaphragmatic herniation: Antenatal detection and outcome. *Br J Radiol* 2000;73:360–5.
109. Guibaud L, Filiatrault D, Garel L, et al. Fetal congenital diaphragmatic hernia: Accuracy of sonography in the diagnosis and prediction of the outcome after birth. *AJR Am J Roentgenol* 1996;166:1195–202.
110. Colvin J, Bower C, Dickinson JE, Sokol J. Outcomes of congenital diaphragmatic hernia: A population-based study in Western Australia. *Pediatrics* 2005;116:e356–63.
111. Lund DP, Mitchell J, Kharasch V, Quigley S, Kuehn M, Wilson JM. Congenital diaphragmatic hernia: the hidden morbidity. *J Pediatr Surg* 1994;29:258–62; discussion 62–4.
112. Muratore CS, Kharasch V, Lund DP, et al. Pulmonary morbidity in 100 survivors of congenital diaphragmatic hernia monitored in a multidisciplinary clinic. *J Pediatr Surg* 2001;36:133–40.
113. Harrison MR, Bressack MA, Churg AM, de Lorimier AA. Correction of congenital diaphragmatic hernia in utero. II. Simulated correction permits fetal lung growth with survival at birth. *Surgery* 1980;88:260–8.
114. Harrison MR, Langer JC, Adzick NS, et al. Correction of congenital diaphragmatic hernia in utero. V. Initial clinical experience. *J Pediatr Surg* 1990;25:47–55; discussion 6–7.
115. Harrison MR, Adzick NS, Flake AW, et al. Correction of congenital diaphragmatic hernia in utero: VI. Hard-earned lessons. *J Pediatr Surg* 1993;28:1411–7; discussion 7–8.
116. MacGillivray TE, Jennings RW, Rudolph AM, Ring EJ, Adzick NS, Harrison MR. Vascular changes with in utero correction of diaphragmatic hernia. *J Pediatr Surg* 1994;29:992–6.
117. Porreco RP, Chang JH, Quissell BJ, Morgan MA. Palliative fetal surgery for diaphragmatic hernia. *Am J Obstet Gynecol* 1994;170:833–4.
118. Wilson JM, DiFiore JW, Peters CA. Experimental fetal tracheal ligation prevents the pulmonary hypoplasia associated with fetal nephrectomy: Possible application for congenital diaphragmatic hernia. *J Pediatr Surg* 1993;28:1433–9; discussion 9–40.
119. DiFiore JW, Fauza DO, Slavin R, Peters CA, Fackler JC, Wilson JM. Experimental fetal tracheal ligation reverses the structural and physiological effects of pulmonary hypoplasia in congenital diaphragmatic hernia. *J Pediatr Surg* 1994;29:248–56; discussion 56–7.
120. DiFiore JW, Fauza DO, Slavin R, Wilson JM. Experimental fetal tracheal ligation and congenital diaphragmatic hernia: A pulmonary vascular morphometric analysis. *J Pediatr Surg* 1995;30:917–23; discussion 23–4.
121. Hedrick MH, Estes JM, Sullivan KM, et al. Plug the lung until it grows (PLUG): A new method to treat congenital diaphragmatic hernia in utero. *J Pediatr Surg* 1994;29:612–7.
122. VanderWall KJ, Skarsgard ED, Filly RA, Eckert J, Harrison MR. Fetendo-clip: A fetal endoscopic tracheal clip procedure in a human fetus. *J Pediatr Surg* 1997;32:970–2.
123. Chiba T, Albanese CT, Farmer DL, et al. Balloon tracheal occlusion for congenital diaphragmatic hernia: Experimental studies. *J Pediatr Surg* 2000;35:1566–70.
124. Flake AW, Crombleholme TM, Johnson MP, Howell LJ, Adzick NS. Treatment of severe congenital diaphragmatic hernia by fetal tracheal

- occlusion: Clinical experience with fifteen cases. *Am J Obstet Gynecol* 2000;183:1059–66.
125. Harrison MR, Mychaliska GB, Albanese CT, et al. Correction of congenital diaphragmatic hernia in utero IX: Fetuses with poor prognosis (liver herniation and low lung-to-head ratio) can be saved by fetoscopic temporary tracheal occlusion. *J Pediatr Surg* 1998;33:1017–22; discussion 22–3.
  126. Harrison MR, Keller RL, Hawgood SB, et al. A randomized trial of fetal endoscopic tracheal occlusion for severe fetal congenital diaphragmatic hernia. *N Engl J Med* 2003;349:1916–24.
  127. Heling KS, Wauer RR, Hammer H, Bollmann R, Chaoui R. Reliability of the lung-to-head ratio in predicting outcome and neonatal ventilation parameters in fetuses with congenital diaphragmatic hernia. *Ultrasound Obstet Gynecol* 2005;25:112–8.
  128. Cortes RA, Keller RL, Townsend T, et al. Survival of severe congenital diaphragmatic hernia has morbid consequences. *J Pediatr Surg* 2005;40:36–45; discussion 6.
  129. Deprest J, Gratacos E, Nicolaidis KH. Fetoscopic tracheal occlusion (FETO) for severe congenital diaphragmatic hernia: Evolution of a technique and preliminary results. *Ultrasound Obstet Gynecol* 2004;24:121–6.
  130. Deprest J, Jani J, Gratacos E, et al. Fetal intervention for congenital diaphragmatic hernia: The European experience. *Semin Perinatol* 2005;29:94–103.
  131. Dzakovic A, Kaviani A, Jennings RW, Wilson JM, Fauza DO. Positive intrapulmonary oncotic pressure enhances short-term lung growth acceleration after fetal tracheal occlusion. *J Pediatr Surg* 2002;37:1007–10; discussion 10.
  132. Fauza DO, Barnewolt C, Brown SD, Jennings RW. Ultrasound-guided fetal tracheal occlusion. *J Pediatr Surg* 2002;37:300–2.
  133. Garza JJ, Downard CD, Clayton N, Maher TJ, Fauza DO. *Clostridium botulinum* toxin inhibits myometrial activity in vitro: Possible application on the prevention of preterm labor after fetal surgery. *J Pediatr Surg* 2003;38:511–3.
  134. Muensterer OJ, Till H, Bergmann F, et al. Lung growth induced by prenatal instillation of perfluorocarbon into the fetal rabbit lung. *Pediatr Surg Int* 2004;20:248–52.
  - 134a. Kunisaki SM, Barnewolt CE, Estroff JA, et al. Ex utero intrapartum treatment with extracorporeal membrane oxygenation for severe congenital diaphragmatic hernia. *J Pediatr Surg* 2007;42:98–104.
  135. Hunt GM. The Casey Holter lecture: Non-selective intervention in newborn babies with open spina bifida: The outcome 30 years on for the complete cohort. *Eur J Pediatr Surg* 1999;9(Suppl 1):5–8.
  136. Heffez DS, Aryanpur J, Hutchins GM, Freeman JM. The paralysis associated with myelomeningocele: Clinical and experimental data implicating a preventable spinal cord injury. *Neurosurgery* 1990;26:987–92.
  137. Meuli M, Meuli-Simmen C, Hutchins GM, Seller MJ, Harrison MR, Adzick NS. The spinal cord lesion in human fetuses with myelomeningocele: Implications for fetal surgery. *J Pediatr Surg* 1997;32:448–52.
  138. Korenromp MJ, van Gool JD, Bruinese HW, Kriek R. Early fetal leg movements in myelomeningocele. *Lancet* 1986;1:917–8.
  139. Meuli M, Meuli-Simmen C, Hutchins GM, et al. In utero surgery rescues neurological function at birth in sheep with spina bifida. *Nat Med* 1995;1:342–7.
  140. Paek BW, Farmer DL, Wilkinson CC, et al. Hind-brain herniation develops in surgically created myelomeningocele but is absent after repair in fetal lambs. *Am J Obstet Gynecol* 2000;183:1119–23.
  141. Adzick NS, Sutton LN, Crombleholme TM, Flake AW. Successful fetal surgery for spina bifida. *Lancet* 1998;352:1675–6.
  142. Bruner JP, Richards WO, Tulipan NB, Arney TL. Endoscopic coverage of fetal myelomeningocele in utero. *Am J Obstet Gynecol* 1999;180 (1 Pt 1):153–8.
  143. Bruner JP, Tulipan NB, Richards WO, Walsh WF, Boehm FH, Vrabcak EK. In utero repair of myelomeningocele: A comparison of endoscopy and hysterotomy. *Fetal Diagn Ther* 2000;15:83–8.
  144. Tulipan N, Bruner JP. Myelomeningocele repair in utero: A report of three cases. *Pediatr Neurosurg* 1998;28:177–80.
  145. Bruner JP, Tulipan N, Paschall RL, et al. Fetal surgery for myelomeningocele and the incidence of shunt-dependent hydrocephalus. *JAMA* 1999;282:1819–25.

146. Sutton LN, Adzick NS, Bilaniuk LT, Johnson MP, Crombleholme TM, Flake AW. Improvement in hindbrain herniation demonstrated by serial fetal magnetic resonance imaging following fetal surgery for myelomeningocele. *JAMA* 1999;282:1826-31.
147. Farmer DL, von Koch CS, Peacock WJ, et al. In utero repair of myelomeningocele: Experimental pathophysiology, initial clinical experience, and outcomes. *Arch Surg* 2003;138:872-8.
148. Johnson MP, Sutton LN, Rintoul N, et al. Fetal myelomeningocele repair: Short-term clinical outcomes. *Am J Obstet Gynecol* 2003;189:482-7.
149. Tulipan N, Sutton LN, Bruner JP, Cohen BM, Johnson M, Adzick NS. The effect of intrauterine myelomeningocele repair on the incidence of shunt-dependent hydrocephalus. *Pediatr Neurosurg* 2003;38:27-33.
150. Tubbs RS, Chambers MR, Smyth MD, et al. Late gestational intrauterine myelomeningocele repair does not improve lower extremity function. *Pediatr Neurosurg* 2003;38:128-32.
151. Aaronson OS, Tulipan NB, Cywes R, et al. Robot-assisted endoscopic intrauterine myelomeningocele repair: A feasibility study. *Pediatr Neurosurg* 2002;36:85-9.
152. Hornberger LK, Sanders SP, Rein AJ, Spevak PJ, Parness IA, Colan SD. Left heart obstructive lesions and left ventricular growth in the midtrimester fetus: A longitudinal study. *Circulation* 1995;92:1531-8.
153. Andrews R, Tulloh R, Sharland G, et al. Outcome of staged reconstructive surgery for hypoplastic left heart syndrome following antenatal diagnosis. *Arch Dis Child* 2001;85:474-7.
154. Bove EL. Current status of staged reconstruction for hypoplastic left heart syndrome. *Pediatr Cardiol* 1998;19:308-15.
155. Maxwell D, Allan L, Tynan MJ. Balloon dilatation of the aortic valve in the fetus: A report of two cases. *Br Heart J* 1991;65:256-8.
156. Lopes LM, Cha SC, Kajita LJ, Aiello VD, Jatene A, Zugaib M. Balloon dilatation of the aortic valve in the fetus: A case report. *Fetal Diagn Ther* 1996;11:296-300.
157. Kohl T, Sharland G, Allan LD, et al. World experience of percutaneous ultrasound-guided balloon valvuloplasty in human fetuses with severe aortic valve obstruction. *Am J Cardiol* 2000;85:1230-3.
158. Tworetzky W, Wilkins-Haug L, Jennings RW, et al. Balloon dilation of severe aortic stenosis in the fetus: potential for prevention of hypoplastic left heart syndrome: Candidate selection, technique, and results of successful intervention. *Circulation* 2004;110:2125-31.
159. Marshall AC, Tworetzky W, Bergersen L, et al. Aortic valvuloplasty in the fetus: Technical characteristics of successful balloon dilation. *J Pediatr* 2005;147:535-9.
160. Mahle WT, Rychik J, Gaynor JW, Spray TL, Rome JJ. Restrictive interatrial communication after reconstructive surgery for hypoplastic left heart syndrome. *Am J Cardiol* 2001;88:1454-7:A9.
161. Marshall AC, van der Velde ME, Tworetzky W, et al. Creation of an atrial septal defect in utero for fetuses with hypoplastic left heart syndrome and intact or highly restrictive atrial septum. *Circulation* 2004;110:253-8.
162. Quintero RA, Huhta J, Suh E, Chmait R, Romero R, Angel J. In utero cardiac fetal surgery: Laser atrial septotomy in the treatment of hypoplastic left heart syndrome with intact atrial septum. *Am J Obstet Gynecol* 2005;193:1424-8.

## Kevin Giordano

*Time is that wherein there is opportunity,  
and opportunity is that wherein there is no  
great time.*

Hippocrates (c. 460–377 B.C.E.)

*Precepts* (ch. 1)

W. H. S. Jones (trans.)

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Pregnancy, labor, and delivery expose the mother to a considerable risk of complications, resulting in the potential for injury or even death. During pregnancy, advancements made in prenatal testing, including ultrasound techniques, have created the opportunity to diagnose many fetal injuries in utero and provide a greater understanding of the development of the fetus. Improving the capability of diagnosing fetal abnormalities has also raised the specter of increased maternal risk when intervention or treatment of the fetus is possible. With advanced knowledge of the development of the fetus while in the womb comes a greater understanding of the impact of maternal behavior on fetal development [1].

Most pregnant women are willing to accept remarkable levels of bodily intrusion or invasion, increased costs, and time-consuming medical interventions to ensure the most successful outcome for their infants. As observed by the American Academy of Pediatrics, “women almost always are willing to accept self-sacrifice to the benefit of their fetus” [2]. The proposition that a pregnant woman will undergo self-sacrifice and expose herself to remarkable intrusion is not universal, however. Not only are some women unwilling to go to such lengths to give their babies the best chance, it is also true that many women practice risky behaviors that jeopardize the health of their fetuses. According to the Office of Applied Studies’ report on drug use during pregnancy, in 2002 to 2003 it was found that approximately 4% of pregnant women ages 15 to 44 years used illicit drugs during their pregnancy [3]. Additionally, approximately 4% of pregnant women reported binge drinking (consuming five drinks or more at a single sitting), with nearly 10% reporting that they had consumed alcohol within the month prior to being involved in the study. Of pregnant women between the ages of 15 and 25 years enrolled in the study, 27.6% had smoked during pregnancy, whereas 10.8% of pregnant women ages 26 to 44 smoked cigarettes. In a separate study undertaken by the National Advisory Council on Drug Abuse, the researchers estimated that 5.2% of the 1,632



pregnant women from four areas across the country (Los Angeles, CA; Des Moines, IA; Tulsa, OK; and Honolulu, HI) had used methamphetamines at some point during their pregnancy [4].

When a patient presents who is unwilling to go to the lengths of accepting any recommendations to give her baby the best chance, or worse is practicing risky behaviors jeopardizing the infant's health, ethical and legal quandaries are presented. Although rare, maternal-fetal conflicts create divided physician obligations to mother and fetus and capture the most poignant of ethical and legal controversies. At issue is the right of any person to refuse invasive medical treatment derived from the right to privacy, bodily integrity, and, in certain situations, religious freedom. In situations of maternal refusal of recommended care, however, this debate is whether the impact to the fetus, the dependent variable in the conflict, has any legal relevance.

Maternal rights advocates argue that attempts by the state to regulate the mother's behavior to protect the fetus involve enormous and unacceptable intrusions into personal privacy. These advocates cite *Roe v. Wade* as establishing a women's constitutional right to be free from state interference and unwarranted bodily intrusion, as well as rely on other legal precedent that protects privacy rights in regard to procreation and the family [5,6]. In *Roe*, the Supreme Court held that a fetus is not a "person" within the meaning of the Fourteenth Amendment of the Constitution, even though the court recognized the state's interest in protecting the fetus in the third trimester. Consequently, proponents of maternal rights argue that pregnant women have the same rights to bodily integrity and personal autonomy as any other citizen and should be allowed the right to refuse care even if that refusal might result in harm to the fetus. In furthering their argument, they cite that courts have refused to order immediate family members or other relatives to submit to organ donation procedures even when such a procedure will save the life of who has instituted the lawsuit. For instance, in the case of *McFall v. Shrimp* [7], the plaintiff suffered from aplastic anemia. After investigation and testing, the only suitable donor was the plaintiff's first cousin. Although he found it morally indefensible for the defendant to withhold consent, the judge refused to order the defendant to submit to a bone marrow aspiration procedure to serve as a donor to the

plaintiff, despite evidence that the plaintiff would die without the donation. In the decision, the judge wrote, "For our law to compel the defendant to submit to an intrusion of his body would change the very concept and principle upon which our society is founded. To do so would defeat the sanctity of the individual, and would impose a rule which would know no limits, and one could not imagine where the line would be drawn." [7]. Consequently, advocates of maternal rights argue that equal protection would support that similarly, a mother has no legal duty to rescue her fetus through medical intervention.

Fetal rights advocates distinguish the maternal-fetal relationship as unique, and thus a distinct obligation exists, removing the situation from the ordinary duty to rescue. Interestingly, proponents of fetal rights similarly rely on *Roe v. Wade* and also the Supreme Court's decision in *Planned Parenthood v. Casey* that altered the trimester framework under *Roe* and emphasized the state's interest in the promotion of potential life [8]. In both *Roe* and *Casey*, the Supreme Court recognized "...that the State has a legitimate interest from the outset of the pregnancy in protecting the health of the woman and the life of the fetus that may become a child". (See Appendix 1 for a more thorough analysis of *Roe v. Wade* and its progeny.) These proponents argue that the legal principles that are more applicable than the line of cases cited by the maternal rights advocates are those that arise out of child abuse and neglect laws and abortion law. They cite that child abuse and neglect statutes are frequently used by obstetricians, prosecutors, and juvenile courts as the basis for compelling a pregnant woman to submit to treatment to preserve the life of the fetus [9]. In this setting, the problematic issue is whether a fetus falls within the state's definition of a "child" or "minor." Recognizing that absent a legal duty, no person is required to come to the aid of another, the advocates for fetal rights point to the fact that the long-held principle is not without qualification. In circumstances in which a special relationship exists (i.e., parent and child), a person does have certain responsibilities.

The legal landscape for delineating maternal versus fetal rights is far from clear. Analysis of the maternal-fetal relationship that is provided by the courts is still evolving. The most publicized cases in recent years have involved court-ordered obstetric interventions on behalf of the fetus and criminal

prosecutions of pregnant women for drug addiction or alcohol abuse. At issue is whether the state's interest in the life or health of the fetus supersedes the autonomy of the pregnant woman. Courts therefore struggle with the question of whether a mother's right to refuse treatment is abrogated because of potential harm to the fetus. This issue has been considered with varied results. *Jefferson v. Griffin Spalding County Hospital* was one of the first cases to review this conflict [10]. Two weeks before her delivery date, the plaintiff was diagnosed with a placenta previa. The plaintiff was refusing a recommended cesarean delivery because of her religious belief. The hospital petitioned the court for an order to perform a cesarean. The court ruled in favor of the hospital and held that the intrusion was outweighed by the state's interest to protect a live but unborn human being from dying. The case was appealed to the Supreme Court of Georgia, which affirmed the decision in favor of the hospital, although by the time the appeal was decided, the placenta had apparently migrated and the patient delivered her child naturally.

A similar rationale was followed in the case of *Pemberton v. Tallahassee Memorial Regional Hospital* [11]. *Pemberton* involved a patient who refused a cesarean delivery and in whom vaginal birth would pose a substantial risk of uterine rupture, resulting in the death of the baby. Following a court ruling, a cesarean operation was performed, resulting in delivery of a healthy baby boy. The mother suffered no complications. Following the events, the mother filed a civil lawsuit seeking an award of damages against the hospital. The plaintiff claimed that the forced cesarean violated her substantive constitutional rights and that the procedure that led to entry of the order violated her right to procedural due process. In dismissing her claims, the Court stated that the balance tipped far more strongly in favor of the state because the full-term baby's birth was imminent, and more important, the mother sought to avoid only a particular procedure for giving birth, not to avoid giving birth altogether. Weighing the respective interests against the *Roe* framework, the court held that bearing an unwanted child is a greater intrusion on the mother's constitutional interests than undergoing a cesarean delivery to deliver a child that the mother affirmatively desires. Relying on the trimester framework, the Court determined that because the fetus was in the

third trimester, the state's interest in preserving the life of the fetus was greater than the maternal interest of the right to choose between a vaginal delivery and a bodily intrusion through the cesarean operation.

In the *Matter of Angela C.*, the appellate court for the District of Columbia came to a different result, supporting patient autonomy [12]. The case arose in 1987, when a pregnant woman at 26 weeks of gestation was dying of cancer. The hospital obtained a court order to perform a cesarean delivery, over the objections of the mother's family. The mother had given conflicting statements about her desire to have the surgery. The patient's competency was compromised by her advanced disease and sedation at the time the hearing was conducted in the hospital. During the hearing, expert testimony established that the fetus's chance of survival was declining daily. The judge ordered the surgery to proceed. The child was delivered but died 3 hours after birth. The mother died 2 days later.

The case was subsequently heard on appeal by the Court of Appeals for the District of Columbia. The court noted that at the time the case was heard, the decision was not capable of altering the events but proceeded with rendering a decision because of the potential that the situation was "capable of repetition." The appellate court determined that issues such as the patient's competency, informed consent, and substituted judgment were not adequately considered by the trial judge at the time the cesarean delivery was ordered. The court determined that the cesarean should not have been performed, holding that the highest value must be placed on the right to bodily integrity. Specifically in regard to the weighing of the maternal-fetal interests regarding a court-ordered cesarean delivery, the court stated that "... a fetus cannot have rights in this respect superior to those of a person who has already been born." In the aftermath of that decision, the survivors of Angela C. filed a lawsuit against the hospital on behalf of her estate. The hospital settled the case for an undisclosed amount and promised to enact policies protecting pregnant women's rights.

Similarly, in a case that occurred in Chicago in 1993, Cook County officials sought a court order to compel a 22-year-old woman to undergo a cesarean delivery because of placental insufficiency [13]. The mother was a born-again Pentecostal Christian and did not agree to a cesarean delivery for religious

reasons. The parties to the case agreed to certain facts, including that

1. The fetus was 36½ weeks and capable of living outside the womb without the assistance of medical technology.
2. Because of medical complications, the chances of the unborn child surviving natural childbirth were close to zero.
3. If the child were able somehow to survive childbirth, he would be severely impaired.

The Illinois Appellate Court upheld a lower court ruling that the patient could not be forced to submit to a cesarean delivery. The court recognized that the fetus has the legal right to begin life with a sound mind and body, assertable against third parties after it has been born alive. The court stated that such right, for unintentional infliction of prenatal injuries, is not assertable against its mother, however. Thus the court reasoned that although third parties might have a legal obligation toward a fetus, a woman is under no duty to guarantee the mental and physical health of her child at birth, and thus she cannot be compelled to do or not do anything merely for the benefit of her unborn child [13].

Similarly, there are contradictory outcomes in cases in which maternal behavior during pregnancy places the fetus at risk. For instance, the Supreme Court of the United States refused to hear a mother's appeal, thus keeping in place her murder conviction pursuant to a state statute for killing her child through the use of cocaine while pregnant [14]. The Supreme Court also refused to consider cases on South Carolina's definition of a fetus as a person for the purposes of criminal prosecution in nonabortion cases. By not hearing the cases described, the Supreme Court in effect upheld the state's interest in the life of the fetus in nonabortion cases. In its denial to hear the case regarding the definition of a fetus as a person, the Court stated, "... we do not think any fundamental right of [the patient's] – or any right at all, for that matter – is implicated under the present scenario." The Court noted that the use of crack cocaine is not a protected fundamental right; therefore, it saw no reason to attach an additional criminal penalty to an already illegal act because of its effect on the fetus [15]. In *Ferguson v. City of Charleston*, however, the Supreme Court was asked to consider the specific

issue of whether a fetus has a right to be protected against prenatal injury resulting from the mother's conduct, specifically illicit drug use [16]. In *Ferguson*, a hospital had instituted a policy of testing the urine of pregnant women for cocaine use, with the primary goal of referring the patient to substance abuse treatment. Under certain circumstances, test results were provided to law enforcement officials. The Supreme Court ruled that testing urine samples from pregnant patients for cocaine and reporting positive results to the police constituted a "search" within the meaning of the Fourth Amendment. The court rejected the view that the searches were justified by a public policy "special need," that need being to coerce pregnant women to participate in substance abuse treatment. The Supreme Court concluded that although the ultimate goal of the program might well have been to get the women in question into substance abuse treatment and off drugs, the immediate objective of the searches was to generate evidence for law enforcement purposes to reach that goal.

The legal issues present in the conflict between maternal rights and fetal rights clearly have not been fully resolved. Advances in medical technology are most likely only to increase this conflict. The American College of Obstetricians and Gynecology (ACOG) Ethics Committee has reviewed maternal-fetal conflict and charges the responsible clinician to be certain that the mother (as a patient) has a realistic understanding of any proposed procedures and its potential benefits and risks [17]. There is a further requirement to explore the potential unspoken fears, concerns, or outside pressures on the woman to correct errors or assumptions that have led her to refuse treatment. In this sense, the ACOG Committee emphasizes the need for medical caregivers to be sure that all elements of autonomy are present. Specifically, they must ensure that the patient can correlate, consider, communicate, and evaluate the quality-of-life differences, in the absence of undue outside interference.

The interaction between patient and physician has multiple dimensions, and these affect the framework from which the physician or caregiver presents the information as well as the ability of the patient to choose rationally rather than from intuition [18]. Caregivers must recognize these issues. Recruiting a second opinion for another point of view is often helpful. Although the mother's decision holds

primacy, the physician has the obligation to clarify or correct erroneous or eccentric views. Informed consent does not necessarily imply that the ultimate choice that the patient reaches is the one that is viewed as either "good" or "reasonable" by the physician [19]. Appeal to the courts for a court order, which seems on the surface to be a "fair" resolution to a difficult situation, usually provides a vehicle for approval of the physician's choice [20]. This is not necessarily the most ethical or legally acceptable outcome. The use of the legal system to force maternal compliance is discouraged by ACOG, except in compelling circumstances.

### SURGERY IN PREGNANCY

Most surgical conditions that occur in the non-pregnant patient also occur in pregnancy. Pregnant women suffer from acute abdominal conditions, suffer trauma, and develop neoplastic diseases. Among the difficulties that confront the practitioner in the clinical evaluation of gravid patients is that pregnancy induces a variety of mechanical, hormonal, and chemical alterations that can confuse and mislead even the most experienced physician. Most often delay occurs, however, because when faced with a pregnant patient experiencing nonspecific pain, some practitioners could have a misconception about the risk of injury to the fetus that clinical investigation or surgical intervention poses, and thus are reluctant to pursue the standard course of evaluation [21].

It is important to put these risks into perspective. Since the early 1990s, the number of live births in the United States has ranged from 3.88 to 4.12 million per year [22]. Appendicitis and cholelithiasis are the two most common surgical problems during pregnancy. Appendicitis occurs in 0.05% to 0.1% of pregnant women, but the rate of occurrence is the same when pregnant women are compared with nonpregnant women and is roughly equal [23]. The incidence is approximately the same in all three trimesters. Although pregnant women are not any more likely to suffer from appendicitis than the non-gravid, actual rupture of the appendix occurs two to three times more frequently during pregnancy, largely from atypical presentation and delays in intervention [24]. Furthermore, although the maternal mortality rate is quite low, of all surgical prob-

lems during pregnancy, appendicitis causes the most fetal loss [25].

After appendectomy, cholecystectomy is the most common nonobstetric surgery performed in pregnant women. Cholecystectomy is performed between three and eight times per 10,000 pregnancies, and the incidence of symptomatic gallstones in women is approximately twice that in men [26, 28]. The incidence and prevalence of cholelithiasis vary greatly among geographic regions and ethnic groups [27, 28]. In the United States, cholesterol stones account for 70% to 80% of the 475,000 cholecystectomies performed per year for cholelithiasis [29].

Malignancies, although the occurrence is quite uncommon, are nonetheless an important consideration. The incidence of cancer in pregnant women ranges from 0.07% to 0.1%, although benign neoplasms are also seen. Within malignancies, those most often seen in pregnancy include cervical, breast, melanoma, lymphoma, and leukemia. As the trend for women becoming pregnant in later reproductive years continues, it is likely that the incidence of cancer complicating pregnancy will increase [30].

Legal liability often arises in these cases because of the difficulty that exists in sorting out the clinical presentation of a pathologic state from the normal physiologic changes of pregnancy. For example, nausea and vomiting during the first trimester might be attributed to hyperemesis gravidarum and not be recognized as symptoms of cholecystitis or appendicitis. A bowel obstruction might not be recognized in a timely fashion because constipation is deemed either a physiologic event associated with normal pregnancy or the problem is viewed as mechanical compression by the gravid uterus. Delay in diagnosis also occurs because of the perceived risks associated with clinical work-up or treatment. Delays in the diagnosis and treatment of acute abdominal pathology or delayed diagnosis of malignancy leads ultimately to an increased number of unfavorable outcomes and legal difficulties.

Once a diagnosis of a nonobstetric condition is made, a physician has the responsibility to exercise reasonable care in recommending a surgical procedure, meaning that there must be a reasonable basis in the history, physical, or laboratory findings to warrant the recommendation. Complications arising from nonobstetric surgery are relatively uncommon, occurring in only 1% to 2% of pregnancies [31]. As in all medical considerations, the

recommendation of surgery necessitates a risk-benefit analysis.

For a surgical problem that arises during pregnancy, the urgency of surgical treatment must be balanced against the risk that such treatment poses to the mother and the fetus. The decision to recommend a procedure is complicated by a pregnancy, and the timing of surgical intervention becomes an important consideration. Any procedure that is considered urgent in a nonpregnant woman (e.g., appendectomy) should be carried out in the usual timely manner in a pregnant woman, because the risks to both mother and fetus outweigh the risks of miscarriage and preterm labor [25]. Timing becomes much more of a consideration in weighing the decision in the less acute or "semi-elective" and elective procedures, however. During the first trimester, surgical procedures are associated with a miscarriage rate of 12%; during the second trimester, this rate falls to 0% to 5.6%. The incidence of preterm labor with surgical procedures is 5% in the second trimester but rises to 30% to 40% in the third trimester [32]. Consequently, most often less acute procedures are generally best performed during the second trimester, whereas elective procedures are usually delayed until 6 weeks after delivery.

Whenever surgery is performed, there is a potential for injury to viscera or other structures. In pregnancy, this risk is increased because of limited exposure, since the uterus occupies much of the intraabdominal space in the second and third trimesters. Contributing risk factors for injury are obesity, distortion of the anatomy by the pathologic process or adhesions from previous surgery, inadequate anesthesia, haste during surgical emergencies, inexperience of the surgeon, and failure to follow sound surgical technique. Similar to any type of medical injury, prevention or recognition and prompt treatment of the problem at the time of the original surgery are best. Prevention by careful case selection and close attention to detail avoids most surgical misadventures, thereby reducing immediate perioperative and postoperative morbidity, long-term sequelae, and eventual medicolegal consequences.

The rare occurrence of a serious surgical complication leads to the potential argument of preventability had appropriate surgical technique been followed. A surgical complication does not automatically translate into legal liability, however. The

patient must prove that the complication occurred because of the failure of reasonable surgical care. Such proof usually focuses on the failure of the surgeon to take some recognized precautionary measure – a measure intended to minimize the risk of the complication.

Finally, when surgical complication occurs that is a known risk of the procedure, liability can arise for any *additional injury* that results from the failure to recognize and treat the complication in a timely manner. During abdominal surgery, iatrogenic injuries commonly involve the gastrointestinal and urinary tracts. Vascular injuries are uncommon, and even less common are neurologic injuries. Most iatrogenic lesions result from the inadvertent laceration, crushing, ligation, or transection of various structures, or by thermal injury from electrosurgical units [33]. Because the surgeon's duty does not end at the threshold of the operating suite, he/she must remain vigilant during the postoperative period to detect and promptly treat complications.

The surgeon's attention to detail needs to begin in the operating suite and before closing the surgical wound is closed. Initially, a careful inspection of the surgical field with attention to possible persisting bleeding or injury to structures adjacent to the primary surgical site is a requirement. Thereafter, close observation continues during the postoperative period, with appropriate examinations, tests, and instructions to the nursing and house staffs, all of which are documented. If an injury occurs despite careful attention to detail, the risk of complications is significantly lessened by its immediate recognition and repair.

The surgeon has a duty to instruct the patient appropriately on the necessary safeguards to avoid postoperative complications after discharge. From a medicolegal standpoint, failure to anticipate postoperative complications and provide appropriate instruction to a patient who subsequently develops complications will most likely result in legal liability. The instructions should be provided to the patient in writing, not only to promote compliance but also to document the content of the instruction provided. At the time of discharge, the record should document that written instructions were provided to the patient. More important than the documentation, however, is the *process* during which the patient was educated about potential signs and symptoms of postoperative complications.

Many surgeons choose to have a resident, physician's assistant, or even a member of the nursing staff provide ongoing instructions. Such delegation might not insulate the physician from ultimate responsibility for improperly given instructions, however. The person providing the instructions can be considered an agent of the physician, or the responsibility of providing instruction could be deemed a "non-delegatable" duty of the operating surgeon. Either conclusion pulls the operating surgeon back into the chain of culpability.

The obstetrician should consider the following:

- Surgical treatment for pregnant woman is acceptably safe if appropriate attention is given to certain key perioperative considerations – namely, fetal monitoring, radiologic investigation, anesthesia, and the timing of the operation.
  - Liability rarely attaches when the surgical recommendation requires the weighing of competing patient interests, as long as the risks and benefits were appropriately considered. This weighing of risks and benefits, however, requires knowledge of the potential complications of the procedure as well as the reasonable alternatives, including no surgery at all.
  - Gestational age and fetal well-being play a pivotal role in all surgical decision making for a pregnant patient. Preoperative ultrasonography can also approximate gestational age when an accurate history cannot be obtained. Fetal monitoring, including measuring uterine contractions with a tocodynamometer, fetal HR with a Doppler transducer, and fetal movement and tone with ultrasonography, provides potential indicators of fetal health that can be used to evaluate fetal condition.
  - An ancillary component of an appropriate surgical recommendation is informed consent. The patient is entitled to make an informed decision whether to undergo surgery. It is during this process that the obstetrician must be cognizant of the divergent interests between mother and fetus that might be presented by the particular clinical situation.
  - Given the potential risks of whichever course of treatment is taken, the final management decision is ultimately best defended by a note in the medical record documenting the decision-making process.
- If an inadvertent surgical injury occurs, the surgeon must ensure a satisfactory repair. If the damaged viscus or structure is outside the area of expertise of the obstetrician, it is advisable to consult a surgeon with more experience immediately, or one from another specialty for advice and help on how best to manage the problem.
  - In all cases of a perioperative injury, the event must be reported in the operative note, including the mechanism and extent of injury and a description of the repair. Soon after the surgery, the patient and, when appropriate, her family must be informed of the injury. Attempts to conceal an injury are unethical and only lead to more questions, anger, and potential litigation. When appropriate, the surgeon can stress complicating factors that led to the injury and the expected good outcome from the repair. It is always best to begin with an open and frank approach to the patient and seek appropriate consultation, while fully documenting all events.

### INSTRUMENTAL DELIVERY

Instrumentally assisted delivery by either forceps or vacuum extractor remains controversial [35]. Depending on the preference of local practitioners, as few as one delivery in five or ten is now accomplished by forceps or the vacuum extractor. After a considerable period of critical review, there has been a recent resurgence of interest in assisted delivery [34,35,36]. Current obstetric literature and legal case reports reveal that obstetric forceps and the vacuum extractor are coming back into the mainstream of obstetric practice. Instrumental delivery is being considered more frequently owing to the increasing perception that such deliveries are largely a safe and efficacious means of overcoming dystocia in the second stage of labor.

With the use of antibiotics to combat postoperative infection after cesarean procedures and oxytocin to augment the forces of weak labor, members of the obstetric community began to call into question both the safety and the necessity of certain traditional obstetric procedures, especially midforceps operations. By the 1960s and 1970s, there were many articles published in the obstetric literature that condemned midforceps procedures because of the reportedly high incidence of infant morbidity

and mortality. In a survey article entitled "Forceps" published in 1983 in *Clinics in Perinatology*, Varner summarized critical observations concerning instrumental delivery [33]. In response to the authors who condemned obstetric forceps out of hand, a series of articles followed from opposing writers who attempted to place a more benign cast on the adverse outcomes associated with some forceps deliveries. These authors argued that when instrumental deliveries were performed by skilled operators who adhered to rigid protocol, significant fetal injuries were rare. Furthermore, it was suggested that certain types of birth injury were essentially unavoidable, even when an elective cesarean delivery procedure was performed; that is, some instances of perinatal asphyxia or neurologic injury occurred in utero, even before hospital admission, and thus could not be ascribed simply to the delivery method. Despite these rebuttal arguments, midforceps operations declined rapidly in popularity in favor of cesarean delivery, a technically less demanding procedure than a midpelvic forceps operation. Although the total number of midforceps procedures actually performed in this country rapidly declined, the procedure never completely disappeared. By 1981, however, it was uncommon, and in some centers rare, for an obstetrician to perform an elective true midpelvic forceps operation [35].

More recently, obstetricians have come under scrutiny and criticism with respect to the rate of cesarean deliveries. Multiple factors have been identified as causing or contributing to this increase. Among the identified influences driving the cesarean epidemic are the following: uncertainty in the profession about appropriate management for certain obstetric problems (e.g., dystocia, breech presentation), physician fear of lawsuits, maternal anxiety over fetal safety, and the physician's economic advantage. An important issue was the failure of physicians to properly consider the significantly increased maternal morbidity associated with cesarean delivery procedures.

In the wake of such criticism, and following a close reexamination of data from the outcome of instrumental deliveries, modern practitioners have seriously reconsidered the option of instrumental operative delivery by either forceps or vacuum extraction as an alternative to at least some cesarean operations. This interest is evidenced by old forceps

textbooks being published as new editions [34,36] and the fact that ACOG, which had not discussed forceps procedures since 1965, found it appropriate to publish a series of updated technical bulletins on these procedures beginning in 1989 [37-39]. The updated technical bulletins clearly advocate the position that midforceps procedures are still accepted practice in modern obstetrics for properly trained practitioners. Furthermore, the authors of recent ACOG Bulletins have modified terminology previously used to classify or code forceps operations. These new coding guidelines established three categories of operations: outlet, low, and midforceps. Rotational procedures on the pelvic floor (up to 45°) are now defined as *outlet procedures*, and a new category of *low forceps* procedures was added despite the fact that previous coding recommendations had automatically included *all* rotational and nonoutlet procedures in the (presumably) higher risk category of midpelvic procedures. (See Chapter 17, Instrumental Delivery.)

Some critics of this ACOG Bulletin suggest that these redefinitions are merely an effort to inhibit malpractice lawyers from effectively using the old literature condemning midforceps procedures as a basis for critical attack on modern practitioners. The counterargument is that there are actual differences in risk that are procedure specific – differences lost in the previously applied coding definitions. Regardless, ACOG has clearly affirmed instrumental procedures, including midpelvic forceps and vacuum extraction operations, as legitimate operations in modern obstetric practice.

With this increasing trend toward greater numbers of forceps operations and vacuum extractions, it is reasonable to anticipate that the number of traumatic fetal and maternal injuries associated with these procedures will also increase. Despite advances in electronic fetal monitoring and other methods of intrapartum surveillance, most of the factors that caused or contributed to significant fetal injury in the 1960s and 1970s remain unchanged in modern times. The single and most significant exception is physician skill. In a survey of Canadian and United States hospitals' obstetric residency training programs conducted in 1981, Healy and Laufe reported that "... a resident in training may see as few as three to four midpelvic procedures a year even in major university hospital setting" [40]. Furthermore, there is a significant variance in the

depth and experience on the part of the principal instructor(s) in residency programs. Because inadequate skill, training, and experience are undeniably factors that cause or contribute to significant fetal and maternal injury in this procedure, this inadequacy will probably be the focus of future lawsuits involving instrumental delivery.

The fact that various segments of the obstetric community have again embraced instrumental deliveries in general and forceps procedures in particular does not remove the long-standing stigma and suspicion attached to midpelvic operations with any instrument and forceps rotation procedures at all pelvic levels. Some authorities still condemn these procedures as unreasonably dangerous, yet many competent obstetric surgeons testify as to their continued clinical necessity. Midforceps operations with appropriate indications cannot be judged as malpractice per se. Nonetheless, such procedures almost certainly will be subjected to critical scrutiny whenever a mother or newborn is seriously injured by instrumental trauma. Vacuum extractor operations, which have gained grudging but increasing acceptance with American obstetricians, probably will continue to be evaluated and critically analyzed for evidence of as-yet unconfirmed long-term neurologic and developmental complications [35,36].

Obstetricians should consider the following:

- The indications for operative vaginal delivery are either maternal or fetal. The principal maternal indications include either an arrested descent, prolongation of the second stage of labor, or shortening of the second stage for maternal benefit. The principal fetal indication is the suspicion of fetal compromise (immediate or potential).
- The most significant and pivotal aspect of using either a forceps or vacuum delivery occurs during clinical judgment making, when the obstetrician is determining if a trial of instrumental delivery should be undertaken, and if so, the plan for its implementation. The physician's failure to gather clinical information properly, assess its significance, and judge the feasibility and safety of implementation is the predicate to serious maternal/fetal injury or death. This critical turning point is also the central focus of most lawsuits in which the physician is found liable for negligent conduct.
- The importance of clear, precise, and detailed documentation of an instrumental operative procedure is most keenly experienced when the physician must explain and justify his/her actions as the defendant in a malpractice suit. This documentation should include but is not limited to a precise and detailed description/discussion of
  - Indication(s) for the procedure
  - Anesthesia used
  - Personnel present
  - Instrument(s) used and in which order
  - Course of labor, before the application of the instrument
  - Application of the instrument
  - Station, position, and deflection of the fetal head at commencement of the operation
  - Amount of effort exerted by the operator: quantifying the force and duration of traction (with vacuum extractors this should include the amount of vacuum force applied and duration of use) and the progress of the fetal head after traction efforts.
  - Condition of the infant at delivery and all steps taken in neonatal resuscitation
- All critical comments detailing actions taken and conditions observed during the labor/delivery should have dated and timed entries. The well-executed operative report should accurately reflect what the operating physician did and why. The documentation should not be self-serving; however, the facts should be detailed to establish the decision-making process that justified performing the instrumental delivery.
- The weakness of current obstetric residency training programs, which have arguably failed to provide adequate training in instrumental delivery techniques to many young obstetricians, could continue to be the Achilles heel of a new generation of practitioners who choose to practice vaginal operative obstetrics. Academic institutions must identify and develop methods to train obstetric residents in the art of instrumental delivery. (See Chapter 25, Education and Certification).



## CESAREAN DELIVERY

Cesarean delivery has been a major tool to assist the obstetrician in improving pregnancy outcome. In modern obstetrics, with the development of regional anesthesia and improved techniques, cesareans have been remarkably safe, particularly in nonemergency circumstances. Consequently the rate of cesarean delivery has dramatically increased in the last 20 years. The factors contributing to this increase include the increasing number of elective repeat cesarean deliveries and the observation that prior cesarean delivery is a major risk for placental previa and abnormal placenta adherence. Other important reasons for cesarean delivery include poor progress in labor, perceived fetal jeopardy, and suspected fetal macrosomia. There is, however, wide and inconsistent variation in identifying criteria for performing a cesarean. This is due in part to cesarean deliveries that occur during the course of delivery often arise from data that has limited predictive value. With regard to cesarean deliveries in which the decision is made before labor, the decision is based upon perceived risk factors that often are not predictive of outcome in any one particular case.

Recently, another controversy has surrounded cesarean delivery. This controversy can be broken down in two issues: 1) whether a woman having no medical indications for a cesarean should nonetheless be given the option of cesarean as an alternative treatment to vaginal delivery during the informed consent discussion of the birth plan, (patient choice or elective cesarean delivery) and 2) the physician's responsibility to support a patient's choice of elective surgery as the mode of birth for a healthy pregnancy. ACOG's Opinion 289 states that if the physician believes that performing a cesarean delivery ". . . promotes the overall welfare of the woman and her fetus more than vaginal birth, he or she is ethically justified in performing a cesarean delivery" [41]. This opinion has been construed by some to support a patient's choice to have a cesarean even though vaginal delivery presents no increased risk.

The movement toward elective cesareans is based on the belief that maternal morbidity might be improved by surgical delivery instead of a spontaneous or instrumentally assisted vaginal delivery. It is argued that normal vaginal delivery damages the pelvic floor and genital tract and in contrast,

that instrumental vaginal delivery is associated with slower recovery. In contrast, a long-term benefit of cesarean delivery is protection of the maternal pelvic floor from parturition-related injury [42,43]. In addition, there is evidence to suggest that vaginal delivery is an important risk factor for the increasing stress urinary incontinence and fecal incontinence in the aging female population [44].

Proponents of "cesarean on demand" also argue that the increased resort to cesarean delivery will reduce the fetal morbidity associated with vaginal delivery. The claim is that intrapartum events account for about 10% of all babies with cerebral palsy, and although elective cesareans cannot guarantee a normal baby, it does avert the birth trauma caused by intrapartum events.

Opponents meanwhile argue that cesareans, despite being relatively safe procedures are still a major abdominal operation that is not without complications and consequences. Maternal risks include hemorrhage, infection, ileus, wound-healing problems, and potentially dangerous complications such as pulmonary embolism among others. Estimated blood loss in vaginal delivery is believed to be 500 ml, whereas estimated blood loss in a cesarean postpartum is 1000 ml [45]. In addition, they cite the prevalence of hysterectomy because of hemorrhage after a cesarean as ten times that after vaginal delivery. Further, the risk of maternal death is increased up to 16-fold [46].

Adversaries of "cesarean on demand" also argue that cesarean sections can compromise future obstetric performance [46,47]; a primary cesarean usually means repeat cesareans in the future, which increases associated risks of the procedure [48], and furthermore, with each cesarean the incidence of placenta previa and placenta accreta increases [49].

Although it is true that the failure to discuss risks of proceeding with vaginal delivery and offering cesareans as an alternative have been the source of medical malpractice litigation, this has not been in the context of a normal healthy pregnancy. In fact, courts have been clear in not articulating a standard that can set a precedent for requiring cesarean on demand. Consider the case of *Schreiber v. Physicians Ins.* [50]. In *Schreiber*, the patient was a gravida 3 para 2, and both prior deliveries were by cesarean. The indication for performing the cesarean delivery in her first pregnancy was insufficient progress

during a 17-hour labor. The indication for performing a cesarean in the second delivery was that, at the time, it was standard procedure to perform an elective repeat operation because of the patient's prior cesarean. When the plaintiff became pregnant with her third baby, vaginal trials after cesarean delivery (VBAC) were becoming more accepted in the medical community. The obstetrician discussed the alternative modes of delivery and the risks of each procedure. The patient elected VBAC. The obstetrician testified at trial that the patient was advised that her vaginal delivery would be treated like any other labor, and that a cesarean would be performed if medically indicated.

At term, she began labor and was admitted to the hospital shortly after 04:00. The defendant first evaluated her at about 08:00, and at that time the plaintiff told him that she had changed her mind and wanted a cesarean. The obstetrician did not grant her request. At about 08:30, the doctor performed an amniotomy, after which the plaintiff began to experience severe upper abdominal pain unlike any she had felt in her prior deliveries, and that she did not associate with her contractions. Analgesia was provided, with limited success.

At 13:00 the defendant concluded that the plaintiff was making insufficient progress in labor; he failed to discern the cause of her pain but concluded from his examination that there was not any impending danger to the mother or child. He recognized that he could not completely rule out uterine rupture or placental separation, however. Because of her intolerance to the pain, the patient again requested a cesarean. The defendant responded to the effect that if he "...gave a section to every woman who was in labor who wanted one, they'd all do it." He testified that although he knew that his patient was experiencing abdominal discomfort and irregular contractions, and he understood that she would have chosen a cesarean if given the choice, he did not grant her request because he felt that a cesarean was not medically indicated at the time. About 14:00 the patient was in a hypotonic uterine contraction pattern. Despite the recent request for a cesarean, the doctor encouraged to her to continue with labor. Pitocin was administered in increasing amounts until 15:40, when the fetal heartbeat dropped. Shortly after 16:00, an emergency cesarean was performed. The baby was injured and was eventually diagnosed with a spas-

tic quadriplegia. Given the medical evidence, it was stipulated at trial that had the baby been born by cesarean prior to 15:29, she would have been born healthy and normal.

The court concluded that based on the specific facts of this case, the defendant violated his duty to obtain informed consent in refusing to comply with the patient's request for a cesarean. In deciding the case, however, the court was clear to articulate: "We do not believe our decision today will lead us... toward the perceived dangers of a treatment-on-demand system." Instead, what was important to the court was the fact that although the VBAC trial was accepted, cesarean was an alternative that was appropriately recommended when the physician discussed the original delivery plan. The fact that she chose one alternative but then reconsidered her decision because of her perception of events is not rising to the level of "cesarean on demand." As the court saw it: "This is a case involving a patient who [had] been given a free choice by her doctor between two medically viable treatment options prior to labor, initially chooses one, but then changes her mind in the face of an unexpected change of circumstances that is inconsistent with or outside the patient's previous experience in similar circumstances." It is noted in the court's opinion that the patient was requesting a medically viable alternative that had been offered to her by this doctor earlier, and the physician had the capability to follow the patient's wishes; thus, in essence, it was concluded that he ignored his patient and substituted his own choice for hers.

The important factor to the court is not that a patient has the right to demand certain care, but that when recognized alternative exists, it is the patient's right to determine the path taken. It cannot be overemphasized that *the law holds the right to informed consent/refusal, particularly for pregnant women, as paramount*. Courts are critical of any course that abrogates the patient's right of self-determination. Furthermore, it is a misconception that informed consent cases arise only out of the failure to recommend a medical or surgical procedure. Specific to labor and delivery, issues of informed consent arise not merely in the context of failing to advise a patient of the option for a cesarean delivery either before the onset of labor due to the existence of risk factors, or during the course of labor due to some change or manifestation of an acute risk.

Conversely, lawsuits have stemmed from instances in which an elective cesarean was performed, and the patient alleged that the baby should have been delivered vaginally. In one reported case, *Meador v. Stahler and Gheridian* [51], a woman successfully sued her obstetricians for not providing her the option of vaginal delivery. The lawsuit alleged that the plaintiff was pregnant with her fourth child. Her most recent past pregnancy had resulted in a cesarean, and she wanted to avoid that mode of delivery for her current pregnancy. She sought and found an obstetrician who agreed to assist in vaginal delivery. Two weeks before her due date, however, the physician advised her that he was going on vacation and wanted to schedule a cesarean. When she objected, the defendant told her that labor would be risky to her and the baby. It was also alleged by the plaintiff that the obstetrician had compared her uterus to a "hydrogen bomb." The defendant denied ever mentioning a "hydrogen bomb" but agreed that he was concerned that if she did not go into labor soon, the baby being might be endangered by going past the due date. Between her last office visit with the obstetrician and her date of confinement, the plaintiff unsuccessfully sought to find a physician who was willing to take over her care and deliver her baby vaginally. When she was admitted to the hospital for delivery, she claimed that she asked the covering physician to deliver her vaginally but that that request was denied.

Although the baby was delivered healthy, the mother developed severe complications from the cesarean procedure. The jury found in favor of the plaintiff and awarded her \$400,000. Juries are very sensitive to issues surrounding patient decision making. In this case, the jury believed that the patient and the doctor had agreed on a mode of delivery, and that there were no significant events that occurred that were unforeseeable to the physician at the time he agreed to that mode of delivery. Thus, there was no justifiable reason for the physician to unilaterally change the plan of care, except perhaps inconvenience to the physician.

As the cases of *Schreiber* and *Meador* illustrate, it is of utmost importance, particularly in determining the mode of delivery, for the patient and the physician to have been on the same page, particularly in the event of an adverse outcome. In most cases, physicians and their patients agree on the treatment plan. In fact, it has been noted that patients agreed

with physicians' recommendations 98% of the time [52]. That study is hollow if the agreed-upon treatment plan was not obtained with proper informed consent. (See Appendix 1 for a more detailed discussion of informed consent.) The discussion should include alternatives and the risks of each alternative and should be undertaken in an unbiased and uncoerced fashion. The process of informed consent that was undertaken in any particular case is most significant in defending cases in which informed consent is the issue. In cases in which the mode of delivery elected and undertaken is a central issue, informed consent properly obtained establishes that the patient accepted the risk of the course that was followed. The jury will disregard the patient's consent if it was obtained through coercion, improper influence, or if it determines that the evidence supports that the patient was uninformed. This was the issue that was presented in the *Schreiber* case discussed previously. In *Schreiber*, a defense was raised by the physician that the harm that occurred to the plaintiff was a recognized complication and had been explained to her prior to performing the procedure. The evidence during trial established that after her request for vaginal delivery was denied, the patient agreed to proceed with a cesarean. The physician's defense also relied upon the fact that the patient did not dispute that she was advised of the risks of cesarean delivery, or that her written consent was obtained. The physician's defense that the injury was the result of a complication to which the patient had given informed consent was rejected, however. In characterizing the situation in which the plaintiff found herself, the plaintiff's expert, a highly regarded obstetrician, testified that he thought it was "sad" that the patient had no alternatives when her agreement with her doctor broke down and she was then in a situation where there was no offered alternative. Thus the jury could conclude that that her decision was not the product of her autonomous right to make healthcare choices but instead was made reluctantly, with no alternative available.

The obstetrician should consider the following:

- Primarily, accepted indications for cesarean delivery arise from clinical conditions that are identified prior to the onset of labor or arise due to conditions that arise during labor. In the setting where the conditions are identified before parturition, potential indications include a previous cesarean,

a multiple gestation, or a malpresentation, among others. Perinatally, indications include presumed fetal jeopardy, intrapartum hemorrhage, cord prolapse, and failure to progress, to name a few.

- VBAC trial is still a safe and available method of delivery. It can be argued that the philosophy of “once a cesarean, always a cesarean” deprives the patient of her right to self-determination unless other factors precluding a trial of labor exist. In the event of a VBAC trial, the physician should be able to provide appropriate technical support to monitor labor and to have experienced staff available should a prompt cesarean delivery become necessary. The physician must be immediately available at all times.
- Purely elective cesarean delivery (cesarean on demand) remains controversial. The large percentage of pregnant women prefer vaginal delivery. The physician’s bias, or that of the institution, should not supplant the patient’s right of self-determination.
- Informed consent requires providing adequate information to the patient, ensuring that she understands that information, and that ultimately it is her voluntary decision to choose a course of treatment.
- In the event that a physician must employ either negotiation methods or respectful persuasion, the obstetrician cannot substitute his or her judgment for that of the patient. Undue influence or coercion merely creates disharmony and is a potential source of discontent should there be a bad outcome.
- The prenatal period is not static and therefore the discussion of strategies and risks cannot be either. On-going dialogue can serve to continually educate the patient as well as to identify areas of patient concern. Any concerns should be addressed in a timely manner; these concerns can also provide the physician with important information should an emergency arise.
- In the event that elective cesarean delivery is an option, the obstetrician should describe the risks/benefits associated with the operation. Furthermore, to avoid patient misconception, it should be emphasized that a cesarean delivery

does not necessarily ensure a healthy baby. In the event that VBAC is considered, or in instances of breech presentation, the physician should outline both the documented risks and benefits for the available delivery options and explain any bias or beliefs that he/she might have about certain obstetric approaches.

- Documentation is very important, with particular attention to instances when the patient chooses a riskier course or refuses certain care.

### UROLOGIC COMPLICATIONS

Urologic injuries occurring during the course of pregnancy or more commonly during surgical or instrumental delivery, can result in serious and potentially life-threatening complications to both the mother and the unborn infant. Anatomically, the bladder is situated immediately adjacent to the uterus, whereas the ureters are located close to the female reproductive organs throughout their course from the pelvic brim to the bladder. Both cesarean delivery and abdominal hysterectomy necessarily involve procedures and instrumentation that require the obstetrician to work close to the bladder and ureters. Because the physiologic changes of pregnancy distort the anatomy, there can be considerable difficulty in the accurate identification of these structures. (See Chapter 19, Urologic Complications.)

Most urologic injuries from vaginal or abdominal surgical procedures on pregnant women involve some form of direct mechanical injury or compromise to the bladder or ureters. Although surgical trauma to the lower urinary tract (urethra) is well documented, it is far less common and is generally associated with a favorable prognosis. Predictably, the overall incidence of urinary tract injuries as the result of forceps procedures has significantly decreased with the decline in popularity of the more difficult midpelvic and rotational forceps operations during the past three decades. Pelvic lacerations resulting from forceps rotations remain an uncommon but well-documented potential source of trauma to the bladder, ureters, and lower urinary tract, however.

Vacuum extraction, a relatively newer procedure in American obstetric practice, also can be associated with traumatic injury to the lower urinary

tract. This is particularly true when the obstetrician fails to remove redundant maternal tissues from beneath the vacuum extractor cup as the instrument is applied to the fetal head. It should be noted, however, that both the incidence and severity of urinary tract injuries are consistently reported to be substantially less with vacuum extraction when compared with forceps rotation and extraction procedures.

Although the statistical incidence of seriously debilitating or lethal complications is rather small, urologic injuries remain one of the most common reasons for medical negligence actions being brought against obstetricians and gynecologic surgeons. A review of available data from medical publications, legal publications, and court files indicates that the types of injuries that have led to malpractice claims include

- Renal damage (compromise or failure) resulting from unrecognized ureteral injury or obstruction, with possible complications of uremia, shock, or maternal death
- Injuries to the urethra and adjacent tissues resulting in incontinence
- Fistula formation involving the bladder, ureters, or lower urinary tract
- Severe infection (e.g., sepsis, septic shock, or death)
- Bladder injuries (resulting in compromised elimination function or incontinence)

More often than not, the allegations of negligence that are brought against the defendant physician critique the technique that was employed during delivery. The difficulty for the plaintiff in establishing liability is the ability to prove substandard care. Certainly it must be acknowledged that urinary tract injuries can be caused by negligent conduct of the physician in performing the surgery; however, injury to one or more of these structures is a well-described risk and can occur even in the hands of the most skilled provider. Often in such cases it is impossible to point to any particular aspect of care to distinguish the injury caused by negligence from the injury caused by known morbidity. As the burden of proof lies with the plaintiff to establish breach of

duty, the plaintiff's experts can face a difficult challenge in any given case. In the nonemergent case, that difficulty is amplified if there is evidence that the patient was aware of the risks of surgery. Consequently, plaintiffs employ a circular argument that ultimately sounds like *res ipsa loquitur* ("the thing speaks for itself"). Thus, despite the injury's being a known risk, had the obstetrician been more diligent in identifying the specific structures, the injury would not have occurred (for a more detailed discussion of *res ipsa loquitur*, please see Appendix I). To assist in proving their case, the plaintiff will look for errors in dictation, untimely dictation, or misstatements of technique.

It is also common that in addition to surgical technique, plaintiffs will allege untimely diagnosis. This is especially true if the complication is not identified at the time of surgery or appropriate consultation was not obtained in a timely fashion, thereby delaying treatment and resulting in a detrimental impact on outcome. An example of the twofold approach is seen in the case of *Seats v. Lowery* [53]. In *Seats*, the plaintiff had undergone a hysterectomy and salpingoophorectomy. During the course of this operation, the surgeon inadvertently ligated a ureter. As a result, it was necessary for the plaintiff to undergo several subsequent medical and surgical procedures and to incur considerable medical expenses for repair of this injury.

At trial, in responding to the questions from the plaintiff's counsel, the plaintiff's expert testified as follows:

Q: Does the standard of care – tell me whether or not in your opinion the standard of care requires the surgeon to anticipate that the ureter itself may be adhered to other structures or itself may not be in its normal location?

A: Absolutely. Flat out yes.

Q: Tell me whether or not –

A: Let me dispel something. A surgeon is a little bit more than a mechanic. He's also a doctor. So he's got to think a little. . . . We're not considered right bright in the staff, you know. You still got to think a little.

Q: Tell me whether or not the standard of care requires the surgeon at the beginning of the case, at the beginning of the surgery to be conscious of the fact that it may become

necessary during the surgery to actually physically identify the ureter?

A: Yes, sir, it does. This operation should never be done by anyone unwilling to do that and anyone who doesn't have the judgment to know when to do that.

Q: Tell me, doctor, whether or not the standard of care requires that the surgeon be aware and conscious of the ureter and its location throughout this entire surgical procedure?

A: Yes. And after his gloves are off and he's standing in the room, he has to think about it then. Is there any way in the world that I could have hurt one of those ureters? He's got to say that to himself. And if there is any suggestion or thought or fleeting thought that that might have occurred, he should get a urologist shoot that scope up there and take two pictures or shoot some dye and look and make absolutely sure that [it] hasn't [been] hurt.

Q: As far as preparation for the case and thinking about having to find the ureter, was there a statement made by the defendant in his deposition?

A: Yeah. He said he doesn't ever look for it, that you don't have to look for it, that you do this procedure this way, and based on doing this procedure this way, this doesn't very often happen but it can happen. Well, isn't that nice?

Q: Does this happen very –

A: If you're careful this does not have to happen at all. It does not at all, ever. And if it does happen, there's something wrong with you when you're doing it. I don't know what it is and don't care what it is, but something's wrong with you. Slop talk like that, lift something up, put a clamp on, cut it out – I don't want any part of it.

The *Seats* case exemplifies that frequently the issue of negligence becomes a battle of the experts. Most often, however, it is the defendant's attention to detail and diligence in identifying complications that affects the outcome. Using the surgical note, which is the defendant's ability to recreate the events during surgery, including the visualization of important anatomy, is an important component to the defense of such cases.

No one can seriously dispute that prompt recognition of any injury is the surgeon's greatest ally in performing a successful repair and avoiding significant and at times potentially lethal complications. The principal danger in delayed diagnosis and treatment in a ureteric injury is the risk of permanent impairment or complete loss of renal function. Ureteral obstruction once surgically relieved is not without complications including urosepsis or fistula formation. In the unusual instance when ureteral compromise is bilateral or the patient has only one functioning kidney to start with, a delayed diagnosis can result in uremia, shock, and even death. Unfortunately, a diagnosis of ureteral injury is usually made postoperatively and establishing that there is an injury may be difficult. Symptoms of fever, flank pain, and abdominal distension occurring within 48 to 72 hours after surgery however, should alert the clinician to possible ureteral injury.

The important principles for practice include the following:

- Meticulous surgical technique and a high index of suspicion are the surgeon's greatest allies in avoiding surgical trauma and making appropriate and timely diagnoses of urologic compromise.
- When surgical trauma to urinary structures is essentially unavoidable, clear and precise documentation within the operative records is essential to the effective defense of any potential malpractice suit.
- The obstetrician must be vigilant in postoperative patient evaluation. Obstetricians should never place themselves or their patients at risk by assuming that they have sufficient knowledge and skill to address the entire range of complex and esoteric demands of urologic surgery, infectious disease, and renal physiology. Timely referral is important in the defense of these matters, both in establishing the existence of any injury and in its repair.
- Given that these cases become a battle of the experts, the defendant's presentation is very important. A timely surgical note, one that is complete and without the appearance of being self-serving, is important in defending a case of urinary tract damage. Even in the routine case, the surgical note should describe the anatomy and discuss

the visualization of important structures when the surgery either involves or is adjacent to the ureters.

### FETAL SURGERY

Innovations in ultrasound technology, fetoscopy, and related procedures have had a dramatic impact on neonatal and child health. The inefficacy and safety are well established. The benefits these techniques have, however, are in establishing a diagnosis. Most are not therapeutic [54]. The efficacy of in-utero therapeutic intervention is much less established. Open fetal surgery, although possible, is accompanied by substantial risks of fetal mortality and often serious maternal morbidity. (See Chapter 20, Fetal Surgery.)

Fetal endoscopic surgery shows promise for selected problems. This procedure offers several distinct advantages when compared with open fetal surgery; however, the endoscopic approach has significant drawbacks including difficulty performing an operation in a tiny working space. Some of the more complex tasks are very difficult with currently available instruments. Percutaneous fetal therapies typically can play an important role in draining space-occupying, fluid-filled structures, such as the pleural space and bladder. Not all intervention has proved beneficial, however. The performance of a number of neurosurgical procedures has been either curtailed or abandoned due to a combination of associated procedure-related deaths and the inability to demonstrate improved outcomes from these interventions.

In-utero surgical treatment procedures have been performed for various fetal afflictions. For spina bifida, in-utero repair has been performed in the attempt to decrease nerve damage and improve outcomes at birth, with uncertain success. In cases of diaphragmatic hernia, procedures have been performed involving surgical repair of the herniated diaphragm or temporary ligation of the fetal trachea. Percutaneous vesicoamniotic fetal shunting, open fetal surgery, and, more recently, endoscopic fetal surgery are possible fetal interventions for lower urinary tract obstruction. In multiple gestations, laser ablation of anastomotic vessels in severe twin-twin transfusion syndrome has been successfully performed when the condition is severe enough to warrant intervention. With continued improvements in

technology, there is optimism that the ability to correct at least some congenital anomalies in utero will have a significant impact on neonatal and child health.

Presently, except for prenatal steroids to prevent hyaline membrane disease, and antiviral treatment for the prevention of perinatal HIV, the standard of care for most congenital anomalies identified during the prenatal period is either abortion or early postnatal intervention. Furthermore, for most prenatal interventions, efficacy has not been proved nor the risks fully defined. Part of the problem is that the natural history of many uncommon conditions has not been sufficiently studied, and in many cases an adequate experimental animal model has not been identified or the available models do not mimic human physiology closely enough to be helpful. Much more basic work must be done before most in-utero surgical procedures exceed their experimental status.

To date, the courts have not fashioned any meaningful or definitive legal criteria to allow physicians to assess when a particular experimental or investigational therapy has reached the status of standardized medical therapy. Essentially, this is a matter determined by the shared or common understanding of practicing physicians. Because this consensus of collective physician opinion accrues informally and over time, it is usually impossible to identify a precise moment in history when a particular treatment or technique achieves the status of standard medical therapy (for a more detailed discussion of this topic, see Appendix I).

During the "twilight zone" that exists between the time that experimental treatment is recognized as having a potential benefit and when it is considered no longer experimental, complex legal issues can arise. Of all the issues, however, most important is the need for informed consent before implementing any fetal procedure that is not generally accepted. Many innovations have not been subjected to prospective clinical trials. Thus, often there are uncertainties such as lack of data, imprecision in diagnosis, and lack of experience on the part of the surgeon. Institutional bias can also prejudice a provider's recommendations. Meanwhile, a patient's ability to weigh the available information objectively can be affected by the clinical situation, particularly when the treatment is considered a "last resort." Ultimately, the best and least biased

information, although critical to a good decision, is difficult to obtain mostly because the technology is new and rapidly evolving [55].

Because of this, is the standard of care for informed consent any different in the clinical setting when experimental treatment is being considered? This issue has been the object of considerable debate, and there is no clear consensus. This issue surrounds whether a physician is required to discuss the experimental nature of the procedure if those risks that are material to the procedure itself have been adequately explained. One legal commentator framed the debate this way:

**The attempt to delineate research and treatment has deep implications for the informed consent requirement. If the distinction [of a procedure as innovative or experimental] is viewed as superfluous, then a uniform standard of disclosure arguably would apply in both contexts. On the contrary, if they remain distinct, then there is reason to believe that the nature of the intervention will dictate different standards, according to the level of protection needed [56].**

Another legal commentator, however, framed the debate differently; advocating for the notion that withholding the experimental nature of the procedure violates the principles of informed consent, he advocated for the idea that some more fundamental legal duty would be violated as well. This commentator stated that "... the individual doctor trying out new techniques is undeniably engaged in medical experimentation. It is unacceptable... to place the burden of this experimentation upon the patient by confining his right of recovery in relation to consent to the tort of negligence" [57].

This debate was addressed in the case of *Adams v. Arthur* [58]. The specific issue was whether the physician's failure to disclose the use of an experimental technique created a cause of action for anything other than lack of informed consent. The reason for the plaintiff's determination to draw a distinction was that the statute of limitations had run on any informed consent claim. The plaintiff therefore was seeking to articulate an alternate theory of recovery, so as not to be barred from bringing the lawsuit. In *Adams*, the patient claimed that the physician should have disclosed that he intended to

use a novel or experimental graft material in her spinal surgery. The physician had informed her that he would use a "synthetic material" from Switzerland as a graft material, that after it was implanted "everything would grow together," and that he had not had any problems in prior surgeries with the material. The plaintiff argued that the act of not advising her of the experimental nature of the "synthetic material" constituted a fraudulent concealment, thus providing an avenue around the statute of limitations. The court refused to frame the issue in anything other than the physician's legal obligation to provide adequate informed consent. It concluded the physician did not make any statements that misrepresented the appropriateness of the product's use in the human spine. In its holding, the court determined that the merits of whether the failure to disclose the experimental nature of the material was determined by the legal principles involved in an informed consent claim and nothing more. Thus the plaintiff was incapable of pursuing her case.

The *Adams* decision has been criticized by some. As one judicial authority commented: "... [the *Adams* decision] creates no distinction between the garden-variety informed consent cases and the situation in which, as in the instant case, a medical provider, without the knowledge or consent of a patient, in effect conducts an experiment on the patient... [M]edical providers are now free to conduct experimental medical procedures upon their patients, without any disclosure whatsoever of the experimental nature of the treatment, and, unless any resulting harm is manifested within the two-year medical malpractice limitations period, without any concern for the consequences..." [59].

As innovative technology continues to develop expeditiously, issues will come more to the forefront of cases that have had an adverse medical outcome after the use of an innovative or experimental procedure. Most jurisdictions are expected to resolve these issues by applying the elements that comprise an informed consent case. The harsh result that some legal commentators have argued can occur by applying only the principles that are used in "garden variety" informed consent cases to cases involving experimental medical procedures will probably be an issue in only a few cases. The same harsh result can occur even in a garden variety case if the



harm remains unknown throughout the applicable period during which a patient can sue for breach of informed consent claim. Other potential claims, such as intentional misrepresentation as in the *Adams* case, require a much higher burden of proof. The cause of action, when considering disclosure in such cases, will rest with informed consent. The primary issue, apart from the risks and available alternatives, is the materiality of advising a patient that the therapy is experimental or novel.

The obstetrician should consider the following:

- The patient has a legal right to receive material information concerning the available therapies, including detailed information about the risks and benefits of therapeutic alternatives to the treatment option that is being recommended.
- When obtaining an informed consent for new and innovative procedures, the physician should be conservative in his or her statements and conduct extensive discussions with the patient. The FDA guidelines for obtaining an informed consent are a reasonable framework for physician use. In addition, providing the patient with literature that discusses and explains the procedure and its potential risks is often an effective means of combating potential allegations of improper or inadequate disclosure.
- Some physicians are reluctant to continue using the word *experimental* in consent forms once a particular procedure has progressed to the level of investigational status, for fear that it might cause reluctance on the part of potential patients and because this label implies that there is no reliable evidence of efficacy.

The prudent course is to indicate candidly within the written consent form that although some investigators are still of the opinion that this therapy remains experimental, the physician provider thinks that the recommended therapy has demonstrable safety and efficacy.

## REFERENCES

1. Coutts MC: Maternal-fetal conflict: Legal and ethical issues. Scope Notes No.14. Washington, DC, National Reference Center for Bioethics Literature, 1990.
2. American Academy of Pediatrics Committee on Bioethics: Fetal therapy: Ethical considerations. *Pediatrics*. 1988 Jun;81(6):898-9.
3. Office of Applied Studies: Results from the 2003 National Survey on Drug Use and Health: National findings (DHHS Publication No. SMA 04-3964, NSDUH Series H-25). Rockville, MD, Substance Abuse and Mental Health Services Administration, 2004.
4. Grella CE: Women in residential drug treatment: Differences by program type and pregnancy. *J Health Care Poor Underserved*. 1999 May;10(2):216-29.
5. *Roe v. Wade*, 410 U.S. 113 (1973).
6. Gallagher J: Prenatal invasion and interventions: What's wrong with fetal rights? *Harvard Women's Law Journal* 10:9, 1987.
7. *McFall v. Shrimp*, 10 Pa. D. & C. Alleghany County Ct. (1978).
8. *Planned Parenthood of Southeastern Pa. v. Casey* 505 U.S. 833 (1992).
9. Blank RH: Maternal-fetal relationship. *J Legal Med* 14:73-92, 1993.
10. *Jefferson v. Griffin Spalding County Hospital*, 274 S.E.2d 457 (Ga. 1981).
11. *Pemberton v. Tallahassee Memorial Regional Hospital* 66 F.Supp. 2d 1247 Fl. 1999).
12. *In Re A.C.* 573 A.2d 1235 (1990).
13. *In Re Brown* 689 N.E.2d (Ill. App. 1997).
14. *McKnight v. South Carolina*, 124 S. Ct. 101 (2003).
15. *Whitner v. State*, 492 S.E.2d 777 (1997), cert denied, 523 U.S. 1145 (1998).
16. *Ferguson v. City of Charleston* 532 U. S. 67 (2001).
17. American College of Obstetricians and Gynecologists: Maternal decision making, ethics and the law. Committee Opinion Number 321, November 2005. Washington, DC, American College of Obstetricians and Gynecologists.
18. Jonsen A, Siegler M, Winslade W: *Clinical Ethics*, ed 3. New York: McGraw-Hill, 1992.
19. Redelmeier DA, Rozin P, Kahneman D: Understanding patient decisions: Cognitive and emotional perspectives. *JAMA* 1993 Jul 7;270(1):72-6.
20. Steinbock B: *Life Before Birth: The Moral and Legal Status of Embryos and Fetuses*. Boston: Oxford University Press, 1992.
21. Barron WM: The pregnant surgical patient: Medical evaluation and management. *Ann Intern Med* 101: 683, 1984.
22. National Vital Statistics Report, Vol. 49. Hyattsville, MD, National Center for Health Statistics, 2001.

23. Andersen B, Nielsen T: Appendicitis in pregnancy: Diagnosis, management and complications. *Acta Obstet Gynecol Scand* 1999 Oct;78(9):758–62.
24. Adams DH, Fine C, Brooks DC: High-resolution real-time ultrasonography: A new tool in the diagnosis of acute appendicitis. *Am J Surg* 1988 Jan;155(1):93–7.
25. Parungo C, Brooks DC: The pregnant surgical patient. ACS Surgery, WebMD, 2006.
26. Hill LM, Johnson CE, Lee RA: Cholecystectomy in pregnancy. *Obstet Gynecol* 1975 Sep;46(3):291–3.
27. Diehl AK, Stern MP, Ostrower VS, Freidman PC: Prevalence of clinical gallbladder disease in Mexican-American, Anglo, and Black women. *South Med J* 1980 Apr;73(4):438–41, 443.
28. Kern F Jr.: Epidemiology and natural history of gallstones. *Semin Liver Dis* 1983 May;3(2):87–96.
29. Trotman BW, Soloway RD: Pigment versus cholesterol cholelithiasis: Clinical and epidemiological aspects. *Am J Dig Dis* 1975 Aug;20(8):735–40.
30. Pavlidis NA: Coexistence of pregnancy and malignancy: *Oncologist* 2002;7(4):279–87.
31. Tarraza H, Moore R: Gynecologic causes of the acute abdomen and the acute abdomen in pregnancy. *Surg Clin North Am* 1997 Dec;77(6):1371–94.
32. Shay DC, Bhavani-Shankar K, 31 S: Laparoscopic surgery during pregnancy. *Anesthesiol Clin North America* 2001 Mar;19(1):57–67.
33. Varner MW: Neuropsychiatric sequelae of midforceps deliveries. *Clin Perinatol* 1983 Jun;10(2):455–60.
34. Dennen, PC: Dennen's Forceps Deliveries, Third Edition. Philadelphia: FA Davis Company, 1989.
35. O'Grady JP: Modern Instrumental Delivery. Baltimore: Williams & Wilkins, 1988.
36. Laufe LE, Berkus MD: Assisted Vaginal Delivery: Obstetrical Forceps and Vacuum Extraction Techniques. New York: McGraw-Hill, 1992.
37. American College of Obstetricians and Gynecologists: Obstetric forceps. Committee Opinion No. 71. Washington, DC: American College of Obstetricians and Gynecologists, 1989.
38. American College of Obstetrics and Gynecologists: Operative vaginal delivery. Technical Bulletin No. 152. Washington, DC: American College of Obstetricians and Gynecologists, 1991.
39. American College of Obstetricians and Gynecologists (ACOG): Operative vaginal delivery. Practice Bulletin No 17, Jun 1, 2000. Washington (DC): American College of Obstetricians and Gynecologists (ACOG).
40. Healy DL, Laufe LE: Survey of obstetrical forceps training in North America in 1981. *Am J Obstet Gynecol* 1985 Jan 1;151(1):54–8.
41. American College of Obstetricians and Gynecologists: Surgery and patient choice. Committee Opinion No. 289. Washington, DC: American College of Obstetricians and Gynecologists, 2003.
42. Glazener CMA, Abdalla M, Stroud P, Naji S, Templeton A, Russell IT: Postnatal maternal morbidity: Extent, causes, prevention and treatment. *Br J Obstet Gynaecol* 1995 Apr;102(4):282–7.
43. Minkoff H, Chervenak FA: Elective primary cesarean delivery. *N Engl J Med* 2003 Mar 6;348(10):946–50.
44. Sultan AH, Stanton SL: Preserving the pelvic floor and perineum during childbirth – elective caesarean section? *Br J Obstet Gynaecol* 1996 Aug;103(8):731–4.
45. Pritchard JA, Baldwin RM, Dickey JC, Wiggins KM: Red blood cell loss and changes in apparent blood volume during and following vaginal delivery, cesarean section, and cesarean section plus total abdominal hysterectomy. *Am J Obstet Gynecol* 1962;84:1271.
46. Hemminki E, Merilainum J: Long-term effects of cesarean sections: Ectopic pregnancies and placental problems. *Am J Obstet Gynecol* 1996 May;174(5):1569–74.
47. Greene R, Gardeil F, Turner MJ: Long-term effects of cesarean sections. *Am J Obstet Gynecol* 1997 Jan;176(1 Pt 1):254–5.
48. Scott JR: Putting elective cesarean into perspective. *Obstet Gynecol* 2002 Jun;99(6):967–8.
49. Clark SL, Koonings PP, Phelan JP: Placenta previa/accreta and prior cesarean section. *Obstet Gynecol* 1985 Jul;66(1):89–92.
50. *Schreiber v. Physicians Ins.*, 579 N.W.2d 730 (1998).
51. The Boston Globe, *Meador v. Stahler and Gheridian*, Wednesday, June 16, 1993.
52. Chervenak FA, McCullough LB: Neglected ethical dimensions of the professional liability crisis. *Am J Obstet Gynecol* 2004 May;190(5):1198–200. Review.
53. *Seats v. Lowery*, 930 S.W. 2d 558 (1996).

54. Kimber C, Spitz, L, Cuschieri A: Current state of antenatal in utero surgical interventions. *Arch Dis Child Fetal Neonatal* Ed 1997 Mar;76(2):F134-9.
55. King N: The line between clinical innovation and human experimentation, 32 *Seton Hall Law Rev* 2003;32(3):571-80.
56. Giesen D: Civil liability of physicians for new methods of treatment and experimentation: A comparative examination. *Med Law Rev* 1995 Spring; 3(1):22-52.
57. Morin K: The standard of disclosure in human subject experimentation, 19 *J. Legal Med* 157 (1998).
58. *Adams v. Arthur*, 969 S.W.2d 598 (1998).
59. *Johnson v. Arthur*, 986 S.W.2d 874 (1999).

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# SPECIAL ISSUES

## Chapter 22 FETAL ASSESSMENT

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*If significant gains are to be made . . . a reliable means of accurately determining reversible 'fetal distress' must be found . . . It is hoped that the use of modern instrumentation methods may aid in the elucidation of clinical fetal distress.*

Edward H.G. Hon (1917–)

Fetal monitoring can be understood as gathering and interpreting information from the fetus about its well-being. This chapter focuses primarily on surveillance during the intrapartum period using electronic fetal heart rate (FHR) monitoring and, to a lesser extent, ancillary techniques of fetal evaluation, including capillary blood sampling, pulse oximetry, and automated electrocardiographic (ECG) analysis of ST waveform changes (STAN<sup>®</sup>, Neoventa Medical AB, Molndal, Sweden) in refining the evaluation of moderately abnormal FHR pattern. The discussion also covers the controversy over the value of fetal monitoring and the pitfalls that interfere with realizing its benefits.

Since its introduction into clinical obstetric care in the early 1970s, electronic FHR monitoring (EFM) has become an almost universally applied technology during labor. By 2004, more than 85% of parturients were so monitored [1]. The introduction of EFM over 30 years ago was accompanied by the anticipation that by the timely detection and intervention in the presence of certain FHR features, EFM would reduce the intrapartum still-birth rate, enhance neonatal condition, diminish the risk of long-term disability, and reduce the rate of cesareans performed because of fetal distress [2]. Although indeed the fetal and neonatal death rates have fallen considerably, the risk and severity of neurologic handicap could be increasing [3,4], and the cesarean delivery rate has risen considerably. Furthermore, both maternal and infant mortality rates appeared to have leveled off and could be rising (according to the *Morbidity and Mortality Weekly Report*). Not all the benefits of EFM envisioned by its early purveyors have been realized, which has sparked considerable controversy.

Reservations about the value of EFM have been fostered by the comparisons to intermittent auscultation (IA) in several randomized controlled trials (RCTs) [5,6,8]. The study designs, the numbers of patients studied, the nomenclatures of FHR

patterns, the diagnostic and management for fetal distress, and the results have varied so widely that they permit almost any conclusion about the benefit or lack thereof of EFM. For example, in one large “randomized” trial, there was no description of the FHR “abnormalities” used to determine fetal distress [9]. Universally lacking from these studies are evaluations of the appropriateness and timeliness of intervention and, most important, the determination of fetal well-being or normalcy at the outset of monitoring.

In retrospect, it seems clear that undertaking the RCTs was premature [10,11]. They were conducted before meeting criteria of reliability for the identification, interpretation, and management of patterns. There had been no validation, except generally, that there was an association between specific FHR patterns and the adverse outcomes (neurologic or otherwise) to be prevented. It was assumed that hypoxic injury occurred as a result of progressive hypoxia and that early intervention would be beneficial. There was no validation of the ability of specific interventions to effect improvement, however; nor was there any understanding of the rapidity or the contributions of other mechanisms of hypoxic injury. With regard to intra- and interobserver error, there was a disappointingly low agreement in both interpretation and management, even among experts and even when the classification was agreed upon [12–15]. Intraobserver reliability for the interpretation of FHR patterns using kappa statistics is about 0.7, reflecting fairly substantial but by no means complete agreement. For case management, the kappa value is about 0.58. For intraobserver error, the kappa values are 0.4 for pattern interpretation and 0.37 for case management [1,16].

These problems notwithstanding, there is extraordinary agreement on the ability of the “reassuring” FHR pattern to define the absence of hypoxia. Alternative strategies for the evaluation of the abnormal tracing have been introduced in this setting, including fetal scalp blood sampling for pH, fetal pulse oximetry to determine fetal oxygen saturation, and computerized evaluation of the fetal ECG to reveal fetal myocardial stress (Table 22.1) [1,17–19]. Each of these techniques relies on the interpretation of FHR patterns to determine their application; none aspires to replace EFM as the primary mode of surveillance.

**TABLE 22.1 Measurement Objectives of Fetal/ Neonatal Surveillance Techniques\***

Technique	Physiologic Events
FHR patterns	End-organ response (heart, brain)
pH	Acidosis – metabolic/respiratory
O <sub>2</sub> Saturation	Acidemia, blood flow
ST-ECG analysis	Myocardial hypoxia, stress
Doppler analysis	Blood flow, resistance
NRBC count	Hypoxia, anemia, growth abnormalities, etc.
Placental anatomy	Nutrition, anemia, hypoxic stress
Neuroradiology	Location, timing, severity, function
Neurologic examination	Function, disability

FHR, fetal heart rate; ST-ECG, stress test-electrocardiograph; NRBC, neonatal red blood cells

\*See text for details.

Despite the obvious questions about the relevance of EFM, the reproducibility and meaning of FHR patterns, and some articles calling for its outright abandonment, this technique is still almost universally applied during labor, with no obvious practical alternative [20].

## **METHODS OF SURVEILLANCE**

### **Intermittent Auscultation (IA)**

Prior to the advent of EFM, fetal surveillance during labor was limited to IA of the FHR and noting the presence of meconium. The information was difficult to ascertain accurately, and the data collected are not reproducible [15,21–26]. The auscultatory criteria for fetal distress (i.e., detection of a FHR above 160 beats/min or below 100 beats/min) had not been modified for more than half a century. Based on a study of more than 25,000 deliveries, IA was simply inadequate for the early detection of fetal distress [27]. The intrapartum fetal death rate was high, but more important, apparently many deaths occurred without warning. Thus, until the fetus was actually delivered, there could be no reassurance that it was healthy, modestly compromised and grateful for the rescue, hopelessly moribund, or dead. IA has never been subjected to any randomized trial comparing it with a program of no heart rate surveillance, and the practice is difficult to implement clinically [28]. This fact would

seem to make the recommendations about the comparability of EFM and IA less than certain. Equally disturbing is the notion that EFM is implemented so widely not because of any perceived benefit but because of “legal vulnerability” [16,20].

Notwithstanding its limitations, electronic monitoring has several practical and clinical advantages over auscultation. Electronically obtained data give a continuous record of heart rate information. EFM is more easily and reliably obtained than IA. Its major clinical advantage, which was unsatisfactorily tested in the RCTs, appears to be that it restricts the use of potentially compromising strategies (e.g., augmentation/stimulation of labor, regional anesthesia) to the demonstrably normal fetus. Indeed, some RCTs have shown benefits in reductions in neonatal seizures and asphyxial deaths [5,6,29]. The authors are unaware of any example of a fetus dying in labor without revealing significant and prolonged abnormalities of the FHR pattern beforehand [30]. With EFM, no data are lost, trends are easily identified, and even subtle changes become recognizable with experience. The monitor creates a hard copy of the data for real-time collaborative analysis, as well as retrospective analysis, teaching, and research, in which the tracings can be interpreted in light of known outcomes. These resources can only assist in the development of rational clinical policies.

### Electronic Fetal Monitor Functions

Before the interpretation and management of individual EFM tracings are presented, it is necessary to describe what the device does and how it is used.

#### *Heart Rate*

The fetal monitor obtains two channels of information by either external or internal transducers and records them continuously on a moving strip chart recorder or electronically. With a direct scalp electrode, the fetal ECG complex acts as a trigger to time the fetal heartbeat. External devices use ultrasound signals to detect the motion of the fetal heart, electronically converting the processed electronic signal into a heartbeat. Regardless of the transducer, the device then calculates the interval between consecutive beats and then plots a “rate” on the moving

strip chart recorder, as if all intervals in one minute were exactly the same as the detected interval. If the device determines that the interval between consecutive beats is 1 second, it calculates that there would be 60 beats in 1 minute – as if all the beats in 1 minute were 1 second apart. When it detects a new interval, it discards the last interval. If the next interval were 0.8 seconds, the device calculates a rate of 75 beats/min as if all the intervals in 1 minute were the same; plotting an “instantaneous rate” for each interval on a graph without averaging reveals the rhythm of the FHR (or the FHR *pattern*). Although an “average rate” can be discerned easily with the naked eye, *the interpretation of EFM strips is essentially unrelated to the rate, but to the pattern or rhythm of the changes*. If the pattern were shifted upward on the graph (higher rate) or lower (lower rate), there would be no difference in the appearance or, for the most part, the interpretation of the pattern.

#### *Uterine Contractions*

The second channel of the monitor strip provides information about the duration and intensity of uterine contractions and, to some extent, fetal and maternal activity. Uterine contractions are measured either directly with a fluid-filled or transducer-tipped catheter placed into the uterus, or indirectly by a tocodynamometer placed on the mother's abdomen. The internal catheters record changes in intrauterine pressure; the tocodynamometer responds to alterations in the contour of the mother's abdominal wall. Although external devices permit the detection of only relative differences in baseline tone and intensity, under most circumstances this suffices clinically. When more precise information about contractility is required, an intrauterine catheter can provide quantitative information about baseline uterine tone and amplitude of contractions.

#### *The Effects of Contractions*

Detecting contractions is an important part of the fetal monitoring scheme. Uterine contractions exert several physiologic effects. Most important and consistent is the adverse effect of contractions on uterine blood flow (UBF), in which maximal flow is obtained with low resting tone between contractions [31,32]. The contraction decreases UBF in

direct proportion to the intensity and duration of the contractions. Indeed, above about 50 mm Hg of intrauterine pressure, UBF essentially stops and there is no option for further exchange between mother and fetus of oxygen, carbon dioxide, nutrients, and heat across the placenta. This reduction in oxygen availability probably occurs for a short time during most normal uterine contractions in active labor. In a healthy fetus, the effect is generally well tolerated and not cumulative. *Excessively prolonged or frequently occurring contractions can jeopardize even the most robust fetus, however.* More important, a fetus whose placental reserves are already impaired by some preexisting problem (e.g., anemia, infection, abruption, preeclampsia) can develop considerable hypoxemia during even normal uterine contractions.

Contractions also stimulate the fetus and increase intracranial pressure within the fetal head, especially as descent occurs, but also in the breech presentation. To counteract the effect of increased intracranial pressure with contractions, the fetus's blood pressure increases proportionally to the uterine contraction. As a result, during a contraction, umbilical and cerebral blood flows (CBF) are normally maintained. An active fetus signals the onset of a contraction by making truncal movements and frequently, especially at high stations, accelerations. These diminish as the contraction peaks. At the end of the contraction, the fetus often exhibits "breathing movements," reflected as a brief diminution of the FHR with somewhat increased variability. There are FHR patterns associated with various aspects of fetal behavior (behavioral states), including breathing, sucking, and mouthing movements [33–35]. When contractions are excessive, or especially in association with pushing or descent, their effects are augmented, potentially to the embarrassment of CBF. Under these circumstances, contractions, abetted by forceful maternal pushing, can further increase intracranial pressure, reduce CBF below critical values, and induce heart rate decelerations. This is especially common in the presence of fetal malposition, such as the occiput posterior position or face presentation [36–41,44].

By influencing the station or position of the fetus, contractions can also facilitate compression the umbilical cord, thus reducing umbilical blood flow proportional to degree and duration of the cord compression.

Comparing the surveillance systems used for the fetus with the adult in a coronary care unit (or the fetus monitored by IA) offers useful perspective. In the coronary care unit, the patient is usually at rest. The role of all the attached devices is to detect any perceived abnormality of homeostasis, rhythm, or ECG pattern quickly and alert the qualified health-care provider so that proper intervention is as timely and appropriate as possible. The FHR-based system is quite different. The fetus is not at rest; rather, it is obliged to deal with the provocations and impositions of the frequently recurring contractions and maternal bearing-down efforts, with their manifold effects on blood flow and other systems. EFM therefore represents the fetus's answer to the recurrent question: "How did you like that contraction?" It would doubtless enhance the sensitivity of EFM if there were an arrangement with the fetus in such a way that decelerations or some other telling sign would appear only when the fetus was truly in trouble. Unfortunately for those conducting the surveillance, nature provided the mature fetus with too many resources for manipulating its heart rate and cardiac output. Although the fetus does not fail to respond to the least hypoxemic stress during labor, it also produces decelerations and changes in variability under a bewildering array of behavioral conditions mentioned previously that have nothing to do with any threat to health or well-being. This happens to such an extent that any notion of using a single short-lived feature of the tracing to assess fetal well-being can result in undesirable interventions and terror in both the providers and the mother.

### ***Principles of Fetal Heart Rate Monitoring***

Anyone using the technique must understand the physiologic principles of FHR monitoring, as well as the technical and logistical pitfalls that impede the ability to realize the objectives originally vouchsafed for EFM. FHR patterns before and during labor depend on numerous factors, including gestational age and behavioral state of the fetus, as well as medication, infection, anomalies, arrhythmias, and maternal disease [42,43]. Beyond these, FHR patterns during labor are also influenced considerably by contractions and expulsive efforts of the mother, as well as the station, position, attitude, molding of the fetal head, and a host of other local factors such as infection (Table 22.2) [44–47].

TABLE 22.2 Factors Influencing Fetal Heart Rate Pattern

Gestational age	Behavioral state – stimulation
Hypoxia/ischemia	Drugs
Infection/fever	Anemia
Arrhythmias	Anomalies
Maternal bearing-down efforts	Uterine contractions
Position of the head	Seizures, other CNS events

CNS, central nervous system

### BASELINE HEART RATE

The normal baseline FHR is defined as the persistently *stable* rate in the absence of contractions or other provocations [45]. The baseline rate can be considered a very narrow range, perhaps 5 to 10 beats/min at best and is usually characterized according to the rate to the nearest 5 beats/min, the stability of the rate, and the presence of short- and long-term variability in the interval between contractions [45]. As gestation progresses, maturation of the autonomic nervous system occurs, with increasing vagal dominance in control of the heart and a gradual decrease in rate. The mean heart rate is somewhat higher early in gestation than at term, but rates above 160 beats/min are unusual in healthy fetuses at any time after 24 weeks of gestation. The normal fetus establishes a unique baseline rate at any particular time during pregnancy, usually contained within the population-derived normal range between 110 and 160 beats/min [46]. For the purposes of monitoring the individual fetus during labor, its baseline rate (and variability) should be established in early labor (hereinafter referred to as the *basal rate*) and retained as the reference point for comparisons with later determinations of the baseline rate.

### Tachycardia and Bradycardia

The definitions of baseline *tachycardia* (rapid heart rate) and *bradycardia* (slow heart rate) are frequently described as single values outside the range of “normal” (e.g., 110–160 beats/min) that last for at least 10 minutes [47]. A more physiologic and clinically important approach uses the fetus as its own control and assesses changes in the baseline rate over time, regardless of whether the rise or fall in the

rate has exceeded a unique threshold. For example, a fetal rate that begins at 155 beats/min in early labor and rises to 160 beats/min (tachycardia by the previous definition) represents much less of a problem than a rise from 110 beats/min to 155 beats/min (technically does not qualify for tachycardia) from a baseline of 110 beats/min. The authors refer to these changes as *relative tachycardia* or *bradycardia*. For this purpose, it is necessary to use the term *basal heart rate*, established on admission, as the reference point for subsequent changes, and not simply to allow the baseline to increase or decrease over time with periodic revision of “the baseline rate.” Thus, if the basal fetal heart rate on admission were about 135 beats/min, a rise to a persistent 150 beats/min should be considered a relative tachycardia, whereas a fall in heart rate to a persistent 120 beats/min, even with normal or increased variability, would represent relative bradycardia. Neither should be considered part of the normal range of this particular fetus (see Table 22.2).

Several factors, some benign, can increase the heart rate, including fever, cardioactive drugs, maternal dehydration, arrhythmia, and hypoxia (with decelerations). In a mother with a fever, the fetus has both a higher temperature and an elevated rate proportional to the maternal fever. If the fetal tachycardia persists despite the reduction in maternal fever, then the fetal tachycardia is no longer passive but likely reflects fetal infection.

There are dramatic differences in the speed with which tachycardia or bradycardia can develop. The authors attempt to both classify and illustrate these differences in the ensuing tables, illustrations, and text (Tables 22.3 and 22.4). The development of tachycardia during labor can represent the slow, progressive rise in baseline that has occurred over many hours and, without decelerations, can be related to the development of dehydration, maternal fever, use of epidural anesthesia, medications (e.g., terbutaline, atropine). *Fetal tachycardia developing in association with fetal hypoxia invariably is associated with decelerations before the rise in the heart rate.* It is unlikely, however, that the rise in rate accompanying tachycardia will be above about 160 to 170 beats/min. When the tachycardia reaches higher levels, maternal fever or drugs such as ephedrine are usually implicated. Conversely, a sudden, sustained increase in rate could represent either an arrhythmia, or, especially in the second stage of labor with



TABLE 22.3 Potential Causes of Fetal Tachycardia\*

Causes	Comment
<b>Decelerations absent</b>	
• Evolutionary – over many minutes/hours	
Maternal fever	Passive tachycardia
Fetal infection	Unrelated to maternal temperature
Medications	Narcotics, tranquilizers, barbiturates, betamimetics, ephedrine – saltatory pattern
Anemia (chronic)	Sinusoidal – highly variable response
• Progressive – over minutes	
Maternal anxiety	
Atropine administration	
• Abrupt	
Tachyarrhythmia	
Neurologic injury	
Terminal tachycardia	Can be preceded by decelerations, but no decelerations with tachycardia
<b>Decelerations present</b>	
• Hypoxia	Associated with decreased variability
• Anemia	Recent, more severe loss but not exsanguination

\*See text for details.

a problematic tracing, or a neurologic injury separate from any effects of fetal hypoxia [48,49]. The authors have identified these tracings as *terminal tachycardias*.

Sustained, low baseline rates, in the range of 50 to 80 beats/min, especially when lacking variability, can be the consequence of profound asphyxia or the manifestation of an arrhythmia, most commonly congenital heart block (Table 22.4). Congenital or acquired heart block, which can be diagnosed by ultrasonography or direct evaluation of the fetal ECG obtained from the scalp electrode, is seen as a stable baseline rate in the range of 50 to 80 beats/min with no decelerations. About 20% of affected fetuses have structural congenital heart disease, and a considerable number of moth-

TABLE 22.4 Potential Causes of Fetal Bradycardia\*

Causes	Comments
Asphyxial – severe	Previously normal rate or tachycardia and decelerations
• Cord prolapse	Prolonged decelerations, often abrupt onset
• Uterine rupture	
• Medication – anesthetics (intoxication)	
• Severe fetal hemorrhage	
• Maternal hemorrhage or profound hypotension	
• Uterine tetany/abruptio placentae	
Other causes	
• Maternal hypothermia	
• Congenital heart block	Absent FHR variability
• Head compression	Usually with prolonged pushing – second stage
• Cord compression	Increased variability – second-stage bradycardia

FHR, fetal heart rate

\*See text for details.

ers have a connective tissue disease, most commonly systemic lupus erythematosus [50–56]. Perinatal infections such as cytomegalovirus as well as hypoglycemia have also been implicated as causes of fetal bradyarrhythmias [57–60]. When bradycardia represents severe asphyxia (really a prolonged deceleration) then, in addition to absent variability, the fetus is often unable to maintain a stable heart rate, and decelerations might not be present or might be too difficult to classify in such situations.

The irregular fluctuations observed in heart rate pattern recordings reflect the variability of the FHR around the baseline rate. The seemingly random irregularity of the heart rate pattern results from the various inputs competing at different frequencies, influencing the rate through autonomic pathways [61–64]. Autonomic input and control of the heart comes from the medulla oblongata controls. The heart functions in response to a complex array of inputs from the hypothalamus and cortex. As the fetus matures, variability increases, and periodic fluctuations in heart rate, fetal activity, and other features of the fetal state become more complex and better organized into epochs of rest and activity

that are generally known as *behavioral states* [35,65–68]. These biologically programmed variations in fetal activity are most obvious in antepartum monitoring, but they persist into labor in most fetuses and can be recognized despite competing inputs from uterine activity and other factors associated with labor [34,35]. Recognition of these spontaneous shifts in the character of the FHR pattern (which are accompanied by state changes in fetal movements, EEG patterns, and other physiologic events) is an important aspect of the clinical interpretation of heart rate patterns, one that has not previously received sufficient emphasis. This understanding of the impact of behavior on the FHR pattern anticipates the use of these patterns to infer abnormal fetal behavior from immaturity, sedation, or injury in the absence of hypoxia [69–72].

Superimposed on the intrinsic rhythm of the sinoatrial node (which can itself be altered by catecholamines, body temperature, pharmacologic manipulation, hypoxia, and other factors) are servocontrol mechanisms mediated through *baroreceptors* and *chemoreceptors*. These receptors are located in several areas of the central circulation and alter heart rate through brain or spinal cord reflex mechanisms. In this manner, respiratory sinus arrhythmias, intrinsic oscillatory mechanisms in the baroreflex loop, and peripheral resistance all influence FHR variability [62–64]. Temporary or permanent brain injury can reduce variability, as can neurotoxic agents, hypoxia, and perhaps hypercapnea and acidosis [61]. The responses of variability to hypoxia are complex. Acute decreases in PO<sub>2</sub> can increase it, but chronic hypoxia reduces variability [62,63].

Although it has been recommended that only a single measurement of variability be used clinically, in fact, at least two components of baseline variability can be identified (Table 22.5) [73,74]. *Short-term variability* is a consequence of the irregularity in intervals between consecutive pairs of heartbeats (average perhaps 1–2 beats/min/beat). This variability is produced primarily by small, seemingly random fluctuations in efferent vagal tone [75,76]. *Long-term variability* represents the broader, more predictable oscillations in heart rate that occur with an amplitude of 5 to 15 beats/min and a frequency of approximately 2 to 6 cycles/min. These oscillations are probably influenced primarily by changes

TABLE 22.5 Classification of Variability

Classification	Interpretation
Absent	Amplitude range undetectable
Minimal	Amplitude range detectable, but 5 beats/min or fewer
Moderate (normal)	Amplitude range 6–25 beats/min
Marked	Amplitude range greater than 25 beats/min

in sympathetic activity and numerous other inputs that do not influence short-term variability per se. Increasing the heart rate (acceleration or tachycardia) diminishes variability.

In circumstances in which both sympathetic tone and vagal tone are increased, the effect of the vagus will predominate and produce a lower rate and increased variability. Indeed, it is likely, at least in cephalic presentation, that contractions increase both sympathetic tone and parasympathetic tone [77].

Although long- and short-term variability generally change simultaneously, sometimes they can vary independently. *Short-term variability is the more important indicator of fetal well-being*. Variability should be viewed clinically as a reflection of the ability of the fetus to adjust heart rate to the need for subtle changes in cardiac output. Variability should not be interpreted in isolation but rather as a component of longer periodic changes related to fetal state (rest-activity cycles), fetal breathing movements, and circadian or ultradian rhythms [78–81].

Decreased variability is a consequence of an aberration in the normal autonomic control of heart rate and has several causes (Table 22.6). Premature fetuses normally have reduced variability compared with those at term. All barbiturates, tranquilizers, anesthetic agents, and narcotics, as well as parasympathetic blocking agents, have the capacity to reduce fetal heart rate variability when they are given to the mother [82].

This is a predictable pharmacologic response, the duration and extent of which vary in direct proportion to the amount of drug administered. Whereas acute hypoxemia can transiently increase baseline variability, significant chronic reductions in PO<sub>2</sub> are associated invariably with diminished

TABLE 22.6 Abnormalities in Fetal Heart Variability\*

Observation: Possible Causes	Comment
<b>Decelerations absent:</b>	
• Neurologic injury	
• "Sleep states"	Reflect normal variation in behavioral state
• Maternal fever	Associated with tachycardia
• Fetal infection	Unrelated to maternal temperature
• Medications	Narcotics, tranquilizers, barbiturates, betamimetics, Atropine
• Anemia – chronic	Sinusoidal pattern: highly variable response
• Anxiety	Usually with tachycardia
• Tachycardia	Any cause
• Congenital anomaly	Common with neurologic, cardiac anomalies
• Arrhythmia	Including tachyarrhythmia with heart block during deceleration
<b>Decelerations present:</b>	
• Hypoxia	Associated with decelerations, and rise in FHR baseline of some duration
• Agonal pattern	Unstable baseline, nondescript decelerations, electromechanical dissociation
<b>Increased variability:</b> (saltatory pattern)	
• Ephedrine effect	
• Active labor	
• Second stage	

\*See text for details.

short-term variability. Without accompanying decelerations, however, decreased baseline variability during labor should not be interpreted as oxygen deprivation.

As emphasized previously, *the most common explanation for diminished variability in well-oxygenated fetuses is a period of reduced arousal, part of the normal sequence of state changes.* Stimulation of such fetuses by pressing on their head during a vaginal examination, pushing on them through the mother's abdominal wall, or performing a scalp blood sample often provokes a transient increase in variability and sometimes a FHR acceleration. These responses indicate that the diminished variability is

probably not indicative of either severe hypoxia or extant brain injury.

A fetus entering labor with persistent lack of variability cannot be defined as having normal neurologic potential. If there has been no preceding testing, then the differential diagnosis is broad and includes drug effect, placental insufficiency, infection, genetic disorders, anemia, neurologic injury (e.g., infarction, hemorrhage, asphyxia), congenital anomaly, or CNS tumor. If the preceding FHR pattern has been reactive, then, absent any other clinical condition or medication, there is a presumptive diagnosis of neurologic injury.

Patterns with increased baseline variability (>25 beats/min) are sometime referred to as *saltatory* or *jumping patterns*. These patterns appear in about 4% of term tracings and are singularly uncommon in preterm fetuses. They are most commonly seen during active labor, after high-dose parenteral ephedrine administration (>30 mg), with uterine hyperstimulation, or too-frequent expulsive efforts in the second stage. Normal variability is usually demonstrated both before and after the saltatory pattern episode. Increased variability (the saltatory pattern) is no more "normal" than is decreased variability. It should be viewed as a period of stress for the fetus and can auger obvious decelerations. In the absence of abnormal periodic FHR changes, and in conjunction with short-term and long-term variability, the saltatory FHR pattern is considered "benign" from an acid-base standpoint (see discussion later) [83]. With abnormal features, including no variability, decelerations, and tachycardia, they represent additional adverse commentary [84]. In some instances, markedly increased or bizarrely exaggerated variability can represent fetal seizures (Table 22.6).

*In the absence of decelerations, loss of variability during labor is most likely a response to drugs, dehydration, or a behavioral state change and very unlikely to represent fetal hypoxia.*

When only short-term variability is present, however, the tracing is usually considered acceptable. When only long-term variability is present, at its extreme, it represents a "sinusoidal" pattern and can represent something ominous, such as severe anemia or neurologic injury. Whereas most attention has been paid to the abnormalities associated with decreased (<5 beats/min) or absent (<2 beats/min) long-term variability, increased variability

(>25 beats/min – frequency >5 cycles/min or saltatory) is no more normal than is decreased variability. The National Institute of Child Health and Human Development (NICHD) classifies variability only as long term but considers the sinusoidal pattern separately from long-term variability [45].

### HEART RATE PATTERNS AND FETAL WELL-BEING

The normal term FHR pattern reveals numerous features corresponding to various behavioral states (Figure 22.1A and B). These include a stable



FIGURE 22.1.

*Reactive tracings. A, Normal variation in FHR pattern: accelerations, normal variability, and cyclic changes reflecting the fetal behavioral state (sequence: quiet sleep, REM sleep, and active sleep). B, EFM tracing with coalescence of accelerations and a "sinusoidal" pattern. These sinusoidal oscillations correspond to fetal sucking and are benign. See text for details.*

baseline rate and no decelerations and alternating epochs of 1) decreased variability and absent accelerations (sleep/rest), and 2) average variability and accelerations associated with fetal movement (awake/active) [66,74,85]. These features, including the cyclic activity, permit the assessment of both normal fetal behavior (neurologic integrity) and the absence of hypoxia. They represent the fundamental property of the normally functioning fetus that will not deteriorate from hypoxia, sepsis, or trauma without obvious changes in baseline rate or variability, often but not invariably associated with decelerations. As mentioned previously, not all changes in rate and variability represent hypoxia or deterioration, and if only brief segments of the tracing are analyzed, some of these normal features of the rest cycle could be interpreted as indicating adverse conditions. Thus, brief periods of no variability (fetal sleep) and sinusoidal heart rate pattern (fetal sucking) should not be confused with fetal abnormality.

With no decelerations during labor, these patterns can be observed for reasonable periods before one considers intervention.

In estimation of fetal well-being, the two most important considerations of the baseline are *stability* and the *presence of normal variability* (not increased or decreased) – even in the presence of decelerations. In any tracing review, look at the beginning of the tracing to establish the basal rate, the variability, and any epochal changes associated with normal fetal behavior.

### FETAL DETERIORATION WITHOUT HYPOXIA

As the fetus deteriorates before the onset of labor, the behavioral cycles become disrupted with lengthening sleep cycles; the variability disappears, and the baseline can rise somewhat (Figure 22.2A). Only then do accelerations disappear; decelerations



FIGURE 22.2.

*The spectrum of nonreactive patterns. A, Note nonreactive pattern with occasional accelerations, minimal variability, and no coalescence of accelerations or obvious cyclicality. B, The tracing becomes quite flat, without accelerations or variability. Note that decelerations are absent.*

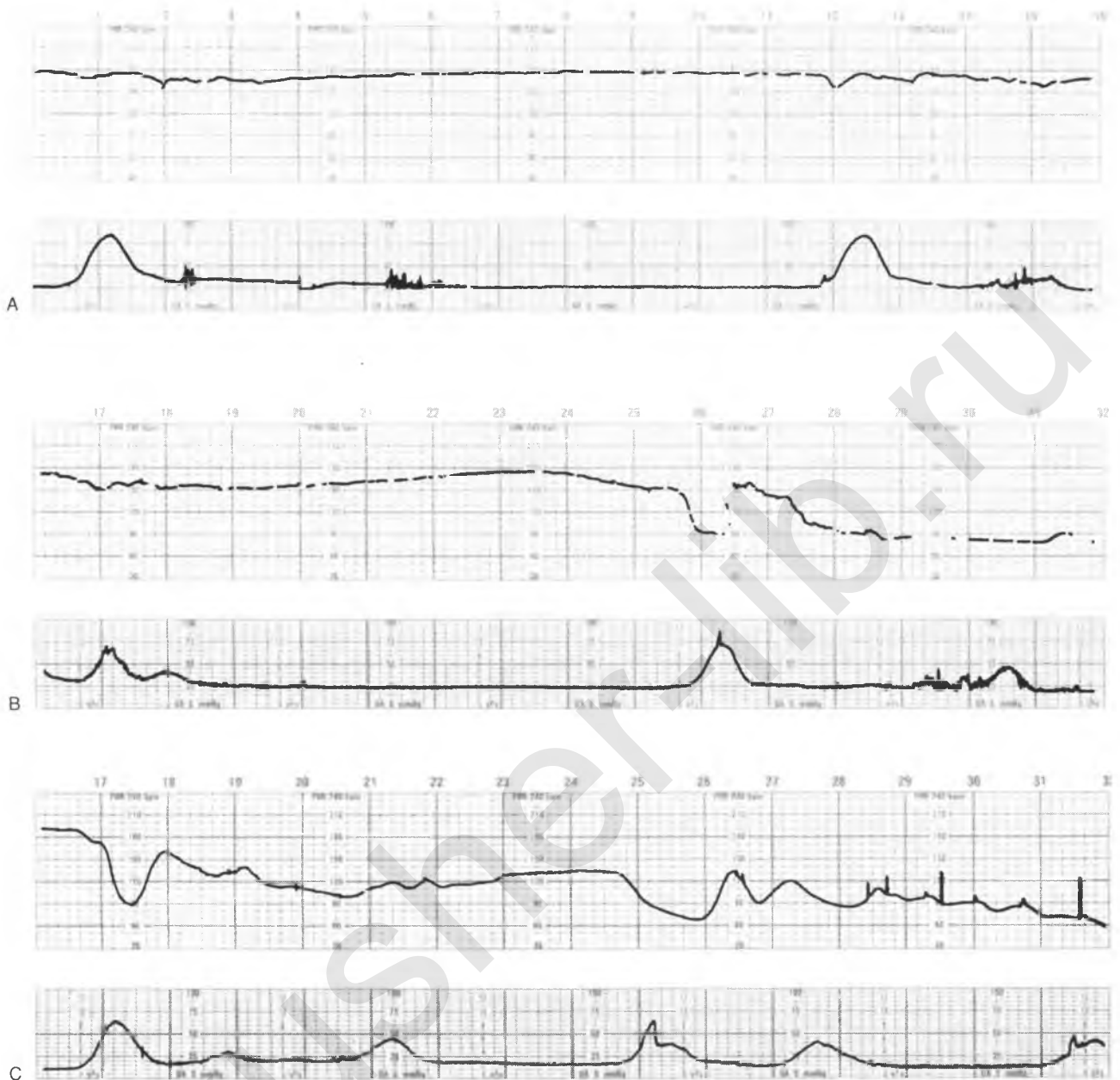


FIGURE 22.3.

*Agonal patterns: Tracing, A, Long-standing asphyxia with absent variability, unstable baseline rate, and bradycardia occurring just prior to death. B & C, The baseline is unstable and falls with nondescript periodic changes (electromechanical dissociation and shock). The infant was profoundly hypoxic and died shortly after birth. See text for discussion.*

appear with contractions (Figure 22.3A), the baseline becomes unstable, it falls, and the fetus dies (Figure 22.3B and C) [78]. The fetus represented in Figure 22.3 had a true knot in the umbilical cord. An emergency cesarean delivery at tracing outset is highly unlikely to have resulted in a different outcome.

#### FETAL DETERIORATION FROM HYPOXIA

The classification of decelerations and their proposed mechanism appears in Tables 22.7 and 22.8. It is critical to the understanding of FHR patterns that in the presence of uterine contractions, any fetal hypoxia is reflected by the appearance of

TABLE 22.7 Patterns of Fetal Heart Rate Decelerations\*

**Early deceleration**

- In association with a uterine contraction, a gradual (onset – nadir >30 sec) decrease in FHR with return to baseline
- Nadir coincident with the peak of the contraction

**Late deceleration**

- In association with a uterine contraction, a gradual (onset – nadir >30 sec) decrease in FHR with return to baseline
- Onset, nadir, and recovery occur after the beginning, peak, and end of the contraction, respectively.

**Variable deceleration**

- An abrupt (nadir <30 sec), decrease in the FHR below the baseline
- Decrease in FHR of >15 beats/min, with a duration >15 sec but <2 min

**Prolonged deceleration**

- Decrease in the FHR below the baseline
- Deceleration is >15 bpm, lasting >2 min, but <10 min – onset to return

FHR, fetal heart rate

\*See text for details.

TABLE 22.8 Mechanism and Physiologic Responses with Decelerations

Deceleration Type	Deceleration Type	
	Lates	Variable/Prolonged
Stimulus	Systemic hypoxia	Regional ischemia
“Dive reflex”	Absent, BP – decreased	Present, BP – increased
Baseline/variability change	Present and required	Depends on hypoxia/injury
Relation to injury	Uncommon, late	Common, can be early
Clinical examples	Frequent uterine contractions	Cord compression
	Maternal hypotension	Head compression

decelerations before a rise in the baseline rate or decrease in variability (Figure 22.4) [86,87]. With continued mild-to-moderate hypoxia, the decelerations persist, accompanied by a rising baseline heart rate and a diminution in baseline variability (and developing acidosis). As the hypoxia increases, it eventually leads to a fixed elevated rate (rarely above 160 beats/min; Figure 22.5).

As the fetus approaches death, the baseline falls and becomes unstable; decelerations can be less obvious and less easily separable into type, that is, late or variable. With severe or profound hypoxia, the initial response can be a prolonged deceleration or bradycardia (see Figure 22.3C).

To indicate fetal hypoxia, therefore, transient decelerations must provoke a rise in a previously normal baseline rate and a decrease in baseline variability between contractions, and not in the deceleration itself. If the fetal baseline rate is already increased or the initial baseline variability is diminished, these features might not change. *Regardless of the deceleration pattern (i.e., late, variable, prolonged), transient decelerations unaccompanied by these baseline changes cannot represent developing tissue hypoxia or troublesome ischemia.* Late-appearing decelerations associated with a stable baseline rate and normal variability often represent a totally benign behavioral pattern of normal fetal breathing (Figure 22.6) [88]. Thus, in the absence of changes in baseline rate and variability, there is no meaningful distinction among late, variable, even prolonged decelerations (Figure 22.7). Prolonged decelerations arising from a previously normal FHR patterns are rarely of (acid–base) consequence if the deceleration stabilizes at 80 beats/min or above, and especially if variability is maintained within the deceleration (see later discussion and Figure 22.8) [89,90]. In clinical circumstances, such as occur in this case, responsible care requires cessation or modification of pushing, and curtailment of oxytocin. It is reasonable to anticipate a normal outcome. Similar patterns occur in the first stage of labor following epidural anesthesia and uterine hyperstimulation related to oxytocin or abruptio placentae.

*If decelerations are accompanied by a rising baseline and decreasing variability, the prognosis becomes guarded regardless of the type of decelerations, variable or late (Figure 22.9).* In this case, at the start of the second stage of labor, the FHR pattern was normal, without decelerations. With pushing, variable decelerations with progressively decreasing variability and tachycardia occur. These decelerations represent recurrent ischemic events without specific diagnosis of injury. The frequency of contractions should be diminished and expulsive efforts moderated. A similar approach is taken with prolonged decelerations that fall below 80 beats/min and lose variability (Figure 22.10A



FIGURE 22.4.

*Late decelerations. A, The previously normal fetus develops late decelerations following epidural anesthesia and maternal hypotension. B, This pattern recovers rapidly once the hypotension resolves. Note the rising baseline and decreasing variability.*

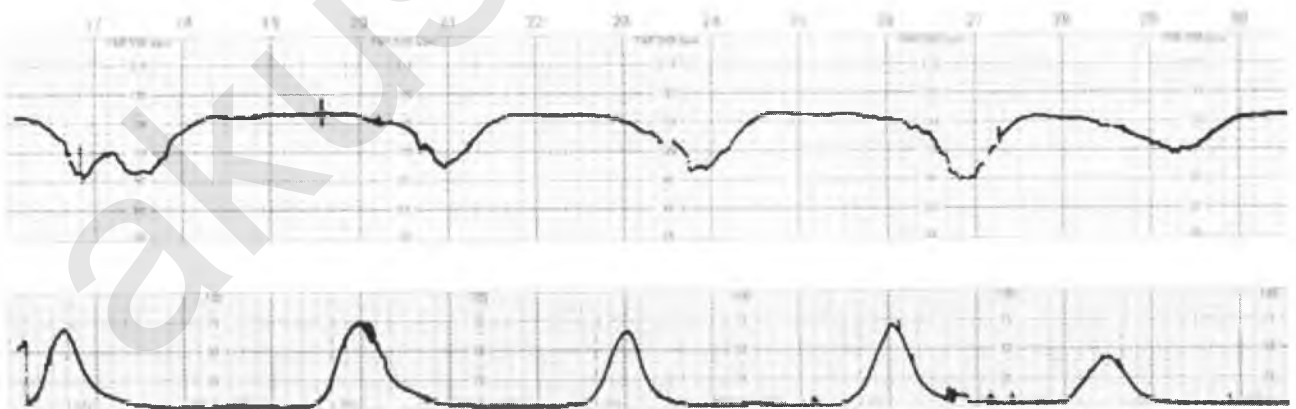


FIGURE 22.5.

*Late decelerations. Decelerations are combined with tachycardia and absent variability.*



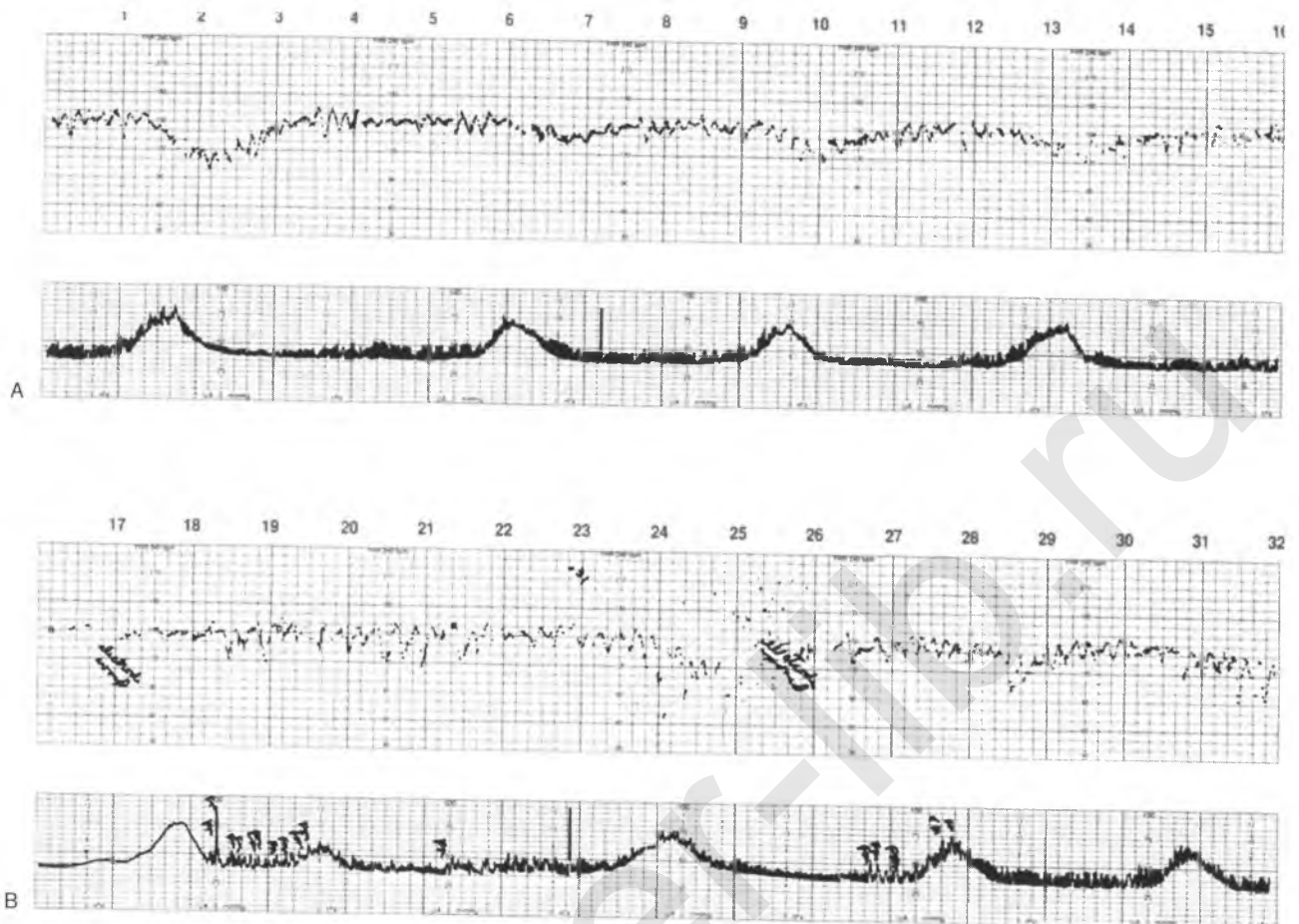


FIGURE 22.6.

*Spurious late decelerations. A, These changes represent breathing movements with induced contractions. B, Fetal breathing movements are reflected on the uterine contraction channel as high-frequency, low-amplitude oscillations. The pattern is entirely benign.*



FIGURE 22.7.

*Variable decelerations. The baseline rate remains stable, and variability is maintained despite decelerations. See text for additional discussion.*

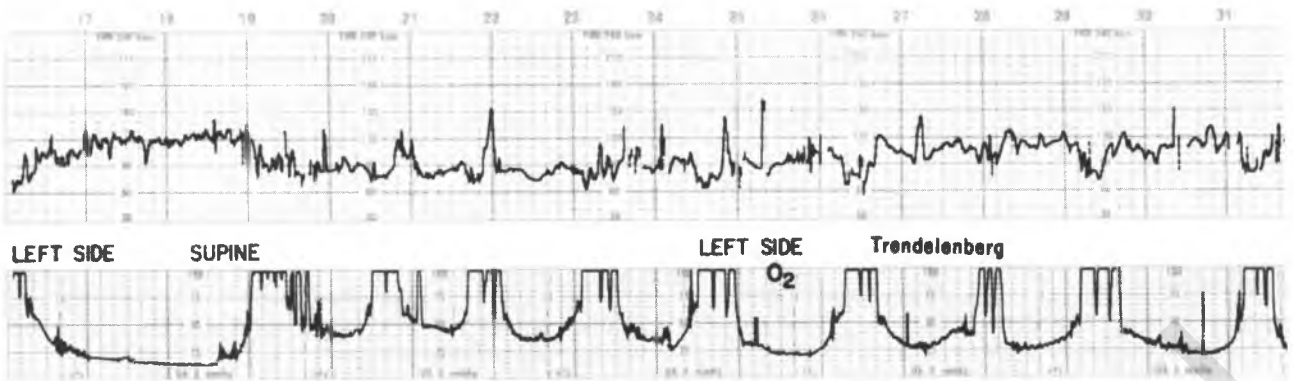


FIGURE 22.8.

*Prolonged decelerations. The pattern is initially normal, but excessive uterine activity occurs because of oxytocin and compulsive second-stage pushing. Notice the maintenance of variability.*



FIGURE 22.9.

*Variable decelerations – deteriorating status.*

and B). Especially in Figure 22.10C, note that in this essentially normal tracing, with the onset of pushing, recurrent variable decelerations appear that extend beyond the contractions. Relentless pushing precipitates prolonged decelerations that do not recover. Urgent delivery (*rescue*) is indicated. This tracing does not permit the diagnosis of injury, only that an acute and rather profound ischemic event just before delivery has occurred. More responsible control of the pushing probably would have eliminated both the ischemic assault and the need to rescue the fetus.

The *pattern of decelerations* provides important insights into the mechanisms and physiologic responses of hypoxia: The fetus can suffer hypoxia that is related either to 1) a decreased availability of oxygen (*hypoxemic hypoxia*), or 2) a decreased blood supply related to interference with blood flow to the brain or placenta, and only secondarily to inter-

ference with oxygen availability (*ischemic hypoxia*). The authors consider those changes associated with late decelerations hypoxemic, whereas those associated with variable or prolonged decelerations of sudden onset ischemic (see Table 22.8). The bases for this distinction are derived from numerous clinical and experimental research publications [86,87,91–96,97–109]. The distinction is important because of the markedly different ways that the fetus responds to such oxygen limitations, in both the speed of deterioration and relationship to subsequent injury.

Late decelerations represent an early response to fetal hypoxemia, even before the rise in baseline rate and diminution in baseline variability. When associated with a rising baseline rate and decreasing variability, these decelerations are accompanied by a transient hypotension during the deceleration and a slow decrease in oxygen availability and CBF [110,111]. They can be tolerated for many hours

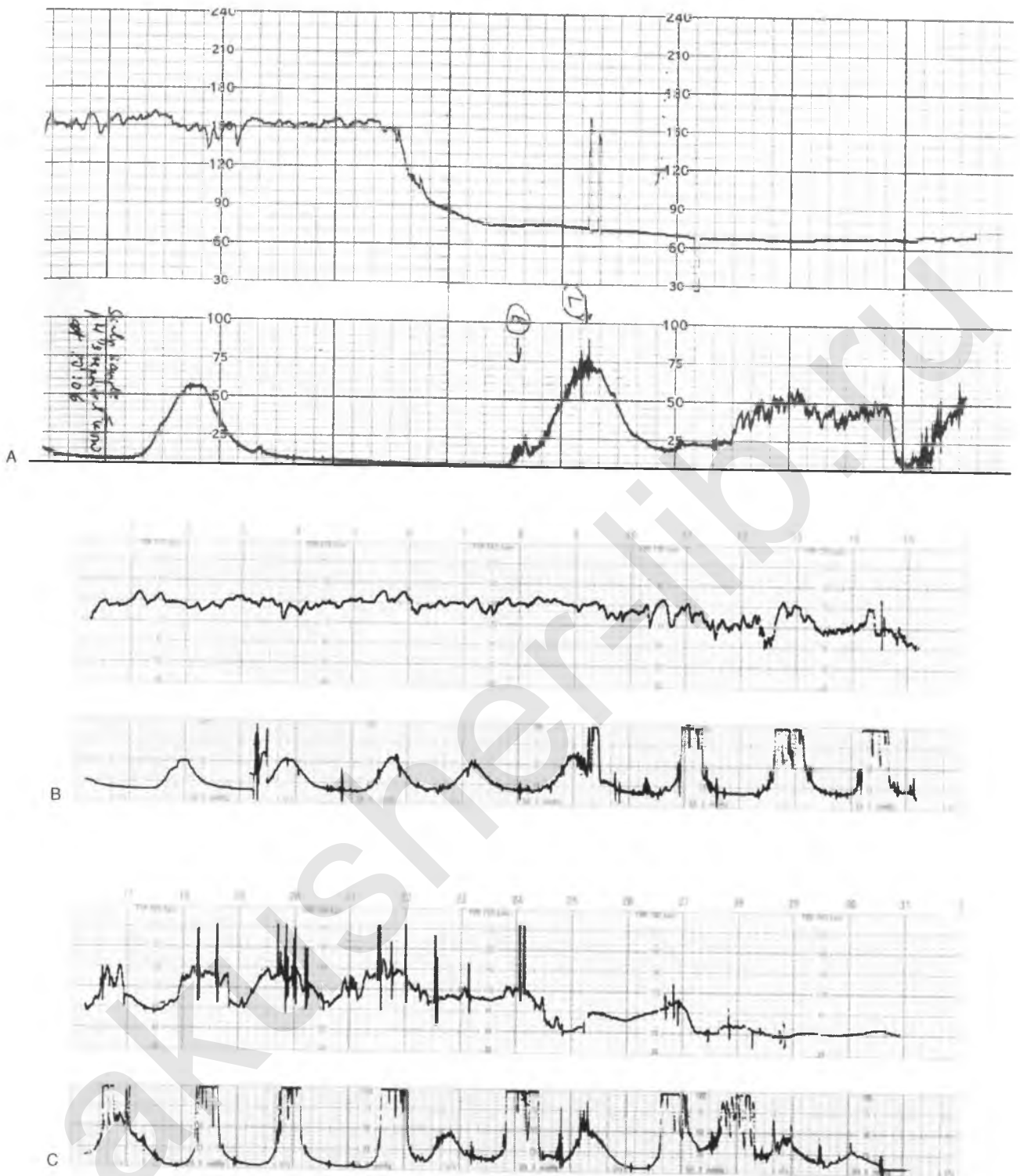


FIGURE 22.10.

*Prolonged decelerations. A, This previously normal EFM tracing develops a prolonged deceleration without either stabilization or variability. This is an acute ischemic/hypoxic event, for which intervention is mandatory. B, C, In this tracing, variable decelerations progress to a prolonged deceleration during second-stage labor. Note the frequent contractions (11 in 15 minutes!) and exuberant pushing. Note also the initially stable baseline rate, average variability, and no decelerations in the upper panel. See text for details.*

while the fetus accumulates a systemic metabolic acidosis [112]. As the hypoxia and acidosis increase, the FHR becomes fixed (no variability) usually with tachycardia (relative) and continuing decelerations. The decelerations at this time might be quite minimal in amplitude but are usually discernible because of the marked reduction in the amount of both long- and short-term variability.

Eventually the severity of the acidosis and the loss of energy substrate conspire so that the fetus can no longer maintain cardiac output. As a result, perfusion of the brain and other vital organs diminishes, and injury supervenes as the fetus approaches death. The monitor shows this as an unstable, diminishing heart rate, less obvious decelerations with no variability, and ultimately asystole (so-called agonal pattern). This is the “pure” sequence of hypoxic changes that most likely result in severe acidosis at birth, prolonged low Apgar scores, and severe neonatal encephalopathy [113]. The initial appearance of late decelerations is an early marker of hypoxia, even before the appearance of acidosis. As such, it is, early in the course, a poor marker for subsequent poor outcome at delivery or long-term outcome [114]. Factors associated with the early appearance of late decelerations include fever, frequent or prolonged uterine contractions, maternal hypotension or hypovolemia, placental disease, and anemia. *Late decelerations with persistently diminished variability appearing in a fetus early in labor suggest a previously injured fetus unable to tolerate the stresses of uterine contractions.*

Conversely, variable and prolonged decelerations are acute anticipatory responses to any diminution in cerebral or umbilical blood flow (ischemia). The rapidity of the onset and the depth of the deceleration result from an exuberant increase in fetal peripheral resistance and a rise in blood pressure, with redistribution of blood flow within the fetus so that flow to the vital structures in the organism (i.e., the brain, the adrenals, the heart, and the placenta [99,115,116]), are maintained, and flow to less critical areas (i.e., the gut, skin, and muscles) are sacrificed. This situation is analogous to the dive reflex and is similar to that found in a diving mammal, including humans [117,118]. Whereas with late decelerations there is no interference with the circulation, no redistribution of blood flow, and hypotension during the deceleration, with variable decelerations the fetal blood pressure is elevated

(to overcome the obstruction to flow), and there is a redistribution of blood flow. Under these manifold responses and redirection of blood flow, specific areas of the fetus from which blood has been shunted or where flow has been inadequate can suffer injury in a short time, even without ongoing systemic hypoxia [119,120]. Variable decelerations deteriorate with much the same baseline changes as late decelerations (i.e., decreasing variability and rising baseline rate) but the time is much more variable.

For these physiologic adaptive reflexes to protect the brain, meaningful obstructions to blood flow must be overcome. As the result of frequent contractions, expulsive efforts, malposition of the fetal head, and nonprogressive labor, CBF can sometimes be diminished beyond the capability of the fetus to adapt. The functional embarrassment of the fetus during variable decelerations is more likely to occur with contractions occurring frequently and are revealed by a host of “atypical” features. “Typical” responses include pre- and postdeceleration accelerations, when variable decelerations with normal baseline variability are preceded and followed by small accelerations, sometimes termed *shoulders* (see Figure 22.7). In these circumstances, the fetus maintains a stable baseline rate and normal variability. In the study by Kazandi, for example, there was no danger of a deteriorating fetal hemodynamic status when decelerations were typical and contraction frequency was normal [91]. The risk of fetal hypoxia/damage and admission to a neonatal intensive care unit [NICU] was quite high when contractions came more frequently or atypical variable decelerations were present [91]. Atypical features include 1) slow return of the fetal heart rate to the baseline; 2) loss of variability during the decelerations; 3) loss of initial and/or secondary accelerations; 4) persistence of secondary acceleration (overshoot); 5) continuation of the baseline FHR at a lower level; and 6) biphasic decelerations. These atypical features, however, can be grouped more simply by determining whether the deceleration has an observable impact on either the baseline rate or its variability. For example, the erratic accelerations that precede or follow variable decelerations can be considered benign when the baseline rate and variability are maintained. Conversely, smooth accelerations after variable decelerations or *overshoots* associated with persistently absent

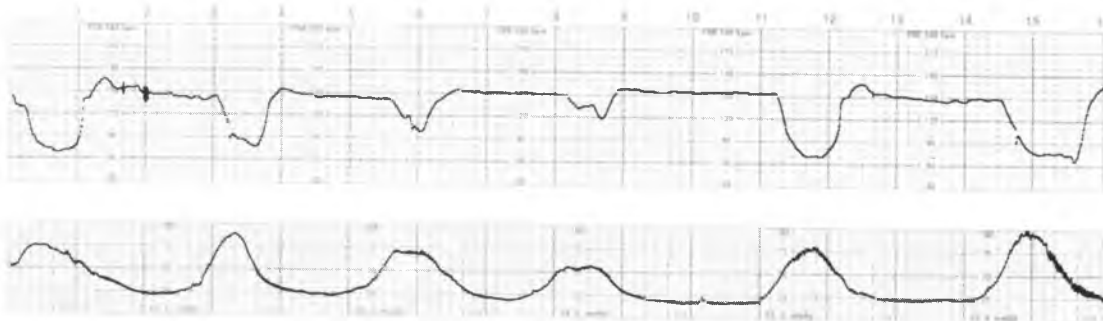


FIGURE 22.11.  
*Variable decelerations, "overshoot," and no variability.*

variability and no preceding accelerations are clearly associated with adverse outcome, both immediate and long-term drop in heart rate [91–94,121]. In the latter situation, the overshoot following a variable deceleration of any amplitude connotes an abnormality of neurologic control over FHR and can be seen in the neurologically injured fetus or after atropine administration (Figure 22.11) [121]. Referring to the illustration, notice the stable baseline rate during the tachycardia. This ominous pattern in a fetus with a previously reactive pattern represents fetal injury and is associated with poor outcome. The degree of acidosis is highly variable, and intervention at anytime is unlikely to alter outcome.

When the rate at the nadir of a variable or prolonged FHR deceleration falls below about 90 beats/min, cardiac conduction defects, in the form of nodal or junctional rhythms, are common. Transient episodes of fetal cardiac asystole lasting several seconds can also develop in this context [122]. Nodal rhythms produce flat heart rates at the nadir of the deceleration. A brief period of loss of variability at this time does not imply more severe oxygen deprivation; it is simply a function of the usually transient change in cardiac pacemaker from the sinoatrial node to the area of the atrioventricular node. If the duration of this flat heart rate at the bottom of the deceleration exceeds 1 to 2 minutes (prolonged deceleration), however, preparations for intervention should be begun.

Recovery of prolonged decelerations is usually associated with a prompt resumption of variability, but there sometimes are late decelerations or tachycardia during the recovery period. The extent and duration of these reactive tachycardias reflect the severity of the hypoxemia that developed during the period of reduced heart rate. In other words, the

more rapidly the heart rate returns to its previous rate and variability, the more limited the hypoxic insult has been during the deceleration. The authors have observed sustained tachycardia out of proportion to the duration or severity of the deceleration in the presence of acute fetal intracranial hemorrhage and other cerebral injuries during labor [48].

Epidural anesthesia can induce late or prolonged decelerations and decreased variability [123–126]. These changes occur most frequently when maternal hypotension or excessive uterine activity develops and are largely preventable through adequate prehydration and proper maternal positioning to avoid aortocaval compression by the uterus, or by withholding an epidural in the case of preexisting pathologic decelerations or uterine hyperstimulation. Even without the development of a documentable fall in blood pressure, the sympathetic blockade that accompanies epidural anesthesia can result in a redistribution of maternal cardiac output, which will preserve flow to vital organs at the expense of the uteroplacental unit. This effect is most obvious if intravascular volume is marginal or reduced. In most patients with FHR changes following an epidural, the tracing will return to normal even without therapy [126].

As mentioned previously, a prolonged deceleration can sometimes evolve into a serious acute asphyxial (ischemic) emergency. In such cases, the FHR is flat after several minutes, and the rate spirals subsequently gradually downward. These situations often have a clinical explanation for the problem, such as extensive placental abruption, fetal exsanguination from a vasa previa, or a massive fetal-maternal hemorrhage. Persistent bearing-down efforts in the presence of a malposition can produce a similar heart rate pattern. When prolonged

decelerations occur in the terminally asphyxiated fetus, they usually have been preceded by a long sequence of late or variable decelerations and no variability. Under these circumstances, bradycardia is a very late sign of fetal hypoxia.

*When recurrent decelerations coexist with reduced variability, a major degree of fetal oxygen deprivation very probably exists.* Relative baseline tachycardia is common but is rarely above 160 beats/min unless maternal fever is present. Ominous patterns occur in the late stages of acute oxygen deprivation but can also be observed when central neurologic injury already exists. Although using all possible efforts to optimize uterine blood flow and to release cord compression is appropriate, recovery from these patterns is unlikely, and most often expedient delivery is necessary. Urgent intervention does not ensure delivery of a child that will survive or be neurologically intact, however.

### DECELERATION RECOVERY

In the evaluation of decelerations, recovery from a deceleration sequence means a return to the previously normal baseline rate *and* variability. When the fetus recovers from even significant hypoxia or ischemia, first the decelerations diminish in amplitude, then disappear; only then do the baseline rate and variability return to normal. Disappearance of decelerations alone or the apparent maintenance of FHR variability of the baseline is insufficient to declare the fetus "recovered." Regardless of the type or duration of the deceleration, recovery, as defined here, appears to make it highly improbable that brain injury has occurred during the episode. Abnormalities in the recovery of the *individual* variable or prolonged deceleration, whether because of late recovery or increased variability (i.e., atypical) variables, represent yet-uncompensated responses to the hemodynamic changes associated with head or umbilical cord compression.

Many classify the severity of decelerations based on their duration and the amplitude but not on recovery [47]. These former features have not been shown to be predictive of adverse outcome, and although some have suggested a benefit to measuring the duration or amplitude of the decelerations [127], for the most part, there appears to be little clinical benefit to making these distinctions. Paul and colleagues showed that the sever-

ity of the acidosis with late decelerations was primarily a function of the amount of variability in the baseline and not reasonably of the amplitude or the duration of the deceleration [128]. Zalar and Quilligan and Visser and others showed that pH was almost invariably normal in fetuses with poor variability, providing there were no decelerations [129]. Kubli and colleagues showed a similar dichotomous division of pH results with fetal tachycardia [130]. Those fetuses with tachycardia and no late decelerations had normal pH values, whereas those with late decelerations were more likely to be acidotic. Fetuses with persistent bradycardia during the second stage of labor are usually normal [62,75,90,131–135]. Thus, in the spirit of preventive care, there seems no reason in waiting until significant decelerations (those with early abnormalities of baseline rate or variability) reach a certain amplitude or duration before undertaking (usually conservative) methods of intervention.

Those experienced in reading EFM tracings are invited to look again at Figures 22.4, 22.7, and 22.9. Instead of looking at the entire tracing, however, imagine that the dotted boxes are opaque and that they begin with the onset of the contraction and cover the entire contraction along with the associated decelerations. Based only on the limited information available with the opacification of the fetal response to the contraction, the reader should attempt to answer the question of whether the description of the amplitude, duration, and type of deceleration makes a meaningful impact on the interpretation of the tracing. In Figure 22.7, the decelerations are variable and occur frequently, with every contraction. Indeed, the fetus spends more time decelerating than it does at baseline (see recommendation about pushing later). Notice also that despite the high frequency and amplitude of these variable decelerations, the baseline rate and the variability, which can be determined *after* each and every contraction, are maintained. Under these circumstances, what is being observed is probably the effects of intermittent head compression (in this case in an occiput posterior fetus), in which there is no basis to believe that the fetal condition is deteriorating or that the fetus is acidotic. These intermittent ischemic events are well tolerated by the fetus. No intervention is required as long as there is good progress in labor and reasonable expectation of safe vaginal delivery.

In Figure 22.9, there are recurrent late decelerations without variability and (relative) tachycardia. Does information about the duration, amplitude, or type of deceleration affect the interpretation or the need for intervention? In the authors' opinion, once decelerations are present in such a baseline pattern, intervention is required regardless of the type, amplitude, or duration of the decelerations. In this example, the fetus is hypoxic, acidotic, and might be injured. Immediate delivery is indicated but might not prevent a devastating outcome.

The tracing in Figure 22.4 is of a fetus with variable decelerations and a rising baseline with decreasing variability. The reader should ask, "Which feature in the deterioration of this tracing appears first? Is it the loss of variability, the rise in baseline rate, or the extension of the deceleration beyond the time of pushing?" The authors think that each feature is important, and that in the presence of any one of them, the deceleration has not been recovered. In the authors' opinion, when any of these features is encountered during the second stage of labor, pushing should be temporarily suspended, rather than attempting to "outrace" the fetal distress by requesting even more forceful pushing from the mother. (See Figure 22.10, B, C.) As an aid to recognizing these important features of the tracing, the authors encourage the user to duplicate the strategy of opacification of the events of the contraction as explained earlier by using a straight edge to highlight the previous baseline and then having the observer use his/her fingers to cover the contraction to determine how much if any residual deceleration or change in variability persists after the contraction or pushing.

According to these concepts, most decelerations developing during labor either represent no significant difficulty for the fetus or are remedial with conservative care (especially, in the second stage, with the temporary cessation of pushing). Thus, careful control of contraction frequency, avoiding excessive uterine activity (with or without oxytocin or prostaglandin [PG]), and regardless of any adverse response by the fetus, minimizing supine hypotension associated with maternal position or epidural anesthesia, and observing the fetal response to pushing in the second stage of labor can forestall decelerations from appearing in the first place. Even when decelerations are progressive, the change from a developing and usually recoverable hypoxia to persistent unrecoverable hypoxia is more or less grad-

ual, partly related to the strength and frequency of contractions and the amount of head or umbilical cord compression from descent and maternal expulsive efforts. In most instances, therefore, decelerations are correctable or failing that, permit timely intervention. Occasionally, most especially in the second stage, the transition can be abrupt, especially if the previous caveats have not been observed (see Figure 22.10A).

There are two patterns of "fetal distress" from which clinical recovery has not been documented and for which there can be no optimism about a salutary outcome: the pattern of injury and the agonal pattern preceding death. The *pattern of injury* consists of persistently absent variability and small variable decelerations with overshoot (see Figure 22.11) when there has been a previously reactive or reassuring nonstressed test (NST). The reversion of this pattern to a normal pattern is an extraordinarily rare event, although it has been seen in the presence of severe maternal ketoacidosis. The pattern appears in about 0.2% of all patients undergoing monitoring; however, it occurs in about 50% of fetuses (in cases reviewed for allegations of malpractice) subsequently developing neurologic disability (cerebral palsy) [136]. Even in a group of patients evaluated for malpractice, about one half of the fetuses will have suffered injury prior to labor [121]. Most patients with this pattern will have normal pH values; only those whose patterns show obvious decelerations also show acidosis. Thus, when one encounters this pattern under the circumstances described previously, it seems reasonable to anticipate a significant problem with the baby and to understand that even the most aggressive and timely management might not change the outcome.

The *agonal* pattern (see Figure 22.3B and C) demonstrates absent variability, an unstable baseline rate, and decelerations of varying type and severity. This pattern normally requires intervention as rapidly as possible with reasonable safety for mother and fetus [47]. Although failing to intervene surely results in fetal death, the fetus suffering from this pattern might die following delivery or be found to already be seriously handicapped regardless of the speed of intervention.

Distinguishing between the features of the sudden bradycardia or prolonged deceleration (see Figure 22.10A) and the agonal pattern (Figure 22.3C) is important. In the prolonged deceleration pattern

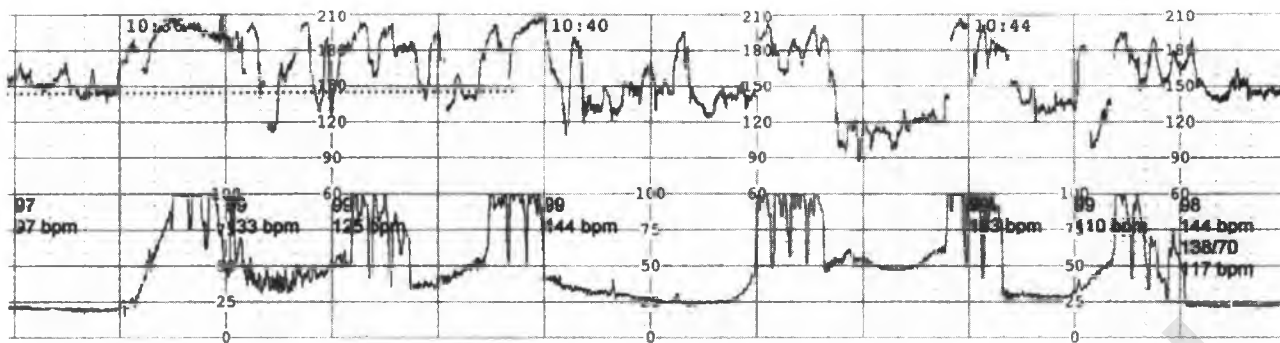


FIGURE 22.12.

*Second-stage EFM changes with a fetus in occiput posterior position. See text for discussion.*

as seen on Figure 22.10A, the preceding fetal heart rate pattern was normal or contained minimal decelerations unassociated with a rising baseline rate or decreased variability. The appearance of the sudden prolonged deceleration does confirm an acute ischemic stress, but it might not by itself be used to infer injury. At least as important, prompt intervention in the face of a sudden deceleration of this type could produce an intact child. As the legends for the individual tracings in this chapter emphasize, *the sudden appearance of a dramatic bradycardia in the absence of obvious predisposing factors (i.e., hypotension, uterine tetany) represents the only pattern in which the “crash” cesarean delivery seems justified without qualification.* In all other situations, there might be reason to anticipate recovery with correction of a predisposing factor. If the tracing is agonal, all in attendance must be reminded that although expedited delivery is reasonably dictated by the standard of care, intervention might be futile, and death or injury are inevitable and probably not preventable.

## THE SECOND STAGE OF LABOR

The second stage of labor begins when the cervix has reached full dilation, that is, it has retracted around the fetal head. Too frequently, however, this is accompanied by instructions to the patient to begin compulsive, closed-glottis pushing (bearing down). There is universal agreement that the frequency of decelerations increases dramatically in the second stage of labor, probably because of the effects of frequent uterine contractions and maternal expulsive forces on the fetal head and the cord.

In the first stage of labor, variable decelerations often reflect umbilical cord compression; in the second stage it is more likely to result from head compression [44,100]. These decelerating patterns are far more common in the occiput posterior position. Intermittent variable decelerations in the first stage of labor, the recovery of which should be anticipated, tend to recur with greater frequency and severity in the second stage.

Although no pattern seems absolutely unique to the second stage, increased variability, prolonged decelerations (i.e., second stage, or end-stage bradycardia), and rising baseline are especially common, as are variable decelerations (Figure 22.12, 22.10C) [75,90,131–135,137]. In Figure 22.12, an example of an occiput posterior-presenting fetus, variability, exaggerated accelerations, and recurrent variable decelerations accompany active second-stage pushing. In certain instances, a baseline cannot be established between contractions and pushing. Immediate modification of the pushing strategy is needed and will probably moderate these potentially confusing changes.

Alterations of the baseline, especially tachycardia, increase the likelihood of associated acidosis [90,133,138–141], findings that are confirmed in the bovine model [142]. Baseline bradycardia is not uncommon in the second stage; it is frequently related to the pushing strategy and only infrequently associated with adverse outcome or acidosis, especially if variability is maintained.

It is widely believed that FHR patterns during the second stage of labor are more common and more “abnormal” than those in the first stage. In the Dublin randomized control trial of monitoring, 40%



of second-stage patterns could not even be classified [5]. Indeed, several investigators have even suggested that second-stage FHR patterns require their own classification [143–145].

The authors' opinion is that the principles of interpretation of FHR patterns apply regardless of the timing of the tracing (i.e., antepartum, first stage, second stage). FHR patterns, the authors believe, including accelerations and decelerations during the second stage, are made more difficult to classify, not because of the inherent changes in their appearance but because of the frequency of contractions, but most important with the onset and conduct of maternal pushing [143]. In the first stage of labor, decelerations usually induce treatment to diminish the frequency of contractions and avoid additional stresses. In the second stage of labor, maternal pushing is often sustained despite decelerations. In this circumstance, there might be no opportunity for the fetus to recover from one contraction before the next one begins. It might not even be possible to determine the fetal baseline rate between contractions. As a result, decelerations pile one on top of the other, creating virtually any type or duration of deceleration pattern, including prolonged deceleration. The authors think that the need to assess the baseline rate and the variability after *each* deceleration is a critical part of monitoring, especially during the expulsive phase of the second stage of labor. *In the interests of fetal safety, there can be no excuse for the maintenance of pushing when there is an unrecovered deceleration or the baseline rate or variability is either abnormal or undetermined.*

Focusing specifically on the second stage helps to draw attention to the role of such factors as head position, compression, and reduction in CBF, as well as pushing strategy in the etiology of fetal cardiac decelerations during this period. Fetuses in the occiput posterior position, for example, demonstrate an increased likelihood of decelerations and adverse outcomes [64].

### Excessive Uterine Activity

Observations of the frequency and regularity of contractions also assist in the estimation of the feasibility of safe vaginal delivery (see later discussion) and whether there is excessive uterine activity. There are unfortunate and unnecessary disagreements about nomenclature and the need for intervention with

TABLE 22.9 Diagnosis: Excessive Uterine Activity\*

Parameter	Definition
Frequency	>5 UC in 10 min or preferably (>7 UC in 15 min)
Duration of UC	Contractions longer than 90 seconds
Interval between UC	<2 min from peak to peak
Tonus	With intrauterine pressure catheter – tone between contractions >20 mm HG With tocodynamometer – coupling, tripling of contractions or baseline tone <60 sec between UC
Duty cycle <sup>†</sup>	<50%

\*Note: Any parameter is sufficient to establish the diagnosis. See text for details.

<sup>†</sup>Duty cycle: term borrowed from mechanical engineering referring to the cycle of operation, starting, running, and stopping, that a motor on intermittent duty performs each time it runs. In this instance, the percentage of time (in 10 minutes) that the intrauterine pressure is above the resting tone.

excessive uterine activity [1]. For the purposes of this chapter, the authors do not use terms such as *hyperstimulation* (excessive uterine activity with or without an adverse fetal response), *tachysystole*, *polysystole*, or *hypertonus* but instead strictly define the several potential components of excessive uterine activity (Table 22.9) with the understanding that any of the abnormalities represents excessive uterine activity, and regardless of any fetal response should be scrupulously avoided if at all possible. Excessive uterine activity threatens the oxygen supply to the fetus, subjecting the fetus to excessive mechanical forces that could result in molding, intracranial hemorrhage, and trauma, and does not reasonably make the labor go any faster [146,147].

Many of the cases in which the fetus deteriorates during the second stage are related to excessive frequency of contractions and unrestrained maternal pushing. Safety requires that contractions appear no more often than every 2 minutes, or more preferably, no more than seven contractions in 15 minutes, and that pushing with the contraction should be resumed only if the baseline heart rate and variability have recovered to their previously normal level (see previous definition of recovery). Determining the significance of second-stage decelerations is best

approached by analyzing the decelerations without maternal pushing. To do this requires curtailing both the frequency of contractions (by reducing the oxytocin infusion rate) and the mother's pushing. If the deceleration lasts beyond the end of the contraction or the baseline rate rises (or cannot be determined) between contractions, the mother must modify her expulsive efforts temporarily. This might require considerable effort and discipline, especially if the mother is not receiving analgesia or epidural anesthesia.

Preferably, if contractions are close together, it is best in this situation to encourage the patient to push only with alternate contractions. As mentioned previously, without attending to the deceleration recovery, practitioners sometimes resort to even more exuberant pushing, trying to "outrace" the fetal distress. Such a strategy often precipitates either prolonged decelerations or the decision to perform a difficult and perhaps unsuccessful operative delivery. The first objective of fetal surveillance is to eliminate the fetal distress and then decide on the urgency and route of delivery. It is far more appropriate to keep the fetus out of harm's way than to rescue it afterward.

### Complications and Risks

In the absence of factors that place the mother at high risk for infection, internal monitoring probably plays a minimal role in fetal or maternal infection. Although monitoring might not be an important contributor to intrapartum or postpartum uterine infection, it seems reasonable to assume that pathogenic bacteria could be introduced into the uterine cavity by insertion of these devices and produce or promote infection in susceptible patients. Uncommonly, trauma to the uterus or the placenta has also been reported, with induction of maternal bleeding, abruption, or, most rarely, amniotic fluid embolism. Multiple attempts of insertion should be avoided, and restraint is advised in advancing the catheter too firmly into the uterus. There have also been wayward applications of the scalp electrode. Its application should be limited to the occiput, rarely the buttock, and not over the face or to an extremity. The authors emphasize that *in most instances, the external devices are quite sufficient for satisfactory monitoring of both FHR and uterine contractions*. When tracings are inadequate, the invasive

devices should be used, not for better quantification of any feature, but for the purpose of obtaining a reliable tracing. A direct electrode along with direct, continuous recording of maternal pulse is mandatory if there is any question of whether the pattern on the tracing is maternal or fetal [69,148–150].

More important than infection or direct injury from fetal monitoring devices are the consequences of interventions prompted or provoked by misinterpretation of monitoring data. Overreaction to abnormal but not serious FHR pattern features can lead to unnecessary and ill-advised surgical delivery. Similarly, failure to appreciate FHR pattern abnormalities can result in a failure to intervene appropriately and can contribute to perinatal mortality and morbidity. Clinicians willing to commit to understanding the nuances and pitfalls of fetal monitoring will be able to interpret monitoring data, minimizing inappropriate interventions and maximizing the probability of good obstetric outcomes.

### Pitfalls

Given all the insights that fetal monitoring provides about the condition of the fetus, why is it subjected to so much criticism and skepticism about its value? Rather than further analysis of RCTs, the merits and limitations of which have been discussed elsewhere [10,11,151,152], instead the discussion now focuses on those relevant technical and philosophical issues that have received broad discussion in the literature.

### NOMENCLATURE

Although several attempts have been made to standardize nomenclature, diverse systems of limited compatibility are in vogue throughout the world [44,153–156]. It is also clear that these classifications function primarily to direct attention to phenomena about the tracing (e.g., decelerations, variability, tachycardia). In each case, the classification is deemed relevant only to the detection of and response to the hypoxia and acidosis, eschewing available insights into fetal behavior and neurologic injury (see later discussion).

These drawbacks have doubtless contributed to the current official disparagement of the term *fetal distress*. As the term is commonly employed by clinicians, *fetal distress* is an abnormal FHR pattern thought to represent oxygen deprivation. The

TABLE 22.10 Electronic Fetal Monitoring: Technical Problems

Problem	Comment
Half-count	Generally rates over 180–200 beats/min, external Rates over 210–240 beats/min, depending on device used
Double-count	Generally rates below 80–90 beats/min
Variability	Can be diminished with external transducers or increased with older transducers
Modified counting	
• With ultrasound scan	Arrhythmia detection in edit mode: Monitor will not detect rapid changes in beat to beat if interval difference >30 beats/min/beat
• With scalp electrode	Complex fetal ECG Maternal-fetal conduction: normally about one fourth (Maternal ECG always present on fetal ECG tracing)
Misidentification	
• With ultrasound scan	Misdirection of transducer to record second twin or, less frequently, a maternal vessel. Common in early labor: usually there is an obvious distinction between maternal and fetal (much higher) rates
• With fetal scalp monitoring	Maternal heart rate displays as fetal heart rate with a dead fetus, under unique circumstances

ECG, electrocardiograph

distress is seldom qualified further or quantified at all. In addition, there are clearly other kinds of stress, some potentially as devastating to the fetus as systemic hypoxia, including infection, anemia, arrhythmia, anomaly, and localized ischemia of the brain or other organs related to perfusion or mechanical problems. The terms *fetal intolerance to labor* and *nonreassuring fetal status*, however, surely underscore the vagrancy of current classifications to provide insight into the mechanism(s) of the problem, the likelihood of recovery, the interaction among various FHR patterns or a basis on which to relate the events of labor to subsequent outcome. This lack of uniformity in classification also explains why there are no computerized models for the interpretation of intrapartum FHR patterns and even why “alarm” systems once installed are so readily abandoned [68].

The problem of nomenclature is best illustrated by a brief discussion of the requirements for a *reactive NST*. In designating a pattern as “reactive,” physicians do not mention the specific baseline rate if it is in the normal range, or the amount of variability, or the fact that there are periods when the baseline variability is poor or absent. The entire pattern has a name. Except perhaps for the agonal pattern, no other combination of features has a specific name; the pattern is simply described by its various features, including the amplitude and duration of accelerations or decelerations [1].

### Technical Issues

Technical issues must be understood as well (Table 22.10). Tracing abnormalities are found predominantly during the expulsive phase of labor (second stage), when technical difficulties are most likely to interfere with proper registration. The source of the FHR and UC signals (internal or external) influences both the accuracy of the interpretation and the potential for misrepresentation of the baseline rate and the variability. The monitor might even misrepresent the maternal heart rate pattern for the fetal pattern, with potentially catastrophic results (Figures 22.13 and 22.14) [69,148–150,157]. In the latter instance, a low Apgar fetus underwent a vacuum extraction after prolonged second-stage labor. Subgaleal and subarachnoid hemorrhages were diagnosed after delivery. The otherwise normal, 3,000-g infant died at 10 days of age. On review of the tracing, several experts opined that the tracing was normal in every respect and that no obvious explanation of the injuries was forthcoming. This tracing is indeed normal – for the mother. The normal fetus descending through the birth canal in the second stage is unlikely to have symmetric accelerations such as are noted on this tracing; much more likely, it would show significant varying decelerations. The nurse’s notes of the mother’s vital signs confirmed that her heart rate was 140 to 160 beats/min, coinciding with the values on the fetal



FIGURE 22.13.

*Fetal/maternal/double count (upper panel). Two different rates are recorded. This is a single fetus with single tachycardia (180 beats/min). A, The excursions to 90 beats/min represents a half-count, an artifact with an external transducer. The diagnosis was confirmed after the application of a direct scalp electrode with simultaneous maternal heart rate monitoring with a pulse oximeter. The increased rate, lack of decelerations, and frequent contractions seen in A suggest a nonhypoxic mechanism – probably maternal fever. B, Indicates a sudden shift from maternal to fetal heart rate due to readjustment of the external monitor. See text for discussion.*

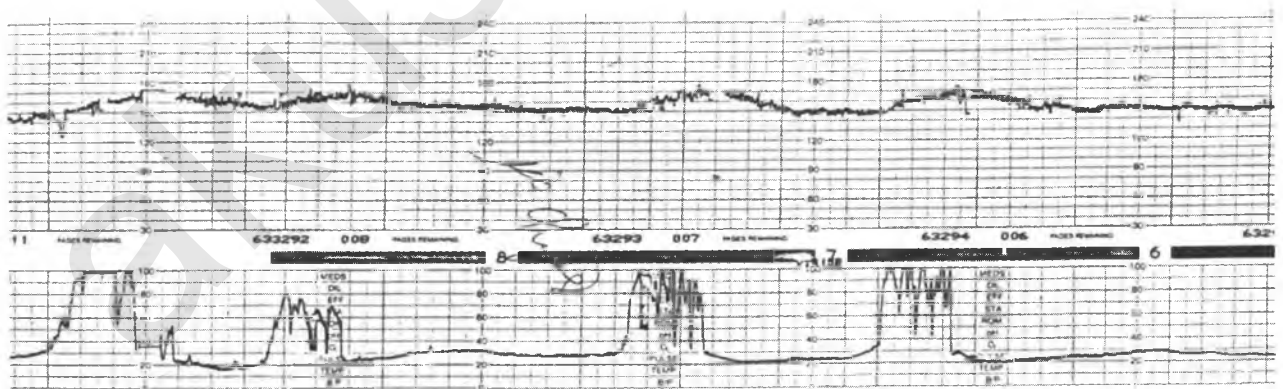


FIGURE 22.14.

*Artifact: Maternal tracing believed, in error, to be fetal. This was associated with a fetus born with remarkably low Apgar scores after prolonged second-stage labor and vacuum delivery. Case was also complicated by subgaleal and subarachnoid hemorrhages. See text for additional details.*

monitor. Notice that the baseline is quite stable and that the smooth symmetric accelerations in the tracings coincide with the mother's pushing efforts. The difficult to discern and occasional snatches of only a second heart rate trace on the tape probably represent the true FHR – in a pattern of deceleration or prolonged bradycardia.

To understand the relationship of abnormal outcomes and FHR tracings, the clinician must be certain that the recorded tracing is indeed that of the fetus and not the mother. This cannot be done reliably by palpating the maternal pulse. Both tracings must be affirmatively demonstrated as should easily be demonstrated on a modern monitor. As a minimum, if there is any question about the source of the tracing, the mother must cease pushing until the true state of affairs is established.

Note in particular Figure 22.13 and Figure 22.14. Both panels reflect artifactual patterns, one involving the maternal heart rate. Figure 22.13 reflects a technical problem, or *half count*. Note Figure 22.13, in which the tracing initially records the maternal heart rate pattern then reverts to the FHR following readjustment. The appearance of the maternal heart rate pattern on the fetal tracing is a common occurrence during fetal monitoring. It is more common and potentially dangerous in the second stage, when alterations of transducer position are common as the result of pushing efforts. In this circumstance, the sudden shift from one stable baseline to another with a markedly different rate and a different pattern of normal variability establishes the correct diagnosis. If the rates were closer, the distinction might not be as obvious. Indeed, the authors' experience suggests that the misinterpretation of the maternal heart rate pattern in the second stage of labor is becoming one of the more common allegations of failure in malpractice claims.

### Fetal Blood Sampling

In the adult, tissue pH is maintained normally within a narrow range by both pulmonary and renal mechanisms. The fetus must rely on placental function for respiratory gas transfer and elimination of bicarbonate and organic acids, however. Under normal conditions, gradients for hydrogen ion and carbon dioxide across the placenta are small. As a consequence of factors discussed previously (i.e., enhanced affinity of fetal blood for oxygen, high fetal cardiac output and systemic blood flow rates, anatomic uniqueness

of the fetal circulation), oxygen availability to fetal tissues is maintained under a wide range of circumstances. Despite its low prevailing plasma  $PO_2$  level, tissue oxygen consumption in the fetus is thus sustained at rates that are as high as or higher than those of an adult.

Acute interruptions in placental respiratory gas transfer occur most commonly during labor because of interruption of uterine or umbilical blood flow. Brief impairment in flow on either side of the placenta results in a primarily respiratory acidosis. More prolonged oxygen restriction provokes anaerobic metabolism and the production of lactate, resulting in a combined respiratory and metabolic acidosis. Respiratory acidosis is generally rapidly reversible once placental perfusion resumes, or, if delivery has occurred, respiration is initiated in the newborn. Metabolic acidosis resolves more slowly and can linger following a hypoxic insult even after the fetal  $PO_2$  has returned to normal.

The most direct information about fetal acid-base homeostasis is obtained by fetal capillary blood sampling. This technique was first introduced by Saling in the 1960s to screen fetuses for acidosis [83]. It is now used infrequently, primarily as a corollary technique to FHR monitoring. When heart rate patterns are equivocal, or further information is required to determine the trend in fetal condition, scalp blood sampling can be useful. The technique is not absolutely required for the clinical interpretation of FHR patterns, but some practitioners find it refines their ability to identify hypoxemia that requires intervention. Experienced personnel and properly maintained equipment must be available for accurate and reproducible results.

### *Fetal Blood Sampling Technique*

Blood is obtained from the presenting part of the fetus, generally the scalp, which is visualized with a conical vaginal amnioscope. To obtain the blood sample, membranes must be ruptured and the cervix dilated more than 2 cm. After a site for puncture away from fontanelles and sutures is identified, the skin is dried with a cotton swab and a thin layer of silicone lubricant is applied over the anticipated puncture site. The incision is made with a guarded lancet, which prevents penetration beyond 2 mm. Blood is collected in a heparinized capillary tube, and measurements of pH and blood gases are

determined by standard microtechniques. To obtain the most meaningful results, the sample should be taken between contractions, with the patient in the lateral position. After the blood is obtained, it is prudent to maintain pressure over the incision at least until the next contraction has abated. The site is then observed to ensure that bleeding has stopped.

The measurement of pH in fetal capillary blood samples is the most reproducible measurement. If the sample is of sufficient volume and laboratory facilities are available, a complete set of blood gas analyses is desirable; this allows one to distinguish the respiratory from the metabolic component of acidosis and reduces the risk of unnecessary intervention.

During labor, most normal fetuses have scalp blood pH values between 7.25 and 7.40. It is, however, a mistake to assume that pH values below 7.25 indicate significant central acidosis and risk of neurologic compromise. In fact, it is quite unusual to see asphyxial tissue injury with a cord artery pH above 7.15 at delivery, and there is no pH value at which injury is universal [84,85]. The pH tends to remain stable or to diminish minimally during the first stage of labor. Although some studies suggest that the pH drops much more rapidly during the second stage of labor, this fall can be prevented or minimized by having the mother avoid the supine position during the second stage [86].

Most obstetricians consider fetal scalp capillary blood values less than 7.20 to be abnormal and requiring further evaluation, although the exact level is controversial [87]. Low or equivocal results should be repeated immediately and at subsequent intervals if confirmation of a trend is important to guide decision making. Occasionally, a simultaneous maternal blood gas sample is necessary to rule out passive acidemia resulting from placental hydrogen ion transfer during maternal acidosis [88]. If fetal blood gas analysis indicates that a nonremedial primary fetal metabolic acidosis is present, or if the pH is found to fall despite efforts to improve the situation, prompt delivery is indicated.

Scalp blood sampling is necessary in only a small percentage of cases. It is of little value to obtain a baseline scalp blood pH when the FHR pattern is normal, even if meconium is present or the pregnancy is otherwise at high risk. Most fetuses whose acid-base status deteriorates during labor have normal pH values at the onset. In addition, ominous

FHR patterns that do not respond promptly to intrauterine resuscitation maneuvers do not require confirmation by scalp sampling. Expedient delivery is preferable.

For those who use scalp sampling, remember that transient fetal respiratory acidosis is common during labor, especially with varying decelerations. A low pH obtained in the presence of variable decelerations is of limited value unless it has been obtained just prior to the deceleration or well after it has recovered. A sample obtained during the deceleration might yield a decreased pH that simply represents a respiratory acidosis that will resolve promptly as soon as umbilical blood flow returns to normal or once delivery and ventilation of the newborn are accomplished.

### **LABOR AND SUBSEQUENT NEUROLOGIC HANDICAP**

The correlation of FHR patterns with neonatal outcome was originally confined to immediate outcome based on hypoxia, death, acidosis of umbilical cord blood, or admission to the NICU [10–13]. A normal FHR pattern virtually precludes fetal hypoxia. Although abnormal patterns show an increased likelihood of hypoxia, acidosis, and adverse outcome, most abnormal patterns fail to show any correlation with either.

Regarding FHR patterns and long-term neurologic outcome, a normal pattern, especially normal fetal behavior, correlates strongly with normal outcome. “Abnormal” FHR patterns are indeed associated with abnormal neurologic activity (seizures) and injury, including subsequent cerebral palsy both before and during labor. Paneth and colleagues found a pooled relative risk for handicap with abnormal patterns of about two – a modest relationship [10]. Others have found similar results [114,158]. Nevertheless, most such abnormal patterns based on hypoxia fail to show any correlation with adverse outcome. These generalizations apply despite differences in classification of FHR pattern.

Before highlighting the use of EFM in the prospective diagnosis of fetal neurologic injury and its potential value in neonatal neuroprotection, we should review some basic principles of fetal injury. Acute progressive intrapartum asphyxia sustained until delivery is usually associated with severe acidosis and inevitably results in obvious disturbances of neonatal function and adaptation, including

low Apgar scores; disordered consciousness; abnormalities of movement, tone, and posture; and frequently seizures [159]. These are typical constituents for the neonatal syndrome of hypoxic-ischemic encephalopathy (HIE). Most asphyxial injuries developing during labor, however, do not present with this constellation of findings.

Numerous pitfalls await those who attempt to define the severity of intrapartum asphyxia or the risk of subsequent injury on the basis of an umbilical pH or other neonatal criteria suggested by various guidelines [69,113,160–164]. No umbilical pH value seems to distinguish those babies who will suffer injury [165–167].

When the ischemic injury is the result of more long-standing intermittent focal or even catastrophic event, or when mechanical or traumatic factors are present, the neonate might not meet the criteria usually considered essential to the diagnosis of perinatal asphyxial brain injury [136,162,167–170]. In studies by Shields and Schifrin and Korst and others, only about 20% of those fetuses injured during labor satisfied all of the “essential” criteria of perinatal asphyxia [136,169], whereas several neonates, injured prior to delivery and unlikely candidates for neuroprotection in the immediate neonatal period, would have met the required criteria [121,136,171].

### Neuroradiologic Studies

Neuroradiologic studies support the notion that these modalities can reasonably determine the distribution of the lesions in the brain, their timing, mechanism, and severity of insult, and are reliably predictive of later neurodevelopmental syndromes. They also suggest that neuroprotection strategies might preferentially improve certain types of lesions [172,180–183].

Most hypoxic injury in the newborn appears to be of recent (perinatal) origin [184], regardless of clinical presentation or prenatal risk factors. Focal ischemic lesions, arterial or venous “neonatal strokes,” generally are not associated with severe encephalopathy at birth but probably occur at or near the time of birth [170,185–188]. Neither the appearance of cerebral edema nor the timing of seizures resolves the timing of the injury with necessary precision [189–191]. Rutherford and coworkers found that hypothermia decreased basal ganglia

and thalamic lesions [173]. This decrease was significant in infants with a moderate EEG finding but not in those with a severe EEG finding. Finally, MRI examinations of newborns born in good condition but who later suffer seizures reveal a high incidence of hemorrhagic or ischemic lesions almost certainly acquired around the time of birth [192].

### Fetal Heart Rate Patterns: Diagnosis of Neurologic Injury

As originally conceived, EFM was predicated on the benefits of the early detection of and timely intervention for fetal hypoxia. The test of hypoxia was the presence of certain specific decelerations. In 1967, the individual decelerations were thought to be so unique that they were named according to the presumed mechanism [51]. Thus, late decelerations were thought to represent uteroplacental insufficiency (UPI), variable decelerations were thought to represent cord compression (CC), and early decelerations were thought to represent head compression (HC).

Figure 22.5 illustrates the importance of understanding the provenance of the patterns. It illustrates a pattern of late decelerations and no variability with tachycardia. If the pattern *preceding* this tracing is a normal reactive pattern, then the progression of the pattern from normal to ominous represents neglect. If conversely the fetus were admitted with diminished variability, even without decelerations, then it is likely that the fetus was injured on admission. In this latter situation, immediate intervention, although seemingly required, is unlikely to modify the outcome.

To understand the potential for using FHR patterns for detecting neurologic injury, it is helpful to adopt a principle that is widely used for the neurologic evaluation and prognosis of an infant or adult after a near-drowning accident. Asphyxiation is now understood to have an obvious, immediate impact on neurologic function, but regardless of the severity of asphyxia and immediate neurologic manifestations, the diagnosis of permanent neurologic injury must await the clearing of acidosis, hypoxia, and cerebral edema. Only then does a neurologic prognosis become reasonable.

In the studies of Ikeda and coworkers on fetal lambs, the umbilical cord circulation was interrupted completely to a level of profound hypoxia

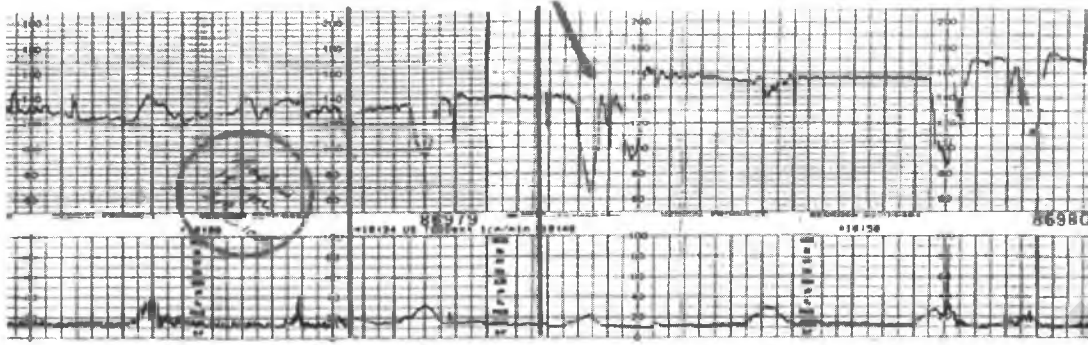


FIGURE 22.15.

*Conversion pattern. Normal EFM pattern for first half of trace. After variable decelerations, the baseline suddenly rises to a new stable rate with no variability (arrow). (Note paper rate at 1cm; normally this is 3 cm.)*

and acidosis (a pH <6.9, and a base deficit >20 mmol) [193]. This was accompanied, as expected, by a profound fetal bradycardia. The occlusion was then relieved, and the lambs were allowed to recover. This was accompanied by recovery of the fetal bradycardia. Later the animals were sacrificed and their brains examined. The authors found no correlation between the severity of the histologic brain damage and the duration of the fetal bradycardia or the severity of the hypoxia or acidosis. Rather, it was the duration of hypotension, its attendant ischemia, and the FHR patterns after recovery of the bradycardia that best correlated with the severity of neuropathologic injury. Several features of this study have critical relevance to the analysis of intrapartum events and injury. The mechanism of injury was an acute, abrupt ischemic assault reaching profound levels of acidosis and base deficit. Neither the severity of the acidosis or the duration of the response (i.e., bradycardia) was directly related to the severity of neurologic injury, however. The factor that was clearly related to the severity of injury was the duration of the diminished perfusion of the brain (i.e., the ischemia), which could be measured in the experiment but cannot be measured directly in the human fetus. The only clinically relevant factor that could be related to the severity of injury was the appearance of the FHR pattern after the event. Incidentally, despite the duration and the severity of the assault, when the obstruction to blood flow in the umbilical cord was removed, the pH and the base deficit returned to normal, but the injury remained.

These experiments reveal the potential for acid-base recovery from an obvious and severe asphyxial

insult, the variability of neurologic injury – regardless of severe asphyxia and the potential relevance of the FHR pattern – after the insult. No estimate of severity could be gleaned from the type or duration of the FHR pattern during the acute episode.

These findings would seem to contradict the strict requirements for abnormalities in Apgar score, pH, and neonatal encephalopathy that have been promulgated. Such findings apply to a model of hypoxic injury as progressive, unrelenting, and systemic, in which the hypoxia is accompanied by a relentless deterioration of the pH and severe systemic manifestations of hypoxia at the end of labor. This progressive injury model assumes that a threshold of acidosis is required for a fetus to suffer neurologic injury and makes no accommodation for a profound hypoxic event from which the fetus has recovered metabolically but not neurologically, as in the experiments of Ikeda and colleagues.

Numerous EFM tracings illustrate that injury can occur during an hypoxic/ischemic episode from which the fetus recovers from hemodynamic and metabolic standpoints but not from a neurologic one (Figure 22.15, 22.16) [48,194]. In these circumstances the decelerations may disappear, and the baseline rate may return to normal, but the variability usually remains absent. In some other instances, neurologic injury is associated with increased “variability” and an unusual respiratory drive in the fetus (Figure 22.17) [195]. Behavioral cycles in the fetus, however, are usually not maintained. On occasion, the transition is obvious and dramatic, as in the sudden development of ischemic or hemorrhagic injury [48]. Figure 22.15 illustrates the dramatic



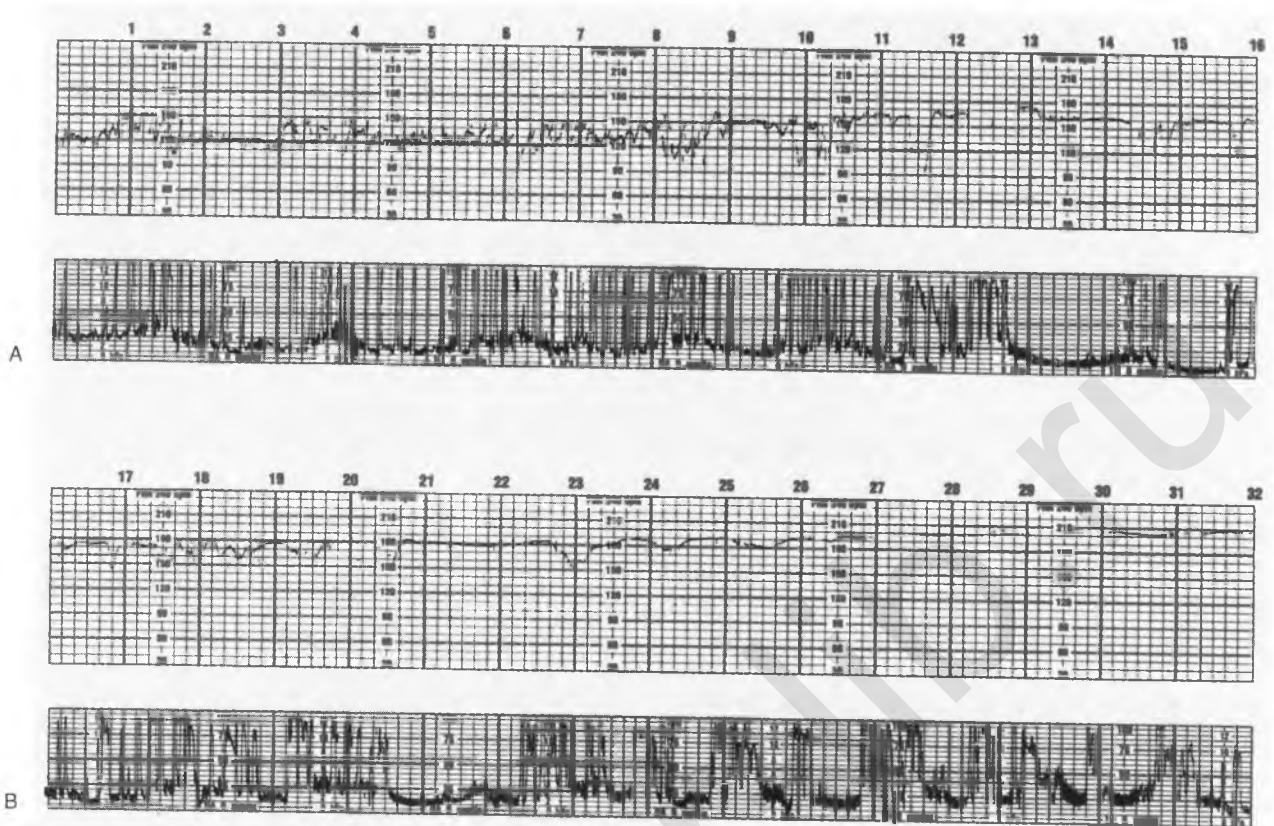


FIGURE 22.16.

*Terminal tachycardia. Occiput posterior presentation and second-stage labor. In panel A note the sudden appearance of sustained tachycardia and no variability. The tachycardia is preceded by recurrent varying decelerations, increased variability, and decreased baseline heart rate during maternal pushing. After minute 18 (panel B) and disappearance of decelerations, a persisting tachycardia with no variability is observed. This infant was born with massive intracranial hemorrhage but normal acid–base balance.*

conversion of a previously normal tracing to one of absent variability in a fetus suffering a stroke during labor. In the *conversion pattern* depicted, the EFM pattern is initially normal; then, in the middle of the tracing, an acute change occurs. The baseline rises acutely, and the variability disappears. In a similar fashion, the rapid pattern change seen in Figure 22.16 strongly suggests acute ischemic neurologic injury during labor unassociated with systemic hypoxia or acidosis, in this instance doubtless predisposed to by malpresentation and active second stage labor. The severity of the fetal injury, however, cannot be predicted from these data alone.

These events, far more common than realized, are compatible only with a regional ischemic event, not one of progressive systemic asphyxia. The appearance of such tracings further underscores the limi-

tations of trying to identify neurologic problems in the fetus using a nosology of decelerations designed to uncover systemic hypoxia.

Although the pattern of persistently absent variability and varying decelerations with overshoot shows an extraordinary correlation with subsequent adverse neurologic outcome, it cannot be used in any way to ascertain the severity of injury, or that its continuation necessarily represents ongoing deterioration of the fetal condition (Figure 22.11) [121]. The determination of injury under these circumstances is not based solely on the presence of a tracing with no variability and other features. To define injury or the potential for it, one must either demonstrate or have reason to believe that the pattern of injury represents the conversion from a previously normal FHR pattern [121]. This distinction is

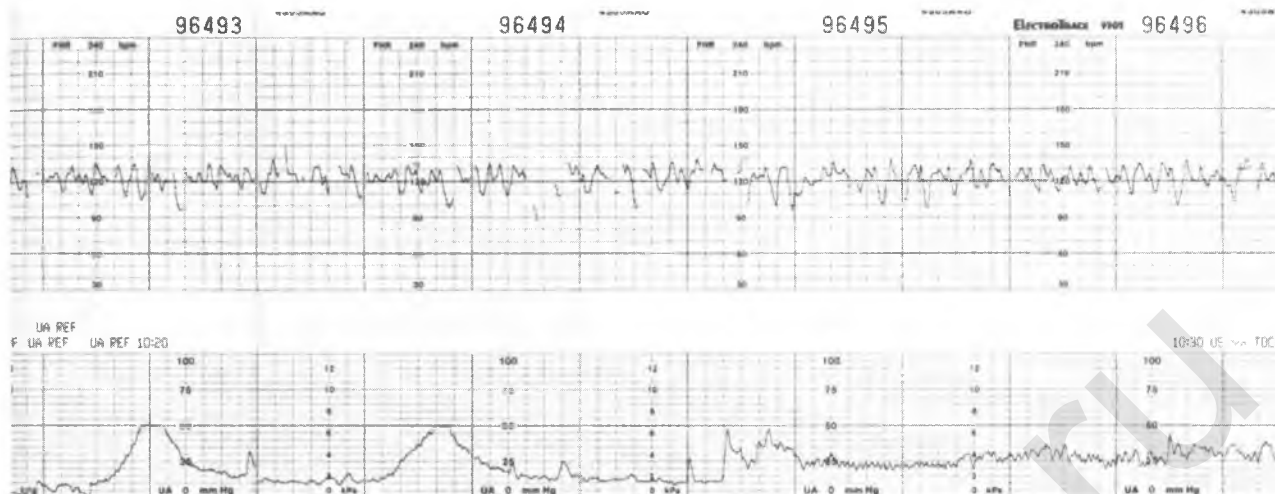


FIGURE 22.17.

*Fetal gasping (convulsions) in a previously normal fetus. Notice stable baseline rate, persistently exaggerated variability, and no decelerations. This was accompanied by obvious fetal breathing (gasp) activity as visualized on a concomitant ultrasound scan. When this pattern is persistent and associated with extraordinary fetal body movements, as in this case, a neurologic abnormality (seizures) is strongly suggested.*

important because this pattern of injury can be emulated in all respects by the administration of atropine to the mother or fetus [196–199]. This pattern also appears in the noninjured fetus as a result of prematurity, extreme tachycardia from any cause, including adrenergic drugs or a congenital anomaly. Abnormalities of variability in the heart rate pattern are also seen in older children with cerebral palsy and in brain-damaged adults [200].

### CESAREAN DELIVERY RATE AND ANCILLARY DEVICES

Most studies suggest that EFM increases the cesarean rate without apparent benefit for the fetal outcome because of the high incidence of abnormal FHR patterns that do not reflect severe acidosis. As a result, there has been considerable interest in techniques that increase the sensitivity interpretation of the nonreassuring pattern. Pulse oximetry was shown to reduce the number of hypoxia-related anesthetic deaths in adults [201], and it appeared logical that, despite considerable technical problems, it could be profitably applied to fetal surveillance. For this purpose, the U.S. Food and Drug Administration (FDA) approved the marketing of the Nellcor N-400 fetal pulse oximeter as an adjunct to FHR monitoring. In the largest study

of the value of pulse oximetry. Fetal intrapartum oxygen saturation was monitored in singleton term fetuses with a nonreassuring heart rate pattern. This does indeed result in a reduction in cesarean delivery rates for nonreassuring fetal heart rate patterns, but there is no difference in the immediate neonatal outcomes or the overall cesarean delivery rates, because of increased abdominal deliveries for dystocia. Given these data, one has difficulty understanding the net value of the device for its stated purpose, and its routine clinical use has been discouraged. Perhaps a broader perspective is necessary. Specific changes in FHR pattern were originally intended to assess fetal oxygenation; that is, unique decelerations from contractions would allow early detection of fetal hypoxia and early intervention (or rescue). Because it is not a direct measure of fetal oxygenation and is considered as having insufficient specificity and sensitivity to identify the fetus in oxygen debt correctly, EFM cannot fulfill this promise.

Attempts at intrauterine resuscitation during episodes of variable FHR decelerations should invoke the same procedures to maximize uterine blood flow as described previously for treating late decelerations. In addition, clinicians should employ maneuvers to reduce or eliminate cord entrapment or head compression. These maneuvers include changing the mother's position and, especially

during the second stage, modulating or ceasing maternal bearing-down efforts. Placing the patient in knee-chest position sometimes alters the relations of the fetus, cord, and uterine wall and thereby eliminates umbilical cord compression. If there is no response to these standard maneuvers in the presence of recurrent severe variable decelerations, consider elevation of the presenting part before surgical intervention [202]. This maneuver should usually be undertaken in an operating suite, in the event of cord prolapse. Occasionally, an occult prolapse or another form of entrapment can be alleviated by this approach.

### TIMING OF INTERVENTION

Many obstetricians and the early purveyors of EFM will be disappointed that, except in rare instances, the FHR pattern by itself is not the major determinant of the timing of intervention. Indeed, most interventions in labor are performed for dystocia or dysfunctional labor. To optimize the timing of intervention, clinicians must be aware of the types of FHR patterns and mechanisms that produce injury, the speed of deterioration, and the options for recoverability. As detailed previously, these parameters have received little attention. Clearly, some patterns develop slowly with ample, noncritical opportunities for intervention with the expectation of normal outcome. Other patterns progress more rapidly, and the timing of intervention becomes critical if the fetus is to be rescued. Still other patterns develop so rapidly and unpredictably that timely intervention is improbable, and no matter how fast the intervention, the fetus might not escape injury or death. In other circumstances, the opportunity for timely intervention has long passed, and intervention, required by the standard of care, might not change the inevitable handicap or death. There has been little discussion of these issues. More common, however, are discussions of the propriety of a "30-minute rule," a concept that holds that with "certain nonreassuring" FHR patterns, the decision-incision interval for the performance of cesarean must be within 30 minutes. *The authors are unaware of any study that correlates this classification of urgency/benefit with the speed of intervention.*

Perhaps the benefits of EFM can be better understood if the perspective of its role is changed from

*rescue to prevention.* Realizing this role for EFM requires that the medical personnel attending the parturient continually answer three questions:

- Does anything have to be done for the mother?
- Does anything have to be done for the fetus?
- What is the feasibility of safe vaginal delivery?

It seems axiomatic that regardless of any abnormality of the FHR pattern; labor can be permitted only if there is reasonable expectation of the feasibility of safe vaginal delivery. The decelerations that can be tolerated at full dilation of the cervix with a head ready to deliver cannot reasonably be tolerated in early labor before descent of the head, rupture of the membranes, and the potential for cord compression – factors likely to increase the decelerations further. Indeed, the lower the feasibility of safe vaginal delivery, the lower should be the threshold for intervention for abnormal FHR patterns, regardless of the severity of the FHR pattern or the amount of acidosis.

In the patient with a nonprogressive labor and a fetus demonstrating a rising baseline with decreasing variability and recurrent moderate-to-severe decelerations, what is the benefit of waiting for an abnormal fetal oxygen saturation or critically low pH? Abnormal FHR patterns, in fact, anticipate difficulty in labor and the need for cesarean delivery for dystocia. Perhaps decelerations occur for a reason. The increased frequency of decelerations, operative intervention, and subsequent adverse neurologic outcome with fetuses in the occiput posterior positions have been known for some time. It stands to reason that the longer and more difficult the labor, the greater the chance of decelerations from fetal head or cord compression and for subsequent harm.

Continued, unproductive labor can be associated with increased molding and deeper lodging of the fetal head in the pelvis. Unproductive labor can provoke protracted fetal bradycardia or make ultimate operative delivery, by either vaginal or abdominal, that much more difficult. The question then arises: what is the value of delaying delivery in the face of changes in FHR pattern that can only represent increasing fetal stress or distress (e.g., recurrent variable decelerations with a rising baseline and decreasing variability, one of the options for using pulse oximetry in prior studies) in a protracted labor with

the fetus in occiput posterior position because the fetal pulse oximetry or the fetal scalp pH is normal? Delaying intervention, waiting until the fetal condition deteriorates or becomes critical before intervention, seems to violate one of the fundamental tenets of obstetric care – keeping the mother and fetus out of harm's way.

The responsibility of the obstetrician is to identify those deviations from normal FHR patterns that signal developing metabolic or behavioral problems in the fetus and to apply this interpretation to the maternal condition and the feasibility of safe vaginal delivery. The fetal situation can be improved by intrauterine treatment, or failing that, by timely delivery if this interpretation is applied. The more problematic the feasibility of safe vaginal delivery, the less “distress” that is tolerable without intervention. Similarly, patterns that might provoke intervention in the first stage of labor can be reasonably tolerated in anticipation of safe vaginal delivery in the very near future. Except rarely, it is difficult to use specific FHR patterns to define the need for emergency delivery.

When the FHR pattern is reactive, there is reasonable diagnostic certainty that there is no fetal indication for intervention. This does not exclude intervention on the basis of fetal size, presentation, or position; pelvic size; or a maternal condition. The evaluation of the nonreassuring FHR pattern therefore cannot be carried out in isolation. The clinician, focusing on the three questions, must estimate the probability that an intervention could benefit the outcome, considering that what could benefit the mother might not benefit the baby, and vice versa. The strength of such evaluations is modified by the reliability and predictability of the information, the skill of the interpreter, and the clinical context. The approach taken here emphasizes the need to examine and codify trends in FHR patterns to identify the boundaries that separate the normally oxygenated and normally behaving fetus from the hypoxemic one, to distinguish types of hypoxia and ischemia, and, with these data, better identify potentially injurious processes from others of lesser risk.

The authors have tried to avoid discussing tracings as a “snapshot” of various patterns without discussion of their evolution. Such limited perspective cannot permit an accurate assessment of the fetal condition or thoughtful deliberation about the

urgency and route of delivery. Assessing nonreassuring patterns would seem to require less attention to specific features of the types and timing of decelerations or variability, but greater attention to changes in these values over time – to use the fetus as its own control. The normal tracing with a stable baseline, cyclic accelerations and variability, and no decelerations defines absent hypoxia and normal fetal behavior. Fetuses do not deteriorate without first modifying their FHR baseline and variability, even in the presence of decelerations.

If patterns thought to represent fetal compromise were present at the outset of labor, then any adverse outcome most likely represents an antepartum insult. If such patterns appear after an initially normal FHR, however, then less severe signs of “distress” were certainly present. Rescue at this time might not allow a normal outcome.

The authors' opinion is that much more is known about the determinants of FHR patterns than is currently used clinically in their analysis or classification. Perinatal asphyxia does not mean that the fetus was injured. Perinatal injury does not mean that the fetus was asphyxiated or that it was preventable. The evaluation of these cases, therefore, requires that testing must be performed properly and expeditiously; the results must be interpreted and communicated correctly by an agreed-on widely understood classification, and the interpretation must provoke an appropriate response in the larger clinical context relating to the condition of the mother and the feasibility of safe vaginal delivery. To a greater or lesser extent, all parts of this “package” must be included in any evaluation of the reproducibility of EFM, to its comparison with alternatives, and finally to the evaluation of its role in the allegation of obstetric negligence.

Finally, all of these modalities (e.g., EFM, fetal pulse oximetry, pH) should at least anticipate, if not prevent, fetal death in labor, because this outcome, unlike neurologic injury, is universally associated with progressive hypoxia and acidosis. Whether any of these modalities, by a single value or feature or even a combination thereof, can dictate the critical time of delivery of the fetus with a problematic FHR pattern and a problematic labor is yet unresolved and seems improbable. There still is a place for skilled interpretation of FHR patterns, for understanding the expected course of labor, and for the art of obstetrics in caring for the parturient in labor.

## REFERENCES

1. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 70: Intrapartum Fetal Heart Rate Monitoring. *Obstet Gynecol* 2005; 106(6):1453–1460.
2. Paul RH, Gauthier RJ, Quilligan EJ. Clinical fetal monitoring: The usage and relationship to trends in cesarean delivery and perinatal mortality. *Acta Obstet Gynecol Scand* 1980;59(4):289–295.
3. Visser GHA, Narayan H. The problem of increasing severe neurological morbidity in newborn infants: Where should the focus be? *Prenatal Neonatal Med* 1996;1:12–15.
4. Stanley FJ. Cerebral palsy trends: Implications for perinatal care. *Acta Obstet Gynecol Scand* 1994;73(1):5–9.
5. MacDonald D, Grant A, Sheridan-Pereira M, Boylan P, Chalmers I. The Dublin randomized controlled trial of intrapartum fetal heart rate monitoring. *Am J Obstet Gynecol* 1985;152(5):524–539.
6. Vintzileos AM, Antsaklis A, Varvarigos I, Papas C, Sofatzis I, Montgomery JT. A randomized trial of intrapartum electronic fetal heart rate monitoring versus intermittent auscultation. *Obstet Gynecol* 1993;81(6):899–907.
7. Shy KK, Luthy DA, Bennett FC, Whitfield M, Larson EB, van Belle G, Hughes JP, Wilson JA, Stenchever MA. Effects of electronic fetal-heart-rate monitoring, as compared with periodic auscultation, on the neurologic development of premature infants. *N Engl J Med* 1990;322(9):588–593.
8. Shy KK, Olshan AF, Hickok DE, Luthy DA. Electronic fetal monitoring during premature labor and the occurrence of perinatal mortality in very low birthweight infants. *Birth* 1988;15(1):14–18.
9. Leveno KJ, Cunningham FG, Nelson S, Roark M, Williams ML, Gozick D, Dowling S, Rosenfield CR, Buckley A. A prospective comparison of selective and universal electronic fetal monitoring in 34,995 pregnancies. *N Engl J Med* 1986;315(10):615–619.
10. Paneth N, Bommarito M, Stricker J. Electronic fetal monitoring and later outcome. *Clin Invest Med* 1993;16(2):159–165.
11. Chalmers I. Continuous fetal heart rate monitoring. *J R Soc Med* 2001;94(5):258.
12. Donker DK, van Geijn HP, Hasman A. Interobserver variation in the assessment of fetal heart rate recordings. *Eur J Obstet Gynecol Reprod Biol* 1993; 52(1):21–28.
13. Bernardes J, Costa-Pereira A, Ayres-de-Campos D, van Geijn HP, Pereira-Leite L. Evaluation of interobserver agreement of cardiocograms. *Int J Gynaecol Obstet* 1997;57(1):33–37.
14. Devoe L, Golde S, Kilman Y, Morton D, Shea K, Waller J. A comparison of visual analyses of intrapartum fetal heart rate tracings according to the new National Institute of Child Health and Human Development guidelines with computer analyses by an automated fetal heart rate monitoring system. *Am J Obstet Gynecol* 2000;183(2):361–366.
15. Simpson N, Oppenheimer LW, Siren A, Bland E, McDonald O, McDonald D, Dabrowski A. Accuracy of strategies for monitoring fetal heart rate in labor. *Am J Perinatol* 1999;16(4):167–173.
16. Parer J, Nageotte M. Intrapartum Fetal Surveillance. In: Creasy RK, Resnick R, eds. *Maternal Fetal Medicine: Principles and Practice*, 5th ed. Philadelphia: WB Saunders; 2004; pp. 403–427.
17. Luttkus AK, Noren H, Stupin JH, Blad S, Arulkumar S, Erkkola R, Hagberg H, Lenstrup C, Visser GH, Tamazian O, Yli B, Rosen KG, Dudenhausen JW. Fetal scalp pH and ST analysis of the fetal ECG as an adjunct to CTG: A multi-center, observational study. *J Perinat Med* 2004;32(6):486–494.
18. Rosen KG. Fetal electrocardiogram waveform analysis in labour. *Curr Opin Obstet Gynecol* 2005; 17(2):147–150.
19. Noren H, Blad S, Carlsson A, Flisberg A, Gustavsson A, Lilja H, Wennergren M, Hagberg H. STAN in clinical practice – the outcome of 2 years of regular use in the city of Gothenburg. *Am J Obstet Gynecol* 2006;195(1):7–15.
20. Lent M. The medical and legal risks of the electronic fetal monitor. *Stanford Law Rev* 1999;51(4):807–837.
21. Schifrin BS, Amsel J, Burdorf G. The accuracy of auscultatory detection of fetal cardiac decelerations: A computer simulation. *Am J Obstet Gynecol* 1992;166(2):566–576.
22. Goodwin L. Intermittent auscultation of the fetal heart rate: A review of general principles. *J Perinat Neonatal Nurs* 2000;14(3):53–61.
23. Menticoglou SM, Harman CR. Problems in the detection of intrapartum fetal asphyxia with intermittent auscultation. *Aust N Z J Obstet Gynaecol* 1999;39(2):218–222.
24. Feinstein NF. Fetal heart rate auscultation: Current and future practice. *J Obstet Gynecol Neonatal Nurs* 2000;29(3):306–315.

25. Gilles MT, Norman M, Dawes V, Gee V, Rouse I, Newnham J. Intermittent auscultation for the intrapartum assessment of fetal well-being in Western Australia. *Aust N Z J Obstet Gynaecol* 1997;37(2):143–148.
26. Miller FC, Pearse KE, Paul RH. Fetal heart rate pattern recognition by the method of auscultation. *Obstet Gynecol* 1984;64(3):332–336.
27. Benson RC, Shubeck F, Deutschberger J, Weiss W, Berendes H. Fetal heart rate as a predictor of fetal distress: A report from the collaborative project. *Obstet Gynecol* 1968;32(2):259–266.
28. Morrison JC, Chez BF, Davis ID, Martin RW, Roberts WE, Martin JN Jr, Floyd RC. Intrapartum fetal heart rate assessment: Monitoring by auscultation or electronic means. *Am J Obstet Gynecol* 1993;168(1 Pt 1):63–66.
29. Vintzileos AM, Nochimson DJ, Guzman ER, Knuppel RA, Lake M, Schifrin BS. Intrapartum electronic fetal heart rate monitoring versus intermittent auscultation: A meta-analysis. *Obstet Gynecol* 1995;85(1):149–155.
30. Cetrulo CL, Schifrin BS. Fetal heart rate patterns preceding death in utero. *Obstet Gynecol* 1976;48(5):521–527.
31. Borell U, Fernstroem I, Ohlson L, Wiqvist N. Influence of uterine contractions on the uteroplacental blood flow at term. *Am J Obstet Gynecol* 1965;93:44–57.
32. Borell U, Fernstroem I, Ohlson L, Wiqvist N. Effect of uterine contractions on the human uteroplacental blood circulation: An arteriographic study. *Am J Obstet Gynecol* 1964;89:881–890.
33. Yarkoni S, Hobbins JC. Intrapartum fetal activity. *J Perinat Med* 1987;15(3):316–22.
34. Spencer JA, Johnson P. Fetal heart rate variability changes and fetal behavioural cycles during labour. *Br J Obstet Gynaecol* 1986;93(4):314–321.
35. Mulder EJ, Visser GH, Bekedam DJ, Prechtel HF. Emergence of behavioural states in fetuses of type-1-diabetic women. *Early Hum Dev* 1987;15(4):231–251.
36. Sokol RJ. Occiput posterior position and FHR patterns. *Obstet Gynecol* 1981;57(2):266–267.
37. Ponkey SE, Cohen AP, Heffner LJ, Lieberman E. Persistent fetal occiput posterior position: Obstetric outcomes. *Obstet Gynecol* 2003;101(5 Pt 1):915–920.
38. Fitzpatrick M, McQuillan K, O'Herlihy C. Influence of persistent occiput posterior position on delivery outcome. *Obstet Gynecol* 2001;98(6):1027–1031.
39. Watson WJ, Read JA. Electronic fetal monitoring in face presentation at term. *Mil Med* 1987;152(6):324–325.
40. Sherer DM, Miodovnik M, Bradley KS, Langer O. Intrapartum fetal head position I: Comparison between transvaginal digital examination and transabdominal ultrasound assessment during the active stage of labor. *Ultrasound Obstet Gynecol* 2002;19(3):258–63.
41. Benedetti TJ, Lowensohn RI, Truscott AM. Face presentation at term. *Obstet Gynecol* 1980;55(2):199–202.
42. van Geijn HP, Copray FJ, Donkers DK, Bos MH. Diagnosis and management of intrapartum fetal distress. *Eur J Obstet Gynecol Reprod Biol* 1991;42:S63–S72.
43. Van Geijn HP. Developments in CTG analysis. *Baillieres Clin Obstet Gynaecol* 1996;10(2):185–209.
44. Ingemarsson E, Ingemarsson I, Solum T, Westgren M. Influence of occiput posterior position on the fetal heart rate pattern. *Obstet Gynecol* 1980;55(3):301–304.
45. The National Institute of Child Health and Human Development Research Planning Workshop. Electronic fetal heart rate monitoring: Research guidelines for interpretation. *J Obstet Gynecol Neonatal Nurs* 1997;26(6):635–640.
46. Nicolaides KH, Sadovsky G, Visser GH. Heart rate patterns in normoxemic, hypoxemic, and anemic second-trimester fetuses. *Am J Obstet Gynecol* 1989;160(5 Pt 1):1034–1037.
47. National Institute of Child Health and Human Development Research Planning Workshop. Electronic fetal heart rate monitoring: Research guidelines for interpretation. *Am J Obstet Gynecol* 1997;177(6):1385–1390.
48. Asakura H, Schifrin BS, Myers SA. Intrapartum, atraumatic, non-asphyxial intracranial hemorrhage in a full-term infant. *Obstet Gynecol* 1994;84(4 Pt 2):680–683.
49. Schifrin BS, Ater S. Fetal hypoxic and ischemic injuries. *Curr Opin Obstet Gynecol* 2006;18(2):112–122.
50. Wax JR, Emmerich M, Eggleston MK, Jr. Intrapartum fetal atrial bigeminy – diagnostic and

- therapeutic role of the fetal scalp stimulation test. *Am J Obstet Gynecol* 1996;174(5):1649–1650.
51. Vesel S, Završnik T, Podnar T. Successful outcome in a fetus with an extremely low heart rate due to isolated complete congenital heart block. *Ultrasound Obstet Gynecol* 2003;21(2):189–191.
  52. Nakamura S, Maeno Y, Matsuishi T. Complete atrioventricular block associated with atrial flutter in fetus with positive maternal anti-SSA antibody. *Pediatr Cardiol* 2003;24(2):182–183.
  53. Koike T, Minakami H, Shiraishi H, Sato I. Fetal ventricular rate in case of congenital complete heart block is increased by ritodrine: Case report. *J Perinat Med* 1997;25(2):216–218.
  54. Klein V, Repke JT. Supraventricular tachycardia in pregnancy: Cardioversion with verapamil. *Obstet Gynecol* 1984;63(3 Suppl):16S–18S.
  55. Breur JM, Oudijk MA, Stoutenbeek P, Visser GH, Meijboom EJ. Transient non-autoimmune fetal heart block. *Fetal Diagn Ther* 2005;20(2):81–85.
  56. Begg L, East C, Chan FY, Brennecke S. Intrapartum fetal oxygen saturation monitoring in congenital fetal heart block. *Aust N Z J Obstet Gynaecol* 1998;38(3):271–274.
  57. Bjorklund AO, Adamson UK, Almstrom NH, Enocksson EA, Gennser GM, Lins PE, Westgren LM. Effects of hypoglycaemia on fetal heart activity and umbilical artery Doppler velocity waveforms in pregnant women with insulin-dependent diabetes mellitus. *Br J Obstet Gynaecol* 1996;103(5):413–420.
  58. Kaneko M, Sameshima H, Ikeda T, Ikenoue T, Minematsu T. Intrapartum fetal heart rate monitoring in cases of cytomegalovirus infection. *Am J Obstet Gynecol* 2004;191(4):1257–1262.
  59. Matias A, Xavier P, Bernardes J, Patricio B. Fetal heart-rate monitoring during maternal hypoglycaemic coma: A case report. *Eur J Obstet Gynecol Reprod Biol* 1998;79(2):223–225.
  60. Wen LZ, Xing W, Liu LQ, Ao LM, Chen SH, Zeng WJ. Cytomegalovirus infection in pregnancy. *Int J Gynaecol Obstet* 2002;79(2):111–116.
  61. van Ravenswaaij-Arts CM, Kollee LA, Hopman JC, Stoeltinga GB, van Geijn HP. Heart rate variability. *Ann Intern Med* 1993;118(6):436–447.
  62. Parer JT, Livingston EG. What is fetal distress? *Am J Obstet Gynecol* 1990;162(6):1421–1425; discussion 5–7.
  63. Martin CB, Jr. Physiology and clinical use of fetal heart rate variability. *Clin Perinatol* 1982;9(2):339–352.
  64. Ross BA. Control of heart rate in infancy. *J Perinatol* 1989;9(2):188–190.
  65. Visser GH, Mulder EJ, Stevens H, Verweij R. Heart rate variation during fetal behavioural states 1 and 2. *Early Hum Dev* 1993;34(1–2):21–8.
  66. Nijhuis JG. Fetal behavior. *Neurobiol Aging* 2003;24 Suppl 1:S41–46; discussion S7–S9; S51–S52.
  67. Devoe LD, Murray C, Faircloth D, Ramos E. Vibroacoustic stimulation and fetal behavioral state in normal term human pregnancy. *Am J Obstet Gynecol* 1990;163(4 Pt 1):1156–1161.
  68. Abrams RM, Gerhardt KJ, Burchfield DJ. Behavioral state transition and local cerebral blood flow in fetal sheep. *J Dev Physiol* 1991;15(5):283–288.
  69. Schifrin BS. The CTG and the timing and mechanism of fetal neurological injuries. *Best Pract Res Clin Obstet Gynaecol* 2004;18(3):437–456.
  70. Horimoto N, Koyanagi T, Maeda H, Satoh S, Takashima T, Minami T, Nakano H. Can brain impairment be detected by in utero behavioural patterns? *Arch Dis Child* 1993;69(1 Spec No):3–8.
  71. Koyanagi T, Horimoto N, Maeda H, Kukita J, Minami T, Ueda K, Nakano H. Abnormal behavioral patterns in the human fetus at term: Correlation with lesion sites in the central nervous system after birth. *J Child Neurol* 1993;8(1):19–26.
  72. Horimoto N, Koyanagi T, Nakano H, Ueda K. The in utero determination of lesion site affecting the central nervous system assessed by abnormal behavioral patterns in the human fetus. *No To Hattatsu* 1992;24(2):147–151.
  73. Ayres-de-Campos D, Costa-Santos C, Bernardes J. Prediction of neonatal state by computer analysis of fetal heart rate tracings: The antepartum arm of the SisPorto multicentre validation study. *Eur J Obstet Gynecol Reprod Biol* 2005;118(1):52–60.
  74. Nijhuis IJ, ten Hof J, Mulder EJ, Nijhuis JG, Narayan H, Taylor DJ, Visser GH. Fetal heart rate in relation to its variation in normal and growth-retarded fetuses. *Eur J Obstet Gynecol Reprod Biol* 2000;89(1):27–33.
  75. Gaziano EP, Freeman DW, Bendel RP. FHR variability and other heart rate observations during second stage labor. *Obstet Gynecol* 1980;56(1):42–47.

76. Maulik D, Saini V, Zigrossi ST. Clinical significance of short-term variability computed from heart-rate waveforms. *J Perinat Med* 1983;11(5):243-248.
77. Caldeyro-Barcia R. Mechanisms and significance of the variations registered in the heart rate of the human fetus during labor. *Tokoginecol Pract* 1964; 23:387-433.
78. Visser GH, Bekedam DJ, Ribbert LS. Changes in antepartum heart rate patterns with progressive deterioration of the fetal condition. *Int J Biomed Comput* 1990;25(4):239-246.
79. Visser GH, Goodman JD, Levine DH, Dawes GS. Diurnal and other cyclic variations in human fetal heart rate near term. *Am J Obstet Gynecol* 1982; 142(5):535-544.
80. de Vries JI, Visser GH, Mulder EJ, Prechtl HF. Diurnal and other variations in fetal movement and heart rate patterns at 20-22 weeks. *Early Hum Dev* 1987;15(6):333-348.
81. Patrick J, Carmichael L, Chess L, Probert C, Staples C. The distribution of accelerations of the human fetal heart rate at 38 to 40 weeks' gestational age. *Am J Obstet Gynecol* 1985;151(2):283-287.
82. Petrie RH. Effect of drugs and anesthetics on the fetal heart rate. *Semin Perinatol* 1978;2(2):147-153.
83. O'Brien-Abel NE, Benedetti TJ. Saltatory fetal heart rate pattern. *J Perinatol* 1992;12(1):13-17.
84. Cibils LA. Clinical significance of fetal heart rate patterns during labor. I. Baseline patterns. *Am J Obstet Gynecol* 1976;125(3):290-305.
85. Nijhuis IJ, ten Hof J, Nijhuis JG, Mulder EJ, Narayan H, Taylor DJ, Visser GH. Temporal organization of fetal behavior from 24-weeks gestation onwards in normal and complicated pregnancies. *Dev Psychobiol* 1999;34(4):257-268.
86. Murata Y, Martin CB, Jr., Ikenoue T, Hashimoto T, Taira S, Saqawa T, Sakata H. Fetal heart rate accelerations and late decelerations during the course of intrauterine death in chronically catheterized rhesus monkeys. *Am J Obstet Gynecol* 1982;144(2):218-223.
87. Itskovitz J, Goetzman BW, Rudolph AM. The mechanism of late deceleration of the heart rate and its relationship to oxygenation in normoxemic and chronically hypoxemic fetal lambs. *Am J Obstet Gynecol* 1982;142(1):66-73.
88. Schiffrin BS, Artenos J, Lyseight N. Late-onset fetal cardiac decelerations associated with fetal breathing movements. *J Matern Fetal Neonatal Med* 2002; 12(4):253-259.
89. Herbert CM, Boehm FH. Prolonged end-stage fetal heart rate deceleration: A reanalysis. *Obstet Gynecol* 1981;57(5):589-593.
90. Krebs HB, Petres RE, Dunn LJ. Intrapartum fetal heart rate monitoring. V. Fetal heart rate patterns in the second stage of labor. *Am J Obstet Gynecol* 1981;140(4):435-439.
91. Kazandi M, Sendag F, Akercan F, Terek MC, Gundem G. Different types of variable decelerations and their effects to neonatal outcome. *Singapore Med J* 2003;44(5):243-247.
92. Westgate JA, Bennet L, de Haan HH, Gunn AJ. Fetal heart rate overshoot during repeated umbilical cord occlusion in sheep. *Obstet Gynecol* 2001; 97(3):454-459.
93. Tortosa MN, Acien P. Evaluation of variable decelerations of fetal heart rate with the deceleration index: Influence of associated abnormal parameters and their relation to the state and evolution of the newborn. *Eur J Obstet Gynecol Reprod Biol* 1990;34(3):235-245.
94. Saito J, Okamura K, Akagi K, Tanigawara S, Shintaku Y, Watanabe T, Akiyama N, Endo C, Sato A, Yajima A. Alteration of FHR pattern associated with progressively advanced fetal acidemia caused by cord compression. *Nippon Sanka Fujinka Gakkai Zasshi* 1988;40(6):775-780.
95. Murata Y, Quilligan EJ, Ninomiya Y, Wakatsuki A, Masaoka N, Oka S, Fujimori K. Variable fetal heart rate decelerations and electrocortical activities. *Am J Obstet Gynecol* 1994;170(2):689-692.
96. Richardson BS, Carmichael L, Homan J, Johnston L, Gagnon R. Fetal cerebral, circulatory, and metabolic responses during heart rate decelerations with umbilical cord compression. *Am J Obstet Gynecol* 1996;175(4 Pt 1):929-936.
97. Itskovitz J, LaGamma EF, Rudolph AM. Effects of cord compression on fetal blood flow distribution and O<sub>2</sub> delivery. *Am J Physiol* 1987;252(1 Pt 2):100-109.
98. Itskovitz J, LaGamma EF, Rudolph AM. Heart rate and blood pressure responses to umbilical cord compression in fetal lambs with special reference to the mechanism of variable deceleration. *Am J Obstet Gynecol* 1983;147(4):451-457.
99. Ball RH, Espinoza MI, Parer JT, Alon E, Vertommen J, Johnson J. Regional blood flow in asphyxiated



- fetuses with seizures. *Am J Obstet Gynecol* 1994; 170(1 Pt 1):156–161.
100. Ball RH, Parer JT. The physiologic mechanisms of variable decelerations. *Am J Obstet Gynecol* 1992;166(6 Pt 1):1683–1688; discussion 8–9.
  101. Ball RH, Parer JT, Caldwell LE, Johnson J. Regional blood flow and metabolism in ovine fetuses during severe cord occlusion. *Am J Obstet Gynecol* 1994;171(6):1549–1555.
  102. Ozden S, Demirci F. Significance for fetal outcome of poor prognostic features in fetal heart rate traces with variable decelerations. *Arch Gynecol Obstet* 1999;262(3–4):141–149.
  103. Yagel S, Anteby E, Lavy Y, B Chetrit A, Palti Z, Hochner-Celnikier D, Ron M. Fetal middle cerebral artery blood flow during normal active labour and in labour with variable decelerations. *Br J Obstet Gynaecol* 1992;99(6):483–485.
  104. Tadmor O, Bocker Y, Rabinowitz R, Aboulafia Y, Yagel S, Stark M, Diamant YZ, Nitzan M. Analysis of umbilical artery flow parameters during fetal variable decelerations using computerized Doppler waveforms. *Fetal Diagn Ther* 1999;14(1): 2–10.
  105. Fujii EY, Takahashi N, Kodama Y, Roman C, Ferrero DM, Parer JT. Hemodynamic changes during complete umbilical cord occlusion in fetal sheep related to hippocampal neuronal damage. *Am J Obstet Gynecol* 2003;188(2):413–418.
  106. Llanos AJ, Riquelme RA, Sanhueza EM, Herrera E, Cabello G, Giussani DA, Parer JT. Regional brain blood flow and cerebral hemispheric oxygen consumption during acute hypoxaemia in the llama fetus. *J Physiol* 2002;538(Pt 3):975–983.
  107. King T, Parer J. The physiology of fetal heart rate patterns and perinatal asphyxia. *J Perinat Neonatal Nurs* 2000;14(3):19–39; quiz 102–103.
  108. Llanos A, Riquelme R, Sanhueza E, Gaete C, Cabello G, Parer J. Cardiorespiratory responses to acute hypoxemia in the chronically catheterized fetal llama. *Comp Biochem Physiol A Mol Integr Physiol* 1998;119(3):705–709.
  109. Mueller-Heubach E, Battelli AF. Variable heart rate decelerations and transcutaneous  $P_{O_2}$  during umbilical cord occlusion in fetal monkeys. *Am J Obstet Gynecol* 1982;144(7):796–802.
  110. Aldrich CJ, D'Antona D, Spencer JA, Delpy DT, Reynolds EO, Wyatt JS. Fetal heart rate changes and cerebral oxygenation measured by near-infrared spectroscopy during the first stage of labour. *Eur J Obstet Gynecol Reprod Biol* 1996;64(2):189–195.
  111. Aldrich CJ, D'Antona D, Spencer JA, Wyatt JS, Peebles DM, Delpy DT, Reynolds EO. Late fetal heart decelerations and changes in cerebral oxygenation during the first stage of labour. *Br J Obstet Gynaecol* 1995;102(1):9–13.
  112. Fleischer A, Schulman H, Jagani N, Mitchell J, Randolph G. The development of fetal acidosis in the presence of an abnormal fetal heart rate tracing. I. The average for gestational age fetus. *Am J Obstet Gynecol* 1982;144(1):55–60.
  113. American College of Obstetricians and Gynecologists. Neonatal encephalopathy and cerebral palsy: Defining the pathogenesis and pathophysiology. Washington, DC: American College of Obstetricians and Gynecologists; 2003.
  114. Nelson KB, Dambrosia JM, Ting TY, Grether JK. Uncertain value of electronic fetal monitoring in predicting cerebral palsy. *N Engl J Med* 1996;334(10):613–618.
  115. Giussani DA, Spencer JA, Moore PJ, Bennet L, Hanson MA. Afferent and efferent components of the cardiovascular reflex responses to acute hypoxia in term fetal sheep. *J Physiol* 1993;461:431–449.
  116. Jensen A, Hanson MA. Circulatory responses to acute asphyxia in intact and chemodenervated fetal sheep near term. *Reprod Fertil Dev* 1995;7(5): 1351–1359.
  117. Liggins GC, Qvist J, Hochachka PW, et al. Fetal cardiovascular and metabolic responses to simulated diving in the Weddell seal. *J Appl Physiol* 1980; 49(3):424–430.
  118. Zapol WM, Liggins GC, Schneider RC, Qvist J, Snider MT, Creasy RK, Hochachka PW. Regional blood flow during simulated diving in the conscious Weddell seal. *J Appl Physiol* 1979;47(5):968–973.
  119. Clapp JF, Peress NS, Wesley M, Mann LI. Brain damage after intermittent partial cord occlusion in the chronically instrumented fetal lamb. *Am J Obstet Gynecol* 1988;159(2):504–509.
  120. Clapp JF 3rd, Mann LI, Peress NS, Szeto HH. Neuropathology in the chronic fetal lamb preparation: Structure-function correlates under different environmental conditions. *Am J Obstet Gynecol* 1981; 141(8):973–986.
  121. Schifrin BS, Hamilton-Rubinstein T, Shields JR. Fetal heart rate patterns and the timing of fetal injury. *J Perinatol* 1994;14(3):174–181.

122. Kates RB, Schifrin BS. Fetal cardiac asystole during labor. *Obstet Gynecol* 1986;67(4):549–555.
123. Boehm FH, Woodruff LF, Jr., Growdon JH, Jr. The effect of lumbar epidural anesthesia on fetal heart rate baseline variability. *Anesth Analg* 1975; 54(6):779–782.
124. Thomas G. The aetiology, characteristics and diagnostic relevance of late deceleration patterns in routine obstetric practice. *Br J Obstet Gynaecol* 1975;82(2):121–125.
125. Amano K, Nishijima M, Arai M. Effects of local anesthetics on fetal heart rate patterns and neonatal neurobehavior. *Nippon Sanka Fujinka Gakkai Zasshi* 1985;37(11):2291–2299.
126. Schifrin BS. Fetal heart rate patterns following epidural anaesthesia and oxytocin infusion during labour. *J Obstet Gynaecol Br Commonw* 1972;79 (4):332–339.
127. American College of Obstetricians and Gynecologists. Practice Bulletin. Clinical Management Guidelines for Obstetrician-Gynecologists, Number 70, December 2005 (Replaces Practice Bulletin Number 62, May 2005). Intrapartum fetal heart rate monitoring. *Obstet Gynecol* 2005;106 (6):1453–1460.
128. Paul RH, Suidan AK, Yeh S, Schifrin BS, Hon EH. Clinical fetal monitoring. VII. The evaluation and significance of intrapartum baseline FHR variability. *Am J Obstet Gynecol* 1975;123(2):206–210.
129. Zalar RW Jr, Quilligan EJ. The influence of scalp sampling on the cesarean section rate for fetal distress. *Am J Obstet Gynecol* 1979;135(2):239–246.
130. Kubli FW, Hon EH, Khazin AF, Takemura H. Observations on heart rate and pH in the human fetus during labor. *Am J Obstet Gynecol* 1969; 104(8):1190–1206.
131. Katz M, Shani N, Meizner I, Insler V. Is end-stage deceleration of the fetal heart ominous? *Br J Obstet Gynaecol* 1982;89(3):186–189.
132. Sheiner E, Hadar A, Hallak M, Katz M, Mazor M, Shoham-Vardi I. Clinical significance of fetal heart rate tracings during the second stage of labor. *Obstet Gynecol* 2001;97(5 Pt 1):747–752.
133. Gilstrap LC 3rd, Hauth JC, Toussaint S. Second-stage fetal heart rate abnormalities and neonatal acidosis. *Obstet Gynecol* 1984;63(2):209–213.
134. Pregazzi R, Venuleo V, Levi D'Ancona R, Bouche C, Ricci G, Toffoletti FG. Fetal bradycardia during the second stage of labor: Study of 72 cases. *Minerva Ginecol* 1992;44(1–2):5–8.
135. Roberts JE. Managing fetal bradycardia during second stage of labor. *MCN Am J Matern Child Nurs* 1989;14(6):394–398.
136. Shields JR, Schifrin BS. Perinatal antecedents of cerebral palsy. *Obstet Gynecol* 1988;71(6 Pt 1): 899–905.
137. Loghis C, Salamalekis E, Panayotopoulos N, Vitoratos N, Zourlas PA. The effect of early second-stage bradycardia on newborn status. *Eur J Obstet Gynecol Reprod Biol* 1997;72(2):149–152.
138. Low JA, Cox MJ, Karchmar EJ, McGrath MJ, Pancham SR, Piercy WN. The prediction of intrapartum fetal metabolic acidosis by fetal heart rate monitoring. *Am J Obstet Gynecol* 1981;139(3): 299–305.
139. Strachan BK, Sahota DS, van Wijngaarden WJ, James DK, Chang AM. Computerised analysis of the fetal heart rate and relation to acidemia at delivery. *BJOG* 2001;108(8):848–852.
140. Bernardes J, Costa-Pereira A. Maternal fever in term labour in relation to fetal tachycardia, cord artery acidemia and neonatal infection. *Br J Obstet Gynaecol* 1998;105(2):241; author reply -2.
141. Honjo S, Yamaguchi M. Umbilical artery blood acid-base analysis and fetal heart rate baseline in the second stage of labor. *J Obstet Gynaecol Res* 2001; 27(5):249–254.
142. Jonker FH, Van Geijn HP, Chan WW, Rausch WD, Van der Weijden GC, Taverne MA. Characteristics of fetal heart rate changes during the expulsive stage of bovine parturition in relation to fetal outcome. *Am J Vet Res* 1996;57(9):1373–1381.
143. Piquard F, Hsiung R, Mettauer M, Schaefer A, Haberey P, Dellenbach P. The validity of fetal heart rate monitoring during the second stage of labor. *Obstet Gynecol* 1988;72(5):746–751.
144. Van Lierde M, De Muylder X, Thomas K. Fetal heart rate in the second stage of labour: Method of interpretation and parameters of the newborn. *Gynecol Obstet Invest* 1979;10(2–3):119–126.
145. Melchior J, Bernard N. Second-stage fetal heart rate patterns. In: Jad S, ed. *Fetal Monitoring, Physiology and Techniques of Antenatal and Intrapartum Assessment*. Tunbridge Wells, UK: Castle House Publications; 1989.
146. Effer SB, Bertola RP, Vrettos A, Caldeyro-Barcia R. Quantitative study of the regularity of uterine contractile rhythm in labor. *Am J Obstet Gynecol* 1969;105(6):909–915.

147. Oppenheimer LW, Bland ES, Dabrowski A, Holmes P, McDonald O, Wen SW. Uterine contraction pattern as a predictor of the mode of delivery. *J Perinatol* 2002;22(2):149–153.
148. Klapholz H, Schifrin BS, Myrick R. Role of maternal artifact in fetal heart rate pattern interpretation. *Obstet Gynecol* 1974;44(3):373–378.
149. Murray ML. Maternal or fetal heart rate? Avoiding intrapartum misidentification. *J Obstet Gynecol Neonatal Nurs* 2004;33(1):93–104.
150. Sherman DJ, Frenkel E, Kurzweil Y, Padua A, Arieli S, Bahar M. Characteristics of maternal heart rate patterns during labor and delivery. *Obstet Gynecol* 2002;99(4):542–547.
151. Parer JT. Obstetric technologies: What determines clinical acceptance or rejection of results of randomized controlled trials? *Am J Obstet Gynecol* 2003;188(6):1622–1625; discussion 5–8.
152. Parer JT. Electronic fetal heart rate monitoring: A story of survival. *Obstet Gynecol Surv* 2003;58(9):561–563.
153. FIGO Study Group on the Assessment of New Technology. International Federation of Gynecology and Obstetrics. Intrapartum surveillance: Recommendations on current practice and overview of new developments. *Int J Gynaecol Obstet* 1995;49(2):213–221.
154. Bernardes J, Pereira AC. Some concerns about the new research guidelines for interpretation of electronic fetal heart rate monitoring. *Am J Obstet Gynecol* 1998;179(2):560–561.
155. Ayres-de Campos D, Bernardes J, Garrido A, Marques-de-Sa J, Pereira-Leite L. SisPorto 2.0: A program for automated analysis of cardiotocograms. *J Matern Fetal Med* 2000;9(5):311–318.
156. Amer-Wahlin I, Bordahl P, Eikeland T, Hellsten C, Noren H, Sornes T, Rosen KG. ST analysis of the fetal electrocardiogram during labor: Nordic observational multicenter study. *J Matern Fetal Neonatal Med* 2002;12(4):260–266.
157. Schifrin B, Harwell R, Hamilton-Rubinstein T, Visser GH. Maternal heart rate pattern – a confounding factor in intrapartum fetal surveillance. *Prenat Neonat Med* 2001;6:75–682.
158. Spencer JA, Badawi N, Burton P, Keogh J, Pemberton P, Stanley F. The intrapartum CTG prior to neonatal encephalopathy at term: A case-control study. *Br J Obstet Gynaecol* 1997;104(1):25–28.
159. Perlman JM, Risser R. Can asphyxiated infants at risk for neonatal seizures be rapidly identified by current high-risk markers? *Pediatrics* 1996;97(4):456–462.
160. MacLennan A. A template for defining a causal relationship between acute intrapartum events and cerebral palsy: International consensus statement. International Cerebral Palsy Task Force. *Aust N Z J Obstet Gynaecol* 2000;40(1):13–21.
161. MacLennan A. A template for defining a causal relationship between acute intrapartum events and cerebral palsy: international consensus statement. *BMJ* 1999;319(7216):1054–1059.
162. Perlman J. Induced hypothermia: A novel neuroprotective treatment of neonatal encephalopathy after intrapartum hypoxia-ischemia. *Curr Treat Options Neurol* 2005;7(6):451–458.
163. Pomerance J. Umbilical cord blood gas casebook: Interpreting umbilical cord blood gases, IX. *J Perinatol* 2001;21(7):469.
164. Westgate J, Garibaldi JM, Greene KR. Umbilical cord blood gas analysis at delivery: A time for quality data. *Br J Obstet Gynaecol* 1994;101(12):1054–1063.
165. Socol ML, Garcia PM, Riter S. Depressed Apgar scores, acid-base status, and neurologic outcome. *Am J Obstet Gynecol* 1994;170(4):991–998; discussion 8–9.
166. Sehdev HM, Stamilio DM, Macones GA, Graham E, Morgan MA. Predictive factors for neonatal morbidity in neonates with an umbilical arterial cord pH less than 7.00. *Am J Obstet Gynecol* 1997;177(5):1030–1034.
167. Dear P, Newell S. Establishing probable cause in cerebral palsy: How much certainty is enough? *BMJ* 2000;320(7241):1075–1076.
168. Amiel-Tison C, Sureau C, Shnider SM. Cerebral handicap in full-term neonates related to the mechanical forces of labour. *Baillieres Clin Obstet Gynaecol* 1988;2(1):145–165.
169. Korst LM, Phelan JP, Wang YM, Martin GI, Ahn MO. Acute fetal asphyxia and permanent brain injury: A retrospective analysis of current indicators. *J Matern Fetal Med* 1999;8(3):101–106.
170. Wu YW, March WM, Croen LA, Grether JK, Escobar GJ, Newman TB. Perinatal stroke in children with motor impairment: A population-based study. *Pediatrics* 2004;114(3):612–619.
171. Shankaran S, Luptook AR, Ehrenkranz RA, Tyson JE, McDonald SA, Donovan EF, Fanaroff AA, Poole WK, Wright LL, Higgins RD, Finer NN,

- Carlo WA, Duara S, Oh W, Cotten CM, Stevenson DK, Stoll BJ, Lemons JA, Guillet R, Jobe AH, National Institute of Child Health and Human Development Neonatal Research Network. Whole-body hypothermia for neonates with hypoxic-ischemic encephalopathy. *N Engl J Med* 2005;353(15):1574–1584.
172. Barnett A, Mercuri E, Rutherford M, Haataja L, Frisone MF, Henderson S, Cowan F, Dubowitz L. Neurological and perceptual-motor outcome at 5–6 years of age in children with neonatal encephalopathy: Relationship with neonatal brain MRI. *Neuropediatrics* 2002;33(5):242–248.
  173. Rutherford MA, Azzopardi D, Whitelaw A, Cowan F, Renowden S, Edwards AD, Thoresen M. Mild hypothermia and the distribution of cerebral lesions in neonates with hypoxic-ischemic encephalopathy. *Pediatrics* 2005;116(4):1001–1006.
  174. Inder TE, Hunt RW, Morley CJ, Coleman L, Stewart M, Doyle LW, Jacobs SE. Randomized trial of systemic hypothermia selectively protects the cortex on MRI in term hypoxic-ischemic encephalopathy. *J Pediatr* 2004;145(6):835–837.
  175. De Haan HH, Gunn AJ, Williams CE, Gluckman PD. Brief repeated umbilical cord occlusions cause sustained cytotoxic cerebral edema and focal infarcts in near-term fetal lambs. *Pediatr Res* 1997;41(1):96–104.
  176. Selzer ME, Myers RE, Holstein SB. Unilateral asphyxial brain damage produced by venous perfusion of one carotid artery. *Neurology* 1973;23(2):150–158.
  177. Vannucci RC, Vannucci SJ. Hypoglycemic brain injury. *Semin Neonatol* 2001;6(2):147–155.
  178. Ogundipe OA, Kullama LK, Stein H, Nijland MJ, Ervin MG, Padbury J, Ross MG. Fetal endocrine and renal responses to in utero ventilation and umbilical cord occlusion. *Am J Obstet Gynecol* 1993;169(6):1479–1486.
  179. Falkowski A, Hammond R, Han V, Richardson B. Apoptosis in the preterm and near-term ovine fetal brain and the effect of intermittent umbilical cord occlusion. *Brain Res Dev Brain Res* 2002;136(2):165–173.
  180. Sherer DM, Hearn B, Woods JR, Jr. Possible intermittent umbilical cord occlusion. *J Ultrasound Med* 1990;9(3):182.
  181. Pasternak JF, Gorey MT. The syndrome of acute near-total intrauterine asphyxia in the term infant. *Pediatr Neurol* 1998;18(5):391–398.
  182. Cowan FM, Pennock JM, Hanrahan JD, Manji KP, Edwards AD. Early detection of cerebral infarction and hypoxic ischemic encephalopathy in neonates using diffusion-weighted magnetic resonance imaging. *Neuropediatrics* 1994;25(4):172–175.
  183. Sie LT, van der Knaap MS, Oosting J, de Vries LS, Lafeber HN, Valk J. MR patterns of hypoxic-ischemic brain damage after prenatal, perinatal or postnatal asphyxia. *Neuropediatrics* 2000;31(3):128–136.
  184. Cowan F, Rutherford M, Groenendaal F, Eken P, Mercuri E, Bydder GM, Meiners LC, Dubowitz LM, de Vries LS. Origin and timing of brain lesions in term infants with neonatal encephalopathy. *Lancet* 2003;361(9359):736–742.
  185. Mercuri E, Rutherford M, Cowan F, Pennock J, Counsell S, Papadimitriou M, Azzopardi D, Bydder G, Dubowitz L. Early prognostic indicators of outcome in infants with neonatal cerebral infarction: A clinical, electroencephalogram, and magnetic resonance imaging study. *Pediatrics* 1999;103(1):39–46.
  186. Govaert P, Matthys E, Zecic A, Roelens F, Oostra A, Vanzielegheem B. Perinatal cortical infarction within middle cerebral artery trunks. *Arch Dis Child Fetal Neonatal Ed* 2000;82(1):59–63.
  187. Lynch JK, Hirtz DG, DeVeber G, Nelson KB. Report of the National Institute of Neurological Disorders and Stroke workshop on perinatal and childhood stroke. *Pediatrics* 2002;109(1):116–123.
  188. Ferriero DM. Neonatal brain injury. *N Engl J Med* 2004;351(19):1985–1995.
  189. Ahn MO, Korst LM, Phelan JP, Martin GI. Does the onset of neonatal seizures correlate with the timing of fetal neurologic injury? *Clin Pediatr (Phila)* 1998;37(11):673–676.
  190. Vannucci RC, Christensen MA, Yager JY. Nature, time-course, and extent of cerebral edema in perinatal hypoxic-ischemic brain damage. *Pediatr Neurol* 1993;9(1):29–34.
  191. Soul JS, Robertson RL, Tzika AA, du Plessis AJ, Volpe JJ. Time course of changes in diffusion-weighted magnetic resonance imaging in a case of neonatal encephalopathy with defined onset and duration of hypoxic-ischemic insult. *Pediatrics* 2001;108(5):1211–1214.
  192. Mercuri E, Cowan F, Rutherford M, Acolet D, Pennock J, Dubowitz L. Ischaemic and haemorrhagic brain lesions in newborns with seizures and normal

- Apgar scores. *Arch Dis Child Fetal Neonatal Ed* 1995;73(2):67-74.
193. Ikeda T, Murata Y, Quilligan EJ, Parer JT, Theunissen IM, Cifuentes P, Doi S, Park SD. Fetal heart rate patterns in postasphyxiated fetal lambs with brain damage. *Am J Obstet Gynecol* 1998;179(5):1329-1337.
194. Freeman RK. Problems with intrapartum fetal heart rate monitoring interpretation and patient management. *Obstet Gynecol* 2002;100(4):813-826.
195. Westgate JA, Bennet L, Gunn AJ. Fetal seizures causing increased heart rate variability during terminal fetal hypoxia. *Am J Obstet Gynecol* 1999;181(3):765-766.
196. Renou P, Newman W, Wood C. Autonomic control of fetal heart rate. *Am J Obstet Gynecol* 1969;105(6):949-953.
197. Parer JT. The effect of atropine on heart rate and oxygen consumption of the hypoxic fetus. *Am J Obstet Gynecol* 1984;148(8):1118-1122.
198. Mendez-Bauer C, Poseiro JJ, Arellano-Hernandez G, Zambrana MA, Caldeyro-Barcia R. Effects of atropine on the heart rate of the human fetus during labor. *Am J Obstet Gynecol* 1963;85:1033-1053.
199. Dalton KJ, Dawes GS, Patrick JE. The autonomic nervous system and fetal heart rate variability. *Am J Obstet Gynecol* 1983;146(4):456-462.
200. Lowensohn RI, Weiss M, Hon EH. Heart-rate variability in brain-damaged adults. *Lancet* 1977;1(8012):626-628.
201. Johnson N. Development and potential of fetal pulse oximetry. *Neonatal Intensive Care* 1992;5(5):43-50.
202. Cohen WR, Schifrin BS, Doctor G. Elevation of the fetal presenting part: A method of intrauterine resuscitation. *Am J Obstet Gynecol* 1975;123(6):646-649.

## Chapter 23 BIRTH INJURIES

John P. O'Grady

*Life is short; Art is long; Opportunity fugitive;  
Experience delusive; Judgment difficult.*

Hippocrates (460–377 B.C.E.)  
*Aphorisms of Hippocrates*  
T. Coar (trans.)  
London, A.J. Valpy, 1822, I, pg 1.

Most intrapartum injuries occurring to mothers and babies are inconsequential; however, a small percentage are serious, and rarely, some prove fatal. This chapter presents an overview of both maternal and infant birth injuries, considering their etiology, potential methods of avoidance, and critiques of current obstetric practices.

The initial difficulty in discussing birth injuries is that of definition. In this text, a *birth injury* is simply defined as an abnormality of the mother or baby found present at the time of delivery. Birth injuries potentially include damage to either the fetus/neonate or the mother from a wide range of causes, both natural and iatrogenic, and are attributable both to fetal development and to the process of parturition. Although damage to the mother is usually not considered a birth injury, maternal trauma during parturition is common. Further, injuries to the mother often accompany injuries to her infant, and a maternal injury during parturition is a potential cause for medicolegal entanglements.

Modern obstetric and neonatal practices are successful in reducing perinatal mortality. Unfortunately, reducing the incidence of permanent neurologic injuries remains an elusive goal [1–2]. The outcome of serious newborn abnormalities, their relationship to events of parturition, and the neurodevelopmental and behavioral problems that these infants can develop are of the greatest concern within and outside of the profession. In recent decades, much has changed in physicians' understanding of fetal injury. Despite prior beliefs, it is now understood that approximately 80% or more of the serious or permanent fetal/neonatal neurologic abnormalities are sustained *prior* to birth. These abnormalities are largely attributable to chromosomal abnormalities, adverse effects of in-utero inflammation, infection, poorly understood toxic exposures, or other currently unknown developmental or hereditary problems [1–8].

Because of the etiology of permanent neurologic injury, neither complete avoidance of instrumental deliveries nor a 100% cesarean delivery rate would

avoid all damaged infants. Permanent neurologic impairment of infants, such as cerebral palsy (CP) or intellectual deficits, are rare after mechanical birth trauma, unless an injury is combined with serious birth asphyxia or prematurity. This is not to deny that poor obstetric technique, prolonged labor, or difficult delivery cannot result in potentially serious birth trauma; rather, current data emphasize the much greater importance of events occurring before the onset of labor in the mechanisms responsible for producing neurologically deficient children [3,7,8]. In essence, how fetal or maternal problems develop during pregnancy is thought to be more important to long-term neonatal outcome than how any resulting peripartum complications are managed.

Unfortunately, the etiology of many birth injuries remains obscure even after close review of the clinical record and the events of parturition. Whenever serious injuries or abnormalities are observed in a newborn, especially those involving neonatal neurobehavioral abnormalities, a full evaluation is required. This evaluation includes critical review of the obstetric and neonatal medical records, a histologic examination of the placenta, a review of the family history, and, on occasion, chromosomal analysis of the placenta, infant, or parents, as well as other tests. Gross and microscopic placental examination by an experienced pathologist can often identify evidence of previously unsuspected chronic infection or other conditions that predate both labor and delivery and that have adversely affected the pregnancy. The clinical events of labor and delivery, immediate neonatal complications, and laboratory and imaging data require close scrutiny to understand what caused an observed abnormality or group of abnormalities.

### **MATERNAL BIRTH INJURIES: OVERVIEW**

Some degree of maternal injury, such as bruising or small birth canal tears, is normal following either spontaneous or assisted delivery. This minor trauma might not be avoidable and is clinically inconsequential. Nonetheless, a small percentage of maternal injuries occurring at parturition prove serious or rarely, even life threatening (Tables 23.1 and 23.2).

The more significant maternal complications of parturition include birth canal lacerations, episiotomy extensions, other perineal or rectal injuries,

**TABLE 23.1 Fetal/Neonatal Birth Injuries**

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Bruising, superficial injuries
Lacerations
Fractures:
Skull, longbones, clavicle, etc.
Nerve injuries:
Spinal cord, brachial plexus
Phrenic, facial nerves, etc.
Visceral injury:
Liver, kidney, other
Scalp injuries:
Subgaleal hemorrhage
Cephalohematoma
Local pressure necrosis/laceration
Infection:
Viral, bacterial, parasitic
Unusual injuries:
Eye, iatrogenic lacerations, etc.
Intracranial injuries:
Subarachnoid, subdural
Intraventricular, parenchymal or posterior fossa hemorrhage

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**TABLE 23.2 Maternal Birth Injuries**

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Birth canal lacerations:
Cervical injuries
Episiotomy
First-, second-, third-, fourth-degree perineal lacerations
Bladder or urethral injury/fistula formation
Uterine rupture, scar dehiscence
Disruption of abdominal wound/wound infection/dehiscence
Rupture of pubic symphysis
Coccygodynia
Infection:
Urinary tract, soft tissue/cellulitis, endometritis
Uterine prolapse
Intrapartum or postpartum hemorrhage
Uterine atony, inversion, rupture
Amniotic fluid or pulmonary embolism
Venous insufficiency, thrombosis

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**TABLE 23.3 Factors Predisposing to Fetal Birth Injury**

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Fetal macrosomia
Fetopelvic disproportion
Preterm labor/delivery
Emergency delivery: cesarean or operative vaginal
Fetal malpresentation/malpositioning
Face/brow/breech presentation
Marked deflection/occiput posterior presentation
Abnormal labor:
Prolongation or descent disorders
Obstetric procedures:
Instrumental or cesarean delivery
Version and extraction
Scalp sampling
Amniocentesis
Percutaneous blood sampling
Multiple gestation
Abruptio placentae
Uterine rupture/scar dehiscence
Maternal trauma
Placenta previa/vasa previa

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and various degrees of intrapartum and postpartum hemorrhage. Although these conditions result in substantial morbidity, associated mortalities are rare. Maternal mortalities associated with birth are usually due to uncommon complications of anesthesia or to obstetric complications such as severe hypertension, exsanguinating hemorrhage, or to the effects of rare catastrophes such as amniotic fluid embolism or manifestations of inherent structural defects such as Marfan's syndrome. Certain clinical settings predispose to birth injury, including labor stimulation, dystocia/macrosomia, preterm delivery, the diagnosis of acute fetal jeopardy from any cause, and instrumental or cesarean delivery (Table 23.3).

Adverse outcomes do not necessarily result from poor or tardy care. Serious and even fatal maternal (and fetal) injuries do result from spontaneous birth processes, despite the best intentions and efforts of competent clinicians; nonetheless, many injuries are preventable by following well-established obstetric techniques, timely intervention, avoidance of unnecessary or ill-timed obstetric procedures, exercise of operator skill, and the judicious use of force. The next sections review important categories of

birth injuries, discuss their etiology, and consider principles of general management.

### Cesarean Delivery

Current high rates of cesarean delivery and new considerations of what has been termed *patient choice cesarean* are controversial [9–17]. Cesarean delivery is a major operative procedure with substantial risk for maternal morbidity, and the risk for emergency procedures is greater than that for elective operations. Operative deliveries are often performed on women with a “full stomach” and can follow an exhausting labor with multiple pelvic examinations, or be associated with other problems such as hypovolemia, coagulopathy, or sepsis. There is an enhanced risk for maternal hemorrhage, endometritis, and other potentially serious complications, such as wound breakdown, venous thrombosis, pelvic thrombophlebitis, and abscess formation associated with cesareans. Dystocia in its various forms is the most common indication for primary operations, and prior cesarean the most common reason for a repeat operation [18]. Many factors affect cesarean delivery rates, including patterns of local practice, changing recommendations for best practice by learned bodies, medicolegal concerns, uncertainties in the specialty about the appropriate management of certain clinical difficulties, the desire to avoid difficult vaginal operations, and patient or physician preference. (See Chapter 18, Cesarean Delivery and Surgical Sterilization.)

### Dystocia/Macrosomia

Dystocia is an important risk factor for birth injury. A discussion of dystocia requires consideration of a poorly clarified clinical entity: *fetopelvic disproportion*. This term refers to a complex obstetric problem, with fetal size being only one of its components. True disproportion, meaning the anatomic inability of the fetus to pass through the pelvis, is quite uncommon. Unless there is a history of congenital anomaly or trauma, an anatomically restricted bony pelvis is a rarity in modern practice. If an element of disproportion is present, it can be due to a large fetus in an otherwise adequate pelvis, or more likely to malpositioning of the head of a normal-sized infant in a birth canal of normal diameter. In the unusual case of true cephalopelvic



disproportion, or with certain malpresentations, (e.g., brow or transverse lie), labor is obstructed, and cesarean delivery is required. In most instances, once absolute disproportion is excluded, poor labor progress results from inadequate uterine activity or, as noted, various forms of cranial malpositioning (e.g., cranial deflection, occiput posterior), which slow the labor process. In many cases, these factors are combined.

Serious maternal and fetal injuries are possible in certain cases of relative disproportion. Injury can occur when dilation and descent proceed far enough to tempt the unwary clinician into an ill-advised instrumental trial in a case of true disproportion, or when cranial delivery occurs but a shoulder dystocia ensues. (See Chapter 17, Instrumental Delivery.)

Cranial presentation, pelvic architecture, the effects of analgesia/anesthesia, adequacy of the spontaneous powers, fetal size, and use of the uterotonics are among the factors known to influence labor outcome [18]. (See Chapter 10, Labor.) Infants weighing more than 4000 g are more likely to be injured in the birth process than smaller ones, although most of these large neonates are delivered atraumatically [19, 20]. Unfortunately, in recent decades, mean birthweights have risen. At present, more than 11% of infants weigh 4000 g or more at birth, and some 2% reach or exceed 4500 g. Avoidance of difficult or traumatic vaginal delivery for these infants is obvious but at times difficult. Despite the best efforts of clinicians, macrosomic infants are surprisingly difficult to identify prospectively [20,21]. Although various clinical, historic, and ultrasonic techniques have been suggested as methods for antepartum identification of the large infant, all have proved to be disappointingly inexact. (See Chapter 14, Shoulder Dystocia.)

Physical examination, even by experienced clinicians, is often inaccurate in estimating fetal weight, despite the fond hopes of many traditionalists. Ultrasonic measurements at or near term are also notoriously imprecise. In the third trimester, ultrasonic weight estimates have a mean absolute error of  $\pm 8\%$  to  $16\%$ , with  $32\%$  to  $69\%$  of these estimates falling within  $\pm 10\%$  of the actual fetal weight [21]. Based on questionable data, elective induction of infants before term or arbitrary ultrasonic weight estimate limits for vaginal trials are occasionally employed in clinical management [22]. Unfortunately, such schemes fail to select those for either a vaginal trial

or for cesarean delivery properly, owing to the inability of current methods to estimate fetal weight correctly. Conducting inductions of labor for "impending macrosomia" increases rather than diminishes the likelihood for a cesarean, mostly because of failed inductions leading to additional abdominal deliveries.

Maternal risks accompany the delivery of macrosomic infants. When the baby is large, the most important of the maternal risks is postpartum uterine atony and resultant hemorrhage. Hematomas, birth canal lacerations, and rectal injuries, the latter mostly from episiotomy extensions, are additional potential problems. In extreme cases, rupture of the pubic symphysis, or even uterine rupture is possible. If a postpartum hemorrhage occurs after the delivery of a large infant, a complete examination of the birth canal (i.e., vagina, cervix, perineum) and a manual uterine exploration are mandatory. This is especially true if a difficult delivery involving a shoulder dystocia or an instrumental assist has occurred. Such a complete examination, with special attention to potential secundines or occult uterine rupture, as well as the prompt postexamination administration of uterotonics, is mandatory. (See Chapter 11, The Third Stage.)

### Instrumental Delivery

When an instrumental delivery is indicated, and the mother is incapable of expelling the fetus, or other important reasons necessitate an expedited or assisted delivery, by definition the labor/delivery is no longer normal. Further, a risk for both maternal and fetal injuries accompanies any instrumental delivery, regardless of the instrument or the technique employed [23].

Forceps and the vacuum extractor share a common mechanism: cranial traction to the fetus aided by voluntary maternal bearing-down effects to overcome maternal soft-tissue resistance. Except in outlet operations, when either instrument is used, the lower vaginal tissues have not been subjected to the slow, progressive distension that normally accompanies descent of the presenting part during a spontaneous delivery. Both instruments therefore share a potential for injuries to the fetal head or its contents as well as to the birth canal, especially to the rectal sphincter apparatus and the supporting muscle and connective tissues of the pelvis. These injuries

result from the rapid descent of the presenting part and the inability of the pelvic tissues to accommodate the mass of the fetal body that rapidly.

Although both the vacuum extractor and forceps can inflict injuries on the perineum or vagina, forceps use is more likely to result in damage to the upper birth canal and to the rectum. Because the vacuum extractor cup is applied solely to the fetal scalp, it normally does not come into direct contact with maternal tissues, unless the maternal sidewall is inadvertently drawn into the instrument. Thus, with vacuum extraction, even if rotation of the presenting part occurs, direct injury to the maternal vaginal vault is less common than with forceps deliveries.

To reduce both maternal and fetal injury, difficult instrumental procedures, especially midpelvic rotational procedures, should not be performed except by experienced clinicians. Reliance on vacuum extraction in more difficult cases is best for most practitioners, especially for trials of midpelvic delivery. If the circumstances are such that a difficult procedure is contemplated, one should probably reconsider labor management by extending the second stage, changing the degree of analgesia/anesthesia, administering oxytocin, or repositioning the mother to determine if further progress is possible by propulsion and not traction. A higher incidence of injured mothers and babies results when forceps are applied in these complex cases by all but the most experienced of practitioners. (See Chapter 17, Instrumental Delivery.)

### Fetal Monitoring

*Fetal monitoring*, as the term is commonly used in the United States, refers principally to electronic fetal monitoring (EFM) [24,25]. This technique uses an electronic device to detect and record instantaneous fetal heart rate (FHR) patterns and uterine contractions and display them graphically, either on a continuously advancing paper strip or electronically. Less frequently, monitoring of the fetus in labor is by intermittent auscultation of the fetal heart by a birth attendant with a fetoscope or, much more commonly when EFM is not employed, by use of a hand-held Doppler device. For the last decade, EFM has been the focus of persisting controversy about its alleged adverse effects and limited efficacy [25–29]. (See Chapter 22, Fetal Assessment.) Despite

these disputations, most major American hospitals continue to depend on EFM tracings as the principal means of fetal evaluation in labor. There are several reasons for this decision. First is the extensive clinical experience that practitioners and institutional birth attendants have with the application and interpretation of EFM tracings. This fact, and the concept that progressive changes in EFM of heart rate patterns reflect general fetal well-being, are the major influences. There is also a major financial investment by delivery services in various electronic monitoring devices. Finally, EFM frees nursing personnel for other duties, or permits a single nurse to attend several patients in labor simultaneously.

Continuous EFM has not proved to be superior to one-on-one intermittent auscultation – at least insofar as was tested in the various prospective, randomized clinical trials conducted over the last decade [25,27]. When EFM is employed, neonatal outcomes are not worse, and some argue that they are better. Most series evaluating the impact of EFM also have recorded more obstetric interventions when EFM is the primary means for fetal evaluation. The potential adverse effects of EFM are complex. Restricting the mother's activity or labor position and using dense epidural anesthesia probably increases the possibility for poor progress and thus surgical intervention. With careful obstetric management, however, modern protocols for fetal evaluation and maternal anesthesia need not be associated with major changes in the rate of cesarean delivery. (See Chapter 9, Obstetric Anesthesia.)

EFM is best considered as a screening study. Reactive or normal heart rate tracings are reassuring for fetal well-being because the false-negative rate is low. Unfortunately, nonreactive, bothersome, or difficult-to-interpret tracings are common, and their positive predictive value (PPV) is generally poor. Such patterns might or might not reflect fetal hypoxia or acidosis. Certain and specific patterns such as severe, fixed bradycardias require immediate, definite action unless spontaneous delivery is imminent, however. Most clinicians concur that ominous FHR findings by EFM include fixed or intermittent bradycardias, tachycardias with absent variability, nonreactive tracings with recurrent late decelerations, and patterns of severe, recurrent variable decelerations accompanied by progressive decline in baseline variability especially with poor return to the baseline.

Newly configured electronic monitors have recently been introduced, combining the usual EFM tracing with computer analysis of fetal electrocardiographic ST segments. This technique has been claimed to reduce the incidence of false-positive diagnoses of suspected fetal compromise [29]. These claims have yet to be fully verified in clinical applications, however. (See Chapter 22, Fetal Assessment.)

When faced with an acute obstetric problem involving presumed fetal jeopardy, the clinician must promptly assess maternal condition, conduct a meticulous vaginal/pelvic examination, and consider the available options. If less than a fixed bradycardia is present, there remains some latitude for observation and additional testing. Additional testing could include scalp stimulation (e.g., electrode placement, Allis clamp application, or digital scalp pinching), fetal acoustic stimulation, percutaneous oxygen monitoring, or other biophysical testing. Pelvic examination should be repeated and fetal station, and position and the dilation and effacement evaluated. The fetopelvic relationship is also judged and the birth canal carefully examined to exclude a cord prolapse. The clinician should observe the quantity and type of vaginal discharge and review the strength and pattern of the uterine contractions. The mother's ability and willingness to assist in bearing down should also be judged. If repositioning, discontinuation of oxytocin, acute tocolysis, and other maneuvers (e.g., elevation of the presenting part to return the FHR to normal) fail to result in improvement, expeditious delivery becomes the clinician's objective.

## **SPECIFIC MATERNAL BIRTH INJURIES**

### **Episiotomy and Extensions**

Superficial maternal birth canal injuries such as soft-tissue abrasions, ecchymoses, or small lacerations are common enough to be considered normal. Such injuries follow vaginal instrumental delivery with forceps or the vacuum extractor as well as spontaneous parturition [23,30]. The more complex forceps or vacuum extraction operations – especially those from higher station – involve a greater risk for fetal trauma and maternal soft-tissue injury. Extractions from midstation or those involving malpositioned fetal heads – especially occiput posteriors –

require an angle of traction that puts heavy pressure on the perineal body and posterior vaginal vault. Even when care is taken, this can result in complex or deep perineal lacerations with or without an episiotomy. When a midline episiotomy is performed, the likelihood of an extension into the rectal sphincter or mucosa increases substantially. In certain acute situations such as shoulder dystocia, a spontaneous laceration of the perineum or an extension of a previously performed episiotomy can occur as the accoucheur performs vaginal manipulations. In multiparous women, in the occasional nullipara with vaginal relaxation, or when there is sufficient time to “iron out” the perineum, slow and gentle extraction can avoid both episiotomy and laceration.

There is general consensus that the routine use of episiotomy predisposes to third- and fourth-degree lacerations while providing limited protection against injury to the periurethral tissues [31,32,34]. There is limited and dated evidence for the claim that episiotomy reduces injury to pelvic support structures, specifically damage to the levator muscles [33]. No modern studies make this claim, however. Furthermore, episiotomy also results in increased maternal blood loss. In an instrumental delivery, episiotomy does apparently reduce the force required for fetal extraction. In this sense, episiotomy could be protective to the infant, at least insofar as permitting operative fetal extraction with less force. Whether this force reduction significantly reduces the risk of infant injury has never been demonstrated, however. In fact, the preponderance of data collected in the last decade indicates that episiotomy increases the likelihood of serious perineal tears and most likely predisposes to long-term perineal dysfunction rather than providing any degrees of protection [31].

The best technique for the repair of rectal injuries remains controversial. Traditionally, obstetric students were taught an end-to-end repair using polyglycolic acid or chromic suture material, with simple interrupted stitches. New information concerning both rectal anatomy and the importance of internal sphincter incontinence has led to disagreement concerning the best method(s) for rectal repair and subsequent management. For external sphincter repair, an overlapping sphincter repair and routine administration of broad-spectrum antibiotics are among the most commonly suggested alternative techniques [35–37]. Unfortunately, randomized trials do not

indicate any special benefit to the overlapping closure technique for the external sphincter versus the traditional approach, although the data remain controversial [36,37]. Furthermore, administering antibiotics to avoid or to reduce the risk of failure of a primary sphincter repair is not supported by prospective data but is favored by some and is common in European practice [37,38]. It should be noted that determination of best practice for perineal injuries and their repair is an area of active research and the publication of additional studies leading to practice improvements will doubtless occur within the next several years.

Because long-term adverse effects of episiotomy are possible and demonstrated benefits are minimal, obstetric surgeons should avoid routine episiotomy. Although a mediolateral incision has a lower likelihood of associated rectal injury, it comes as no surprise that postpartum, this incision is more uncomfortable for the parturient. Furthermore, mediolateral incisions are more likely to result in anatomic disfigurement of the perineum and long-term dyspareunia. Finally, the mediolateral incision does not guarantee complete freedom from the risk of rectal sphincter injury. When and if to use a mediolateral as the perineal incision of choice is not clear.

Although it is easy to counsel practitioners to revise their delivery technique to avoid incision or tearing of the perineum, best practice concerning elective episiotomy remains uncertain. This is especially true when episiotomy is required during an instrumental delivery or for the relief of a should-

der dystocia. It seems unlikely that American practitioners will follow their European counterparts and return to the mediolateral technique. Additional study of the risk–benefit ratio for the types of elective perineal incision in various clinical settings is a research priority.

There are other issues of importance. Immediate identification of third- and fourth-degree extensions, control of bleeding, and prompt anatomic layered closure minimalizes the risk of long complications from perineal injury. Nonetheless, when serious rectal injuries such as traumatic transection occur, maternal morbidity is increased, as is the long-term risk of rectal dysfunction [38–41]. In addition, when a perineal tear extends into the rectum, there is a small but potentially serious risk of worse complications at the repair, site including incontinence, infection, or fistula formation.

Data do suggest an important relationship between injuries to maternal pelvic support structures and to the rectum in the long-term complications of procidentia and rectal incontinence [42–44]. This information remains controversial but has been used as an argument for prelabor prophylactic cesarean delivery [11,44–48].

Some knowledge of normal rectal anatomy and physiology is germane to an understanding of the problems of perineal injury and long-term rectal dysfunction or incontinence. Cadaver and magnetic resonance imaging (MRI) studies verify the existence of both internal (IS) and external (ES) anal sphincters [49–51] (Figure 23.1). In a recent study

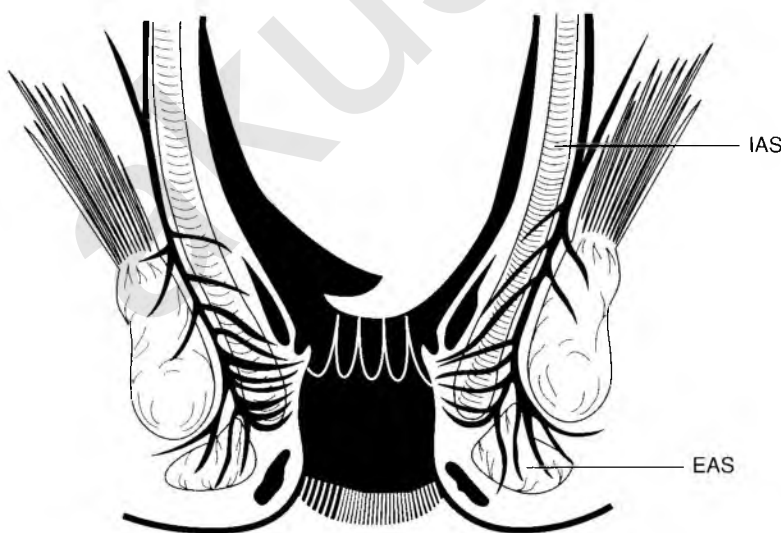


FIGURE 23.1.

*Size and relationship of the internal anal sphincter (IAS) and the external anal sphincter (EAS) in the lower pelvis. (Modified from Delancey JOL, Togli MR, Perucchini D: Internal and external anal sphincter anatomy as it related to midline obstetric lacerations. *Obstet Gynecol*, 1997 Dec; 90(6):924–7; with permission.)*

involving MRI scans of nulliparous women, Hsu and coworkers reported that there are three identifiable parts to the sphincteric mechanism [51]. For the purposes of general discussion, a clear distinction between the IS and the ES is sufficient. The IS is best understood as a downward continuation and thickening of the muscle layers of the lower bowel. Immediately above the rectal verge, the muscle fibers encircling the large bowel increase in size. This results in an anatomic structure that functions as a physiologic sphincter. This IS is anatomically distinct from the familiar and discrete doughnut-shaped ES, which is positioned adjacent to the anal orifice.

Normal continence requires coordinate action among the anal sphincters, the sensory nerve endings, and the smooth muscle of the rectum and the puborectalis muscle [42]. In the physiology of normal defecation, when a bolus of material is first presented to the lower bowel, the IS initially opens by an autonomic reflex, while the ES contracts. This action permits the bolus of material to descend into the lower bowel. The rectal nerves then "sample" the bowel content, determining its nature. If defecation is inappropriate, the ES remains contracted and the puborectalis muscle contracts, acutely changing the rectal angle and forcing the bolus upward. Once this bowel material has moved upward, the IS again contracts to retain continence. Thus, both sphincters act in concert to maintain normal defecatory function. Based on physiologic studies, it is now recognized that the IS is responsible for 50% to 80% of the resting tone of the rectum, and the ES for the remaining 25% to 30% [42]. Because of this complex anatomic relationship, injuries to the IS, the ES, the nerves subserving these structures, and to surrounding tissues can result in varying degrees of dysfunction in normal defecation. Post-delivery, transanal ultrasound studies have indicated lesions of the IS in up to 24% of primiparas and an even higher percentage of multiparas [50]. Even with these demonstrated lesions, however, many if not most affected women remain asymptomatic. Although controversial, such relatively common parturition-related IS injuries are suspected to predispose to eventual anal incontinence when the combined effects of age, menopause, or subsequent childbirth injury further weaken the sphincteric mechanism.

### Vaginal and Cervical Lacerations

Both vaginal vault lacerations and cervical tears can accompany spontaneous deliveries; however, these injuries are most common following vaginal instrumentation or deliveries complicated by precipitate labor or shoulder dystocia. The use of vacuum extraction for selected midpelvic procedures avoids many vaginal injuries, unless the sidewall or cervix is inadvertently drawn into the edge of the cup. Proper application and routine checks for vacuum cup application should avoid such injuries. In forceps procedures, extreme care during rotational procedures is warranted. The technique employed in the wandering of the blades and in rotational deliveries is critical to the avoidance of high vaginal lacerations. For this and other reasons, midpelvic and rotational forceps procedures must remain restricted to experienced practitioners or performed only where immediate expert assistance is possible.

Following any vaginal operative delivery, no matter how simple, a meticulous examination of the entire birth canal is mandatory. (See Chapter 17, Instrumental Delivery.) Vault lacerations are usually easy to repair when adequate light and exposure are provided. Some tears extending high into the lateral fornix are positioned behind the cervix or dissect deeply into the ischiorectal fossa, representing surgical challenges, however. The most potentially serious of these injuries are deep lateral tears of the vagina. These rents can lacerate vessels, which results in either observed external hemorrhage or the formation of pelvic hematomas that can invade the ischiorectal fossa, or rarely, dissect upward in the retroperitoneal space. Instrumental delivery is not always the culprit. The pudendal artery or one of its tributaries can be lacerated or avulsed during spontaneous, delivery unaided by an instrument and without a prior pudendal nerve block. Regardless of the exact vessel injured, such hematomas can enlarge and on occasion become hemodynamically serious.

When an acute vaginal hematoma develops, the usual presenting complaint is severe perineal/vaginal pain and the inability to void. On examination, an exquisitely tender, progressive, unilateral swelling of the labia with a purplish discoloration is noted. Infrequently, blood loss is extensive

enough to require transfusion or to render the mother hemodynamically unstable. An additional and potentially serious complication of deep lateral wall lacerations is a ureteric injury. This injury usually results from injudicious clamping or suturing in the vaginal side wall in the effort to control bleeding. Management of the rare ureteric or bladder injuries is discussed in greater detail in Chapter 19, Urologic Complications.

### Infection

The overall rate of postpartum maternal infection following parturition is estimated to be 1% to 8%. The most common infections seen after delivery involve the endometrial cavity, the respiratory system, and the urinary tract. Although current antibiotic and surgical management cure most obstetric infections, morbidity is common and serious complications are occasionally possible [52,53]. Maternal soft tissue or intrauterine infection following uncomplicated vaginal delivery is surprisingly rare. Considering the frequency of open birth canal wounds, spontaneous and iatrogenic lacerations of the perineum, and the gross contamination of the vagina and perineum by genital tract bacteria, the incidence of infection of the soft tissues of the birth canal and the endometrium is low. Infections involving vaginal lacerations or episiotomy sites are usually superficial and minor. If the perineum becomes heavily infected, the usual outcome is disruption of the episiotomy or the laceration repair. For superficial soft-tissue infections, the best treatment is wound exploration to drain any collections, accompanied by local débridement as required. Broad-spectrum antibiotics should be administered if signs of cellulitis, induration, or gross infection are present. Sitz baths and analgesics usually provide symptomatic relief. Whether to employ immediate versus delayed closure of episiotomy and perineal lacerations is controversial, but in many cases early repair is successful with good results [54,55].

Rarely, a birth-related infection is severe or life threatening. Most post delivery morbidity is associated with obstetric interventions (Table 23.4) [56]. Although most serious cases of postpartum infection result from endometritis, other complications are possible. Septic pelvic thrombophlebitis,

**TABLE 23.4 Risk Factors: Postpartum Endometritis\***

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Invasive fetal monitoring:
Fetal monitoring electrodes
Intrauterine pressure catheters
Prolonged membrane rupture
Multiple pelvic examinations
Prolonged labor
Cesarean delivery:
Manual removal of placenta
Prolonged surgery, excessive blood loss
Antepartum genital tract infection
Limited or no prenatal care
Low socioeconomic status

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\*See text for details and additional discussion.

pyelonephritis with generalized sepsis, peritonitis, or the rare necrotizing fasciitis are potentially fatal postpartum infections demanding early diagnosis and aggressive treatment.

### Urinary Tract Dysfunction

Common urinary tract complications of parturition include postpartum voiding difficulties and urinary tract infection. Serious problems, such as vesicovaginal fistulas, are rare. There is also believed to be a long-term risk of urinary incontinence because of injury to pelvic support structures. Physiologic changes in bladder and ureteric function of pregnancy, with stasis, increased residual volumes, and a degree of obstruction, also predispose gravid women to cystitis and ascending urinary tract infection. At special risk are women with preexisting bladder dysfunction, chronic bacilluria, or anatomic abnormalities of the genitourinary tract that result in reflux or become associated with partial obstruction by the gravid uterus. Early diagnosis, rapid treatment, the selective administration of antibiotics, and avoidance of unnecessary catheterization are best management.

In modern obstetric practice, vesicovaginal fistulas are rare complications of delivery. Management consists of complete urologic evaluation, identification, and excision of the defect, followed by a meticulous closure in layers, with close attention to hemostasis. (See Chapter 19, Urologic Complications.)

In developing regions of the world, serious complications of perineal/ bladder injuries occur much more frequently. The absence of trained birth attendants, high incidences of multiple pregnancies, inability to perform cesarean deliveries, and serious limitations in medical care are associated with many perineal fistulas, overwhelming the capacity of local health services. Vesicovaginal and rectovaginal fistulas resulting from unattended or traumatic deliveries are still shockingly common in sub-Saharan Africa, where the number of women with unrepaired fistulas is estimated to be in the thousands.

### Uterine Infection

Patients with endometritis/parametritis presents with a variety of symptoms, including malaise, uterine tenderness, tachycardia, tachypnea, fever or rigors, purulent vaginal discharge, and rarely, frank sepsis with cardiovascular instability. The route of delivery is the most important risk factor for endometritis. The highest incidence follows cesarean delivery, especially failed vaginal birth after cesarean (VBAC) trials that result in cesareans [56]. When the infection is manifested intrapartum, a prolonged or dysfunctional labor with multiple examinations or postpartum hemorrhage are common associations (Table 23.4). Such infections are usually polymicrobial, with anaerobic bacteria predominating. Treatment consists of parenteral therapy with broad-spectrum antibiotics, removal of any secundines, control of fever, administration of uterotonics, and maintenance of adequate circulating volume. The perioperative administration of antibiotics to high-risk patients (e.g., prolonged membrane rupture, multiple examinations/long labor, cesarean delivery) reduces febrile morbidity and avoids some of these infections. When endometritis is promptly treated, serious complications, although still possible, are rare.

### Uterine Rupture

Uterine ruptures are of two types: dehiscence/rupture of a prior uterine scar, or rupture of a previously unscarred uterus. The uterus can rupture from several causes. A rupture can occur spontaneously during a VBAC. The uterus can also be traumatically ruptured in an operative vaginal delivery, dur-

**TABLE 23.5 Clinical Features Associated with Increased Risk of Uterine Rupture**

---

Cephalopelvic disproportion/dystocia
Oxytocin administration
Grand multiparity
Abruptio placentae
Placenta percreta
Malpresentations (e.g., face, brow, shoulder)
Vaginal operative delivery
Difficult delivery (e.g., shoulder dystocia, internal version)
Trauma
Mullerian anomalies
Labor induction with prostaglandins
Hysterotomy scar
Uterine perforation scan or previous rupture repair
Myomectomy or metroplasty scar

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ing version and extraction, or following other obstetric procedures such as manipulations for relief of a shoulder dystocia. Uncommon causes of spontaneous rupture include obstructed labor, myometrial invasion by placenta percreta, severe abruptio placentae, maternal abdominal trauma, or Mullerian anomalies (Table 23.5). In modern practice, rupture during oxytocin stimulation is essentially restricted to multiparas or to those with a previously scarred uterus. Use of newer, potent uterotonics, such as misoprostol, can be associated with an increased risk of rupture when administered to women with a prior uterine scar during labor induction. (See Chapter 10, Labor.)

The risk of prior uterine scar rupture varies with the type of original incision. The rupture risk for a classic vertical scar is approximately 6%, whereas that for a low transverse incision is less than 1% [57,58]. Rupture rates for low vertical incisions are generally similar to those for low transverse incisions [58,59]. Maternal mortality from transverse scar ruptures is virtually nil, but the fetal loss rate approximates 10%. The fetal mortality rate from rupture of a classical uterine incision is 70% or more, with an accompanying maternal mortality of approximately 5%. Although the precise numbers for these risks vary by series, these averages serve as reasonable approximations. (See Chapter 18, Cesarean Delivery.)

TABLE 23.6 Clinical Features of Uterine Rupture

Clinical Features	Previously Scarred Uterus	Unscarred Uterus	Total
Tachycardia only	1	4	5
Shock	3	24	27
Scar (or abdominal tenderness or pain)	9	14	23
Uterine bleeding (external)	13	40	53
Hematuria	1	1	2
Cessation of contractions	4	4	8
Change in fetal position	2	6	8
Disappearance of fetal heart sounds	3	17	20
Routine examination of scar	9		15
Operation for another reason	6		

Modified from Golan A, Sandhank P, Rubin A: Rupture of the pregnant uterus. *Obstet Gynecol*. 1980. Nov; 56(5):549–54; with permission.

Classic signs of uterine rupture include arrest of labor, loss of station, vaginal bleeding, maternal cardiovascular collapse, fetal distress, and the sudden onset of abdominal pain (Table 23.6) [60–65]. Many ruptures seen in current practice do not include all of these classic signs and symptoms, however. In separations of low transverse scars, the dramatic presentation that often accompanies the spontaneous rupture of an unscarred uterus is frequently absent because the rupture usually occurs in the relatively avascular lower uterine segment. In fact, some transverse scar ruptures are found only incidentally at the time of laparotomy after a failed trial of labor or instrumental delivery.

If a uterine rupture is diagnosed, prompt surgical exploration either to repair the tear or to perform a hysterectomy is required. In contrast, asymptomatic scar dehiscences palpated after delivery, unless associated with hemorrhage, pain, or signs and symptoms suggesting excessive blood loss, are best left untreated. In a case of spontaneous rupture, the surgery can be complex, especially if rupture occurs in a previously scarred uterus. Extension into the broad ligament, major arterial vessels, and occasionally even the bladder is possible.

### Uterine Atony and Inversion

Uterine atony and inversion both can result in exsanguinating hemorrhage [66–71]. For the United States, it is estimated that atony is responsible for 1.4 mortalities per 100,000 live births [72]. There is good evidence that active management of the third stage, including the routine administration of uterotonics, reduces both postpartum blood loss and the incidence of atony [73]. In general, atony is more common after delivery of a macrosomic infant, a multiple gestation, an oxytocin-stimulated labor, chorioamnionitis, history of postpartum hemorrhage, or use of a tocolytic such as magnesium sulfate [69,70]. Infection and abruptio placentae also predispose to atony, as does precipitate labor and – now uncommonly – the use of halogenated anesthesia agents. The treatment for atony includes a variety of techniques: removal of intrauterine blood and clots, uterine massage, administration of potent uterotonics, packing or balloon compression, vessel embolization or ligation [68–70,74–79]. (See Chapter 18, Cesarean Delivery and Surgical Sterilization.)

Uterine inversion requires prompt restoration of the uterus to its usual anatomic position by either physical manipulation or surgery. Inversion occurs when both fundal implantation of the placenta and marked postpartum laxity of the lower uterine segment combine in some unusual manner. The incidence is approximately 1 in 2,000 deliveries and might be more common in nulliparas. Although incompetent midwifery and inappropriate cord traction were classically taught as the causes for uterine inversion, this is now believed to be only partially correct, although this association is still reported in standard textbooks. Up to 40% of inversions occur *without* a history of cord traction or fundal massage/pressure. Spontaneous uterine inversion has been observed at cesarean delivery by the senior author of this chapter. (See Chapter 11, The Third Stage.)

Uterine inversion is treated by prompt vaginal replacement of the prolapse [66,67]. If the inversion is complete and the placenta remains intact, manual replacement of the uterus is best performed before attempting to remove the placenta, in case of unusual placenta adherence (e.g., accreta). The initial use of tocolytics, such as intravenous terbutaline or nitroglycerin, followed by the administration of



one of the potent uterotonics, eases replacement, reduces overall blood loss, and helps the uterus to retain its correct anatomic position. Uncommonly, abdominal exploration or a combined vaginal/abdominal procedure is required to replace the uterus since reversion is a common complication. In the hours immediately after a replacement, serial reexamination by palpation and real-time ultrasound scan should accompany the administration of potent uterotonics. (See Chapter 11, The Third Stage.)

### Amniotic Fluid Embolism

Classic amniotic fluid embolism (AFE) is a syndrome of catastrophic acute cardiovascular collapse, pulmonary dysfunction, and coagulopathy that is unique to pregnant women [80–87]. AFE was first established as a clinical entity in the 1940s, and since that time a large body of literature has developed about this condition and its antecedents, risks, and treatment. AFE is now believed to be a complex immunologic reaction to an intravenous bolus of cardiovascular amniotic fluid or its contents, resulting in serious and potentially fatal cardiovascular and coagulation events. Traditionally, mortality rates have been described as high (50%–80%). As is discussed later, even the confirmation of AFE is problematic. As the classic causes for maternal morbidity decline in incidence with improvements in obstetric care (i.e., hypertension and infection), AFE has become increasingly important as a cause of maternal death. At present, AFE is responsible for approximately 10% of all maternal deaths.

The pathophysiology of this disorder is not well understood. Although it is believed that AFE is initiated by an immunologic event, the exact mechanism remains to be established. The pathogenesis of the often-associated profound uterine atony is likewise unknown. The AFE syndrome is initiated when amniotic fluid and its contents, which include fetal squamous cells, meconium, lanugo hair, and other debris, enter the maternal pulmonary circulation. Fluid entry is thought to occur by venous absorption and in most cases occurs at or about the time of parturition. The open uterine venous sinuses that are normally presented postpartum accelerate this process, facilitated by the usual myometrial contractions. This embolization results in an anaphylactic reaction, possibly to various fetal antigens. Of inter-

est, approximately 40% of the women who develop AFE have a history of allergy and, for unknown reasons, the syndrome is more common when the fetus is male [87]. Because of the suspected association of AFE with hypersensitivity, and its hematologic and hemodynamic similarities to septic shock and anaphylaxis, the term *anaphylactoid syndrome of pregnancy* has been suggested for this condition [87]. This new term reflects the uncertainties of diagnosis, the strong association between this condition and hypersensitivity, and the variation possible in clinical presentation. The term *AFE* has been in use for decades, is well embedded in the literature, and is familiar to clinicians, however. For these reasons, in this discussion the syndrome is referred to by its traditional title.

The initiation of the AFE syndrome is somehow involved with either the unique constituents of amniotic fluid or the effects of this fluid on the maternal cardiopulmonary circulation. Human amniotic fluid is known to have potent effects that mimic thromboplastin, including platelet aggregation and the rapid activation of vasoactive substances [88,89]. Embolization of amniotic fluid could also initiate a local thrombosis, potentially leading to a clinical picture that is essentially indistinguishable from that of a pulmonary embolism. At the membrane and cellular level, it is known that the pathophysiology of AFE involves mast cell degranulation, histamine or tryptase release, or activation of the complement pathway [90–92]. Tissue factor (TF), derived from fetal epithelial cells, is another important vasoactive substance associated with AFE, and could be part of the pathophysiology of the syndrome. TF binds with Factor VII and has the potential to activate the coagulation cascade by the extrinsic pathway [88]. This process can rapidly deplete circulating clotting factors including fibrinogen, as well as prompt activation of the fibrinolytic system. Many vasoactive substances, including bradykinins, prostaglandins, leukotrienes, and the various cytokines, are also involved in the AFE syndrome, perhaps with a mix unique to each case.

Although the risk for AFE is estimated from 1 in 8,000 to 1 in 50,000 pregnancies, the true incidence is unknown. Imprecision in this estimate is due to the lack of established criteria for making the diagnosis, the inclusion in some series of undocumented or suspect cases, and the sporadic nature of its occurrence. In its full-blown manifestation of

**TABLE 23.7 Amniotic Fluid Embolism and Neonatal Survival: Interval from Maternal Cardiac Arrest to Delivery\***

Interval (min)	Neonatal Survivors	Intact Survivors
≤5	3/3 (100%)	2/3 (67%)
5–15	3/3 (100%)	2/3 (67%)
16–25	2/5 (40%)	2/2 (100%)
26–35	3/4 (75%)	1/3 (33%)
36–54	0/1 (0%)	0/1 (0%)
Totals	11/16 (69%)	6/12 (50%)

\*N = 16.

Modified from Clark S, Hankins DG, Dudley DA, Dildy DA, Porter TF: Amniotic fluid embolism: Analysis of the national registry. *Am J Obstet Gynecol*, 1995. Apr; 172(4 Pt.1):1158–67; with permission.

cardiovascular collapse and coagulopathy AFE is uncommon at best. There is no established ethnic or racial predilection. The components of the syndrome that constitute the greatest threat to the mother's life are the acute hypoxia and cardiopulmonary dysfunction. Depending on whether cases of pregnant women with unexplained coagulopathy, severe uterine atony, or transient cardiovascular dysfunction are either accepted or rejected as being part of this disorder, the associated mortal risk can be reported as much lower than the commonly reported 50% to 80% [80,81]. When classic AFE occurs, morbidity is also a major problem. In the severe cases included in the U.S. registry, only 15% of the surviving women were neurologically intact [86]. Intact maternal survival after a cardiac arrest occurring in conjunction with AFE is rare. Fetal risk is likewise high. Of the 28 cases in the registry in which the fetus was alive at the time of the event, only 22 (79%) survived. Unfortunately, fewer than one half of these fetal survivors proved normal (Table 23.7).

The AFE syndrome is not unique to near-term vaginal or cesarean deliveries. AFE has been reported in association with blunt abdominal trauma or after certain obstetric procedures (e.g., amniocentesis, cerclage removal, manual removal of the placenta, forceps delivery, or curettage for mid-trimester fetal demise). AFE has also been suspected in twin gestation with intact membranes and reported in association with saline abortion in the mid-trimester [83–84,89,93–104].

In most cases, AFE occurs in laboring women in the third trimester, most often in the advanced stages of labor [104]. In approximately 80% of reported cases, the membranes are documented to have been ruptured, and in nearly three quarters, the acute symptoms develop intrapartum. In the remaining 25% to 30% of cases, most occur postpartum, with approximately two thirds after a cesarean and one third after a vaginal delivery. Although the AFE syndrome is generally assumed to be an acute reaction, documented cases occurring 30 minutes or more after delivery have been reported. The mechanism of these delayed-onset cases is unclear but could be from parturition-induced changes in maternal circulation or delayed, perhaps leukotriene-mediated, anaphylaxis [86,87].

Several previously reported associations for AFE are no longer thought to be true. Specifically, method of delivery, use of oxytocin, and tumultuous or precipitate labor are apparently unrelated factors [86]. Both severe abruptio placentae and uterine rupture remain as valid risk factors, however.

What causes most maternal deaths is the combination of acute heart failure and hypoxia. Severe and rapid-onset hypotension is among the most consistent observations among women with the AFE syndrome, and death from the syndrome is usually secondary to refractory cardiopulmonary arrest. Once initiated, the process of the syndrome rapidly progresses to a relentless spiral of profound myocardial dysfunction that results in hypoxia and leads to acidosis and then to further depression in myocardial function, which promotes more hypoxia. The principal mechanism for the sudden-onset cardiac dysfunction is not established. Proposed mechanisms include direct effects of hypoxia, coronary artery spasm leading to ischemia, or a direct depressant effect on the myocardium by an unknown circulating substance or substances. Of note, if a cardiac arrest ensues in an AFE patient, the maternal survival rate is 30% or less. Other causes of maternal death associated with AFE include adult respiratory distress syndrome (ARDS), multiorgan failure, exsanguination, and withdrawal of life support secondary to brain death.

In the classic case, the acute clinical symptoms of AFE include dyspnea, tachypnea, cough, seizures, and fetal bradycardia (Table 23.8). Sudden maternal cardiac arrest is also possible. Arterial blood gases reveal hypoxia and variable acidosis. Coagulation

**TABLE 23.8** Frequency of Specific Signs and Symptoms in Cases of Amniotic Fluid Embolism

Events*	N	%
Hypotension	43	100
Fetal distress	30	100
Pulmonary edema, ARDS <sup>†</sup>	28	93
Cardiac arrest	40	87
Cyanosis	38	83
Coagulopathy	38	83
Dyspnea	22	49
Seizures	22	48
Uterine atony	11	23
Bronchospasm	7	15
Transient hypertension	5	11

AFE, Amniotic fluid embolism

\*Note: Some women did not survive long enough to either express specific symptoms or to confirm the diagnosis of AFE.

<sup>†</sup> Adult respiratory distress syndrome.

Modified from Clark S, Hankins DG, Dudley DA, Dildy DA, Porter TF: Amniotic fluid embolism: Analysis of the national registry. *Am J Obstet Gynecol*, 1995. Apr; 172(4 Pt.1):1158-67; with permission.

studies are usually abnormal, although acute thrombocytopenia is rare. In some instances, the presenting symptoms are refractory postpartum uterine atony, with or without coagulopathy, with the cardiopulmonary phase either absent or subclinical [105,106].

When the AFE syndrome is triggered, there is a two-phase response. Phase one, which accounts for approximately 50% of the associated maternal mortality, involves severe pulmonary vasoconstriction of sudden onset. Acute cor pulmonale, hypoxia, cyanosis, hypotension, and eventually, myocardial depression and pulmonary edema, are the principal complications [106]. If the woman survives sufficiently long, a clinical condition mimicking ARDS can develop. Phase two is a rapid-onset coagulopathy, usually compounded by profound and unresponsive uterine atony. Not all cases follow this progression. Many of the women destined to succumb die rapidly of cardiopulmonary dysfunction before the coagulopathy or other complications can develop. One half of the maternal deaths occur within the first hour, and among the survivors, coagulopathy subsequently develops in approximately 50% [81]. There are no data on the likelihood of recurrence, or the possibility that morbidity or mortality could be increased if an extension of the orig-

inal embolus occurs. Of interest, normal pregnancy is apparently possible following an episode of AFE [107,108].

Acutely afflicted women are treated to maintain cardiac function, reduce bronchospasm and pulmonary artery hypertension, and to restore uterine tone as is clinically required. Transfusion of blood and blood products, endotracheal intubation and positive-pressure-controlled respiration, administration of steroids, and at times, heparin, are additional common therapies. The uterine atony associated with AFE is often resistant to standard medical management, and other treatments, including surgical interventions such as vessel ligation, balloon tamponade or gauze packing, placement of uterine compression sutures (i.e., B-Lynch or others), hysterectomy, or vessel embolization can be required for control [105]. If the mother sustains a cardiac arrest, an early perimortem cesarean delivery is indicated. In this setting, maternal recovery is unlikely, and although data are limited, a prompt delivery might improve the prognosis for the infant.

For the treating physician, there is no test or procedure that absolutely confirms the AFE diagnosis. Both clinical experience and review of the literature indicate that the diagnosis of AFE is one of exclusion. A convincing or objective verification of AFE is not possible in many cases; in some the mothers survive and in these and other instances there are no pathologic specimens for review. Also the criteria required for the diagnosis are not exact or consistent. For these reasons, a critical review of the general clinical associations (e.g., coagulopathy, cardiopulmonary dysfunction, etc.) with the obstetric events (e.g., atony, hemorrhage, etc.) is prudent when any specific case is reviewed as a possible example of an AFE. Several other clinical conditions must be considered. The acute pulmonary edema often seen in conjunction with AFE could be a complication of pregnancy-related hypertension or could be secondary to pulmonary embolism, anaphylaxis, septicemia, tension pneumothorax, or severe abruptio placentae, among other conditions.

Standard radiographic studies cannot distinguish between cases of AFE and simple pulmonary embolism. The principal radiographic abnormality on chest films is a nonspecific increased opacity, a finding that is essentially indistinguishable from pulmonary edema from any cause. For the radiologist, the differential diagnoses for such films includes

both aspiration pneumonia and diffuse pulmonary hemorrhage [109,110].

Histologic confirmation of suspected cases is often attempted; however, the identification of amniotic fluid debris in maternal pulmonary vessels varies and so is often nondiagnostic. Further, the isolated finding of squamous cells in the maternal pulmonary circulation is now considered nonspecific and not pathognomonic of the disorder [111]. Although catheter aspiration from the pulmonary artery to identify fetal cells is now discredited, histologic studies of pulmonary vessels in women on autopsy is still helpful in the retrospective assignment of the diagnosis. AFE is strongly suspected as the correct diagnosis if the clinical presentation is typical and subsequent histologic examination of vessels in the maternal lung reveals embolization of amniotic fluid debris such as mucin and not simply squames. In the 1995 Registry, in which strict clinical criteria for inclusion were used, the diagnosis of AFE was confirmed by the observation of fetal debris in the pulmonary vessels of only 73% of the women going to autopsy [86]. For the purposes of the registry report, squamous cells, hair, fat, trophoblast, and nonspecific cellular debris such as mucinous, keratinous, or proteinaceous material were considered as presumed fetal elements and accepted as histologic evidence of embolization. Although animal models for AFE have not proved terribly helpful, a recent goat model of embolization indicates that histologic evidence of specific fetal debris is more likely to be found if the embolization is produced by meconium-stained amniotic fluid, as opposed to meconium-free fluid, raw fluid, or filtered fluid [111].

To confuse the matter further, there are cases of uterine atony, coagulopathy, or transient intrapartum or postpartum cardiopulmonary distress or dysfunction that occur without documented abruptio placentae, hypoxia, or hypotension. It is theorized that these clinical events might be due to a *forme fruste* of AFE [42,97]. When the parturient survives and there is no histologic confirmation, especially if the original presentation was at all atypical, the diagnosis in such instances remains uncertain.

Research has yet to identify a satisfactory animal model for AFE or to suggest therapies based on established pathophysiology. Owing to the unpredictability of the disorder and the problems inher-

ent in establishing the correct diagnosis, there are no randomized treatment trials. Prevention of the syndrome is unknown. In the future, if the triggering mechanism for the hypoxia, atony, and cardiac dysfunction of the syndrome can be elucidated, treatment to slow or arrest the disastrous and all too rapid cascade of vasoconstriction, coagulopathy, and depressed myocardial function might be possible. Given current knowledge, the best chance to reduce the mortality associated with AFE is to train birth attendants in the rapid identification of the characteristic clinical presentation, methods of cardiovascular support, treatment of profound uterine atony with hemorrhage, and the management of acute coagulopathy.

### Pelvic Relaxation Syndrome

*Pelvic relaxation* (also termed *physiologic pelvic girdle relaxation*, *pelvic girdle relaxation*, or *symptom-giving pelvic girdle relaxation*) is a condition of pain and discomfort caused by the softening and separation of ligaments of the pelvic girdle (*os coxae*) associated with pregnancy [113,114]. A degree of relaxation of the pelvic ligaments and separation of the pubic symphysis is a normal and often asymptomatic event during pregnancy. Relaxin and other pregnancy-related hormonal substances are thought to be the cause. This type of physiologic ligament laxity spontaneously resolves postpartum without specific treatment. Surprisingly, the extent of the separation in the pubic symphysis is unrelated to symptomatology. In a substantial number of women, however, a pregnancy induces a more profound relaxation of the *os coxae*, resulting in symptoms and some degree of activity restriction. The incidence of this type of *symptomatic* pelvic relaxation is not accurately known, but in its milder forms it is common [115–119].

In terms of anatomy, both the pubic symphysis and the sacroiliac joints are amphiarthroses; that is, they are slightly movable cartilaginous articulation joints that lack both a synovium and synovial fluid. In the symphysis, a thick interpubic fibrocartilaginous disk is positioned between layers of hyaline cartilage as a cushion. Both the symphysis and the sacroiliac joints are reinforced by a web of external ligaments that normally permit minimal motion, except when they are disrupted by trauma or softened by the hormonal effects of pregnancy. Several

muscles insert over the symphysis, including the falxiform aponeurosis of the rectus abdominus (falx inquinalis), the rectus abdominus, the gracilis, the adductors brevis and magnus, the external obturator, the levator ani, the obturator internus, and the sphincter urethrae. The nerves to the pubis include the pudendal and the genitofemoral. The blood supply is provided by the pudendal, obturator, inferior epigastric, and medial femoral circumflex vessels.

The symptoms of symphysis separation apparently result from mechanical instability of the pelvic girdle. Physiologically, the pelvic bones function as arches, joined at the symphysis, that serve to transfer the weight of the trunk to the hips. During normal ambulation or single-leg standing, shear forces act on the symphysis in opposite directions at the point of the articulation of the pubic bones (i.e., the symphysis). With weakening of the connecting ligaments, the *os coxae* move much more than is normal at the pubic symphysis as well as at the sacroiliac joints, resulting in pelvic instability. With normal changes in position or with ambulation, this motion of the pelvic bones in relation to one another results in the varying distressing symptoms of pelvic relaxation.

Women with pelvic relaxation are reasonably comfortable and pain free while at rest or supine; however, walking up or down stairs or arising from a chair or bed usually results in some degree of discomfort or acute distress. This is primarily reported as pain in the area of the symphysis, lower back, or thigh [113,115]. Pain in the distribution of the sciatic nerve is also common but is not the predominant symptom. On ambulation these women often display a peculiar and characteristic shuffling or waddling gait which Shakespeare described as the "swimming gait" of pregnancy [121]. This distinctive technique of ambulation apparently serves to minimize displacement of the pelvic bones [122].

A characteristic history, combined with the findings on abdominal and vaginal examination, is diagnostic. There is usually moderate-to-acute tenderness to palpation over the pubic symphysis. If the woman is instructed to step up and down on a step or stool while the examiner conducts a transvaginal palpation of the symphysis, an unusual and paradoxical up-and-down movement of the pubic bones can often be appreciated. To establish the correct diagnosis, the history and patient's physical examination must exclude ataxia and common nerve com-

pression syndromes, such as sciatica. Once these conditions have been considered and the diagnosis is established, additional unnecessary testing can be avoided, the patient appropriately counseled, and local relief measures instituted.

Treatment during pregnancy is symptomatic and often less than ideal. Mild analgesia, acupuncture, a pelvic binder, physical therapy, use of walkers, bedrest, and other local treatments provide some relief [120,123,124]. There is an extensive lay literature about this condition, with a plethora of additional potential therapies that include chiropractic or osteopathic maneuvers, among others [123]. Occasionally, direct injection of the symphysis with local anesthetic agents with or without a steroid relieves acute symptoms. The only definitive treatment is delivery, however. In most cases not caused by traumatic or surgical separation of the symphysis, improvement or full recovery can be confidently anticipated as the hormonal effects of pregnancy abate postpartum. It is possible for a chronic pelvic pain syndrome to develop in some cases. If a spontaneous traumatic rupture occurs intrapartum, or if surgical division of the symphysis was performed, the triad of chronic pubic pain, urinary incontinence, and pelvic instability are possible long-term complications.

In a small percentage of women, pregnancy incites a syndrome of pelvic joint pain that persists after pregnancy [125,126]. This chronic and debilitating condition is preceded by what clinically appears to be a routine case of obstetric pelvic relaxation. If the symptoms improve over time, this more severe disorder often recurs with subsequent pregnancies, and signs and symptoms can persist for years. In one prospective study, the likelihood of some degree of persisting discomfort up to two years postpartum occurred in up to 9% of women who initially developed pregnancy-related pelvic joint pain [121–126]. Furthermore, if pain was initially reported in all three pelvic joints, the incidence of the persistence of the pain after six months doubled. There is an association between this long-term syndrome and both a family history of pelvic pain occurring with pregnancy and developmental hip dysplasia, suggesting a more complex etiology [126]. When this long-term disorder develops, it is difficult to treat. Rest, pelvic supports, acupuncture, and rarely and only in selected cases, surgery are potential therapies.

Infrequently, acute symptoms referable to the pubic symphysis and ambulation follow rupture of the symphysis. This is a complication that can accompany either a spontaneous or an instrumental delivery or be associated with obstetric manipulations, such as the McRoberts maneuver for relief of a shoulder dystocia [120,127–129]. When spontaneous rupture of the pubic symphysis occurs, an audible snap or crack accompanied by a sudden lessening of difficulty during fetal extraction or spontaneous delivery is often reported. In other instances, however, a specific traumatic episode is not recalled. On physical examination, palpation elicits pain over the symphysis. The pain in the symphysis usually can be reproduced when both greater trochanters are pressed together toward the midline. The patient is usually unable to flex at the hip when the legs are extended. Clinical associations for spontaneous symphysis rupture include prior symphyseal symptoms, precipitate labor, abnormal presentation, fetal macrosomia, use of the McRoberts maneuver, instrumental delivery, or relative fetopelvic disproportion. In many cases, no specific predisposing factor beyond pregnancy is identified.

Infrequently, the symphysis is intentionally divided at the time of delivery to permit extra pelvic room in cases of cephalopelvic disproportion or shoulder dystocia. This procedure, *symphysiotomy*, is not recommended to the inexperienced physician. Elective symphysiotomy is rare in Western practice; however, it continues to be employed in other parts of the world where it is occasionally employed to avoid the peri- and postoperative morbidity of cesarean delivery [130–133]. (See Chapter 18, Cesarean Delivery.)

Other rare and unusual conditions can have a similar presentation to pelvic relaxation. The most likely is osteomyelitis of the pubic symphysis, a condition that rarely follows vaginal delivery. In these cases, a localized infection disrupts the continuity of the symphysis. Prior incontinence surgery, sports injuries, pelvic malignancy, and intravenous drug use are much greater risk factors than obstetric delivery for infection of the symphysis pubis, however [134–136].

### Coccygodynia

A potentially disabling obstetric injury little discussed in standard obstetric texts is fracture, disloca-

tion, or other trauma to the maternal coccyx, leading to the syndrome of *coccygodynia* [137–150]. This condition includes various distressing symptoms such as poorly relieved perirectal pain, pain on sitting or with pelvic/rectal examination, and chronic dyspareunia [138]. The etiology of chronic pain associated with the coccyx and surrounding tissue is not clear but is believed to arise from several related causes. Hypermobility of the coccyx caused by the hormonal changes of pregnancy could permit more flexion or extension than usual, as well as changing the resting tension of ligaments and muscles, leading to pericoccygeal inflammation. An inflammatory focus that develops in the area of the coccygeal synchondrosis can also extend to other anatomically related structures, including the anococcygeal, sacrospinal, and sacrotuberal ligaments, and the gluteus maximus and levator ani muscles, thus amplifying the original discomfort. Direct delivery trauma leading to coccygeal hypermobility or subluxation is another possibility [138,142].

Anatomically, the coccyx varies in both size and shape and includes three or four vestigial vertebrae bound together by fibrous tissue or bone. At the sacrococcygeal junction, there is usually a movable joint. The coccygeal segments following the first bone display variable development of the vertebral arches and often consist only of small bony nodules of varying size. Neither the number of coccygeal segment nor the extent of joint fusion is related to existence of pain or to its intensity [141]. The primary blood supply to the coccyx is by the medial sacral artery, arising from the abdominal aorta near the bifurcation. The nerve supply is primarily from the fifth sacral root. The nerve roots exit above the first coccygeal vertebra and contribute to the coccygeal plexus, formed from the anterior division of roots L4–5 and the coccygeal nerve.

The differential diagnosis for coccygodynia includes several disorders, including referred pain from rectal disorders, lumbar or sacral disk disease, a variant of the pelvic relaxation syndrome, or a pilonidal cyst. Rarely, a tumor of the cauda equina or the coccygeal region can result in similar symptoms.

The diagnosis is based principally on clinical findings and history. As noted, among the afflicted, chronic debilitating pain in the area of the coccyx and dyspareunia are common complainants. On physical examination, palpation or movement of the coccyx elicits or reproduces intense discomfort.

Although a pelvic radiograph might reveal displacement or unusual angulation of the coccyx, similar findings are also noted in asymptomatic subjects; thus, standard radiographic findings are not usually helpful in confirming the diagnosis. In recent years, more specialized imaging studies, including a dynamic sitting lateral view of the coccyx, have been suggested as better techniques for accurately identifying abnormal anatomy. A pathologic subluxation is defined as the coccyx being displaced by more than 25% from the standing to the sitting view [142–144]. Documented coccygeal hypermobility and the failure to respond to local measures can help to identify those patients who are likely to improve with surgery.

For many years, the syndrome of coccygodynia has presented difficulties as a distinct medical entity [144]. Nonetheless, there are patients who develop symptoms discretely referable to the coccyx or the paracoccygeal tissues after obstetric trauma from both spontaneous and assisted vaginal delivery. Unfortunately, the etiology of the pain associated with coccygeal injury or pericoccygeal inflammation is imperfectly understood, and the literature concerning the pathophysiology and therapy of this condition is long, confusing, and often contradictory. In treating patients with coccygodynia, it is prudent to make haste slowly, evaluate patients carefully, and follow an established protocol. All authors underscore the importance of excluding patients whose symptoms are unrelated to the coccyx or who are emotionally unstable.

Treatment is symptomatic, and several methods might need to be attempted to provide relief [138,143,145–147]. Analgesics, sitting on an air cushion, injection of local anesthetics with or without steroids, various direct manipulations or massage of the coccyx, and acupuncture are the most common treatment methods. In patients with a mobile displaced coccyx, manipulation to restore it to its anatomic position can provide some relief. Other treatment modalities include localized nerve ablation, cryoanalgesia, and steroid therapy by iontophoresis. Unfortunately, recovery can be incomplete and prolonged.

Surgical removal or repair of the coccyx is considered in selected severe cases but should not be contemplated until after several months of conservative treatment [147,148]. Elective surgery should not be performed during pregnancy. Operative treat-

ment must be approached with trepidation, because reflux sympathetic dystrophy, chronic severe pain, and causalgia can follow surgery. Despite these risks, 75% or more carefully selected patients have their symptoms relieved by partial or complete coccygectomy if other forms of nonsurgical treatment fail [143,147–150]. If the coccyx heals, refracture or dislocation can accompany a subsequent pregnancy, with recurrence of the syndrome. Thus, a prior history of coccygodynia can be a potential reason for a cesarean delivery in a subsequent pregnancy.

### Other Causes of Maternal Injury

Medical errors are another potential cause of maternal injury. The magnitude of this risk for obstetric patients is not known, and good data are limited. As all clinicians recognize, errors in the provision of medical care, particularly to hospitalized patients, are possible. Because any injuries caused by error are potentially avoidable, methods of prevention need consideration. In 1999 and in a follow-up study in 2001, The Institute of Medicine published a detailed report on errors in hospitals [151]. It was claimed that errors were responsible for nearly 100,000 deaths a year among hospitalized patients in the United States. These reports and other studies generated in response to these documents have proposed important design changes for the provision of medical care, including revision in information technology, communication between caregivers, coordination of care, and the effective use of performance and outcome measures. In obstetric services, reform is most visible in the operating suite and when treatments or medications are ordered. New methods of surveillance to identify and learn from various “near-miss” events have been instituted. Different systems for patient identification and surgical site verification to avoid surgical and other errors have been introduced for use in the operating suite. The issue of correct patient identification in the operating suite seems at first glance to be of less importance in obstetric practice than in general surgery. Procedures such as cesarean delivery are usually conducted only after the parturient has been hospitalized for some time and physician, midwifery, or nursing personnel have been in immediate attendance. This fact should make misidentification a remote risk. There are other considerations, however. In many if not most busy clinical

services, a varying percentage of the parturients do not speak or understand English. Further, emergency surgery is occasionally required on gravidas rapidly transferred from an emergency service or a triage or prep unit. Some of these women have not had prenatal care; others have had their prenatal care in other institutions or other cities, making their records effectively available. Some patients could be unresponsive owing to their medical conditions or otherwise unable to communicate. Other persons destined for surgery, such as newborns for circumcision, are equally incapable of identifying themselves and must depend on the protocols of the institution to identify and verify their correct surgery. When surgery is looked at in this light, the meticulous identification of any person undergoing surgery and a secondary verification of the intended procedure are necessary requirements, despite the redundancies and limitations of current methods. Another set of changes in hospital practices affecting the obstetric clinician concerns the pharmacy. Hospital pharmacies have markedly changed the manner in which oral and intravenous medications are ordered, stocked, and administered. In order to avoid errors, the addition of various electrolytes and commonly used drugs such as magnesium sulfate and oxytocin to intravenous solutions on nursing or labor and delivery units has been abandoned. Premixed solutions are now prepared and dispensed by a central pharmacy. The use of some drugs is also restricted, based on institutional protocol. Thus, not all drugs preferred by some practitioners are on the formulary. As with surgery, the effort in these changes is to avoid error by the standardization of practice and simplification of routine procedures.

There has also been interest in *crew resource management*. This is a model for handling complex events that was originally developed in the aviation industry in response to accident analysis. In medicine, in which circumstances are also similarly complex and often rapidly changing, crew management involves the organization of multidisciplinary professional teams (med teams). These teams receive special group training. The aim is to ease communication, encourage unrestrained questioning of events up the hierarchy, and introduce various redundancies to better ensure quality and safety. The theory is that the more cooks that watch the pot, the less likely that something will be overlooked or be done incorrectly. More will be heard

concerning this type of organization in the future as these methods are better refined for use in clinical services.

## FETAL INJURIES: OVERVIEW

Fetal/neonatal abnormalities can be divided into those resulting from *mechanical*, *hypoxic/asphyxic*, *congenital*, *developmental*, or *infectious* causes. Hypoxia/asphyxia and mechanical trauma often coexist, especially when more serious injuries are considered. In recent decades, the incidence of mechanical injuries has decreased owing to nonspecific improvements in perinatal care and the avoidance of difficult instrumental procedures and fetal extractions (Table 23.9). A brief review of various types of potential infant injuries associated with parturition illustrates the problems associated with efforts to reduce the incidence of damaged babies.

### Head and Neck Injuries

Birth injuries to the fetal head or neck occur with an overall incidence of approximately 1% [152]. Cephalohematomas are the most common lesions, accounting for 56% of all head/neck injuries in the series reported by Hughes and coworkers. In this study, facial lacerations (12%), facial nerve palsies (8.6%), skull fracture (2.9%), injuries to the nasal septum (0.6%), and the phrenic (1.7%) or laryngeal nerve (0.6%) were also reported. Head or neck injuries can accompany other significant fetal trauma such as brachial plexus or spinal cord injuries; thus, complete evaluations are necessary. Although some of these injuries are of minimal clinical importance or resolve rapidly, others are potentially much more serious.

### Minor Scalp Injuries

Mild scalp abrasions and ecchymoses are common following vacuum extraction, forceps-assisted, and spontaneous delivery. These are not of clinical importance; however, rarely, serious and even life-threatening cranial injuries occur after instrumental delivery with either a vacuum extractor or forceps.

Minor scalp lacerations are common after either fetal scalp sampling or the application of fetal scalp electrodes. The surgeon also occasionally



TABLE 23.9 Incidence of Birth Trauma in Two Studies 20 Years Apart

Injury	Rubin (1964) (n = 15,435)		Scotland (1980) (n = 51,191)		Reduction
	Number	%	Number	%	
Clavicle fracture	43	2.8	28	0.5	5.6
Facial nerve	21	1.4	30	0.6	2.3
Brachial plexus	18	1.2	5	0.1	12.0
Intracranial	13	0.8	35	0.7	1.1
Humerus fracture	7	0.3	3	0.06	5.0
Phrenic nerve	2	0.1	0	0.0	
Spinal cord	1	0.06	1	0.02	3.0
Lacerations	9	0.6	16	0.3	2.0

From Walker CHM: Birth trauma, In: Crawford JW. Risk of Labour. Chichester: John Wiley, 1985. pp 71–93; with permission.

inadvertently lacerates the fetal scalp during a cesarean while incising the myometrium. Potential complications of scalp lacerations include localized scalp abscess, hemorrhage, and localized or rarely systemic infection. Uncommon and rare complications of direct electrode application include osteomyelitis, herpetic infection, necrotizing fasciitis, or inadvertent spinal fluid leak [153–157]. Fetal HIV infection is another potential risk whenever the fetal skin is punctured or lacerated in an at-risk pregnancy.

Simple local treatment occasionally is required for minor injuries to the scalp. If the scalp is lacerated or punctured, cleansing with an antibacterial soap followed by the application of a topical antibiotic ointment is appropriate. Rarely, for larger injuries, suturing with fine (5–0, 6–0) interrupted sutures of polyglycolic acid is required. Such injuries should be promptly discussed with the parents. The pediatrician should also be informed to avoid a potentially uncomfortable situation with the family. Extensive scalp lesions require careful evaluation to exclude underlying subgaleal hematomas, cephalohematomas, or other potentially serious cranial injuries such as subdural hemorrhages, skull fractures, or other trauma.

The *chignon*, a peculiarly shaped, localized collection of caput succedaneum formed by vacuum extractor cups, is the most common type of scalp lesion associated with vacuum extraction (Figure 23.2). Chignon formation is usually minimal when soft-cup extractors are used [158]. Although



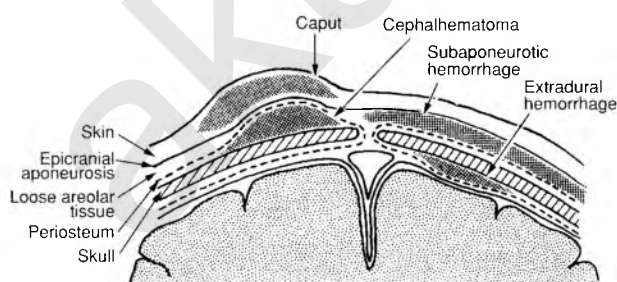
FIGURE 23.2.  
*Chignon produced by rigid-cup vacuum extractor. (Bird's modification; see Chapter 17, Instrumental Delivery, for discussion.)*

cosmetically unpleasant, the chignon is simply a localized form of the normal process of caput formation or scalp edema that generally disappears within 12 to 24 hours and results in no permanent injury. The importance of the chignon is the reaction of others to it. This distortion of the cranial outline is unusual and even frightening to the uninitiated, who have not been properly forewarned of its benign and transient nature. The common development of the chignon with rigid-cup vacuum devices contributed to the initially slow acceptance of vacuum extraction in American practice. (See Chapter 17, Instrumental Delivery.)

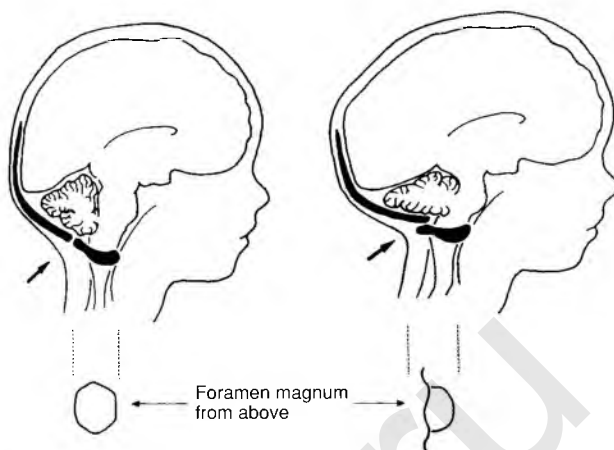
Serious scalp injuries are possible but quite uncommon. As discussed later, these lesions include partial scalp necrosis, and more frequently and of greater importance, the formation of cephalohematomas and subaponeurotic (subgaleal) hematomas. Jaundice and anemia, and much less often, infection, coagulopathy, vascular collapse, or even death can follow such injuries [159].

### Cephalohematoma

A *cephalohematoma* is the most common cranial injury among newborns, occurring in approximately 2.5% of live births [160]. A cephalohematoma is a collection of blood beneath the pericranium (periosteum), resulting from the laceration of subperiosteal vessels (Figure 23.3). The cause is thought to be shearing of the soft tissues of the scalp as they are displaced back and forth over the more rigid underlying bone during a spontaneous or instrumentally assisted delivery. There are small communicating vessels bridging between the veins in the diploic space, which lies beneath the inner and outer



**FIGURE 23.3.**  
*Sites of cranial and extradural hemorrhage in the cranium of the newborn. (From Pape WE, Wigglesworth JS: Hemorrhage, Ischaemia and the Perinatal Brain. Philadelphia: JB Lippincott, 1979; with permission.)*



**FIGURE 23.4.**  
*Occipital osteodiastasis, proposed mechanism of injury. See text for details. (From Wigglesworth JS: Textbook of Fetal and Perinatal Pathology. Boston: Blackwell Scientific Publishers, 1991; with permission.)*

tables of the fetal skull, and both the pericranium and meningeal veins positioned on either side of the calvarium. With scalp trauma, these superficial venous connections rupture, leading to the formation of a hematoma. The size of the resulting hemorrhage is limited by the area of the involved cranial bone. Cephalohematomas occur more frequently in instrumental than in spontaneous deliveries and are more common especially after midforceps and operative vacuum extraction procedures, in which, presumably, scalp trauma is more marked [161].

Immediately after delivery a large cephalohematoma can be difficult to distinguish from the normal caput succedaneum (Table 23.10). There are however several important clinical distinctions. In a caput succedaneum, the associated scalp edema varies from soft to moderately firm, and most importantly, extends *across suture lines* because it is not confined by the periosteum of the bone. In contrast, in a cephalohematoma, the hemorrhage is confined in size to the limits of a single cranial bone owing to the firm attachments of the periosteum at the periphery of the bony plate, which restrict the expansion of the hematoma.

On physical examination, a cephalohematoma is suspected when a soft-to-firm but well-circumscribed cranial swelling, restricted to a specific cranial bone, is palpated. This swelling does not move with the scalp, as is usually true for the simple caput succedaneum. Often, the edge of a

TABLE 23.10 Scalp Birth Injuries: Differential Diagnosis

Injury	Clinical Findings	Expansion Postdelivery?	Crossing Suture Lines	Hemorrhage Possible?
Caput succedaneum	Soft mass, pitting, commonly midline, overlying vertex	No	Yes	No
Cephalohematoma*	Firm to tense, restricted to one cranial bone, usually parietal and unilateral	Yes	No	No
Subgaleal <sup>†</sup> Subaponeurotic hemorrhage	Variable, soft to firm, usually fluctuant; systemic signs possible	Yes	Yes	Yes

\*Underlying skull fracture in 10%–25% of cases.

<sup>†</sup>Can be accompanied by a coagulation defect, cardiovascular collapse, or anemia. See text for details.

Modified from Volpe JJ: Neurology of the Newborn, ed 4. 2001, Philadelphia: WB Saunders; with permission.

cephalohematoma is raised, whereas the center is slightly depressed. As discussed in greater detail in the next section, a large cephalohematoma can also be difficult to differentiate initially from a subgaleal hemorrhage, a potentially much more serious injury. There are several important clinical differences between these conditions, however. First, cephalohematomas are common, whereas subgaleal hemorrhages are not. Although a cephalohematoma can be large, it is self-limited. Thus, a cephalohematoma cannot progress in the same fashion as a subgaleal hemorrhage. Finally, although cardiovascular collapse or hypoperfusion/hypovolemia is relatively common after a subgaleal bleed, such serious complications are rare with cephalohematomas. Occasionally, neonatal anemia or hyperbilirubinemia results from a cephalohematoma or its subsequent resorption. Despite their appearance, cephalohematomas are usually inconsequential, unless they conceal an underlying skull fracture (5%–20%), or, in very unusual circumstances, a coagulopathy is also present [164–175]. For uncomplicated cephalohematomas, no specific therapy is required. Aspiration is specifically contraindicated, because these lesions regress spontaneously and the introduction of a needle unnecessarily risks infection. If a cephalohematoma is accompanied by neonatal neurologic symptoms or signs and symptoms of cardiovascular instability, and a subgaleal hemorrhage has been excluded, a prompt CT scan or MRI study is indicated to exclude an occult skull fracture or other intracranial injury. In this setting, the clinician should also carefully examine the infant for other evidence of trauma. Very

infrequently, cephalohematomas become infected, usually but not invariably after an attempt at aspiration, a history of spiral electrode attachment, or related to systemic seeding from neonatal meningitis or septicemia [165,166]. The responsible organisms are predominately *Escherichia coli* and, to a lesser degree, *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and coagulase-negative Staphylococci [165]. Perhaps the rarest complication is calcification of the cephalohematoma, with resultant craniosynostosis [167].

### Subgaleal (Subaponeurotic) Hemorrhages

In subgaleal hemorrhage, bleeding occurs in the potential space between the periosteum of the skull (pericranium) and the galea aponeurotica (epicranial aponeurosis), owing to rupture of the emissary veins that connect the dural sinuses to veins of the scalp. The aponeurosis is a thin, but remarkably sturdy tendinous sheet that passes over the calvarium, connecting the frontal and occipital portions of the occipitofrontalis muscle. Laterally, the aponeurosis attaches to the zygomatic arch and periauricular muscles. Unfortunately, the underlying tissues and the calvarium are quite plastic in the fetus and provide little effective compression to restrict the growth of a developing hematoma. Thus, a hemorrhage developing beneath the galea can dissect freely throughout the large subfascial space or posteriorly into the suboccipital region [159,163]. This potential space can accept a surprising percentage of the neonatal blood volume, up to 250 ml in some cases or more. Very large and tense hematomas can also

result in secondary extracranial cerebral compression and increased intracranial pressure.

Approximately 50% of subgaleal hemorrhages follow vacuum extraction, whereas 30% occur after spontaneous vaginal deliveries [159,169,170]. About 10% are observed after cesarean delivery, and 14% are the sequelae of forceps deliveries. In some of these cases, a congenital coagulopathy is a predisposing factor. In particularly difficult deliveries, other cranial trauma, such as skull fracture or a subdural or intraparenchymal hemorrhage, is also present. The prevalence of subgaleal hemorrhages at delivery is approximately 1.5 per 10,000 births [159,169,170]. Unfortunately, neonatal administration of parenteral vitamin K has no effect in preventing subgaleal hemorrhages. The increase in coagulation activity after this treatment is not rapid enough to offset the formation of the hematoma.

Most large subgaleal hematomas are easy to diagnose. A subgaleal hematoma is suspected when an unusual diffuse and fluctuant swelling of the neonate's scalp is palpated. On examination, especially early in their progression, a subgaleal hematoma can be indistinguishable from normal scalp edema (caput succedaneum) or mistaken for a cephalohematoma. In contrast to both caput and cephalohematomas, the swelling of a subgaleal hematoma is not fixed and usually shifts dependently when the infant's head is repositioned. On examination, the scalp might have a peculiar feel, as if a viscous liquid were trapped in a plastic bag. In most of these hemorrhages, the scalp indents on palpation. Less often and in the presence of large hemorrhages, the scalp is tense or firm. In the latter case, there are also usually systemic signs present, such as tachycardia, hypotension, or even vascular collapse. Occasionally, blue-black scalp discoloration from blood extravasation appears over the lateral margins of the frontal or occipital bones. Infrequently, the cranial findings are unremarkable, and hypotension and pallor are the only demonstrable signs.

Subgaleal hemorrhages can result in clinical signs and symptoms soon after delivery, and most cases are diagnosed within the first several hours of life. The growth of the hemorrhage can be insidious, however, and its recognition delayed to as long as 48 hours after birth. Thus, even if after an initial examination, the scalp appears normal, this does not exclude the diagnosis. As it resorbs, the hemoglobin

sequestered in the hematoma can result in hyperbilirubinemia and neonatal jaundice.

The diagnosis is usually rapidly established after a brief physical examination of an infant with a suspect history. A prior instrumental delivery, especially by vacuum extraction, evidence of neonatal scalp injury, a rising pulse rate, increased respiratory rate, hypotension, clinical documentation of a rapid drop in hemoglobin/hematocrit, and a suspicious clinical examination collectively suggest a subgaleal bleed. Uncommonly, there are signs of cerebral irritation, including convulsions. These signs might indicate a concomitant intracranial injury or reflect increased intracranial pressure secondary to a large hematoma. Although the diagnosis remains clinical, the hematoma can be confirmed by real-time cranial ultrasound examination or radiographic imaging. These studies reveal a dependent, echo-free space in the scalp that crosses suture lines. In most instances, such confirmation is not required, and treatment should not await such investigations. Both CT scans and MRI studies can establish the diagnosis while also evaluating the calvarium for possible fractures and noting the coexistence of any other CNS injury.

Many cases of subgaleal hemorrhage associated with vacuum extraction are thought to be preventable by close attention to technique and the limitation of effort [168,171–174]. In 1999 and 1998, respectively, both Health Canada and the U.S. Food and Drug Administration (FDA) issued warnings about the association between vacuum operations and subgaleal hemorrhages. The current recommendation is to inform the pediatrician or those responsible for the neonate's care whenever a vacuum extractor has been used, regardless of the child's apparent normalcy, so that appropriate attention can be given to the infant's vital signs, physical examination, and in selected cases, serial laboratory data.

Treatment for a subgaleal bleed is supportive. Fresh-frozen plasma, vitamin K1 (phytonadione), isotonic salt solutions, or packed red blood cells are administered as required. Vigorous cardiovascular support is sometimes necessary, because a substantial percentage of the neonate's blood volume can be lost into the subgaleal space. If the clinical associations for the hemorrhage are atypical, studies to evaluate a possible fetal coagulopathy are also indicated. This is a dangerous condition. In the dated

review of Plauché, 28 of 123 neonates with subgaleal hematomas died, a mortality rate of 22.8% [159]. In more recent reports, Gebremariam, from Addis Ababa, reported 69 cases of subgaleal hemorrhage drawn from 23,353 live, term deliveries [169]. Of these infants, 14% died. In a 2006 report, Kilani and coworkers reported a mortality rate of 11.8% [170]. In this latter Ethiopian study, all survivors who could be subsequently located were neurologically and developmentally normal.

A review of the mortalities associated with subgaleal hemorrhage shows several important associations. First, there is a high incidence of other cranial injuries, such as subarachnoid and subdural hemorrhage [170]. Intraventricular and intraparenchymal bleeds are also possible. Coagulopathy, hemodynamic shock, marked volume depletion, anemia, and the need for transfusion with blood or blood products are frequently observed.

Clinicians must remain vigilant to detect this sometimes insidious and potentially serious condition. In current practice, with early diagnosis and prompt therapy, neonatal mortality from subgaleal hemorrhage should be at best unusual.

### Skull and Facial Fractures

Skull fractures are usually identified after radiographic studies performed for soft-tissue injury to the scalp, such as cephalohematoma or suspected subgaleal hemorrhage [175]. Less often, an obvious deformity of the calvarium is present. These fractures appear either as linear cracks on radiographic, CT, or MRI images, or clinically as "Ping-Pong ball" depressions of the skull. The depressions are a localized inward buckling of a bone of the skull. Although cranial fracture is a clearly uncommon finding, its incidence in neonates is not accurately known, because doubtless many cases are never clinically identified.

Regardless of cause, depressed fractures virtually always involve the frontal or parietal bones and are rarely accompanied by intracranial hemorrhage or cerebral contusion [176]. The most frequent nondepressed fractures found at postmortem examination are linear fractures of the parietal bone, commonly extending along the lines of cleavage. Despite the presence of a fracture, the infant's activity/behavior is usually normal unless an associated intracranial

hemorrhage is present. In the now-rare event of basal skull fracture, drainage of bloody cerebrospinal fluid from ears or nose, shock, or various neurologic abnormalities are possible.

Occasionally, the depressed or "Ping-Pong ball" types of skull fractures are observed without a history of instrumental delivery. Such lesions are thought to be due to a localized in-utero compression, with secondary fetal skull impingement on the promontory of the mother's sacrum. Rarely, such fractures are the result of maternal abdominal trauma.

A depressed fracture usually has minimal long-term consequences as long as intracranial hemorrhage or a brain contusion is not present. Complete recovery is the rule. The presence of the fracture might or might not imply traumatic delivery or the use of excessive force, because some lesions occur spontaneously after precipitate labors. Regardless of clinical association, if a fracture has been diagnosed, it is prudent to search for additional injuries involving the underlying intracranial structures.

When an instrumental delivery has been performed, depressed fractures are essentially restricted to cases in which forceps have been applied or to those involving sequential applications of both forceps and a vacuum extractor. Fractures can follow a vacuum extraction operation alone, but this is unusual. Associated intracranial injuries are more likely when any cranial fracture follows an instrumental, as opposed to a spontaneous, delivery.

Given the frequency of instrumental delivery and the marked cranial molding common to many normal deliveries, cranial fractures might be anticipated to occur at a much higher rate than they actually do. The reason for their rarity relates to the mechanics and physics of cranial plasticity. The fetal skull is normally capable of considerable cranial distortion because the bones are poorly mineralized and easily deformed. In addition, the bones of the calvarium are separated into separate plates, permitting flexion at the suture lines. Collectively, these anatomic features successfully accommodate for the cranial deformation that normally occurs in most cases as the fetal head negotiates the bony pelvis or when it is subjected to traction during an instrumental delivery.

Diagnosis of a cranial bone fracture often requires close study of skull MRI, scans, or standard

radiographs, because these lesions are difficult to identify and artifacts are common. These studies should be referred to an experienced radiologist. "Ping-Pong ball" depressions aside, fractures are rarely evident on physical examination, except in extreme cases.

For simple linear fractures, no specific therapy is required. Depressed fractures can be elevated by digital pressure at the periphery or the application of vacuum by a breast pump or an obstetric vacuum extractor. Spontaneous elevation can occur in smaller lesions. Thus, surgical intervention is not always required [177]. The biggest problem with a fracture is that the attending physician or midwife is open to a claim of excessive force. The magnitude of risk, however, remains low. In the data from the National Collaborative Perinatal Project, only 12 perinatal skull fractures were clinically identified among some 40,000 births. All of these children were neurologically normal at 1 year of age [178].

Rarely, leptomeningeal cysts develop at cranial fracture sites, or most unusually, at a fontanelle [179]. This cyst is formed when the arachnoid herniation protrudes through a traumatic laceration of the dura matter. Fluid subsequently collects, widening the cranial defect. The resulting cystic mass often pulsates, transilluminates, and progressively enlarges. These cysts are associated with underlying brain injury and seizures, and surgical repair is necessary.

Traumatic separation of the squamous and lateral portions of the occipital bone (occipital osteodias-tasis), with a resulting posterior fossa hematoma, is now a rare condition that occasionally proves fatal (see Figure 23.3) [157]. During delivery, excessive suboccipital pressure can rupture the cartilaginous connections between the squamous and posterior segments of the occipital bone, permitting the lower edge of the squamous portion of the bone to rotate forward. The sharp edge of the bone then lacerates the dura or the occipital sinus. Occasionally, contusions of the cerebellum are also present. The important related problem is increased pressure in the enclosed posterior fossa from an expanding hematoma. The extent of compression varies, and a posterior fossa hemorrhage from an occipital bone injury, although serious, is by no means inevitably fatal.

Classically, occipital osteodias-tasis was most closely associated with traumatic breech delivery, but it can also occur after a Kielland forceps rotation for a transverse arrest. An equally rare cause is a normal presentation with prolonged labor and marked cranial molding. Most infrequently, occipital injuries are complicated by the bizarre complication of embolization of cerebellar fragments into the general circulation [157,180,181].

The best management is avoidance. When breech delivery is performed, the accoucheur should carefully follow established protocols and avoid rapid or forceful deliveries. These actions, combined with the conservative use of forceps, mostly avoids this unusual but potentially fatal complication. (See Chapter 12, Breech Presentation, and Chapter 17, Instrumental Delivery.)

### Other Craniofacial Injuries

Delivery-associated facial injuries often involve the nose. In approximately 5% of deliveries, the cartilage of the nasal septum is dislocated [120]. Fracture of the nasion or fracture of the nasal bones or septum is also possible. These complications predominantly occur in association with persistent occiput posterior presentations and assisted delivery. Not surprisingly, there is an association between such injuries and nulliparity, a prolonged second stage of labor, and forceps applications. These injuries are rarely, if ever, of serious clinical consequence, although respiratory embarrassment or neonatal feeding problems are possible.

There is a weak association among malocclusion, mandibular hypoplasia or other dental defects, and a history of difficult instrumental delivery. The available data are weak, and the strength of these associations, if any, remains suspect.

### Eye Injuries

Birth injuries to the eye are usually but not exclusively iatrogenic [182–189]. Fortunately, most resolve without important sequelae. The most common culprit is a forceps delivery, although rarely, inaccurate applications of a spinal electrode or even an injudicious episiotomy incision can injure the eye or the eyelid [154,182–184]. Inexpert rotations, and to a lesser extent, poor cranial placement of the

forceps blades, can result in a blade directly impinging on the infant's orbit. Direct corneal injuries, hyphema, choroidal rupture, retinal hemorrhage, damage to Descemet's membrane, or injuries to the optical or oculomotor nerve are possible outcomes [184–187]. All of these injuries are rare. Although resolution is possible, these injuries can also progress to various abnormalities in vision, such as astigmatism or rarely to complete unilateral loss of vision. Prevention requires the careful application of forceps, especially in cases of marked cranial malpositioning when blade application is difficult and wandering is required. When forceps malapplication is suspected as the cause of an ocular injury, close examination of the neonate might note characteristic marks from the companion blade on the back of the fetal head 180° from the observed facial injury, strengthening the presumed association with instrumentally assisted delivery [189].

#### **Fractures: Clavicle, Humerus, Radius, and Femur**

The likelihood of a long-bone fracture during delivery is low, with an incidence of less than 1%. Excluding syndromes such as osteogenesis imperfecta associated with congenital abnormality of bone structure, the principal clinical associations for fracture of peripheral bones include obstetric maneuvers such as relief of shoulder dystocia [190], or classically, breech delivery by extraction. Difficult fetal extraction at cesarean delivery, maternal abdominal trauma, and external cephalic version are additional but rare causes [191–195]. Prematurity is often an accompanying condition when a fracture is identified.

In prior years, three quarters of humerus and femur fractures occurred in breech presentations, most often after extractions [196]. With the decline in breech deliveries, these injuries are seen less frequently. Fractures of the humerus usually occur in the middle third of the shaft and are either transverse or spiral. A transient radial nerve injury can accompany the fracture but is uncommon. In cephalic presentations, the humerus is most often fractured by oblique traction, now most frequently while disengaging an impacted shoulder or arm during the delivery of the posterior arm in a shoulder dystocia. On occasion, the humerus can be electively fractured to assist delivery in a difficult dystocia case. Often a crack or snap is audible as the

humerus fractures, or the operator feels the bone give as the extremity is grasped and traction applied. Femoral shaft fractures occur in the middle third of the bone and are usually transverse. This injury is normally caused by traction on the extremities during breech delivery, or rarely after vigorous efforts at fetal extraction during a cesarean. Traumatic separation of the upper femoral or humeral epiphysis can occasionally occur under similar clinical settings, mimicking a joint dislocation or fracture.

When long-bone fractures are present at birth, neonatal limb motion is usually reduced or absent. Most often, the neonate simply avoids moving the involved extremity. Less frequently, dislocations or obvious deformities of the limbs are observed. Because of the infant's not moving the extremity, the clinician initially might suspect nerve trauma, such as a brachial plexus or spinal cord injury. Physical examination usually confirms the correct diagnosis, because palpation detects tenderness, swelling, or irregularity along the affected bone. Direct palpation of the suspect site or passive movement of the limb usually elicits cries of pain. Standard radiographs confirm the injury.

Fractures from intrapartum manipulations should be distinguished from pathologic fractures that occur in infants with various congenital abnormalities in bone structure, such as osteogenesis imperfecta. In these unfortunate cases, fractures can occur spontaneously in utero or from minimal manipulation. A family history, prior abnormal ultrasound examination, observation of multiple fractures at the time of delivery, or evidence of old, healing long-bone injuries should prompt the clinician to investigate the possibility of an intrinsic abnormality in bone structure.

Nondisplaced humeral and femoral fractures require no specific therapy beyond immobilization, and associated nerve injuries are rare. The old clinical saw that "any two fetal bones left in the same room will knit" does have validity. Displaced upper extremity fractures are immobilized to prevent potential injury to either the brachial plexus or the pleural cavity. Surgical exploration is rarely required, and recovery is usually rapid and complete as long as there is no nerve involvement.

The problem with long-bone fractures is not in establishing the correct diagnosis or in adverse outcomes. The real difficulty occurs with the families, who usually have pointed questions to ask about the

management that led to the injury. When any such uncommon or potentially serious injuries occur, careful counseling is necessary to avoid inappropriate conclusions about intrapartum management.

The clavicle is the bone most commonly fractured during delivery. Depending on the population studied, the incidence is approximately 6 to 18 per 1,000 singleton infants delivered vaginally in cephalic presentation. This incidence has been stable for many years [197–201]. Although such fractures can occur spontaneously, they are more common in association with delivery of macrosomic infants or during shoulder dystocias [200,201]. Most frequently, a clavicular fracture is an incidental finding after a normal spontaneous delivery. This diagnosis does not necessarily imply either incompetent midwifery or the use of excessive force.

Fracture of the clavicle can result in pseudo-Erb's palsy. This is a clinical state in which the neonate does not use the limb normally, giving rise to a suspicion of brachial plexus or other nerve injury. A physical examination and radiograph rapidly establishes the correct diagnosis. Within several days after a fracture occurs, a mass of localized callus is often palpable at the site of injury, frequently leading to the diagnosis of what otherwise might be an occult injury. On physical examination, an area of obvious induration or tenderness is present, usually at or near the midportion of the bone. In the case of fracture, movement of the fingers and the shoulder girdle is otherwise normal. This potential for normal, spontaneous movement distinguishes a fracture from a brachial plexus or other nerve injury. Although a radiograph easily confirms a clavicular fracture, the clinician must always evaluate the neurologic intactness of the brachial plexus, because both a nerve injury and a bone fracture can coexist. Treatment is immobilization of the arm and expectant observation.

### Spinal Cord Injuries

Most spinal cord injuries occur in the cervical portion of the cord [202–204]. The incidence is unknown, but in modern practice, permanent cord injuries are at best rare. In 30 years of tertiary hospital perinatal practice, this author has seen three cases, one of which did not resolve. The latter followed the vaginal delivery of a breech presentation fetus complicated by a hyperextended head.

Because of the wide range of presentation for these injuries and their rarity, establishing the correct diagnosis is often not easy and an initial erroneous diagnosis is common.

The major birth-related traumatic injuries to the spine include epidural hemorrhages, lacerations or avulsions of nerve roots, and direct traumatic injuries to the cord, including complete transection. With severe cord injury, paralysis of the lower extremities and absent or difficult respiration associated with severe neonatal asphyxia are the common clinical findings. Respiratory distress, intercostal paralysis, a retracted thorax, and a prominent abdomen during diaphragmatically driven inspiration are observed. The extremities are usually atonic and flaccid, the reflexes absent. The bladder might not empty until overflow voiding occurs. If a difficult delivery has occurred, brachial plexus defects, phrenic nerve paralysis, a long-bone fracture, or other serious injuries can also be present. Microscopic examination of the cerebrospinal fluid usually reveals frank blood. Radiographs of the spine are usually normal. The differential diagnosis includes asphyxia neonatorum, an intracranial hemorrhage, transverse myelitis, spinal cord tumors, spinal dysraphia, and amyotonia congenita [204].

Even with severe cord injury, including complete cord transection, a coexisting dislocation or fracture in the vertebral column is usually not present, because of the unique anatomy of the vertebral column and spinal cord. The cord is both delicate and relatively inelastic and is firmly tethered at both its cranial and caudal ends. In contrast, the vertebral column is a much more robust structure capable of considerable stretch owing to the weakness of the intervertebral muscles and the elasticity of the connecting ligaments. Because of these anatomic features, damage to the cord or to its supporting vascular structures from traction or torsion can easily occur, without evidence of either a vertebral dislocation or a fracture.

In the past, approximately 75% of cord lesions followed difficult breech delivery, but these have almost entirely disappeared [202]. Spinal cord injuries do rarely still occur in cephalic presentations after midpelvic forceps rotations, or most unusually, spontaneous cephalic deliveries [203]. The presumed mechanism of cord injury in instrumentally delivered cephalic presentations is forceful cranial traction with concomitant acute angulation of the



vertebral column. The true incidence of cervical lesions after midpelvic rotational forceps deliveries cannot be accurately estimated, but it is at best rare. In 1995, a Canadian series estimated the risk as approximately 1 per 1,000 instrumental rotational deliveries [203]. (See Chapter 17, Instrumental Delivery.)

Classic experiments on fresh stillborn fetuses, simulating footling breech extraction, observed that when sufficient force is applied, the fetal spinal cord ruptures consistently between segments C4 and C7. Direct linear force similar to that occurring in such an experimental study is highly unlikely in modern obstetric practice, however. When severe cord injuries occur, they usually involve a similar partial or complete lesion at a cervical or high thoracic level. During cephalic vaginal delivery, such injuries are thought to be caused by ischemia from acute narrowing or occlusion of vertebral arteries, or to excessive nerve traction perhaps transmitted by the brachial plexus. Dural tears, avulsion of the cervical nerve roots, vertebral fracture, and spinal epidural hemorrhage are also possible in the now-rare instances of *accouchement forcé*. Histologically, the principal lesion is hemorrhage into dorsal and central gray matter, often combined with mechanical disruption from stretching, laceration, and, rarely, complete transection of the cord. Posttraumatic vascular occlusion is also possible, resulting in the infarction of adjacent cord segments, extending the original injury.

At breech delivery, if a "star-gazing" or "flying" fetus is present with a hyperextended head, a peculiar type of spinal injury occurs in approximately 25% of cases when these infants are delivered vaginally. Classically, most of these deliveries were described as uncomplicated or easy, and many were spontaneous. On occasion, clinicians reported an audible snap or crack during the delivery, thought to represent an acute rupture of the spinal dura mater. In this case, the mechanism of injury is mechanical and directly related to the cranial hyperextension. During the delivery of the fetal body a sudden, additional angulation/hyperextension of the fetal head apparently occurs just prior to the head's flexing vertically into a deliverable attitude (i.e., military or flexed). The result is acute and localized cord compression. This injury, with the associated hemorrhage and edema, results in the characteristic

high-cord transection. Cesarean delivery of hyperextended breech presentation fetuses usually but not always avoids this complication. In very unusual circumstances, a similar cord injury occurs in hyperextended breech fetuses despite cesarean delivery. In these cases, difficult fetal extraction or an in-utero injury is sometimes the cause.

Prevention is the best treatment for cord injury. Avoidance of breech extraction in singleton presentations and delivery of selected term breech delivery by protocol, with appropriate ultrasound studies to detect possible cranial hyperextension, reduce risk. Clinicians should particularly avoid traction or forceps-induced rotation, especially in occiput posterior positions, if the fetal head is not well flexed. Furthermore, the position of the fetal spine should be identified. When a forceps rotation is performed, the direction should be chosen to minimize the number of degrees through which the head (occiput) is turned relative to the longitudinal plane of the fetal spine. Thus, if the head is occiput posterior and the fetal spine is positioned to the mother's left, the rotation should proceed counterclockwise. This minimizes angulation and is presumed to reduce the risk or prevent this rare complication. In occiput transverse presentations, if marked deflection is present, disproportion also can be present. Caution in attempting any instrumental procedure in this setting is urged. (See Chapter 17, Instrumental Delivery.)

Treatment for neonates with spinal injuries is largely supportive and often unsatisfactory. A degree of spontaneous recovery occurs in some cases as the traumatic edema resolves [204]. Ultrasound scan can be employed to screen neonates for spinal injury and in expert hands can visualize most abnormalities. MRI or radiographic studies of the spine are required to exclude a potentially treatable lesion, such as a neuroblastoma or spinal dysraphia. Infection, fecal retention, a neurogenic bladder, and skin breakdown are common problems in long-term survivors. In the past, many neonates with serious spinal injury died in infancy, most from complications of urinary tract infection. The long-term prognosis depends on the severity and location of the initial lesion, the extent of spontaneous recovery, the quality of supportive care, and the incidence of infection. Long-term outcome can be fairly reliably predicted by the return of motor function. Absence of

spontaneous breathing on day one and the failure of motor function to return by 12 weeks are poor prognostic signs.

### Facial Nerve Palsy

The incidence of seventh nerve palsy after a spontaneous delivery is unknown but is estimated in unselected cases as 1% or less for live-born singleton infants delivered in a cephalic presentation [205]. Common associations are forceps delivery, prolonged second-stage labor, nulliparity, and fetal macrosomia. The incidence of facial palsy related to forceps deliveries varies greatly – from 0.1% to 12%. Sampling methods, differences in the incidence of certain types of instrumental delivery – particularly rotational operations – and the transient nature of most palsies probably explains this wide range. Permanent seventh nerve palsies are rare and are often unassociated with the usually recognized risk factors. This suggests that these persisting lesions might not be caused by intrapartum events but rather by intrauterine or hereditary factors.

The neurologic deficit is usually unilateral. Observation of obvious facial asymmetry or decreased or absent movements of the eyelid, lip, and facial muscles in the distribution of the seventh nerve suggests the diagnosis. The extent of the lesion is best seen when the child cries. With crying, the eye on the affected side rolls up, but the lid remains open. Touching the cornea does not evoke local lid response; the eye rolls upward and the opposite lid closes. In complete lesions, the affected cheek and mouth edge droop or sag. When a seventh nerve palsy follows a cesarean or a spontaneous vaginal delivery, the facial paresis occurs on the same side of the fetal head as the cranial presentation, suggesting direct nerve compression, perhaps by the mother's sacral promontory [206,207].

Traumatic nerve injury must be differentiated from various congenital anomalies of the facial nerves, muscles, or central nerve nucleus, such as the Möbius syndrome and the rare congenital absence of the depressor anguloris muscle [207,208]. When developmental anomalies are the cause of facial nerve palsy, there are associated structural anomalies of the face or ear, or other cranial nerve defects (especially, nerves III, IV, V, IX, X, and XII). In these cases, various radiographic abnormalities of

the temporal bone and abnormalities in brainstem auditory evoked responses can also be noted. In most of these cases, there is a family history of the same or similar defect. Finally, some of these children also have other congenital anomalies involving the cardiovascular, skeletal, or genitourinary systems.

In differentiating a traumatic from a developmental palsy, electrophysiologic testing is helpful but not infallible. When birth trauma is the cause, facial nerve conduction studies are initially normal and become abnormal only several days later, as Wallerian degeneration progresses. In cases of injury occurring at birth, by 3 to 7 days of age, progressive reduction in the amplitude of the evoked compound motor unit potential is noted, and subsequently, by days 10 to 14, characteristic fibrillation potentials usually are observed.

The developmental anatomy of the seventh nerve predisposes to injury. The nerve in the fetus is more superficial, less protected, and slightly higher position than is normal for adults. In the fetus, the facial nerve emerges directly from the stylomastoid foramen onto the lateral surface of the skull. In its course, the nerve is protected by only the posterior body of the digastric muscle and the overlying sternomastoid muscles [209]. Unfortunately, because the fetal mastoid bone and stylomastoid process are underdeveloped and do not protect the seventh nerve well as it exits the skull, direct compression can easily damage the nerve root. Rarely, the nerve is injured by a basilar skull fracture or a temporal bone injury. Such trauma can compress the nerve within its bony canal. Hemotympanum is an important but not invariable clinical sign of this fracture.

In the absence of a skull fracture or an intracranial abnormality, rapid recovery of seventh nerve function after a compression injury is usual. Recovery is common prior to hospital discharge and virtually always occurs within the first month unless there is a central lesion. Delayed improvement is also possible but less likely. In any event, permanent disability from an intrapartum injury is rare.

Whenever a facial nerve abnormality is diagnosed, the infant should be carefully examined to exclude other congenital malformations. Depending on the setting, an otoscopic examination for hemotympanum and radiographic studies (i.e., CT, MRI) might be indicated to exclude an occult skull

fracture. Supportive measures include facial muscle massage, and less certainly, controlled electrostimulation of the nerve [210]. If congenital neurologic abnormalities are excluded, surgery for the very rare seventh nerve lesion that does not resolve should be approached conservatively because of its uncertain success.

### Other Neurologic Injuries

Isolated intrapartum injuries to the recurrent laryngeal, peroneal, radial, or lumbosacral nerves occur rarely. Their relationship to either in-utero posturing or acute intrapartum events is uncertain. Treatment is conservative, and spontaneous improvement is likely.

Rarely, injury to the laryngeal nerve results in disturbances in swallowing or breathing. Vocal cord paralysis can be unilateral, or more commonly, bilateral. Unilateral lesions are usually on the left and thought to be associated with fetal positioning, either intrauterine or intrapartum, that injures the nerve. An additional cause in premature infants is inadvertent injury to the recurrent branch during surgical repair of a patent ductus arteriosus or other thoracic surgery [217,219]. The usual clinical presentation is stridor or a hoarse cry. Uncommonly, swallowing is affected. Bilateral defects are most often associated with CNS malformation or hemorrhage. Infants with bilateral laryngeal lesions are often in respiratory distress and usually have been seriously injured by asphyxia or a major CNS abnormality such as a brainstem hemorrhage.

The mechanism for nerve injury when there is increased intracranial pressure is thought to be direct nerve compression and ischemia as the primary nerve root exits the skull. Surgical injury to the recurrent laryngeal branch during cardiac procedures or removal of mediastinal tumors are other potential etiologies for laryngeal nerve injury.

Treatment is symptomatic. In cases involving trauma, slow resolution usually occurs. The prognosis in many cases depends more on the presence of accompanying injuries or malformations than the nerve dysfunction.

### Rare Injuries

Infrequently, soft-tissue crush injuries and trauma to various fetal visceral organs, including the kid-

ney, spleen, or liver, occur during delivery. Historically, most of these were associated with difficult breech extractions or forceps deliveries, although some have been reported in spontaneous and unusually precipitate births [197,212,218]. Accidental castration has also been reported because of inadvertent severing of the testicles during episiotomy at breech delivery [211]. Literature from the early part of the twentieth century included rare reports of lacerations to the perineum of female infants. Although hard to believe, these events apparently occurred from forceful digital manipulation of the fetal anus, mistaken by the examiner for a tight or unyielding cervix [196].

### Retinal Hemorrhage

Retinal hemorrhages are the most common ophthalmologic birth injury and occur in 25% to 30% of spontaneous or forceps-assisted deliveries [213–216]. The primary associations for retinal hemorrhage include instrumental delivery and length of labor. Vacuum extraction is an important risk factor. Approximately 40% of infants delivered by the ventouse develop retinal hemorrhages. Cesarean delivery is protective; after an abdominal delivery, the incidence of these hemorrhages is approximately 2%. The reported incidence varies both with the time from birth until the examination is performed and the ophthalmologic technique used. Most hemorrhages are small, flame-shaped lesions radiating from the optic disc, parallel and superficial to the vessels. These generally resorb rapidly, usually within 24 to 48 hours.

There is a poor correlation among retinal hemorrhage incidence, maternal age, and parity. No correlation has been demonstrated with sex of the newborn, birthweight, or mean Apgar scores. Cord complications, including true knots or cord around the neck, as described in older literature, are unrelated. Furthermore, the incidence of retinal hemorrhage is not associated with the type of obstetric analgesia/anesthesia used or to the administration of oxytocin.

The mechanism for retinal hemorrhages is not established. The low incidence of retinal hemorrhage after cesarean delivery indicates that the passage of the fetal head through the birth canal and its resulting compression/elongation is the most

important predisposing factor. Suggestions for the etiology include fluctuations in pressure within the cavernous sinus, increased intracranial pressure, mild-to-moderate fetal asphyxia/hypoxia, disorders of blood coagulation or vitamin K deficiency, cephalic molding, presumed changes occurring with the onset of respiration, and nonspecific obstetric trauma [215,216].

There is general agreement that there is a higher incidence of retinal hemorrhages after vacuum extraction deliveries. The length of time that the vacuum is applied is important. The longer the extraction, the greater the incidence of hemorrhages – at least when classic rigid metal cups are applied. Vacuum extraction is thought to cause temporary impairment of blood flow in the cavernous sinus and to the bridging veins, leading to venous stasis and resultant retinal bleeding [216]. It is also possible that rapid intracranial pressure changes are the cause. The apparent protective effect of outlet forceps in reducing the incidence of retinal hemorrhages might be due to the dampening of such pressure fluctuations within the fetal head.

Retinal hemorrhages are of little clinical importance. These lesions are transient events related to the birth process and result in no permanent ill effects. Specifically, there is no correlation between hemorrhages at birth and subsequent childhood visual problems.

### Shoulder Dystocia

*Shoulder girdle dystocia* occurs when the fetal shoulder (bisacromial diameter) fails to pass easily below the maternal pubic symphysis during a vaginal delivery. The obstruction to spontaneous delivery occurs because of different dimensions between the fetal chest and the maternal pelvis, to malpresentation of the fetal shoulders at the inlet, or to a combination of both mechanisms. Shoulder dystocia is more common when fetal macrosomia is present. Dystocia is also thought to be occur more frequently when the maternal pelvis is either relatively flat or platypelloid in shape – although the evidence for this association is weak. One half or more of dystocias occur in association with normal-sized or even small infants, presumably owing to some type of malpositioning at the moment of delivery. Shoulder dystocia is considered an obstetric emergency, because its resolution

can result in injuries to both the mother and baby [220–226]. (See Chapter 14, Shoulder Dystocia.)

A common clinical observation noted when a shoulder dystocia occurs is cranial recoil. *Cranial recoil* is a characteristic rapid retrograde movement of the fetal head immediately after its spontaneous or assisted delivery, also termed the *turtle sign*. Subsequent traction to the fetal head to deliver the shoulders is either difficult or unsuccessful. Even when a serious dystocia is present, however, classic recoil might not occur. Another possible indicator is the “double-chin” sign that occurs when the head of a large infant is observed pressed firmly against the perineum after cranial delivery. This sign is associated principally with macrosomic infants. Although these signs can be helpful, the definitive diagnosis of dystocia occurs when, after delivery of the fetal head, the normal degree of downward cranial traction fails to deliver the anterior fetal shoulder.

The incidence of shoulder dystocia is approximately 5 in 1,000 deliveries. Poor progress, large infants, and the use of vacuum extraction to assist delivery are often reported as risk factors [221]. This estimate of incidence is not accurate, however. The recording of the diagnosis of shoulder dystocia remains subjective, and only cases requiring special manipulations or resulting in neonatal injury are usually reliably reported. Thus, one clinician's impression of dystocia might fall within another's range of normal. Other more objective measures for the diagnosis, such as head-to-body delivery time, have been suggested. Because of various problems with all of the suggested rigid definitions, the author prefers a simple clinical test: A shoulder dystocia is present when more than usual effort is required for delivery of the shoulders and, for appropriate delivery, ancillary methods for fetal extraction other than simple cranial traction are necessary.

Shoulder dystocia is infrequently associated with neonatal death, but morbidity remains a serious problem. Several fetal injuries are classically associated with dystocia, including soft-tissue injuries to the fetus such as bruising or ecchymoses, fractures of long bones or of the clavicle, brachial plexus injuries, and rarely, other nerve injuries. A retrospective study involving 285 cases of shoulder dystocia reported an infant injury rate of approximately 25%. Of these, brachial plexus injuries (48/285),

clavicular fractures (27/285), and humeral fractures (12/285) were included [227]. Prolonged efforts at delivery also contribute to depressed Apgar scores, varying degrees of hypoxia, asphyxia, and in the most unusual cases, death.

Brachial plexus injury from all causes is reported in approximately 1 of 1,000 live births [220]. Both the actual mechanism of brachial plexus injury and its association with shoulder dystocia is controversial. In terms of the pathophysiology, nerve compression, stretch, and hemorrhage leading to direct nerve damage and edema are the presumed mechanisms of injury. It is certainly possible to damage the nerve roots by stretching if excessive force is applied to the fetal head while the shoulders remain entrapped. Plexus injuries also occur in the absence of heavy traction, however, and some are reported after completely spontaneous deliveries or even a cesarean. (See Chapter 14, Shoulder Dystocia, for further discussion.) These observations imply that some combination of direct compression and stretching of the plexus nerve roots can occur during parturition, independent of cranial traction or perhaps additive to normal traction, either resulting in the characteristic lesion or predisposing to it. In most instances, in terms of pathophysiology there is rupture of the perineural sheath, actual separation of the nerve, and hemorrhage into the nerve trunk. Rarely, the nerves of the plexus are avulsed from the spinal cord. In severe cases, the phrenic or recurrent laryngeal nerves can also be injured.

When the brachial plexus is damaged, the primary defects are motor. The terminology used to describe these lesions is inexact. Two general types of plexus injury are recognized: the *proximal* or *Duchenne-Erb type*, and a *distal* or *Klumpke type*. In cephalic presentation deliveries, the upper nerve roots, specifically the fifth and the sixth cervical roots, are the most vulnerable. Damage to these nerves results in the classic Duchenne-Erb palsy, or the *waiter's tip deformity*, in which the affected arm is rotated inward with extension and adduction. If the lower part of the plexus, which includes the seventh and eighth cervical and first thoracic roots, is damaged, the forearm and hand become affected and the condition is termed a Klumpke palsy. An isolated Klumpke palsy is uncommon, occurring in only 2% to 3% of all brachial plexus injuries. When the lower roots are injured, a mixed pattern involving both upper and lower roots is most often encountered.

Occasionally, complex injuries occur. Damage to cervical sympathetic fibers of the first thoracic root can result in an ipsilateral Horner's syndrome, with ptosis, miosis, anhidrosis, and isolated flushing. Injury to the sympathetic innervation of the iris is also associated with long-term abnormalities in deposition of pigment. Rarely, spinal cord injuries or phrenic nerve palsies accompany brachial nerve injuries. The diagnosis of *Weigart palsy* is made when a Duchenne-Erb-type brachial plexus lesion is observed, combined with diaphragmatic paralysis. Although diaphragmatic palsy is rare, accompanying only 5% of cases of shoulder dystocia or less, it is serious [224–226]. Most cases are related to shoulder dystocia and brachial plexus injury, but others rarely occur in the absence of other demonstrable nerve injury. The diaphragm on the affected side is elevated, and some degree of respiratory embarrassment is common. In the exceedingly rare circumstance of a bilateral palsy, serious respiratory difficulty is present at birth. A *pseudo-Erb* palsy is also possible, from injuries of the shoulder joint with tearing of the capsule, fracture of the clavicle, or fracture, dislocation, or detachment of the upper humeral epiphysis. In a pseudo-Erb palsy, the neonate fails to move the involved extremity normally, giving rise to the erroneous suspicion of a nerve injury. Appropriate neurologic and radiographic investigations promptly establish the correct diagnosis, however.

The literature includes many descriptions of various manipulations to relieve shoulder dystocia. Most of these procedures either reduce the size of the fetal thorax (e.g., clavicular fracture, delivery the posterior arm) or reposition the fetal shoulders into a larger pelvic diameter (shoulder repositioning into a pelvic obliquity) or manipulate the fetal body utilizing physical principles (e.g., Woods screw and Gaskin maneuvers, Table 23.11). In unusually severe cases, complete replacement of the fetal head, with subsequent cesarean delivery (i.e., Zavanelli or cephalic replacement maneuver) or symphysiotomy has been suggested [228,229].

Recovery is the rule, with 75% or more of Erb-Duchenne, anterior brachial plexus injuries spontaneously regressing within 3 to 6 months. A posterior plexus injury or Klumpke paralysis has a much poorer prognosis, with only 40% of cases completely recovering within a year. If diaphragmatic paralysis (Weigart palsy) has occurred in association with the

TABLE 23.11 Shoulder Dystocia: Principal Delivery Techniques<sup>\*†</sup>

Maternal repositioning:
McRoberts maneuver
Gaskin maneuver (all fours)
Suprapubic pressure:
Repositioning of fetal shoulders
Rotational maneuvers:
Woods screw maneuver
Posterior arm extraction
Intrauterine replacement procedures:
Zavanelli maneuver (cephalic replacement)
Upward cranial displacement (combined operations)
Clavicular fracture
Instrumental delivery:
Shute forceps rotation
Vectis blade, Chavis maneuver
Symphysiotomy

\*These and additional techniques are discussed in detail in Chapter 14, Shoulder Dystocia.

†These maneuvers are often used in combination.

brachial plexus injury, the outcome is more guarded, and fatal outcomes are possible, especially in the rare case of bilateral nerve involvement.

In the newborn, treatment for plexus injuries is symptomatic. The aim is to maintain full range of extremity motion and prevent contractures while awaiting spontaneous recovery. Surgical exploration and attempts at repair are not indicated until it is clear that spontaneous improvement has failed to occur. MRI and other studies of the plexus are usually initiated after approximately 3 months of conservative therapy. If surgery is decided on, exploration of the axilla with microsurgical reconstruction and nerve transplantation is usually performed. Multiple operations are sometimes needed, and eventually tendon transplants also can be required for an appropriate cosmetic and functional result.

The major controversy about shoulder dystocia is its predictability [220,221,223]. Accurate identification of a high-risk population is the initial step in designing strategies for shoulder dystocia avoidance. Unfortunately, most reviewers do not think that current methods of analysis permit the accurate antepartum prediction of cases in which cesarean delivery is appropriate to possibly avoid the serious complications of plexus injury or of birth canal trauma. Despite the many articles written about

TABLE 23.12 Shoulder Dystocia Incidence: Labor Course, Type of Delivery, and Fetal Weight

Type of Delivery	Fetal Weight (g)	Incidence Shoulder Dystocia	%
Vertex/vaginal	≤4000	6/7836	0.07
PSS + MPD	≤4000	6/360	1.60
Vertex/vaginal	≥4000	8/638	1.20
PSS + MPD	≥4000	13/56	23.00

PSS, prolonged stage of labor; MPD, midpelvic delivery.

From Benedetti TJ, Gabbe SC: Shoulder dystocia: A complication of fetal macrosomia and prolonged second stage of labor with midpelvic delivery. *Obstet Gynecol*, 1978. Nov; 52(5):526–9; with permission.

dystocia, it is difficult to determine what is best practice when confronted with a possibly at-risk pregnancy. Depending on the situation, the practitioner can use the history, clinical examination, and ultrasound data to conclude that 1) a cesarean is best, or 2) conduct a trial of labor, planning the avoidance of instrumental assistance, with prompt cesarean delivery possible if failure of descent occurs, or, with normal labor progression, making preparations to treat shoulder dystocia if it occurs. (See Chapter 14, Shoulder Dystocia, for an indepth discussion of these important issues.)

The principal identifiable risk factor for shoulder dystocia is fetal macrosomia. Risk to some extent is proportionate to obstetric difficulty and delivery effort, as when a large baby, non-outlet instrumental delivery and a prolonged second stage of labor combine (Table 23.12) [223]. Whereas approximately one half of shoulder dystocia cases occur in infants of ≤4 kg, the remainder occurs in the relatively small fetal population of large and very large infants. How to properly identify these cases is still the issue, however. Unfortunately, ultrasound estimations of fetal size are unreliable. The mean absolute error of ultrasound weight estimates in the late third trimester are approximately ±6% to 18%, with approximately 30% to 60% of these estimates falling within a range ≤10% of actual body weight [21]. Because of this inherent error in the methodology, ultrasound weight estimates cannot be used as the sole basis for reaching obstetric management decisions. Recently, there has been renewed interest in developing new methods of estimating fetal size, incorporating one

or more measures of fetal soft tissue (i.e., upper arm, thigh, or abdominal wall) to improve case identification, but none has yet reached the point of clinical applicability.

An important factor in shoulder dystocia cases is the relative disproportion between cranial and shoulder diameters and the fit of the fetal head to the maternal pelvis. Although methods purporting to test this relationship have been suggested, none has been sufficiently verified for general use. These efforts are a step in the right direction, however, because they attempt to judge the relative size between the fetal head and thorax. Either these or similar techniques eventually might permit better identification of a true at-risk population – at least among larger fetuses.

Because cases at risk for shoulder dystocia can be predicted only imperfectly, and in light of the low incidence of permanent injury even when dystocia occurs (<3%), prevention of brachial injury by cesarean delivery is not a reasonable option. In the study by Rouse and coworkers [230], it was calculated that if a program of routine cesareans for either a 4- or 4.5-kg threshold for estimated fetal weight were instituted, assuming there was a technique for accurate weight estimates, more than 1,000 cesareans would be required to avoid a single, permanent brachial plexus injury. With current knowledge, a better solution is to train practitioners to identify the extreme cases properly, avoid high-risk clinical situations, and to manage dystocia when it occurs. The PPV for techniques purporting to identify shoulder dystocia cases correctly or predict brachial injury cases before actual delivery remains either disappointingly inexact or unproved although new proposals for methods of the identification of “at-risk” cases continue to be developed. Thus, there is no currently available method sufficiently verified by independent analysis that can be confidently recommended as the basis for clinical decision making. (See Chapter 14, Shoulder Dystocia, for a more extensive review of these and related issues.)

### Visceral Injuries

Injuries to intraabdominal organs, including the liver and spleen, are rare but potentially dangerous as possible causes of intraperitoneal hemorrhage [212,231]. A subcapsular hematoma of the liver is the more common lesion. Lacerations of other

viscera are rare. The spleen also can be ruptured, but the frequency of this injury is fivefold lower. Clinical associations for splenic injury are similar to those for hepatic injury, and the presentation is usually soon after birth. There is no sex predilection. Clinical associations include traumatic delivery (usually breech or breech extraction), asphyxia, prematurity, or paradoxically, macrosomia. Postmaturity, hepatomegaly from any cause, coagulation abnormalities, and vigorous resuscitation are additional risk factors. Treatment is supportive and includes administration of blood and blood products to restore coagulation factors, maintain oxygenation, and ensure a good circulating volume. When the infant is premature, hemodynamically serious bleeding can also occur into muscles, usually the buttocks, and this can occur without evidence of superficial bruising.

Subcutaneous fat neurosis is a relatively common finding after delivery. Localized soft tissue trauma during parturition is generally thought to be the cause; however, these lesions are also seen after cesarean or atraumatic births. This condition is sometimes associated with hypercalcemia, suggesting a more complex etiology. The diagnosis is made when circumscribed firm, irregular subcutaneous nodules with an overlying erythematous discoloration can be palpated. Histologically, these lesions include inflammatory and foreign body giant cells, necrotic fat, and fatty acid inclusions in viable fat cells. No treatment is required; spontaneous resolution is the rule. Occasionally a lesion becomes calcified. Subcutaneous fat necrosis must be differentiated from *sclerema neonatorum*, a rare condition involving a difference waxlike hardening of the skin. This disorder is clinically associated with neonatal sepsis, frequently found in infants who ultimately succumb [232].

### Fetal Infection

Fetal infection can occur as a result of membrane rupture and an ascending process, through transplacental transfer, or rarely, directly as a complication of invasive obstetric procedures. Although infection is frequently considered in the differential diagnosis in neonates, actual confirmation by histology (e.g., funisitis) or culture occurs in few cases. A number of different pathogens can potentially affect the fetus in utero and result in significant damage

**TABLE 23.13 Rates of Selected, Potentially Serious Perinatal Infections**

Infection	Estimated Rate/Live Births
Group B streptococcus	1–5/1,000
Cytomegalovirus	2–24/1,000 (10%–20% have overt disease)
HSV (intrauterine)	0.5–1/100,000
HSV (perinatal)	1/2,000–5,000
Toxoplasma	0.1–3.5/1,000
Syphilis	0.05–6.1/1,000*
Rubella	0.02/1,000

\*Modified prevalence varies with definition of infection.

From perinat. <http://home.mdconsult.com/da/book/body/384927164/1209/232>; with permission.

(Tables 23.13 and 23.14). As an example, infection plays an important role in the pathophysiology of peripartum permanent neurologic injury [244–246]. The discussion of these subjects is beyond the scope of the present chapter, and readers are referred to standard sources for additional information.

### Intracranial Hemorrhage

Intracranial hemorrhages have been reported after forceps and vacuum extraction operations, as well as following spontaneous deliveries [157,163,231,238]. Most intracranial hemorrhages occur in premature infants or in association with severe asphyxia; however, classic subdural hemorrhages also have been described after both complicated and uncomplicated vacuum extractions and forceps delivery operations in otherwise apparently normal neonates [240]. The clinical manifestations of intracranial bleeding and the prognosis for recovery depend on the amount and location of bleeding, and the presence or absence of other fetal injuries [234–236].

Intracranial bleeding can be subarachnoid, subdural, intraventricular or intracerebellar, and occasionally intraparenchymal [249]. Intracranial injury has a general association with prematurity, hypoxia/asphyxia, and preexisting coagulation disorders (e.g., thrombocytopenia), as well as mechanical trauma [162,163,231]. Hypoxia/asphyxia, rapid alterations in perfusion pressure or osmolality, and other factors are especially important in influencing the probability of intraparenchymal or intraven-

tricular hemorrhage in the peculiarly fragile intracerebral circulation of premature infants. Intraventricular hemorrhages are nearly always restricted to premature infants. Intracranial bleeding in term to near-term babies is more likely to be traumatic, although tissue hypoxemia and hypercapnia are still common features when intraparenchymal lesions occur. Intracerebellar hemorrhages are uncommon, often serious, and when they occur are more likely to be found in preterm infants.

There is a well-documented association between subdural hemorrhages and difficult delivery [162,163,239]. Thus, breech presentation, macrosomia (weight of 4,000 g or more), instrumental delivery, and protracted labor are the principal clinical associations. Such intracranial lesions are similar to those described by Holland in 1922 in association with traumatic forceps operations or difficult breech extraction deliveries [240]. Such bleeds are believed to occur mostly because of the distortion of the fetal cranium during the birth process. Deformation of the fetal skull with rapid second-stage labor or from the cranial traction in assisted delivery strains or ruptures the fixed cranial sidewall attachments of the unyielding falx and tentorium. The resultant subdural hemorrhage either dissects anteriorly to cover the hemispheres or extends downward into the posterior fossa, compressing brain tissue. Rarely, rupture at the intersection of the falx and tentorium disrupts the connection between the straight sinus and the great vein of Galen, resulting in a serious and often rapidly fatal bleed [231,233].

*Periventricular hemorrhage* is primarily a lesion of premature infants, believed to be due to immaturity and fragility of vessels in the germinal matrix or subependymal plate [162,163,231,249]. Acidosis, coagulation abnormalities, and fluctuations in perfusion pressure, among other factors, predispose to bleeding from germinal matrix vessels. Hemorrhage can extend into the ventricles of the brain, or less commonly, into the brain parenchyma. Serious complications of such bleeding are common, with hydrocephalus caused by fluid obstruction and porencephalic cysts secondary to tissue necrosis being the most serious potential events.

*Subarachnoid bleeding* is usually, but not invariably, a lesion of full-term infants [163,231]. The hemorrhage occurs because of injury to small subarachnoid vessels, or as an extension of intraventricular hemorrhage. The causes are generally



TABLE 23.14 Possible Clinical Features and In-utero Infection

Body Area	
General	Intrauterine growth retardation: all etiologies
	Hydrops fetalis: parvovirus B19, CMV, syphilis, Toxoplasma, HSV, Coxsackie B3 virus
	Placentomegaly: CMV, syphilis
Central Nervous System	Hydrocephaly: CMV, Toxoplasma, enterovirus, varicella
	Microcephaly: CMV, Toxoplasma, rubella, varicella, HSV
	Intracranial calcifications: CMV, Toxoplasma, HSV, rubella, HIV, parvovirus, West Nile virus, lymphocytic choriomeningitis virus
Cardiac	Congestive heart failure: parvovirus B19, syphilis, CMV, Toxoplasma
	Pericardial effusion: parvovirus B19, syphilis, CMV, Toxoplasma
	Cardiac defects: rubella, parvovirus B19, mumps (?)
	Myocarditis: enterovirus
	Calcification of the pericardium and lungs: varicella
Pulmonary	Pleural effusion: parvovirus B19, syphilis, CMV, Toxoplasma
	Pulmonary hypoplasia: CMV
Intra-abdominal	Hepatosplenomegaly: CMV, rubella, Toxoplasma, HSV, syphilis, enterovirus, Parvovirus B19
	Hyperechogenic bowel: CMV, Toxoplasma
	Hepatic calcifications: CMV, Toxoplasma
	Meconium peritonitis: CMV, Toxoplasma
	Ascites: Parvovirus B19, CMV, Toxoplasma, syphilis
	Limb reduction, restriction: VZV

CMV, cytomegalovirus; HSV, herpes simplex virus; VZV, varicella zoster virus.

Modified from perinat. <http://home.mdconsult.com/da/book/body/384927164/1209/232>; with permission.

recognized as trauma and mild-to-moderate hypoxia. Most of these bleeds are of trivial to minimal consequence, and affected infants can be asymptomatic.

The usual clinical indicators of intracranial bleeding are nonspecific changes in behavior. Cere-

bral irritation can result in convulsions, spasticity or rigidity, photophobia, a high-pitched cry, or depressed reflexes [163]. Occasionally, signs of CNS injury are delayed for 24 hours or even later. Neonates with late presentation sometimes show additional nonspecific signs such as weak suckling,

respiratory distress, apnea spells, a bulging fontanel, or lethargy [239].

The combination of events that predisposes to intracranial bleeding in outwardly normal deliveries is unknown. Delivery technique, the effects of labor, prematurity, and preexisting fetal condition(s) such as coagulopathies influence the likelihood of injury. Observations from clinical experience and review of reported cases of traumatic hemorrhage suggest several important associations. When instrumental deliveries are performed, the more lengthy and difficult the operation, the higher the fetal station at its commencement, and perhaps, the greater the speed of descent of the fetal head appear to increase the risk for hemorrhage [238]. Both the degree and the rapidity of cranial distortion also contribute to the extent of subdural hemorrhage. As noted, such rapid changes in cranial shape and tension can rupture the falx or tentorium at their respective attachments to the inner wall of the calvarium, with resultant hemorrhage. This association has been recognized for many years [196,240]. Subdural, intraparenchymal, and cerebellar bleeding are paradoxically more common after either spontaneous precipitate labors or complex difficult instrumental deliveries. The common feature in such cases of intracranial hemorrhages in term infants with normal coagulation mechanisms is rapid fluctuation in intracranial pressure that accompanies sudden or extreme cranial deformation. As previously noted, anatomic and imaging data suggest that small, asymptomatic, subarachnoid, and subdural bleeds occur as a consequence of both spontaneous and instrumental delivery more frequently than is suspected on clinical grounds alone [336].

Although recognized, intracranial hemorrhage is an uncommon and even rare complication at term; clinicians should recall that hemorrhage within the skull is possible even in apparently easy and uncomplicated deliveries. Infants delivered after spontaneous precipitate labor, as well as those subjected to difficult vacuum extraction or forceps operations who then manifest characteristic signs and symptoms should be speedily evaluated for a possible hemorrhage. The possibility of a subgaleal hemorrhage with or without an associated cranial fracture should also not be forgotten, especially in the event of an instrumental delivery. In cases with characteristic neurologic signs and symptoms or notable for otherwise unexplained anemia or cardiovascu-

lar collapse, appropriate ultrasonic, MRI, or radiographic studies of the CNS are indicated.

If hemorrhage has been diagnosed, the treatment is principally supportive. Blood transfusion is provided as required. Neurosurgical consultation is obtained, and coagulopathy is excluded by appropriate testing. Neurosurgical procedures can be performed to evacuate hematomas or relieve intracranial pressure, but such surgery is uncommonly performed [163].

### Permanent Neurologic Injury

There is no more complex and contentious clinical problem in perinatal medicine than the relationship between intrapartum events and permanent neurologic injury [3–8,25,242–243,248,251]. The etiology of most long-term neurologic abnormalities is multifactorial and imperfectly understood. Complications defined as abnormal during labor and delivery are common and are observed in up to 60% of all parturitions. Thus, identification of pregnancies truly at risk for permanent injury remains elusive.

Because of the complexity and medicolegal importance of the relationship of fetal hypoxia to permanent neurologic injury, the American College of Obstetricians and Gynecologists (ACOG) and the American Academy of Pediatrics (AAP) have established clinical criteria for the case evaluation to be applied in judging the strength of the association between CP and evidence of intrapartum hypoxia in a specific instance [248]. This controversial document is helpful because it summarizes existing data, clarifies definitions, and sharpens the techniques for assigning a cause for neurologic injuries.

Part of the problem is terminology. Neurologic injuries have historically received various terms, including *birth* or *perinatal asphyxia*, *postasphyxial encephalopathy*, and *asphyxial birth injury*, among others. Owing to the complexity of these diagnoses, the inconsistency in the use of the various terms, and recent data concerning pathogenesis, new terminology has been suggested. Neither *perinatal* nor *birth asphyxia* are preferred terms. It is usually difficult if not impossible to identify the time of a neurologic injury accurately, and similarly problematic to verify fetal normalcy before the presumed insult. The recent ACOG/AAP monograph suggests the term *neonatal encephalopathy* (NE) [248].

NE is a disorder of various causes, marked by abnormalities in an infant's tone, reflexes, consciousness, feeding, or respiration. Seizures might occur, and the condition might or might not result in permanent impairment. The term *hypoxic ischemic encephalopathy* (HIE) is a subset of NE involving both encephalopathy and intrapartum hypoxia. HIE was introduced to replace other earlier and inexact diagnoses and to describe a clinical state of neonatal encephalopathy thought to be due to asphyxia, while specifically avoiding any specification of the timing of the injury. As described in greater detail later, HIE is thought to be primarily a disorder of cerebral hypoperfusion, notable for the loss of the normal autoregulatory mechanisms for intracranial blood flow.

Accuracy in diagnosis is important. Several conditions can at least in part mimic the signs and symptoms of HIE. These conditions include the effects of maternally administered CNS depressants, including commonly prescribed drugs such as narcotics or magnesium sulfate. Various CNS malformations, septicemia, drug withdrawal syndromes, congenital myopathies, and inborn errors of metabolism (e.g., organic acidurias, pyruvate dehydrogenase deficiency, abnormalities in the urea cycle), and other rare congenital conditions also can have a similar presentation.

NE is not the same disorder as CP. In fact, infants developing CP often lack evidence of neonatal encephalopathy. CP is a chronic disorder of the CNS developing during the prenatal, perinatal, or postnatal periods and involving abnormalities in posture and movement, and often other dysfunctions. CP is considered a static form of encephalopathy [251]. It usually originates early in life, but it is not the result of a progressive neurologic disorder. Epilepsy, mental retardation, or attention deficit/hyperactivity disorder cannot be ascribed to birth-related asphyxial injury unless CP is also present.

Common associations for CP include histologic evidence of perinatal infection (funisitis or chorioamnionitis), preterm delivery, intrauterine growth restriction (IUGR), antepartum hemorrhage, twin gestation, and various genetic disorders [4,242–248]. The associated risk factors for neonatal encephalopathy are advanced maternal age, maternal thyroid disease or hypertension, IUGR, antepartum vaginal bleeding, and infertility.

A related issue is mental subnormality. The prevalence of mental retardation (IQ  $\leq 50$ ) is approximately 3 to 4 in 1,000 school-aged children. Retardation is frequently associated with seizure disorders and other motor handicaps. The incidence of mild mental retardation (IQ 50–69) is highly influenced by socioeconomic status and is approximately tenfold more common than the more severe forms of retardation, with an incidence of 23 to 30 in 1,000 school-aged children.

Etiologies suggested for the severe forms of mental retardation include chromosomal anomalies (40%), inborn errors of metabolism (3%–5%), intrauterine infection (5%), and various complications of postnatal life (10%). The extent to which other toxic brain exposures to alcohol, prescription or illicit drugs, chemical compounds, or other environmental toxic agents contribute to the ranks of the mentally retarded is unknown. Current opinion holds that perhaps 10% to 15% of both mild and severe mental retardation can be ascribed to perinatal events [248].

Incidence figures for birth asphyxia depend on definition and case inclusion. When cases diagnosed as HIE and NE are combined, the estimate for incidence is 1.9 to 3.8 cases in 1,000 births. When HIE is considered alone but defined as intrapartum hypoxia plus NE, and preconceptual and antepartum etiologic factors are excluded, the estimate becomes tenfold smaller, approximating 0.16 in 1,000 births [4,6]. For isolated CP, the incidence is approximately 1.5 to 2.0 in 1,000 births, a rate unchanged for more than 4 decades [241]. The interrelations between these disorders are complex. For example, CP can occur without clinical evidence for either hypoxia or encephalopathy. Most cases involving NE or hypoxia never turn into CP. The classic clinical indicators of fetal distress (e.g., meconium passage, bradycardias, pH abnormalities), or obstetric complications (e.g., breech presentation, abruptio placentae, cord prolapse), are also poorly predictive of the development of CP unless the later Apgar scores are also significantly depressed (i.e., 3 or less at 10 minutes or more) and neonatal depression is present (Table 23.15) [248]. It is also fair to say that the interpretation of such clinical data remains controversial, however. In the attempt to provide guidelines for care analysis, the joint ACOG/AAP document specifies clinical and laboratory features required to link perinatal events and the

**TABLE 23.15 Death and Cerebral Palsy (CP) Rates in Babies >2,500 g with Apgar Scores  $\leq 3$**

Time of Apgar $\leq 3$ (min of life)	No.	Death (%)	CP in Survivors (%)
1	202	5.6	1.5
5	397	15.5	4.7
10	122	34.4	16.7
15	59	52.5	36.0
20	20	59.0	57.1

*Modified from Nelson KB, Ellenberg JH: Antecedents of cerebral palsy: Multivariate analysis of risk. N Engl J Med, 1986. Jul 10; 315(2):81-6; with permission.*

subsequent development of persistent neurologic injuries. Although the specifics outlined in this document are controversial, the report does serve as an important effort to understand the relationship between specific intrapartum events and long-term outcomes. It is safe to predict that much more will be heard on this subject in future.

The central cause of much of the brain injury related to HIE is disordered cerebral circulation [249-251]. Hypoxia/asphyxia and cardiac disease, or induced cardiac dysfunction, lead to adverse effects on cerebral blood flow. In the fetal as in the adult CNS, blood flow is autoregulated. Both hypercapnia and hypoxia can disrupt the CNS autoregulatory system, however, resulting in a situation in which perfusion depends solely on the intravascular perfusion pressure. When this "pressure-passive" state is reached, the cerebral circulation becomes vulnerable. Hypoperfusion and cerebral ischemia in specific parts of the brain become possible. Alternatively, increased perfusion with a concomitant risk for hemorrhage can also occur [249]. When the combination of perfusion abnormalities and localized hypoxia overcome the compensatory mechanisms of the intracranial neurons, progressive damage ensues. When hypoxia/ischemia is severe and prolonged, excitatory neurotransmitters such as glutamate are also released, and oxygen free radicals are produced. These, among other local events, collectively result in increased cellular damage beyond that initiated by the original circulatory and hypoxic insult [250].

Classically, ischemic necrosis develops in the watersheds or border zones between the end branches of major brain vessels, resulting in char-

acteristic patterns of injury. The parasagittal region is the major site of damage in mature to near-mature fetuses. Such injury commonly results in spastic quadriplegia. In preterm infants, injuries at or about the germinal matrix, especially injury to periventricular white matter, is most common, resulting in spastic diplegia [251]. Near-total asphyxia can also damage deeper brainstem structures such as the thalamus or caudate nucleus.

Various methods of neuroprotective treatment have been attempted in neonates suspected to be at high risk for HIE. Treatment with indomethacin, phenobarbital, allopurinol, morphine, and other drugs has been studied, generally with uncertain results [252]. Hypothermia as a preventive therapy is now under investigation based on the theory that lowering temperature reduces the infant's metabolic rate, which could help to stabilize membrane permeability or reduce the local release of excitatory transmitters, thus ameliorating brain cell injury. To date none of these therapies has been sufficiently validated for clinical use; however, this line of investigation is thought to hold promise.

### Electronic Fetal Monitoring and Cerebral Palsy

The appropriate role of EFM during labor and its effectiveness in the avoidance of permanent neurologic injury remain controversial [5,8,24-27,253,255]. This technique has distinct limitations and certain well-defined risks. Despite technical improvements in recent years, monitoring as usually conducted largely restricts the parturient to bed, limiting ambulation and possibly exerting an adverse effect on the course of labor. Maternal or fetal movements also require periodic readjustment of the Doppler monitoring head, consuming the attention of the birth attendants. In addition, the tocodynamometer is notorious for spontaneous displacement and various recordings artifacts. While direct-lead electrodes resolve many technical problems and reduce artifact, they require membrane rupture and intravaginal manipulations to apply.

When EFM is used instead of intermittent auscultation, the overall cesarean rate for suspected fetal stress/distress is increased [25]. EFM is also associated with a greater incidence of instrumental delivery (i.e., forceps and vacuum extraction). Finally, although EFM could reduce perinatal mortality from intrapartum hypoxia, perinatal

morbidity, at least as measured in the various studies performed to date, is unchanged.

To the distress of its original proponents, intensive use of EFM has arguably had essentially no effect on the incidence of CP [7,25]. Over recent decades, many obstetric interventions have occurred based solely on EFM patterns, contributing to the high rate of obstetric intervention. Unfortunately, the classic EFM findings initially thought to have an association with the development of CP, such as repetitive late decelerations and a decrease in beat-to-beat variability, are subject to varying interpretation [25,255–257]. Thus, these patterns have a very high false-positive rate and a poor predictive value.

EFM is best at predicting a normally oxygenated fetus. Thus, a normal FHR with a pattern of accelerations and fetal movement in an alternating pattern, reflecting changing behavioral state, normal variability, and an absence of decelerations, is highly reassuring. These data indicate that the fetus is neither acidotic nor hypoxic. EFM is most helpful to the clinician when such normal and reassuring patterns are observed despite obstetric difficulties such as a prolonged labor or a difficult induction. Alternatively, if the labor begins with a frankly abnormal tracing, this might suggest prior fetal injury not likely to be remedied even by an accelerated or cesarean delivery. Finally, in cases actually involving intrapartum asphyxia, an initially normal EFM tracing might be observed to change. Sentinel events can also be identified when acute heart rate alterations are observed, such as with cord prolapse. Other patterns possibly indicating acute asphyxia include repetitive late or severe variable decelerations and prolonged decelerations, especially when combined with or decreased FHR variability [258]. (See Chapter 22, Fetal Assessment.)

The rapidity of intervention necessary to reduce or avoid fetal injury in the face of evidence that suggests acute hypoxia is unknown. In nonhuman primate experimental models of total asphyxia, brain damage occurs after 10 minutes. When total asphyxia persists for more than 25 minutes, most fetuses in these studies died [259]. This classic rhesus monkey data cannot be fully extrapolated to humans, however. There are additional clinical data to consider. Review of perimortem cesarean deliveries and instances of uterine rupture, when reasonably reliable data exist about the timing of the critical injury to delivery, indicates that deliveries occur-

ring at less than 5 minutes and occasionally up to 15 minutes after injury can result in normal or near-normal survivors [260–263]. Deliveries delayed past 15 minutes have much poorer outcomes. Many of these infants die, and significant numbers of those surviving are seriously injured. Uncommonly, individual cases involving much longer intervals from maternal injury to fetal extraction have been reported [263]. The important variables in these unusual cases include both the nature of the maternal injury (e.g., exsanguination vs. head trauma), the preservation of the mother's vascular volume, the time from injury to the arrest of the maternal heart, and the speed and effectiveness of resuscitation efforts. (See Chapter 18, Cesarean Delivery and Surgical Sterilization.)

Whenever methods of intrauterine evaluation are considered, human fetal outcome depends on many variables. Human data concerning the interval from the time of injury until delivery are often inexact. Furthermore, the degree of asphyxia in individual cases is not necessarily measured by the assigned intervals from insult to delivery. In some instances, especially when the fetus was initially compromised, serious or fatal fetal injury is possible with shorter intervals.

For these reasons, no simple relationship exists between the degree of presumed fetal hypoxia and the observed neonatal injury. This inexactitude does not allow the establishment of clear guidelines about the rapidity of response necessary to avoid fetal injury when acute events such as uterine rupture, cord prolapse, or fatal maternal injuries occur. When hypoxia/asphyxia is both severe and prolonged, the most common outcomes are either death or apparently normal survival. Nonetheless, as the general degree of asphyxia rises, so does the number of surviving but damaged infants (Table 23.16).

Clinicians must refocus on understanding the antecedents of perinatal injury and the importance of antepartum events, while taking a more conservative view toward intrapartum interventions for fetal indications, unless clinical indications are marked. Techniques to detect in-utero abnormalities that might injure infants prior to the onset of labor, rendering them unable to withstand the additional stress accompanying parturition, are sorely needed. When a “bad baby” results from a delivery, the mere presence of complications at delivery is insufficient evidence that obstetric interventions such as

TABLE 23.16 Likelihood of Birth Asphyxia or Trauma Causing Brain Damage in Children with Spastic Cerebral Palsy\*

Infant's Birth Status	Relationship of Birth Events to Observed CP				Number
	Number	Possible	Probable	Definite	
No fetal distress, birth asphyxia, or abnormal neurologic signs	124	0	0	0	124
Abnormal signs of neurologic dysfunction; no birth asphyxia	22	3	5	0	30
Birth asphyxia and abnormal neurologic signs	10	3	7	9	29
	<i>n</i> = 156 (85.2%)	6 (6.6%)	12 (3.3%)	9 (4.9%)	183 (100%)

\*Cases drawn from Western Australia and England, 1975–1983.

Modified from Blair E, Stanley FJ. Intrapartum asphyxia: A rare cause of cerebral palsy. *J Pediatr*, 1988. Apr; 112(4):515–9; with permission.

a cesarean or an expedited instrumental delivery could have avoided or ameliorated the observed injury. Each case requires individual analysis, with close attention to pre-, peri-, and postpartum data, including a review of placental pathology and any specialized testing suggested by the clinical history or other data [264].

The central issues in both intrapartum management and operative delivery are related to maternal and fetal safety. These issues, some of which have been discussed in this review, have long been hotly debated without definitive resolution. All studies relating events of labor/delivery to long-term outcome are flawed in organization, enrollment bias, or failure to control confounding variables or reflect current practice. Thus, conclusions derived from these data are to be interpreted with caution. Virtually all discussants agree on some aspects of this controversy, however. Practitioners generally have become less certain of the value of many obstetric interventions. The demonstrable benefits of continuous EFM as opposed to careful one-on-one intermittent auscultation are limited. As yet, there is no clear consensus about the appropriate methods of evaluating prenatal risk or how such considerations should govern clinical management. Techniques of antepartum management continue to be controversial, despite the concept now gaining strength that prolonging the effects at fetal propulsion is more desirable and less dangerous than immediate fetal extraction in achieving delivery when dystocia ensues. Nonetheless, an important but limited role remains for classic obstetric interventions in obstetric practice, including instrumental and cesarean delivery.

### Environmental Risks and Exposures

A potentially important but controversial contribution to birth injuries includes environmental factors [265–279]. Although there are suspicions, few definitive data link general human exposure to synthetic chemicals and birth injury. Exposure to certain substances is known to be clearly deleterious, however. Although the potential adverse effect of these exposures is either unclear or, in some instances, thought to be insignificant, an in-depth analysis of this subject is well beyond the scope of this chapter. Nonetheless, several general observations on the voluminous literature about the relationship among various environmental events or exposures, in-utero stressors, and adult human disease are appropriate. Interested readers are referred to standard sources and recent reviews for additional information [266,275,277].

Among the many potential sources of environmental risk are maternal drug use, disordered maternal nutrition, and exposure to prescription drugs, various chemicals, and irradiation. There is a complex interaction between the genetic and development components of a pregnancy, the mother's social environment, and the timing and extent of any potentially dangerous antenatal exposures. Principles used when evaluating the strength of the relationship between an environmental exposures and an observed fetal abnormality or injury include

- The dose-response effect
- The relationship to a specific period of embryonic development

- The restricted set of abnormalities that characterize the suspected exposure (syndrome)

Thus, the possibility that any chemical exposure, drug, or event of maternal deprivation will result in a biologic effect depends on a number of factors [265]. These include the dose or strength of the exposure, the properties of the agent or event, the period of gestation when the exposure occurs, and the presence of compounding factors that might either increase or diminish the risk for an adverse effect. Furthermore, adverse effects might not be identified until many years after the initial exposure, vastly increasing the difficulty in establishing associations.

Among the general population in all developed nations, there is widespread exposure to many environmental chemicals that are thought to be endocrinologically active. These agents suspected to be endocrine disrupters, substances with the potential to interfere with hormonal receptor sites, mimic hormones, or trigger inappropriate hormonal responses. The clinical importance of these substances remains to be established, but their potential for mischief could be great.

Caution is necessary when interpreting suspected environmental associations and adverse effects. Some teratogenic syndromes do mimic established genetic syndromes. Furthermore, many subjects have compounding exposures or conditions accompanying the specific exposure in question, such as various medical disorders, chronic infections, or illicit drug use. This makes the assignment of an observed exposure to a specific adverse effect identified in a neonate or, at an interval of years in an adult, at best difficult.

Perhaps the most interesting data on early fetal injury is derived from various epidemiologic studies, which have associated various adult diseases with in-utero events [270,271,273,274,278]. The associations derived from these investigations are thought to reflect a type of in-utero programming. These induced, permanent changes in fetal physiology are thought to subsequently affect adults in terms of their structure, physiology, or metabolism. Several common chronic adult diseases are now suspected to be linked to various physiologic adaptations made by the fetus to permit its continued growth in response to in-utero undernutrition. As is discussed in the, coro-

TABLE 23.17 Estimates of Major Reproductive Risks per 1,000 Pregnancies

Spontaneous abortions: <sup>*</sup>	350
Clinically recognized abortions	150
Genetic disorders:	110
Polygenic/multifactorial	90
Dominant inheritance disorders	10
Aneuploidy	5
Autosomal/sex linked disorders	1.2
New mutations	3
Preterm deliveries	40
Fetal growth disorders <sup>†</sup>	30
Stillborns	2.20

<sup>\*</sup> Recognized clinically and by pregnancy testing.

<sup>†</sup> As variously defined, including intrauterine growth retardation (IUGR).

*Modified from Brent RL. Environmental causes of human congenital malformations the pediatrician's role in dealing with these complex clinical problems caused by a multiplicity of environmental and genetic factors. Pediatrics, 2004. Apr; 113(4):957-68.*

nary artery disease, obesity, abnormalities in ovulation, and diabetes are among the most common associations [268,270,272,277,278]. Obviously, both the nature of the fetal events and their timing during development are critical.

Spontaneous errors in the reproductive process are the principal cause of human fetal wastage. These various disorders account for many more fetal losses than can be ascribed to environmental exposures of all types. Consequential congenital malformations are observed in approximately 3% of human births [265]. Every year in the United States, 120,000 infants are born with serious or significant abnormalities of genetic origin. Fetal wastage from spontaneous abortions, lethal malformations, extreme prematurely infants, and stillbirth are also major contributors to reproductive loss (Table 23.17). (See Chapter 2, Prenatal Genetic Assessment.)

The emotional and financial costs of inherited disease are great. Both clinicians and families struggle to meet the challenges of genetic disease and reproductive loss. Congenital disorders also have medicolegal importance. Preexisting fetal or developmental abnormalities are thought to be major contributors to permanent neurologic disorders, including CP. With the current emphasis on prenatal diagnosis, incorrect predictions of normality or

the failure to evaluate families properly for occult genetically based disorders can result in legal entanglements.

### Developmental Disorders and Adult Disease

There are increasingly strong data linking development in-utero events with both life expectancy and subsequent adult disease including ovulatory dysfunction, atherosclerosis, hypertension, stroke, the metabolic syndrome, and diabetes [268–279].

Specifically disordered fetal growth as reflected in birthweight or body proportions seems to be linked to an increased incidence of metabolic disorders, including impaired glucose tolerance, abnormalities in lipid profile, and Type 2 diabetes mellitus. There is also a strong link to vascular endothelial dysfunction as manifested by coronary artery disease. It appears that the incidence of these adult diseases is also increased among people who are small at birth but become obese as adolescents or adults.

It is hypothesized that developmental plasticity and genetic propensities permit the fetus to develop a predictive adaptive response (PAR) to a difficult in-utero environment; however, these PARs developed in utero, although adaptive very early in life, establish a predisposition to various diseases developing decades later. It is also possible that adverse effects early in development or in early childhood can affect the long-term disease risk for a population, even if the birthweight falls within the normal range. Other features such as maternal smoking, season of birth, and the occurrence of childhood infections also play a role in changing the likelihood of adult disease.

Abnormal fetal growth is thought to impair subsequent organ development and vascular and metabolic functions [249,275,278]. Other evidence indicates that childhood inflammatory disease (infections) also strongly influences adult morbidity and mortality. The implications of these observations are large. If in-utero events weigh so heavily in changing basic metabolic “set-points” that literally persist for decades, and if birthweight reflects the adequacy of nutrition in utero, then the focus of perinatologists should become the uterine environment. This implies a new focus and investigation of potential adverse influences on fetal growth and development from many sources, including basic nutrition.

There are additional implications for the reduction in adult disease. If in-utero programming pre-sets metabolic and vascular endothelial function, attempted modifications of behavior or drug therapy instituted later in life could well be influenced. This fact could partially explain the differences of outcomes seen when poor dietary and other habits are practiced, but the susceptibility to disease varies considerably across a population. It has also been suggested that restriction in prenatal growth influences adult behaviors by effects on personality, emotional response, lifestyle choices, socialization, or even sexuality. Prenatal growth influences could then be reflected in life-long eating patterns, excessive behavior, and even the incidence of marriage.

There are several postulated mechanisms whereby environmental events might alter development. One possibility is the induction of epigenetic change in DNA, thus changing gene expression. Alternatively, the differentiation of tissues could be disrupted or changes induced in homeostatic control mechanisms. In terms of metabolic constraints, if the available nutrient is limited or poor, a change in growth and in glucose utilization could be adaptive, leading to the expression of genes favoring insulin resistance. If this adaptation persists into later adult life, however, the risk is the development of glucose intolerance, the metabolic syndrome, or frank diabetes [272]. Because insulin is a principal fetal growth factor, the expression of such genes in an individual's early development could affect fetal weight gain significantly.

A body of data from animal experimentation supports a link between early perinatal events and adult disease [275]. Most of these studies have involved dietary manipulations (e.g., restricted calories or low-protein, high-fat diets) or the maternal administration of glucocorticoids. It is suggested that pronounced undernutrition suppresses enzymes involved in cortisol degradation (11 $\beta$ -hydroxysteroid dehydrogenase, Type 2), thus exposing the developing fetus to increased concentrations of potent maternal steroids.

In many instances in human pregnancy, poor fetal growth accompanies multiple gestations, chronic hypertension, recurrent abruptio placentae, and other disorders for which treatment is at best limited, and in many instances probably ineffectual. In general clinical terms, fetal growth is assessed by several parameters: maternal weight gain, the



observed increase in fundal height, and selected ultrasonic computations thought to document that the observed fetal growth pattern is within normal limits. While it is not certain, it can be safely assumed that these crude measures are insufficient to identify significantly at-risk pregnancies properly in terms of lifelong influences. Nevertheless, even if such pregnancies could be identified properly, effective treatment is at best uncertain. What physicians now do when fetal growth is tardy or suspect is to attempt to "fix the fixable." A reasonable amount of lateral recumbancy rest is usually suggested and the mother's food and liquid intake and habits such as smoking are reviewed. Occult hypertension or problems with carbohydrate metabolism are investigated. Occasionally under treatment improved fetal growth is observed. When fetal growth is markedly abnormal, Doppler flow studies of umbilical, middle cerebral artery, and other vessels such as the ductus venosus are performed to provide an opportunity to intervene before fetal acidosis or death occurs in utero. How effective this approach is in the avoidance of long-term risk or permanent fetal injury remains uncertain.

Study of these long-term effects of "fetal programming" for events that become manifest only in adult life will doubtless continue. Eventually, the anticipation is that both better identification of cases involving growth disturbances and an understanding of appropriate treatment will become a reality.

### Trauma

Blunt abdominal trauma, and specifically motor vehicle accidents, are major maternal and fetal risk factors for morbidity and mortality [280,282-289]. In modern industrialized societies, two thirds of all trauma that occurs during pregnancy is due to automobile accidents [287]. Between the prime child-bearing ages of 20 to 29 years, approximately 3% of women involved in reported motor vehicle accidents are pregnant [290]. Other common causes of blunt trauma during gestation include domestic violence and accidental falls [282,283]. Approximately 10% of pregnant women admitted for the treatment of trauma have blood tests that reveal elevated alcohol levels or an abnormal toxicology screen. The principal fetal risks associated with maternal trauma are due to premature labor, abruption placenta, and death [307,324,325]. The most common direct fetal injury after maternal blunt trauma

is a cranial fracture. The risk for fetal loss after maternal blunt trauma varies from 3.4% to 38%, depending on the severity of injury and the rapidity of intervention. Because injury leading to fetal loss can be occult, all pregnant women who experience blunt trauma should be promptly evaluated. The related principal causes of morbidity and mortality related to blunt trauma are abruption placenta, hemorrhagic shock, and events resulting in maternal death [287,289,323,326].

If the mother has sustained a cardiac arrest, active resuscitation should be attempted. If the fetus is determined to be potentially viable, a perimortem cesarean must be considered. Although data are limited, in general, the earlier the fetus is removed and resuscitated after the maternal arrest, the greater the chances are for its survival. Cesarean deliveries after 15 minutes of maternal cardiopulmonary arrest have little likelihood of success, although these are several reported exceptions. As a general rule, cesarean delivery should be performed within 4 to 5 minutes of an arrest, but always before 10 minutes if at all possible. (See Chapter 18, Cesarean Delivery and Surgical Sterilization, for a complete discussion of this topic.)

In the common situation of an out-of-hospital injury, the best management is not certain; however, the available data suggest that continued efforts at maternal resuscitation with vigorous replacements of intravenous fluid and oxygenation can improve the possibility for fetal survival even if longer intervals from injury to intervention occur. In some cases, maternal resuscitation becomes possible once the fetus is removed, presumably because delivery improves maternal cardiovascular function.

Fetal deaths associated with maternal trauma are principally from motor vehicle accidents. In a 3-year review of data from 16 states that collectively accounted for approximately 15,000 fetal death registrations year, accident-related fetal deaths actually exceeded the number of infant deaths from motor vehicle accidents in three of the states studied [289]. Compared with nongravid subjects, pregnant women in motor vehicle accidents have a lower incidence of thoracic or head injuries but a higher incidence of abdominal injuries. The most dangerous injuries are pelvic fractures, however.

Pregnant women should use seat belts. Properly applied seat belts protect both mother and child in all but the most severe of accidents [289]. Airbags

are also appropriate and should not be disarmed simply because the passenger is pregnant.

When maternal blunt trauma occurs, there are several appropriate evaluations. Initially, the mother requires prompt evaluation and stabilization before fetal assessments are performed. As a general rule, improving the maternal condition, ensuring normal oxygenation, and restoring cardiovascular stability are the best initial supportive actions for the fetus as well. Oxygen, infusion of balanced salt solutions, and blood or blood products should be provided to the mother as required. Real-time ultrasound scans, laboratory tests (i.e., coagulation factors, blood count, etc.), and continuous EFM are the principal diagnostic tools.

Ultrasound examinations are important despite the fact that scanning is not a reliable test to exclude placental separation. The advantages of ultrasonography lie elsewhere. Scanning documents the FHR, estimates the gestational age, notes the amniotic fluid volume (AFV), and documents placental locale. These data establish whether the pregnancy has reached the period of potential viability, verify that the fetus is alive, and document that a normal volume of amniotic fluid is present, reducing the likelihood of membrane rupture. Less commonly, evidence of placental separation or oligohydramnios can be visualized, and rarely, fetal injuries are identified. Continuous heart rate monitoring is appropriate only when the period of gestation is determined to be within the range of possible neonatal survival. Potential viability is a clinical concept that is assessed by determination of the period of gestation, the fetal weight, and when appropriate, any electronic monitoring data. When the estimated fetal weight is  $\geq 350$  g to 400 g and the composite gestational age is  $>23$  to 24 weeks, potential viability is assumed. In terms of laboratory testing, these parameters can vary to some degree among institutions. Kleihauer-Betke testing has not proven useful for the detection of abruptio and is unnecessary. Rh-negative women should receive Rh immune globulin in the usual dose. The decision for hospitalization hinges on the extent of the maternal injuries and the results of the FHR, as well as uterine contraction monitoring. Abruptio placentae is a problem. Neither the extent of maternal injury nor the ultrasound studies can reliably predict its occurrence. Recurrent contractions and notation of the blood count and differential (WBC  $>20,000$ )

helps to identify patients at increased risk for an abruptio [307,308,323,326]. An abruptio can be delayed by 24 to 48 hours after the initial events. In contrast, when the mother's physical examination is unremarkable, the EFM is normal, and uterine contractions are not recorded, the risk of a major complication, such as an abruptio, is very low.

### Nerve Injury

Nerve injuries are another potential source of morbidity that can occur spontaneously, be due to problems in patient positioning for delivery or surgery, or follow direct surgical trauma [281,290–292,296,300,301]. The principal factors that predispose to iatrogenic surgical injury are 1) improper placement of retractors, 2) improper patient positioning, and 3) radical surgery. Variations in patient anatomy play a lesser role. When obstetric injuries are considered, most are unrelated to regional analgesics or anesthesia. Both nulliparity and a prolonged second stage of labor are common associations [302]. Fortunately, these injuries are rarely permanent but do result in discomfort and difficulty with ambulation or other normal activities. Rarely, these injuries require surgery or specialized therapy to resolve. The most important nerve injuries are those leading to postoperative or postpartum paralysis involving the lower extremities, and *meralgia paresthetica* (MP), a relatively common injury involving a paresthesia of the lateral femoral cutaneous nerve [293,294,297].

The occurrence of nerve injuries is relatively low, but accurate statistics of incidence are not available. Parturition-related peripheral nerve injuries of all types are estimated to occur from 0.8 to 100 in 10,000 deliveries [281,302,303,335]. This wide range of incidence probably reflects mostly how the data were obtained. Retrospective studies report the lower incidences, whereas prospective investigations find the higher numbers. In addition, many injuries are minor or transient and might not be given clinical recognition unless signs and symptoms were carefully sought.

*Postoperative* or *postpartum paralysis* is a descriptive term that includes several different nerve injuries with an outwardly similar presentation. In all of these conditions, a previously normal woman is unable to ambulate or ambulates only with difficulty after a delivery or surgery owing to weakness in

various muscle groups of the lower extremity. The major neurologic injuries that can present in this fashion include:

- Postpartum footdrop
- Lumbrosacral palsy
- Peroneal nerve palsy
- Femoral neuropathy
- Obturator neuropathy
- Sciatic neuropathy

The difficulty in ambulation can go unrecognized in the immediate postoperative or postpartum period. Muscle weakness is normally not appreciated until ambulation is attempted. The diagnosis therefore is made at different times after delivery or surgery. Initially, it can appear that the problem is simply unsteadiness from exhaustion, vasomotor instability, residual drug effects, or anemia. A true sensory deficit or motor nerve injury also can be confused with a lingering motor blockade from a regional anesthetic agent. In women experiencing a vaginal delivery, pain from vaginal or perineal lacerations can also interfere with normal ambulation and be misinterpreted as a motor weakness.

A demonstrable muscle weakness or characteristic paresthesia in a previously normal woman, especially with a history of prolonged labor or difficult delivery, suggests nerve injury. When an iatrogenic neurologic injury is suspected, the extent of the deficit should be evaluated by careful physical and neurologic examination. At times, injuries are complex, with more than a single nerve root involved, potentially resulting in confusing findings.

In obstetric cases, most injuries are due to intrapelvic pressure on an exposed nerve root from descent of the presenting part. At times, pressure or trauma from the fetal head is combined with trauma from a delivery instrument such as forceps. In other instances, patient positioning in stirrups or incorrect second-stage pushing technique results in an injury from direct compression or an acute angulation, stretching the nerve root. A nerve can also be traumatized directly at surgery, usually from an operative instrument, such as a self-retaining retractor.

In the study by Warner and coworkers involving 991 surgical cases performed in lithotomy posi-

tion, the incidence of nerve injury was 1.5% [291]. The nerves injured included the lateral femoral cutaneous nerve, and the obturator, sciatic, and peroneal nerves. As has been noted in most reviews, resolution occurred in more than 90% of cases within 6 months.

The next section reviews both MP and the various major causes of postpartum paralysis and considers the pathophysiology of injury. Although prevention for all nerve injuries is not possible, surgical attention to technique and patient positioning can avoid many of these complications.

### Meralgia Paresthetica

MP (Bernhardt-Roth syndrome) is a relatively common nerve compression syndrome involving the lateral femoral cutaneous nerve [281,290,293–295,297,305,315,316]; 20% or more of all cases are bilateral. The prevalence of this condition is unknown but is estimated at 3 to 4 in 10,000 in the general population [294,316]. Most cases occur in patients from 30 to 65 years of age, but the condition has been described by patients of all ages.

The lateral femoral cutaneous nerve arises from the second and third lumbar root, although it can originate from different combinations of L1–3 nerve roots. In the pelvis, the nerve trunk runs across the iliacus muscle, inferior to the covering iliac fascia. The nerve then exits the pelvis, passing under the inguinal ligament just medial to the anterior superior iliac spine, and penetrates the fascia lata to distribute sensory branches to the skin of the lateral thigh. There is considerable anatomic variation in the course of the nerve. In approximately 30% of cases, the course of the nerve varies, arising partially or entirely from either the femoral or the genitofemoral nerve [295,297]. Operative injury to the nerve has been reported following laparoscopic procedures as well as laparotomy, orthopedic procedures, and even intramuscular injections [281,295,296,298,315,316]. When the condition is obstetrically related, symptoms usually begin in the third trimester. The paresthesia is usually progressive. Patients complain of sensations of tingling or burning (21%), numbness over the lateral thigh (48%), and decreased sensation of pinprick (45%) or pain (33%). There is no associated motor dysfunction and no predilection for either side, and the condition can recur in subsequent pregnancies.

Extension of the hip increases the distress. Symptoms are often exaggerated by walking, continuous standing, or certain actions (e.g., getting in or out of automobiles). Rest in lateral recumbency tends to relieve the discomfort. The condition is more common among the obese, especially during periods of rapid weight gain or uterine enlargement. Exaggerated lordosis is a common finding in symptomatic women.

The diagnosis follows a history review and clinical examination, with the notation of specific clinical features and characteristic signs and symptoms. Imaging or laboratory studies are not helpful in establishing the diagnosis. Electromyography can be performed but is usually unnecessary. Several findings distinguish MP from other disorders:

- Characteristic distribution
- Often unilateral findings, but possibly bilateral
- Absence of motor involvement
- Preservation of patellar and adductor deep tendon reflexes
- Association with pregnancy or recent surgery
- Absence of systemic disease or infection (e.g., multiple sclerosis or herpes simplex)

MP is usually a mild and self-limited disorder. Virtually all cases associated with pregnancy completely resolve within 3 months postpartum. A rare case might persist for years, however [305]. In most cases, no specific treatment beyond reassurance is required. Analgesics or a nerve block with a local anesthetic (e.g., 0.25% bupivacaine) can provide symptomatic relief and serve as a test for the diagnosis. The injection of phenol or other neurotoxins is not recommended owing to possible adverse effects such as dysesthesias. Other modalities of treatment include applications of moist heat, transcutaneous nerve stimulation, phonophoresis, or trigger-point soft-tissue therapy (usually the sartorius muscle).

Surgery for neurolysis or transection of the nerve is rarely indicated and not performed during pregnancy. Rarely, in nonpregnant patients, surgical decompression of nerve root is performed at the site where it exits through its narrow channel under the inguinal ligament.

Nerve transection is rarely done for relief in cases of chronic distress. In severe cases, such surgical exploration might be appropriate because there is a reasonable likelihood of partial or complete relief of symptoms [299,309,315].

In the nonpregnant patient, nonsteroidal anti-inflammatory drugs, tricyclic antidepressants (amitriptyline [Elavil]) or an anticonvulsant (gabapentin [Neurontin]; carbamazepine [Tegretol]) can be administered for relief [305,306,322].

### Femoral Neuropathy

*Femoral nerve palsy*, or *femoral neuropathy* (FNP), is among the causes of postpartum and occasionally postsurgical paralysis [281,300–304,310–314,328–335]. At laparotomy, the femoral nerve has usually been injured by direct compression caused by a self-retaining surgical retractor (Balfour or O'Connor-O'Sullivan type). FNPs also can result from obstetric and gynecologic procedures when a woman has been placed in dorsal lithotomy position in stirrups, or they can occur after prolonged second-stage labor. In general, the prognosis for recovery is good as long as an underlying serious or systemic disease is not present.

Femoral neuropathy is an uncommon complication of pregnancy and vaginal delivery. The incident in modern obstetric practice is not established but is usually reported as less than 1 in 10,000 live births. Al Hakim and Katirji [300] reported an incidence of FNP of approximately 1.5 to 2.0 in 1,000 surgical and obstetric procedures involving use of lithotomy position. Wong reported an incidence of 3.1 in 1,000 in an obstetric population studied prospectively [302].

### Pathophysiology

The femoral nerve (anterior crural nerve) is composed of roots arising from the posterior divisions of lumbar segments 2, 3, and 4. In the pelvis, the nerve passes through the body of the iliopsoas muscle, which normally shields it from direct injury. The nerve exits the pelvis lateral to the major femoral vessels, passing under the inguinal ligament. The femoral nerve provides motor innervations to the iliacus, sartorius, and quadriceps femoris muscles and also conveys sensation from the anterior and medial surfaces of the thigh and medial aspect of the leg by

the anterior femoral cutaneous and saphenous nerve roots, respectively. The nerve does not innervate the iliopsoas muscle, which is supplied by a separate branch from the lumbar plexus (L2–3). Thus, complete iliopsoas paralysis is usually not demonstrated when femoral nerve lesions occur. In terms of diagnosis, a combined femoral and iliopsoas palsy indicates an intrapelvic injury to the nerve roots rather than an isolated femoral nerve weakness without iliopsoas involvement. The latter suggests a peripheral injury to the nerve.

The femoral nerve is usually injured by direct compression in the iliopsoas groove, where the nerve root is covered by a tight fascia [300,328,329]. In gynecologic practice, the most common cause of injury is trauma from the blade of a self-retaining retractor inserted at laparotomy [329–332]. Less frequently in obstetric-related cases, the normal intrapelvic descent of the fetal head or an instrumental delivery can directly damage the nerve root.

Femoral nerve injury to either the portion of the nerve in the abdomen or the distal segment beyond the inguinal ligament during parturition is usually related to maternal positioning and can be unilateral or bilateral [311,312,333,334]. Injury from compression by the presenting part or obstetric manipulations such as instrumental delivery are unlikely causes of nerve trauma. Excessive hip abduction and external rotation either stretch the femoral nerve or interfere with its somewhat tenuous blood supply, resulting in localized ischemia. The portion of the nerve distal to the inguinal ligament can also be directly compressed during positioning of the thigh during second-stage pushing. Marked thigh flexion with external rotation and abduction, such as performed during the McRoberts maneuver, compresses the nerve directly. This position can result in injury when such positioning is combined with active bearing-down efforts.

As with the other obstetric paralysis syndromes, pain is usually not the major complaint; however, variable pain is often initially reported in the hip, buttock, or anterior portion of the thigh. Soon thereafter, the classic triad of 1) quadriceps muscle weakness, 2) absent reflexes, and 3) specific paresthesias becomes evident.

Examination reveals that muscle weakness is present, primarily involving the extensors of the thigh. The weakness is usually of acute onset and is

accompanied rarely by paresthesia over the medial thigh and anteromedial calf. The woman can sometimes walk on flat surfaces but is usually unable to climb stairs and has great difficulty going down stairs or any irregular surface. Unless the knee is kept locked, it can buckle. Arising from a seated position and stepping up are particularly difficult. Approximately 25% of these cases are bilateral. Complete recovery can be delayed but usually begins within 2 to 3 weeks of delivery and occurs within 10 to 12 weeks. The prognosis for full recovery is good [281,290].

On clinical examination alone, a femoral nerve injury can be difficult to differentiate from lumbosacral nerve palsy (i.e., combined footdrop, abductor or quadriceps palsy, and anteromedial leg/thigh paresthesias) or an isolated peroneal nerve injury (footdrop). In nonpregnant subjects with no history of surgical trauma, femoral neuropathy can be a sign of serious systemic disease such as advanced diabetes mellitus or atherosclerosis, but patients with these disorders are easily identified.

A characteristic history of recent abdominal surgery or obstetric vaginal delivery and acute quadriceps palsy can suggest the correct diagnosis. Because the onset of the neuropathy is abrupt, its association to delivery or to a gynecologic procedure is immediately recognized. Rarely, when the primary nerve injury arises from intrapelvic compression, trauma to other nerves with a pelvic course (e.g., obturator, lumbosacral trunk, iliopsoas nerves) is possible, potentially resulting in atypical findings or complex symptoms.

The various potential metabolic, infectious, or toxic causes of neuropathy can be excluded by appropriate testing or review of history. In the nonpregnant patient, MRI or CT scan can be helpful in diagnosis if compression from a pelvic mass is suspected.

Treatment is symptomatic and the prognosis for full recovery is excellent. Physical therapy is useful to maintain range of motion while spontaneous improvement is awaited. Bracing of the knee to prevent buckling and possible injury, and the use of assistive devices for ambulation are frequently required. If the etiology is traumatic, no intervention beyond these measures is usually required, and resolution in more than 90% of cases within 6 months can be anticipated. In the rare case

arising from nerve compression by intrapelvic masses or retroperitoneal tumors or hematomas, surgical or other medical intervention could be required to relieve pressure on the nerve trunk. In these situations, the long-term prognosis is less certain and depends on the progress of the underlying disorder.

At abdominal surgery, femoral nerve injury can be easily avoided by careful placement of the blades of the self-retaining retractor and by the routine use of sponges to pad displaced tissues. Because injuries can occur to patients placed in lithotomy position, owing to acute angulations or stretching of the nerve, proper positioning and avoidance of prolonged pushing with extreme abduction and rotation of the thigh reduces risk.

The recurrence risk from an injury during vaginal delivery is unknown. In addition, the best management for subsequent deliveries in women who have experienced this injury is unclear. Such cases need individual management, because there are little or no data to help in counseling these patients.

### Lumbosacral Palsy

*Lumbosacral palsy* (LSP, postpartum footdrop) is a nerve compression syndrome resulting from an injury to the pelvic lumbosacral trunk nerve (LST) [281,290]. Affected patients are found to have a unilateral footdrop and variable leg pain. This condition must to be distinguished from other causes of puerperal lower limb paralysis and pain, including peripheral peroneal nerve compression, obturator or femoral neuropathies, and lumbar disk disease. With LSP, the prognosis for full recovery is good, but the course can be long. The incidence of this disorder in modern practice is unknown, but it is uncommon. In a prospective study, Wong and coworkers [302] reported six cases in 6,048 deliveries or approximately 1 in 1,000.

The lumbosacral trunk (LST) is a large pelvic nerve root arising from the sacral plexus. The combined L4–5 lumbosacral nerve trunk crosses the sacroiliac articulation, passes medial to the obturator nerve, and joins the sciatic nerve. The principal nerve root within the lumbar sacral trunk is the sciatic nerve, which includes a preaxial component, the tibial nerve (anterior branches L4–S3), and a postaxial component, the common peroneal nerve (poste-

rior branches L4–S2), both contained within a single sheath. The sciatic exits the pelvis through the lower part of the greater sciatic foramen and runs along the inferior border of the piriformis muscle to the lower one third of the thigh, where its two parts separate. The sciatic nerve supplies motor nerves to the piriformis, coccygeus, levator ani, superior gluteal, and biceps and quadratus femoris and inferior gemellus muscles, among others. In the lower limb, the two major branches of the sciatic are the tibia (medial portion) and peroneal nerves (lateral portion).

Just below the fibular head, the peroneal nerve divides into the deep and superficial peroneal nerves, which pass into the lower leg. The superficial peroneal nerve innervates the peroneus longus and brevis muscles, which are foot evertors. The deep peroneal nerve innervates the major muscles extending the toes and dorsally flexing the foot. As it passes the knee, the peroneal nerve also supplies branches to a segment of the biceps femoris, one of the knee flexors.

The tibial is the larger, medial division of the sciatic nerve. The tibial nerve runs through the popliteal fossa, sending auricular branches up to the knee. The nerve then continues into the lower limb, giving rise to the sensory medial sural cutaneous nerve and a series of muscular branches to the various calf muscles and lateral plantar nerves of the foot.

In most cases, direct nerve compression between the fetal head and the ischium is presumed to be the cause of LSP. Usually the part of the nerve trunk giving rise to the common peroneal portion of the nerve is predominantly involved. Nerve injury results in the characteristic lower-limb footdrop palsy, mostly involving dorsoflexors of the lower limb. Presumably during labor, partial deflection of the fetal head during dystotic labor directly traumatizes the exposed nerve trunk as it passes through the pelvis, leading to an inflammatory edema and resultant dysfunction. Occiput posterior or brow position and a straight sacrum or a wide posterior pelvis are suspected risk factors for injury. The patient also might give a history of a difficult delivery. If the injury occurs from a nerve compression restricted to the lower limb, there is neither muscle weakness nor paresthesia superior to the fibular head. This finding can be difficult to detect without nerve conduction studies, however. If the

lumbosacral trunk has been injured, injuries to other pelvic nerves, such as the obturator, are possible. A footdrop syndrome with findings similar to LST palsy can follow from direct compression of the lateral peroneal nerve as it crosses the fibular head. In obstetric patients with such distal injuries, there is either a history of stirrup use during dorsal lithotomy positioning or prolonged second-stage pushing with the parturient grasping her leg over the fibular head ("pushing palsy") [281,318–321].

Patients with classic obstetric palsy also can have other injuries to the sciatic (L4–S3) or to the obturator nerve (L3–4), which might confuse the initial clinical findings [290,314]. Most infrequently, isolated obturator palsies are observed without demonstrable injury to other nerve roots. The obturator is usually compressed against the pelvic sidewall or in the obturator canal, presumably by some unique combination of maternal anatomy and positioning of the fetal head. Obturator nerve injury results in weakness of thigh adductors and the gracilis muscles and paresthesia in the upper inner thigh.

Once other diagnoses are excluded, the treatment for a lumbosacral nerve injury is symptomatic and nonspecific. Bedrest is appropriate for patients having difficulty with ambulation; a footboard and splinting reduce the risk of footdrop. Massage and range-of-motion exercises are also appropriate. There are no data about whether oral or parenteral steroid therapy is helpful in shortening the course of recovery or reducing the risk for permanent injury.

Complete, spontaneous resolution of LSP from obstetric trauma is the rule, generally occurring within 12 weeks or less of the original injury. Appropriate management of subsequent deliveries is unsettled, however, and so the choice is best left to an informed discussion between the woman and her physician.

### Other Nerve Injuries

Much less commonly, transient injuries occur to nerves of the upper extremities. The principal risks are to the nerves of brachial plexus. Ulnar neuropathies are also possible owing to improper arm positioning during surgery. The brachial plexus injuries are due to stretching; ulnar injuries are from direct compression and nerve ischemia. Proper padding and the avoidance of hyperabduction of the

arm by correct positioning on the operating suite table can avoid these injuries [290,292].

### REFERENCES

1. Illingworth R, A paediatrician asks – why is it called birth injury? *Br J Obstet Gynaecol*, 1985. 92:122–30.
2. Illingworth R, Why blame the obstetrician? A review. *Br Med J*, 1979. 1:797–801.
3. Badawi N, Kurinczuk JJ, Keogh JM, Alessandri LM, O'Sullivan F, Burton PR, Pemberton PJ, Stanley FJ, Antepartum risk factors for newborn encephalopathy: The Western Australia case-control study. *Br Med J*, 1998. Dec 5; 317(7172): 1549–53.
4. Nelson KB, Ellenberg JH, Antecedents of cerebral palsy: Multivariate analysis of risk. *N Engl J Med*, 1986. Jul 10; 315(2):81–6.
5. Badawi N, Kurinczuk JJ, Keogh JM, Alessandri LM, O'Sullivan R, Burton PR, Pemberton PJ, Stanley FJ, Intrapartum risk factors for newborn encephalopathy: The Western Australian case-control study. *Br Med J*, 1998. Dec 5; 317(7172): 1554–8.
6. Blumenthal I, Cerebral palsy – medicolegal aspects. *J Royal Soc Med*, 2001. Dec; 94(12):624–7.
7. Clark S, Hankins GD, Temporal and demographic trends in cerebral palsy – fact and fiction. *Am J Obstet Gynecol*, 2003. Mar; 188(3):628–33.
8. MacLennan A, A template for defining a causal relation between acute intrapartum events and cerebral palsy: International consensus statement. *Br Med J*, 1999. Oct 16; 319(7216):1054–9.
9. Nygaard I, Cruikshank DP, Should all women be offered elective cesarean delivery? [editorial]. *Obstet Gynecol*, 2003. Aug; 102(2):217–19.
10. McCusker JHD, Hosmer DW, Association of electronic fetal monitoring during labor with cesarean section rate and with neonatal morbidity and mortality. *Am J Pub Health*, 1988. 78:1170–4.
11. Farrell SA, Cesarean section versus forceps-assisted vaginal birth: It's time to include pelvic injury in the risk-benefit equation. *Can Med Assoc J*, 2002. Feb 5; 166(3):337–8.
12. Lai M, Mann C, Callender R, Radley S, Does cesarean delivery prevent anal incontinence? *Obstet Gynecol*, 2003. Feb; 101(2):305–12.

13. Fogelson N, Menard MK, Hulsey T, Ebeling M, Neonatal impact of elective repeat cesarean delivery at term: A comment on patient choice cesarean delivery. *Am J Obstet Gynecol*, 2005. May; 192(5):1433–6.
14. Sachs BP, Kobelin C, Castro M A, Frigoletto F, The risks of lowering the cesarean-delivery rate. *N Engl J Med*, 1999. Jan 7; 340(1):54–7.
15. Ghetti C, Guise J-M, Physicians' responses to patient-requested cesarean delivery. *Obstet Gynecol Surv*, 2005. Jun; 60(6):348–9.
16. National Institutes of Health: Department of Health and Human Services. In *Cesarean Delivery on Maternal Request*: March 27–29, 2006. Bethesda, Maryland.
17. Minkoff H, Powderly KR, Chervenak F, McCullough LB, Ethical dimensions of elective primary cesarean delivery. *Obstet Gynecol*, 2004. Feb; 103(2):387–92.
18. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin Number 49, December 2003: Dystocia and Augmentation of Labor.
19. Boulet SL, Salihi HM, Alexander GR. Mode of delivery and the survival of macrosomic infants in the United States, 1995–1999. *Birth*. 2006 Dec; 33(4):278–83.
20. Henriksen T, The macrosomic fetus: A challenge in current obstetrics. *Acta Obstet Scand* 2008; 87(2):134–45.
21. Nahum GG, Estimation of fetal weight. [www.emedicine.com](http://www.emedicine.com), 1–61, 2005.
22. Irion O, Boulvain M, Induction of labour for suspected fetal macrosomia. *Cochrane Database Syst Rev*; 2, 1998. CD000938.
23. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin Number 17, June 2000: Operative Vaginal Delivery.
24. American College of Obstetricians and Gynecologists, ACOG Practice Bulletin: Number 70, December, 2005: Intrapartum Fetal Heart Rate Monitoring.
25. Thacker S, Stroup D, Chang M, Continuous electronic heart rate monitoring for fetal assessment during labor. *Cochrane Database Syst Rev*: 2001: 2.
26. Althaus JE, Petersen SM, Fox HE, Holcroft CJ, Graham EM, Can electronic fetal monitoring identify preterm neonates with cerebral white matter injury? *Obstet Gynecol*, 2005. Mar; 105(3):458–65.
27. Nelson D, Dambrosia JM, Ting TY, Grether JK, Uncertain value of electronic fetal monitoring in predicting cerebral palsy. *N Engl J Med*, 1996. 324: 613–18.
28. Vintzileos A, Nochimson DJ, Guzman EF, Knuppel RA, Lake M, Schiffrin BS, Intrapartum electronic fetal heart rate monitoring versus intermittent auscultation: A meta-analysis. *Obstet Gynecol*, 1995. 85:149–55.
29. Ross M, Devoe LD, Rosen KG, ST-segment analysis of the fetal electrocardiogram improves fetal heart rate tracing interpretation and clinical decision making. *J Matern Fetal Neonatal Med*, 2004. Mar; 15(3):181–5.
30. Demissie K, Rhoads GG, Smulian JC, Balasubramanian BA, Gandhi K, Joseph KS, Kramer M, Operative vaginal delivery and neonatal and infant adverse outcomes: Population-based retrospective analysis. *Br Med J*, 2004. July; 329(7456): 24–9.
31. Carroli G, Belizan J, Episiotomy for vaginal birth (Cochrane Review). *Cochrane Database Syst Rev*. 1999.
32. Youssef R, Ramalingam U, Macleod M, Murphy DJ, Cohort study of maternal and neonatal morbidity in relation to use of episiotomy at instrumental vaginal delivery. *Br J Obstet Gynaecol*, 2005. Jul; 112(7):941–5.
33. Gainey H, Postpartum observation of pelvic tissue damage: Further studies. *Am J Obstet Gynecol*, 1955. Oct; 70(4):800–07.
34. Weber AM, Meyn L, Episiotomy use in the United States, 1979–1997. *Obstet Gynecol*, 2002. Dec; 100(6):1177–82.
35. Fitzpatrick M, Behan M, O'Connell PR, O'Herlihy C, A randomized clinical trial comparing primary overlap with approximation repair of third-degree obstetric tears. *Am J Obstet Gynecol*, 2000. Nov; 183(5):1220–4.
36. Briel J, de Boer LM, Hop WC, Schouten WR, Clinical outcome of anterior overlapping external anal sphincter repair with internal anal sphincter imbrication. *Dis Colon Rectum*, 1998. Feb; 41(2):209–14.
37. Buppasiri P, Lumbiganon P, Thinkhamrop J, Thinkhamrop B, Antibiotic prophylaxis for fourth-degree perineal tear during vaginal birth. *Cochrane Database Syst Rev*. 2005; 4.



38. Donnelly V, Fynes M, Campbell D, Johnson H, O'Connell PR, O'Herlihy C, Obstetric events leading to anal sphincter damage. *Obstet Gynecol*, 1998. Dec; 92(6):955-61.
39. Chaliha C, Sultan AH, Bland JM, Monga AK, Stanton SL, Anal function: Effect of pregnancy and delivery. *Am J Obstet Gynecol*, 2001. Aug; 185(2):427-32.
40. Sultan A, Kamm MA, Hudson CN, Thomas JM, Bartram CI, Anal-sphincter disruption during vaginal delivery. *N Engl J Med*, 1993. Dec; 329(26):1905-11.
41. Kearney R, Miller JM, Ashton-Miller JA, DeLancey OL, Obstetric factors associated with levator ani muscle injury after vaginal birth. *Obstet Gynecol*, 2006. Jan; 107(1):144-9.
42. Rorveit G, Daltveit AK, Hannestad YS, Hunskaar S, Urinary incontinence after vaginal delivery or cesarean section. *N Engl J Med*, 2003. Mar 6; 348(10):900-07.
43. Seymour S, Fecal Incontinence. [www.eMedicine.com](http://www.eMedicine.com), 1-28, 2002.
44. Klein MC, Elective cesarean section. *Can Med Assoc J*, 2004. Jul; 171(1):14-5.
45. Wax J, Cartin A, Pinette MG, Blackstone J, Patient choice cesarean: An evidence-based review. *Obstet Gynecol Surv*, 2004. Aug; 59(8):601-16.
46. Sharma G, Is patient-choice primary cesarean rational? *OBG Management*, 2006. May; 18(5):1-5.
47. Minkoff H, Chervenak FA, Elective primary cesarean delivery. *N Engl J Med*, 2003. Mar; 348(10):946-50.
48. MacLennan A, Taylor AW, Wilson DH, Wilson D, The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. *Br J Obstet Gynaecol*, 2000. Dec; 107(12):1460-70.
49. Beets-Tan RGH, Morren GL, Kessels AFG, el Naggar K, Baeten CGMI, van Engelshover JMA, Measurement of anal sphincter muscles: Endoanal U/S, endoanal MR imaging, or phased-array MR imaging? A study with healthy volunteers. *Radiology*, 2001. 220(1):81-89.
50. Delancey JOL, Toggia MR, Perucchini D, Internal and external anal sphincter anatomy as it related to midline obstetric lacerations. *Obstet Gynecol*, 1997. Dec; 90(6):924-7.
51. Hsu Y, Fenner DE, Weadock WJ, DeLancey JOL, Magnetic resonance imaging and 3-dimensional analysis of external anal sphincter anatomy. *Obstet Gynecol*, 2005. Dec; 106(6):1259-65.
52. Shy K, Eschenbach DA, Fetal perineal cellulitis from an episiotomy site. *Obstet Gynecol*, 1979. 54:292-8.
53. Dellinger E, Severe necrotizing soft tissue infections. *JAMA*, 1981. 246:1717-21.
54. Ramin S, Ramus RM, Little BB, Gilstrap LC 3rd, Early repair of episiotomy dehiscence associated with infection. *Am J Obstet Gynecol*, 1992. Oct; 167(4 Pt 1):1104-7.
55. Arona A, al-Marayati L, Grimes DA, Ballard CA, Early secondary repair of third-and fourth-degree perineal lacerations after outpatient wound preparation. *Obstet Gynecol*, 1995. Aug; 86(2):294-6.
56. Wen S, Rusen ID, Walker M, Liston R, Kramer MS, Baskett T, for the Maternal Health Study Group, Canadian Perinatal Surveillance System, Comparison of maternal mortality and morbidity between trial of labor and elective cesarean section among women with previous cesarean delivery. *Am J Obstet Gynecol*, 2004. Oct; 191(4):1263-9.
57. Durnwald C, Mercer B, Vaginal birth after cesarean delivery: Predicting success, risks of failure. *J Matern Fetal Neonatal Med*, 2004. Jun; 15(6):388-93.
58. Shipp T, Zelop CM, Repke JT, Cohen A, Caughey AB, Lieberman E, Intrapartum uterine rupture and dehiscence in patients with prior lower uterine segment vertical and transverse incisions. *Obstet Gynecol*, 1999. Nov; 94(5 Pt 1):735-40.
59. Naef R, Ray MA, Chauhan SP, Roach H, Blake PG, Martin JN, Trial of labor after cesarean delivery with a lower-segment, vertical uterine incision: Is it safe? *Am J Obstet Gynecol*, 1995. Jun; 172(6):1666-73.
60. Golan A, Sandhank P, Rubin A, Rupture of the pregnant uterus. *Obstet Gynecol*, 1980 Nov; 56(5):549-54.
61. Golan A, David MP, Birth Injuries. In *Operative Obstetrics*, ed 2. L. Iffy, Apuzzio JJ, Vintzileos AM (eds). 1992, McGraw-Hill: New York.
62. Landon M, Leindecker S, Spong CY, Hauth JC, Bloom S, Varner MW, Moawad AH, Caritis SN, Harper M, Wapner RJ, Sorokin Y, Miodovnik M, Carpenter M, Peacemen AM, O'Sullivan MJ, Sibai BM, Langer O, Thorp JM, Ramin SM, Mercer BM, Gabbe SG, National Institute of Child Health and Human Development, Maternal and Fetal Units

- Network, The MFMU Cesarean Registry: Factors affecting the success of trial of labor after previous cesarean delivery. *Am J Obstet Gynecol*, 2005. Sept; 193(3 Pt 2):1016–23.
63. Smith G, White IR, Pell JP, Dobbie R, Predicting cesarean section and uterine rupture among women attempting vaginal birth after prior cesarean section. *PLoS Med*, 2005. Sept; 2(9):e252.
  64. Smith G, Pell JP, Pasupathy D, Dobbie R, Factors predisposing to perinatal death related to uterine rupture during attempted vaginal birth after caesarean section: Retrospective cohort study. *Br Med J*, 2004. Aug 14; 329(7462):375.
  65. Norris T, Management of postpartum hemorrhage. *Am Fam Phys*, 1997. 55(2):635–640.
  66. Shah-Hosseini R, Evrard JR, Puerperal uterine inversion. *Obstet Gynecol*, 1989. Apr; 73(4):567–70.
  67. Brar H, Greenspoon JC, Platt LD, Paul RH. Acute puerperal uterine inversion. New approaches to management. *J Reprod Med*, 1989. Feb; 34: 173–7.
  68. Baskett TF, Essential management of obstetric emergencies, ed 3. 1999, Redland, Bristol, England: Clinical Press Limited.
  69. American College of Obstetricians and Gynecologists, ACOG Educational Bulletin: Number 76, October 2006: Postpartum Hemorrhage.
  70. Shevell T, Malone FD, Management of obstetric hemorrhage. *Semin Perinatol*, 2003. 27(1):86–104.
  71. Smith J, Brennan BG, Postpartum hemorrhage 2006. [www.eMedicine.com](http://www.eMedicine.com). topic = 3568.
  72. Chichakli L, Atrash HK, MacKay AP, Musani AS, Berg CJ, Pregnancy-related mortality in the United States due to hemorrhage: 1979–92. *Obstet Gynecol*, 1999. 94(5 Pt 1):721–25.
  73. Prendville W, Elborune D, McDonald S, Active versus expectant management in the third stage of labour (Cochrane Review). *Cochrane Database Syst Rev*, 2000; 3.
  74. Turner J, Embolization, hemorrhage. [www.eMedicine.com](http://www.eMedicine.com). 1–18, 2006.
  75. Clark S, Yeh SY, Phelan JP, Bruce S, Paul RH, Emergency hysterectomy for the control of obstetric hemorrhage *Obstet Gynecol*, 1984. Sept; 64(3):376–80.
  76. Chou Y, Wang PH, Yuan CC, Yen YK, Liu WM, Laparoscopic bipolar coagulation of uterine vessels to manage delayed postpartum hemorrhage. *J Am Assoc Gynecol Laparosc*, 2002. Nov; 9(4): 541–4.
  77. Mazhar S, Yasmin S, Gulzar S, Management of massive postpartum hemorrhage by “B-Lynch” brace suture. *J Coll Physicians Surg Pak*, 2003. Jan; 13(1):51–52.
  78. AbdRabbo SA, Step-wise uterine devascularization: A novel technique for management of uncontrollable postpartum hemorrhage with the preservation of the uterus. *Am J Obstet Gynecol*, 1999. Sept; 171(3):694–700.
  79. Clark S, Managing postpartum hemorrhage: Establish a cause. *OB Management*, 14(11):1–12.
  80. Tuffnell D, Amniotic fluid embolism. *Curr Opin Obstet Gynecol*, 2003. April; 15(2):119–22.
  81. Moore LE, Ware D, Amniotic fluid embolism. [www.eMedicine.com](http://www.eMedicine.com).topic = 122, 2007.
  82. Fletcher S, Parr MJ, Amniotic fluid embolism: A case report and review. *Resuscitation*, 2000. Jan; 43(2):141–6.
  83. Davies S, Amniotic fluid embolism: A review of the literature. *Can J Anaesth*, 2001. Jan; 48(1):88–98.
  84. Clark S, New concepts of amniotic fluid embolism: A review. *Obstet Gynecol Surv*, 1990. 45(6):360–8.
  85. Steiner P, Lushbaugh CC, Maternal pulmonary embolism by amniotic fluid as a cause of obstetric shock and unexpected death in obstetrics. *JAMA*, 1941. 117:1245–54; 1341–5.
  86. Clark S, Hankins DG, Dudley DA, Dildly DA, Porter TF, Amniotic fluid embolism: Analysis of the national registry. *Am J Obstet Gynecol*, 1995. Apr; 172(4 Pt.1):1158–67.
  87. Gilmore D, Wakim J, Secrest J, Rawson R, Anaphylatoid syndrome of pregnancy: A review of the literature with latest outcome and management data. *AANA J*, 2003. Apr; 71(2):120–6.
  88. Lockwood C, Bach R, Guha A, Zhou XA, Miller WA, Nemerson Y, Amniotic fluid contains tissue factor, a potent initiator of coagulation. *Am J Obstet Gynecol*, 1991. Nov; 165(5 Pt 1):1335–41.
  89. Kent K, Cooper BC, Thomas KW, Zlatnik FJ, Presumed antepartum amniotic embolism. *Obstet Gynecol*, 2003. Sept; 102(3):493–5.
  90. Nishio H, Matsui K, Miyazaki T, Tamura A, Iwata M, Suzuki I, A fatal case of amniotic fluid embolism with elevation of serum mast cell

- tryptase. *Forensic Sci Int*, 2002. Mar 28; 126(1): 53–6.
91. Benson M, Lindberg RE, Amniotic fluid embolism, anaphylaxis and tryptase. *Am J Obstet Gynecol*, 1996. 175(3):737–9.
  92. Farrar S, Gherman RB, Serum tryptase analysis in a woman with amniotic fluid embolism: A case report. *J Reprod Med*, 2001. Oct; 46(10):926–8.
  93. Haines J, Wilkes RG, Non-fatal amniotic fluid embolism after cervical suture removal. *Br J Anaesth*, 2003. 90(2):244–7.
  94. Ray B, Vallejo MC, Creinin MD, Shannon KT, Mandell GL, Kaul B, Ramanathan S, Amniotic fluid embolism with second trimester pregnancy termination: A case report. *Can J Anaesth*, 2004. Feb; 51(2):139–44.
  95. Tramoni G, Valentin S, Robert MO, Sergeant MV, Branche P, Duperret S, Clement HJ, Lopez F, Boisson C, Audra P, Rudigoz RC, Viale JP, Amniotic fluid embolism during caesarean section. *Int J Obstet Anesth*, 2005. Apr; 14(2):179–80.
  96. Manchandra R, Sriemevan A, Anaphylactoid syndrome caused by amniotic fluid embolism following manual removal placenta. *J Obstet Gynaecol*, 2005. Feb; 25(2):201–2.
  97. Bastien J, Graves JR, Bailey S, Atypical presentation of amniotic fluid embolism. *Anesth Analg*, 1998. 87(1):124–6.
  98. Grimes D, Cates W Jr, Fatal amniotic fluid embolism during induced abortion, 1972–75. *South Med J*, 1977. Nov; 70(11):1325–6.
  99. Mirchandani H, Mirchandani IH, Parikh SR, Hyponatremia due to amniotic fluid embolism during a saline-induced abortion. *Am J Forensic Med Pathol*, 1988. Mar; 9(1):48–50.
  100. Margaron M, Delayed amniotic fluid embolism following caesarean section under spinal anaesthesia. *Anaesthesia*, 1995. Sept; 50(9):804–6.
  101. Hasaart T, Essed GG, Amniotic fluid embolism after transabdominal amniocentesis. *Eur J Obstet Gynecol Reprod Biol*, 1983. Sept; 16(1):25–30.
  102. Maher J, Wenstrom KD, Hauth JC, Meis PJ, Amniotic fluid embolism after saline amnioinfusion: Two cases and review of the literature. *Obstet Gynecol*, 1994. May; 83(5 Pt 2):851–4.
  103. Lau G, Chui PP, Amniotic fluid embolism: A review of 10 fatal cases. *Singapore Med J*, 1994. Apr; 35(2):180–3.
  104. Locksmith G, Amniotic fluid embolism. *Obstet Gynecol Clin North Am*, 1999. Sept; 26(3):435–445.
  105. Goldszmidt E, Davies S, Two cases of hemorrhage secondary to amniotic fluid embolus managed with uterine artery embolization. *Can J Anaesth*, 2003. 50:917–21.
  106. van Haeften T, van Schijndel RJM, Thijs LG, Severe lung damage after amniotic fluid embolism: A case with haemodynamic measurements. *Neth J Med*, 1989. 35:317–20.
  107. Clark S, Successful pregnancy outcomes after amniotic fluid embolism. *Am J Obstet Gynecol*, 1992. Aug; 167(2):511–2.
  108. Demianczuk C, Corbett TF, Successful pregnancy after amniotic fluid embolism: A case report. *J Obstet Gynaecol Can*, 2005. Jul; 27(7):699–701.
  109. Han D, Soo Lee K, Franquet T, Muller NL, Kim TS, Kim H, Kwon OJ, Byun HS, Thrombotic and nonthrombotic pulmonary arterial embolism: Spectrum of imaging findings. *Radiographics*, 2003. 23:1521–39.
  110. de Rooij G, Gelissen HPMM, Wester JPI, Spijkstra JJ, Go ATJJ, Girbes ARJ, Severe maternal respiratory distress due to the amniotic fluid embolism syndrome in a twin pregnancy. *Neth J Med*, 2003. Oct; 61(10):337–40.
  111. Hankins G, Snyder R, Dinh T, Van Hook J, Clark S, Vandelan A, Documentation of amniotic fluid embolism via lung histopathology: Fact or fiction? *J Reprod Med*, 2002. Dec; 47(12):1021–4.
  112. Stanten R, Iverson LI, Daugharty TM, Lovett SM, Terry C, Blumenstock E, Amniotic fluid embolism causing catastrophic pulmonary vasoconstriction: Diagnosis by transesophageal echocardiogram and treatment by cardiopulmonary bypass. *Obstet Gynecol*, 2003. Sept; 102(3):496–8.
  113. Young J, Relaxation of the pelvic joints in pregnancy: Pelvic arthropathy of pregnancy. *J Obstet Gynaecol Br Emp*, 1940. 47:493–524.
  114. Dietrichs E, Kogstad O, "Pelvic girdle relaxation" – suggested new nomenclature. *Scand J Rheumatol Suppl*, 1991. 88:3.
  115. Rost C, Jacqueline J, Kaiser A, Verhagen A, Koes B, Pelvic pain during pregnancy: A descriptive study of signs and symptoms of 870 patients in primary care. *Spine*, 2004. Nov 15; 29(22):2567–72.
  116. Leadbetter R, Mawer D, Lindow SW, Symphysis pubis dysfunction: A review of the

- literature. *J Matern Fetal Neonatal Med*, 2004. Dec; 16(6):349–54.
117. Owens K, Pearson A, Mason G, Symphysis pubis dysfunction – a cause of significant obstetric morbidity. *Eur J Obstet Gynecol Reprod Biol*, 2002. Nov 15; 105(2):143–6.
  118. Elden H, Ladfors L, Olsen MF, Ostgaard HC, Hagberg H, Effects of acupuncture and stabilising exercises as adjunct to standard treatment in pregnant women with pelvic girdle pain: Randomised single-blind controlled trial. *Br Med J*, 2005. April 2; 330(7494):761–4.
  119. Jain N, Sternberg LB, Symphyseal separation. *Obstet Gynecol*, 2005. May; 105(5 Pt 2):1229–32.
  120. Culligan P, Hill S, Heit M, Rupture of the symphysis pubis during vaginal delivery followed by two subsequent uneventful pregnancies. *Obstet Gynecol*, 2002. Nov; 100(5 Pt 2):1114–7.
  121. Shakespeare, W. Measure for Measure. II, i, 86–95.
  122. Snow R, Neubert AG, Peripartum pubic symphysis separation: A case series and review of the literature. *Obstet Gynecol Surv*, 1997. Jul; 52(7):438–43.
  123. Pubic, [www.plus-size-pregnancy.org/pubic-pain.htm](http://www.plus-size-pregnancy.org/pubic-pain.htm), 2006.
  124. Schwartz Z, Katz Z, Lancet M, Management of puerperal separation of the symphysis pubis. *Int J Gynaecol Obstet*, 1985. Apr; 23(2):125–8.
  125. Albert H, Godsken M, Westergaard J, Prognosis in four syndromes of pregnancy-related pelvic pain. *Acta Obstet Gynecol Scand*, 2001. Jun; 80(6):505–10.
  126. MacLennan A, MacLennan SC, Symptom-giving pelvic girdle relaxation of pregnancy, postnatal pelvic joint syndrome and developmental dysplasia of the hip: The Norwegian Association for Women with Pelvic Girdle Relaxation (Landforeningen for Kvinner Med Bekkenlosningsplager). *Acta Obstet Gynecol Scand*, 1997. Sept; 76(8):760–4.
  127. Heath T, Gherman RB, Symphyseal separation, sacroiliac joint dislocation and transient lateral femoral cutaneous neuropathy associated with McRobert's maneuver: A case report. *J Reprod Med*, 1999. Oct; 44(10):902–4.
  128. Phupong V, Sudjai D, Vaginal delivery with intrapartum pubic symphysis separation: A case report. *J Reprod Med*, 2003. Apr; 48(4):296–8.
  129. Cappiello G, Oliver BC, Rupture of symphysis pubis caused by forceful and excessive abduction of the thighs with labor epidural anesthesia. *J Fla Med Assoc*, 1995. Apr; 82(4):261–3.
  130. Van Roosmalen J, Symphysiotomy – a reappraisal for the developing world. In *Progress in Obstetrics and Gynaecology*, J. Studd (ed). Edinburgh: Churchill-Livingstone, 1991, pp. 149–62.
  131. Mola G, Symphysiotomy or caesarean section after failed trial of assisted delivery. *P N G Med J*, 1995. Sept; 38(3):172–7.
  132. Crichton D, Seedat EK, The technique of symphysiotomy. *S Afr Med J*, 1963. Mar 2; 37:227–31.
  133. Gebbie D, Symphysiotomy. *Clin Obstet Gynaecol*, 1982. Dec; 9(3):663–83.
  134. Ross J, Hu LT, Septic arthritis of the pubic symphysis: Review of 100 cases. *Medicine (Baltimore)*, 2003. Sept; 82(5):340–5.
  135. Eskridge C, Longo S, Kwark J, Robichaux A, Begnaud W, Osteomyelitis pubis occurring after spontaneous vaginal delivery: A case presentation. *J Perinatol*, 1997. Jul–Aug; 17(4):321–4.
  136. Lovisetti G, Sala F, Battaini A, Lovisetti L, Guicciardi E, Osteomyelitis of the pubis symphysis, abscess and late disjunction after delivery: A case report. *Chir Organi Mov*, 2000. Jan–Mar; 85(1):85–8.
  137. Wray C, Easom S, Hoskinson J, Coccydynia: Aetiology and treatment. *J Bone Joint Surg*, 1991. Mar; 73-B(2):335–8.
  138. O'Grady J, Coccygodynia. In *Obstetric Syndromes & Conditions*, J. O'Grady, Burkman RT (eds). 1998, The Parthenon Publishing Group: New York.
  139. Maigne JY, Doursounian L, Chatellier G, Causes and mechanisms of common coccydynia: Role of body mass index and coccygeal trauma. *Spine*, 2000. 25(3):3072–9.
  140. Cooper G, Lee MS, Lutz GE, Fracture of coccyx during childbirth: A case report of an unusual cause of coccygodynia. *Arch Physical Med Rehab*, 2003. Sept; 84(9):E15.
  141. Postacchini F, Massorbrio M, Idiopathic coccygodynia. *J Bone Joint Surg*, 1983. 65A(8):1116–24.
  142. Lyons ML, Coccygodynia. *eMedicine*, topic = 383, 2005.
  143. Maigne J, Management of common coccygodynia. [www.coccyx.org](http://www.coccyx.org), 2005.

144. Nelson D, Coccydynia and lumbar disk disease – historical correlations and clinical cautions. *Perspect Biol Med*, 1991. Winter; 34(2):229–38.
145. Ramsey M, Toohey JS, Neidre A, Stromberg LJ, Roberts DA, Coccygodynia: Treatment. *Orthopediatrics*, 2003. Apr; 26(4):403–5.
146. Hodges S, Eck JC, Humphreys SC, A treatment and outcomes analysis of patients with coccygodynia. *Spine J*, 2004. Mar–Apr; 4(2):138–40.
147. Karalezli K, Iltar S, Irgit K, Karalezli N, Karakoc Y, Aydogan N, Coccygectomy in the treatment of coccygodynia. *Acta Orthop Belg*, 2004. Dec; 70(6):583–5.
148. Wood K, Mehbod AA, Operative treatment for coccygodynia. *J Spinal Disord Tech*, 2004. Dec; 17(6):511–5.
149. Feldbrin Z, Singer M, Keynan O, Rzetelny V, Hendel D, Coccygectomy for intractable coccygodynia. *Isr Med Assoc J*, 2005. Mar; 7(3):160–2.
150. Perkins R, Schofferman J, Reynolds J, Coccygectomy for severe refractory sacrococcygeal joint pain. *J Spinal Disord Tech*, 2003. Feb; 16(1):100–3.
151. Stelfox HT, Palmisani S, Scurlock C, Orav, EJ, Bates DW, The “To Err is Human” report and the patient safety literature. *Qual Saf Health Care*, 2006 Jun; 15(3): 174–8.
152. Hughes C, Harley EH, Milmoc G, Bala R, Martorella A, Birth trauma in the head and neck. *Arch Otolaryngol Head Neck Surg*, 1999. Feb; 125(2):193–9.
153. Davey C, Moore AM, Necrotizing fasciitis of the scalp in a newborn. *Obstet Gynecol*, 2006. Feb; 107(2 Pt 2):461–3.
154. Lauer A, Rimmer SO, Eyelid laceration in a neonate by fetal monitoring spiral electrode. *Am J Ophthalmol*, 1998. May; 125(5):715–7.
155. Wigger H, Influence of perinatal management. In *Textbook of Fetal and Perinatal Pathology*, H Wigger, Singer DB (eds). 1991, Blackwell Scientific Publications: Boston.
156. Curran J, Birth-associated injury. *Clin Perinatol*, 1981. Feb; 8(1):111–29.
157. Wigglesworth J, Singer DB, *Textbook of Fetal and Perinatal Pathology*. 1991, Boston: Blackwell Scientific Publications.
158. Vacca A, *Handbook of Vacuum Extraction in Obstetric Practice*. 1992, London: Edward Arnold.
159. Plauché WC, Subgaleal hematoma. A complication of instrumental delivery. *JAMA*, 1980. Oct; 244:1597–8.
160. Broekhuizen F, Washington JM, Johnson F, Hamilton PR, Vacuum extraction versus forceps delivery: Indications and complications, 1979–1984. *Obstet Gynecol*, 1987. Mar; 69(3 Pt 1):338–42.
161. Bofill J, Rust OA, Devidas M, Roberts WE, Morrison JC, Martin JN Jr, Neonatal cephalohematoma from vacuum extraction. *J Reprod Med*, 1997. Sept; 42(9):565–9.
162. Pape K, Wigglesworth JS, Haemorrhage, ischaemia and the perinatal brain. 1979, London: Heinemann.
163. Volpe J, *Neurology of the Newborn*, ed 4. 2001, Philadelphia: W. B. Saunders.
164. Garcia Aruqueza CG, Aguayo Maldonado J, Almuedo Paz A, Cephalohematoma as the first manifestation of congenital factor XIII deficiency. *An Esp Pediatr*, 2000. Sept; 53(3):241–2.
165. Fan H-C, Hua Y-M, Juan C-J, Fang Y-M, Chen S-N, Wang C-C, Infected cephalohematoma associated with sepsis and scalp cellulitis: A case report. *J Microbiol Immunol Infect*, 2002. 35:125–8.
166. Chang H, Chiu NC, Huang FY, Kao H A, Hsu CH, Hung HY, Infected cephalohematoma of newborns: Experience in a medical center in Taiwan. *Pediatr Int*, 2005. Jun; 47(3):274–7.
167. Kaufmann H, Hochberg J, Anderson RP, Schochet SS Jr, Simmons GM Jr, Treatment of calcified cephalohematoma. *Neurosurgery*, 1993. Jun; 32(6):1037–9.
168. Macleod C, O'Neill C, Vacuum-assisted delivery – the need for caution. *Ir Med J*, 2003. May; 96(5):147–8.
169. Gebremariam A, Subgaleal haemorrhage: risk factors and neurological and developmental outcome in survivors. *Ann Trop Paediatr*, 1999. Mar; 19(1):45–50.
170. Kilani R, Wetmore J, Neonatal subgaleal hematoma: Presentation and outcome – radiological findings and factors associated with mortality. *Am J Perinatol*, 2006. Jan; 23(1):41–8.
171. Vacca A, Vacuum-assisted delivery. *Best Pract Res Clin Obstet Gynaecol*, 2002. Feb; 16(1):17–30.
172. Vacca A, Vacuum-assisted delivery: Improving patient outcomes and protecting yourself against litigation. *OB Management*, 2004. Feb; (Suppl): S1–S12.

173. O'Grady J, Pope CS, Patel SS, Vacuum extraction in modern obstetric practice: A review and critique. *Curr Opin Obstet Gynecol*, 2000. 12:475–480.
174. O'Grady J, Instrumental delivery: A critique of current practice. In *Gynecologic, Obstetric, and Related Surgery*, ed 2. D. Nichols, Clarke-Pearson DL (eds). 2000, Mosby: St. Louis. pp 1081–105.
175. Zelson C, Lee SJ, Pearl M, The incidence of skull fractures underlying cephalohematomas in newborn infants. *J Pediatr*, 1974. 85:371–3.
176. Loeser J, Kilburn HL, Jolley T, Management of depressed skull fracture in the newborn. *J Neurosurg*, 1976. Jan; 44(1):62–4.
177. Raynor R, Parsa M, Nonsurgical elevation of depressed skull fracture in an infant. *J Pediatr*, 1968. Feb; 72(2):262–4.
178. Bercendes H, The epidemiology of perinatal injury. In *Preventability of Perinatal Injury*, F.H.E. Adamson K (ed). 1989, Alan R. Liss: New York.
179. Kingsley D, Till K, Hoare R, Growing fractures of the skull. *J Neurol Neurosurg Psychiatry*, 1978. Apr; 41(4):312–8.
180. Valdes-Dapena M, Arey JB, Pulmonary emboli of cerebral origin in the newborn: A report of two cases. *Arch Pathol*, 1967. Dec; 84(6):643–6.
181. Wannakrairo P, Shuangshoti S, Cerebellar tissue embolism associated with birth injury. *J Med Assoc Thai*, 1984. May; 67(5):290–4.
182. Dorfman M, Benson WH, Marginal eyelid laceration after episiotomy. *Am J Ophthalmol*, 1993. Dec 15; 116(6):778.
183. Holden R, Morsman DG, Davidek GM, O'Connor GM, Coles ED, Dawson AJ, External ocular trauma in instrumental and normal deliveries. *Br J Obstet Gynaecol*, 1992. Feb; 99(2): 132–4.
184. Regis, A, Dureau P, Uteza Y, Roche O, Dufier JL, Ocular injuries and childbirth. *J Fr Ophtalmol*, 2004. Nov; 27(9 Pt 1):987–93.
185. Estafanous M, Seeley M, Traboulsi EI, Choroidal rupture associated with forceps delivery. *Am J Ophthalmol*, 2000. Jun; 129(6):819–20.
186. Lambert S, Drack AV, Hutchinson AK, Longitudinal changes in the refractive errors of children with tears in Descemet's membrane following forceps injuries. *JAAPOS*, 2004. Aug; 8(4):368–70.
187. Honig M, Barraquer J, Perry HD, Riquelme JL, Green WR, Forceps and vacuum injuries to the cornea: Histopathologic features of twelve cases and review of the literature. *Cornea*, 1996. Sept; 15(5):463–72.
188. Angell L, Robb RM, Berson FG, Visual prognosis in patients with ruptures in Descemet's membrane due to forceps injuries. *Arch Ophthalmol*, 1981. Dec; 99(12):2137–9.
189. McDonald M, Burgess SK, Contralateral occipital depression related to obstetric forceps injury to the eye. *Am J Ophthalmol*, 1992. Sept 15; 114(3):318–21.
190. Thompson KA, Satin AJ, Gherman RB, Spiral fracture of the radius: An unusual case of shoulder dystocia-associated morbidity. *Obstet Gynecol*, 2003. Jul; 102(1):36–8.
191. Lysack J, Soboleski D, Classic metaphyseal lesion following external cephalic version and cesarean section. *Pediatr Radiol*, 2003. Jun; 33(6): 422–4.
192. Alexander J, Gregg JEM, Quinn MW, Femoral fractures at caesarean section: Case reports. *Br J Obstet Gynaecol*, 1987. Mar; 94(3):273.
193. Papp S, Dhaliwal G, Davies G, Borschneck D, Fetal femur fracture and external cephalic version. *Obstet Gynecol*, 2004. Nov; 104(5 Pt 2):1154–6.
194. Bhat B, Kuman A, Oumachigui A, Bone injuries during delivery. *Indian J Pediatr*, 1994. Jul–Aug; 61(4):401–5.
195. Garcia Garcia I, de la Vega A, Garcia Fragoso L, Long-bone fractures in extreme low birth weight infants at birth: Obstetrical considerations. *P R Health Sci J*, 2002. Sept; 21(3):253–5.
196. Ehrenfest H, *Birth Injuries of the Child*. 1922, New York: D. Appleton & Company.
197. Turnpenny P, Nimmo A, Fractured clavicle of the newborn in a population with a high prevalence of grand-multiparity: Analysis of 78 consecutive cases. *Br J Obstet Gynaecol*, 1993. Apr; 100(4):338–41.
198. Beall M, Ross MG, Clavicle fracture in labor: Risk factors and associated morbidities. *J Perinatol*, 2001. Dec; 21(8):513–5.
199. Lam M, Wong GY, Lao TT, Reappraisal of neonatal clavicular fracture: Relationship between infant size and risk factors. *J Reprod Med*, 2002. Nov; 47(11):903–8.
200. Kaplan B, Rabinerson D, Avrech OM, Carmi N, Steinberg DM, Merlob P, Fracture of the clavicle in the newborn following normal labor and delivery. *Int J Gynaecol Obstet*, 1998. Oct; 63(1): 15–20.

201. Roberts SW, Hernandez C, Maberry MC, Adams MD, Levano KJ, Wendel GD Jr, Obstetric clavicular fracture: The enigma of normal birth. *Obstet Gynecol*, 1995. Dec; 86(6):978-81.
202. Caird M, Reddy S, Ganley TJ, Drummond DS, Cervical spine fracture-dislocation birth injury: Prevention, recognition, and implications for the orthopaedic surgeon. *J Pediatr Orthop*, 2005. Jul-Aug; 25(4):484-6.
203. Menticoglou S, Perlman M, Manning FA, High cervical spinal cord injury in neonates delivered with forceps: Report of 15 cases. *Obstet Gynecol*, 1995. Oct; 86(4 Pt 1):589-94.
204. Byers R, Spinal-cord injuries during birth. *Dev Med Child Neurol*, 1975. Feb; 17(1):103-10.
205. Bergman I, May M, Wessel HB, Stool SE, Management of facial palsy caused by birth trauma. *Laryngoscope*, 1986. Apr; 96(4):381-4.
206. Hepner WJ, Some observations on facial paresis in the newborn infant: Etiology and incidence. *Pediatrics*, 1951. Oct; 8(4):494-7.
207. McHugh H, Sowden KA, Levitt MN, Facial paralysis and muscle agenesis in the newborn. *Arch Otolaryngol*, 1969. Jan; 89(1):157-69.
208. Palmer CA, Mobius Syndrome. [www.eMedicine.com](http://www.eMedicine.com), topic = 612, 2001.
209. Crelin ES, Anatomy of the newborn: An atlas. 1969, Philadelphia, PA: Lea & Febiger.
210. Kornblut AD, Facial nerve injuries in children. *Ear Nose Throat J*, 1977. Sep; 56(9):369-76.
211. George S. Castration at birth. *Br Med J*, 1988. Nov; 297(6659):113-14.
212. Sokol DM, Tompkins D, Izant RJ Jr, Rupture of the spleen and liver in the newborn. *Pediatr Surg*, 1974. Apr; 9(2) 227-9.
213. Sezen F, Retinal haemorrhages in newborn infants. *Br J Ophthalmol*, 1970. Apr; 55(4):248-53.
214. Maltau JM, Egge K, Moe N. Retinal hemorrhages in the preterm neonate. A prospective randomized study comparing the occurrence of hemorrhages after spontaneous versus forceps delivery. *Acta Obstet Gynecol Scand*, 1984; 63(3):219-21.
215. Schenker JG, Gombos GM. Retinal hemorrhage in the newborn. *Obstet Gynecol*, 1966. Apr; 27(4):521-24.
216. O'Grady J, *Modern Instrumental Delivery*. 1988, Baltimore: Williams & Wilkins.
217. Zbar RI, Smith RJ. Vocal fold paralysis in infants twelve months of age and younger. *Otolaryngol Head Neck Surg*, 1996. Jan; 114(1):8-21.
218. Towbin A, Turner GL. Obstetric factors in fetal-neonatal visceral injury. *Obstet Gynecol*, 1978. Jul; 52(1):113-24.
219. Gentile RD, Miller RH, Woodson GE. Vocal cord paralysis in children 1 year of age and younger. *Ann Otol Rhinol Laryngol*, 1986. Nov-Dec; 95(6 Pt 1):622-5.
220. Mollberg M, Hagberg H, Bager B, Iilja H, Ladfors L, Risk factors for obstetric brachial plexus palsy among neonates delivered by vacuum extraction. *Obstet Gynecol*, 2005. Nov; 106(5 Pt 1): 913-8.
221. Lerner H, Hamilton E. Shoulder dystocia: What if you could see it coming. *Contemp OB/GYN*, 2007. Nov; 52(11):61-70.
222. McFarland L, Raskin M, Daling FR, Benedetti TJ, Erbs/Duchenne's palsy: A consequence of fetal macrosomia and method of delivery. *Obstet Gynecol*, 1986. Dec; 68(6):784-8.
223. Benedetti T, Gabbe SC, Shoulder dystocia: A complication of fetal macrosomia and prolonged second stage of labor with midpelvic delivery. *Obstet Gynecol*, 1978. Nov; 52(5):526-9.
224. Gordon M, Rich M, Deutschberger J, Green M, The immediate and longterm outcome of obstetric birth trauma. I. Brachial plexus paralysis. *Am J Obstet Gynecol*, 1973. Sept; 117(1):51-6.
225. Jennett R, Tarby TJ, Kreinick MA, Brachial plexus palsy: An old problem revisited. *Am J Obstet Gynecol*, 1992. Jun; 166(6 Pt 1):1673-7.
226. Haenggeli CA, Lacourt G. Brachial plexus injury and hypoglossal paralysis. *Pediatr Neurol*, 1989. May-Jun; 5(3):197-8.
227. Gherman R, Ouzounian JG, Goodwin TM, Obstetric maneuvers for shoulder dystocia and associated fetal morbidity. *Am J Obstet Gynecol*, 1998. Jun; 178(6):1126-30.
228. O'Leary JCA, Abdominal rescue after failed cephalic replacement. *Obstet Gynecol*, 1992. Sept; 80(3 Pt 2):514-16.
229. Graham J, Blanco JD, Wen T, Magee KP, The Zavanelli maneuver: A different perspective. *Obstet Gynecol*, 1992. May; 79(5 Pt 1):883-4.
230. Rouse DJ, Owen J, Goldenberg RL, Cliver SP, The effectiveness and costs of elective cesarean delivery for fetal macrosomia diagnosed by ultrasound. *JAMA*, 1996. Nov 13; 276(18):1480-6.
231. Wigglesworth, JS, Singer DB, *Textbook of Fetal and Perinatal Pathology*. 1991, Boston: Blackwell Scientific Publications, p 300.

232. Kellum RE, Ray TL, Breum GR, Sclerema neonatarum. Report of a case of subcutaneous and epidermal-dermal lipids by chromatographic methods. *Arch Dermatol*, 1968. Apr; 97(4):372–80.
233. Hanigan WC, Prenatal magnetic resonance imaging detection of a vein of Galen aneurysm. *Child's Nervous System*, 1993. Nov; 9(7):377–78.
234. Govaert P, Vanhaesebrouck P, De Praeter C, Traumatic neonatal intracranial bleeding and stroke. *Arch Dis Child*, 1992. Jul; 67(7 Spec No):840–45.
235. Hernansanz J, Munoz F, Rodriguez D, et al, Subdural hematomas of the posterior fossa in normal-weight newborns. Report of two cases. *J Neurosurg*, 1984. Nov; 61(5):972–74.
236. Romodanov AP, Brodsky YS, Subdural hematomas in the newborn: Surgical treatment and results. *Surg Neurol*, 1987. Oct; 28(4):253–58.
237. perinat. <http://home.mdconsult.com/da/book/body/384927164/1209/232>.
238. O'Grady JP, Gimovsky ML, McIlhargie CJ, Vacuum Extraction in Modern Obstetric Practice. New York: The Parthenon Publishing Group, 1995.
239. Govaert P, Defoort P, Wigglesworth JS, Cranial haemorrhage in the term newborn infant. *Clin Dev Med*, 1993. 129:1–223.
240. Holland E, Cranial stress in the foetus during labour and on the effects of excessive stress on the intracranial contents: With an analysis of eighty-one cases of torn tentorium cerebelli and subdural cerebral hemorrhage. *J Obstet Gynaecol Br Emp*, 1922. 29:551–71.
241. Thorngren-Jerneck K, Herbst A, Low 5-minute Apgar score: A population-based register study of 1 million term births. *Obstet Gynecol*, 2001. Jul; 98(1):65–70.
242. Wu Y, Escobar GJ, Grether JD, Croen LA, Greene JD, Newman TB, Chorioamnionitis and cerebral palsy in term and near-term infants. *JAMA*, 2003. Nov 26; 290(20):2677–84.
243. Pharoah P, Risk of cerebral palsy in multiple pregnancies. *Obstet Gynecol Clin North Am*, 2005. Mar; 32(1):55–67.
244. Hagberg H, Mallard C, Jacobsson B, Role of cytokines in preterm labour and brain injury. *Br J Obstet Gynaecol*, 2005. Mar; 112(Suppl 1):16–8.
245. Kendall G, Peebles D, Acute fetal hypoxia: The modulating effect of infection. *Early Hum Dev*, 2005. Jan; 81(1):27–34.
246. Garnier Y, Coumans AB, Jensen A, Hasaart TH, Berger R, Infection-related perinatal brain injury: The pathogenic role of impaired fetal cardiovascular control. *J Soc Gynecol Invest*, 2003. Dec; 10(8):450–9.
247. Hankins GD, Koen S, Gei AF, Lopez SM, van Hook JW, Anderson GD, Neonatal organ system injury in acute birth asphyxia sufficient to result in neonatal encephalopathy. *Obstet Gynecol*, 2002. May; 99(5 Pt 1):688–91.
248. Task Force Neonatal Encephalopathy and Cerebral Palsy, Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology. 2003, American College of Obstetricians and Gynecologists and the American Academy of Pediatrics: Washington, DC.
249. Annibale D, Periventricular hemorrhage–intraventricular hemorrhage. [www.imedicine.com; topic = 2595](http://www.imedicine.com/topic=2595), 2003.
250. Calvert J, Zhang JH, Pathophysiology of an hypoxic-ischemic insult during the perinatal period. *Neurol Res*, 2005. Apr; 27(3):246–60.
251. Zeldin AS, Ratanawongsa B, Cerebral palsy. [www.eMedicine.com.topic = 533](http://www.eMedicine.com/topic=533), 2007.
252. Felderhoff-Mueser U, Buhner C, Clinical measures to preserve cerebral integrity in preterm infants. *Early Hum Dev*, 2005. Mar; 81(3):237–44.
253. Schifrin B, Hamilton-Rubinstein T, Shields JR, Fetal heart rate patterns and the timing of fetal injury. *J Perinatol* 1994. May–Jun; 14(3):174–81.
254. Neilson J, Fetal electrocardiogram (ECG) for fetal monitoring during labour (Cochrane Review). *Cochrane Database Syst Rev*, 2003.
255. Nielsen P, Stigsby B, Nickelsen C, Nim J, Intra- and inter-observer variability in the assessment of intrapartum cardiocograms. *Acta Obstet Gynecol Scand*, 1987. 66(5):421–4.
256. Beaulier M, Fabia J, Leduc B, Brisson J, Bastide A, Blouin D, Gauthier RJ, Lalonde A, The reproducibility of intrapartum cardiocogram assessments. *Can Med Assoc J*, 1982. Aug 1; 127(3): 214–6.
257. Helfand M, Marton K, Ueland K, Factors involved in the interpretation of fetal monitor tracings. *Am J Obstet Gynecol*, 1985. 151:737–44.
258. Williams K, Galerneau F, Intrapartum fetal heart rate patterns in the prediction of neonatal



- acidemia. *Am J Obstet Gynecol*, 2003. Mar; 188(3):820-3.
259. Meyers R, Two patterns of perinatal brain damage and their conditions of occurrence. *Am J Obstet Gynecol*, 1972. 112:246-76.
  260. Whitten M, Irvine LM, Postmortem and perimortem caesarean section. *J R Soc Med*, 2000. Jan; 93(1):6-9.
  261. Katz MV, Dotters DJ, Droegemueller W, Perimortem cesarean delivery. *Obstet Gynecol*, 1986. Oct; 68(4):571-6.
  262. Thomas R, Sotheran W, Postmortem and perimortem caesarean section. *J R Soc Med*, 2000. Apr; 93(4):215-6.
  263. Lopez-Zeno J, Carlo WA, O'Grady JP, Fanaroff AA, Infant survival following delayed postmortem caesarean delivery. *Obstet Gynecol*, 1990. Nov; 76(5 Pt 2):991-2.
  264. Phelan J, Martin GI, Korst LM, Birth asphyxia and cerebral palsy. *Clin Perinatol*, 2005. Mar; 31(1):1-17.
  265. Jones KL, *Recognizable Patterns of Human Malformation*, ed 6. 2006, Philadelphia, Elsevier.
  266. Sharpe RM, Irvine DS, How strong is the evidence of a link between environmental chemicals and adverse effects on human reproductive health? *BMJ*, 2004. Feb; 328:447-51.
  267. Doblhammer G, Vaupel JW, Lifespan depends on month of birth. *PNAS*, 2001. Feb 27; 98:2934-39.
  268. Palinski W, Napoli C, The fetal origins of atherosclerosis: Maternal hypercholesterolemia and cholesterol-lowering or antioxidant treatment during pregnancy influence in utero programming and postnatal susceptibility to atherogenesis. *FASEB J*, 2002. Sep; 16(11):1348-60.
  269. Gluckman PD, Hanson MA, Living with the past: Evolution, development, and patterns of disease. *Science* 305, 2004:1733-6.
  270. Barker DJP, Lackland DT, Prenatal influences on stroke mortality in England and Wales. *Stroke*, 2003. Jul; 34(7):1598-602.
  271. Shaper AG, Pocock SJ, Walker M, Phillips AN, Whitehead TP, Macfarlane PW. Risk factors for ischaemic heart disease: The prospective phase of the British Regional Heart Study. *J Epidemiol Community Health*, 1985. Sep; 39(3):197-209.
  272. Desai M, Crowther NJ, Ozanne SE, Lucas A, Hales CN, Adult glucose and lipid metabolism may be programmed during fetal life. *Biochem Soc Trans*, 1995. May; 23(2):331-5.
  273. Lawlor DA, Ronalds G, Clark H, Smith GD, Leon DA, Birth weight is inversely associated with incident coronary heart disease and stroke among individuals born in the 1950s: Findings from the Aberdeen Children of the 1950s prospective cohort study. *Circulation*, 2005. Sep 6; 112(10):1414-8.
  274. Barker DJ, Osmond C, Forsen TJ, Kajantie E, Eriksson JG, Maternal and social origins of hypertension. *Hypertension*, 2007. Sep; 50(3):565-71.
  275. Barker DJ, The origins of the developmental origins theory. *J Intern Med*, 2007. May; 261(5):412-7.
  276. Fleming TP, Kwong WY, Porter R, Ursell E, Fesenko I, Wilkins A, Miller DJ, Watkins AJ, Eckert JJ, The embryo and its future. *Biol Reprod*, 2004. Oct; 71(4):1046-54.
  277. Joseph KS, Kramer MS, Review of the evidence on fetal and early childhood antecedents of adult chronic disease. *Epidemiol Rev*, 1996. 18(2):158-74.
  278. Barker DJ, Fetal programming of coronary heart disease. *Trends Endocrinol Metab*, 2002. Nov; 13(9):364-8.
  279. Lev-Ran A, Human obesity: An evolutionary approach to understanding our bulging waistline. *Diabetes Metab Res Rev*, 2001. Sep-Oct; 17(5):347-62.
  280. American College of Obstetricians and Gynecologists, Educational Bulletin. Number 251, September, 1998. *Obstetric Aspects of Trauma Management*.
  281. Donaldson JO, *Neurology of Pregnancy*. Philadelphia: WB Saunders, 1989.
  282. Connolly AM, Katz VL, Bash KL, McMahan MJ, Hansen WF, Trauma and pregnancy. *Am J Perinatol*, 1997. Jul; 14(6):331-36.
  283. Rogers FB, Rozycki GS, Osler TM, Shackford SR, Jalbert J, Kirton O, Scalea T, Morris J, Ross S, Cipolle M, Fildes J, Cogbill T, Bergstein J, Clark D, Frankel H, Bell R, Gens D, Cullinane D, Kauder D, Bynoe RP, A multi-institutional study of factors associated with fetal death in injured pregnant patients. *Arch Surg*, 1999. Nov; 134(11):1274-7.
  284. Theodorou DA, Velmahos GC, Souter I, Chan LS, Vassiliu P, Tatevossian R, Murray JA, Demetriades D, Fetal death after trauma in pregnancy. *Am Surg*, 2000. Sep; 66(9):809-12.

285. Schiff MA, Holt VL, The injury severity score in pregnant trauma patients: Predicting placental abruption and fetal death. *J Trauma*, 2002. Nov; 53(5):946-9.
286. Ali J, Yeo A, Gana TJ, McLellan BA, Predictors of fetal mortality in pregnant trauma patients. *J Trauma*, 1997. May; 42(5):782-5.
287. Weiss HB, Songer TJ, Fabio A, Fetal deaths related to maternal injury. *JAMA*, 2001. Oct 17; 286(15):1863-8.
288. Poole GV, Martin JN Jr, Perry KG Jr, Griswold JA, Lambert CJ, Rhodes RS, Trauma in pregnancy: The role of interpersonal violence. *Am J Obstet Gynecol*, 1996. Jun; 174(6):1873-7.
289. Hyde LK, Cook LJ, Olson LM, Weiss HB, Dean JM, Effect of motor vehicle crashes on adverse fetal outcomes. *Obstet Gynecol*, 2003. Aug; 102(2): 279-86.
290. Ong BY, Cohen MM, Esmail A, Cumming M, Kozody R, Palahniuk RJ, Paresthesias and motor dysfunction after labor and delivery. *Anesth Analg*, 1987. Jan; 66(1):18-22.
291. Warner MA, Warner DO, Harper C, Schroeder DR, Maxson PM, Lower extremity neuropathies associated with lithotomy positions. *Anesthesiology*, 2000. 93(4):938-42.
292. Irvin W, Andersen W, Taylor P, Rice L, Minimizing the risk of neurologic injury in gynecologic surgery. *Obstet Gynecol*, 2004. Feb; 103(2):374-82.
293. Keegan JJ, Holyoke EA, Meralgia paresthetica: An anatomical and surgical study. *J Neurosurgery*, 1962. Apr; 19:341-45.
294. van Slobbe AM, Bohnen AM, Bernsen RM, Koes BW, Bierma-Zeinstra SM, Incidence rates and determinants in meralgia paresthetica in general practice. *J Neurol*, 2004. Mar; 251(3):294-97.
295. de Ridder VA, de Lange S, Popta JV, Anatomical variations of the lateral femoral cutaneous nerve and the consequences for surgery. *J Orthop Trauma*, 1999. 13(3):207-11.
296. Colachis SC 3rd, Pease WS, Johnson EW. A preventable cause of foot drop during childbirth. *Am J Obstet Gynecol*, 1994. 171:270-2.
297. Schnatz P, Wax JR, Steinfeld JD, Ingardia CJ, Meralgia paresthetica: An unusual complication of post-cesarean analgesia. *J Clin Anesth*, 1999. Aug; 11(5):416-18.
298. Hutchins FL Jr, Huggins J, Delaney ML, Laparoscopic myomectomy – an unusual cause of meralgia paresthetica. *J Am Assoc Gynecol Laparosc*, 1998. Aug; 5(3):309-11.
299. Siu TL, Chandran KN, Neurolysis for meralgia paresthetica: An operative series of 45 cases. *Surg Neurol*, 2005. Jan; 63(1):19-23.
300. al Hakim MA, Katirji B, Femoral mononeuropathy induced by the lithotomy position: A report of 5 cases with a review of literature. *Muscle Nerve*, 1993. Sep; 16(9):891-5.
301. Warfield CA, Obturator neuropathy after forceps delivery. *Obstet Gynecol*, 1984. 64(Suppl 3):47S-8S.
302. Wong CA, Scavone BM, Dugan S, Smith JC, Prather H, Ganchiff JN, McCarthy RJ, Incidence of postpartum lumbrosacral spine and lower extremity nerve injuries. *Obstet Gynecol*, 2003. 101(2):279-88.
303. Vargo MM, Robinson LR, Nicholas JJ, Rulin MC, Postpartum femoral neuropathy: Relic of an earlier era? *Arch Phys Med Rehabil*, 1990. Jul; 71(8): 591-6.
304. Aminoff MJ, Neurological disorders and pregnancy. *Am J Obstet Gynecol*, 1978. Oct; 132(3): 325-35.
305. Kornbluth I. Meralgia paresthetica. *eMedicine*. www.imedicine.com topic = 416, 2006.
306. Pearlman MD, Tintinalli JE, Lorenz RP, A prospective controlled study of outcome after trauma during pregnancy. *Am J Obstet Gynecol*, 1990. Jun; 162(6):1502-10.
307. Pearlman MD, Tintinalli JE, Evaluation and treatment of the gravida and fetus following trauma during pregnancy. *Obstet Gynecol Clin North Am*, 1991. Jun; 18(2):371-81.
308. Pearlman MD, Tintinalli JE, Lorenz RP, Blunt trauma during pregnancy. *N Engl J Med*, 1990. Dec 6; 323(23):1609-13.
309. Ducic I, Dellon AL, Taylor NS, Decompression of the lateral femoral cutaneous nerve in the treatment of meralgia paresthetica. *Reconstr Microsurg*, 2006. Feb; 22(2):113-8.
310. Azuelos A, Coro L, Alexandre A, Femoral nerve entrapment. *Acta Neurochir Suppl* 2005; 92: 61-2.
311. Adelman JU, Goldberg GS, Puckett JD, Postpartum bilateral femoral neuropathy. *Obstet Gynecol*, 1973. Dec; 42(6):845-50.
312. Dar AQ, Robinson A, Lyons G, Postpartum femoral neuropathy: More common than you think. *Anaesthesia*, 1999 May; 54(4):512.

313. Cohen S, Zada Y, Postpartum femoral neuropathy. *Anaesthesia*, 2001 May; 56(5):500–1.
314. Nogajski JH, Shnier RC, Zagami AS, Postpartum obturator neuropathy. *Neurology*, 2004 Dec 28; 63(12):2450–1.
315. Nahabedian MY, Dellon AI., Meralgia paresthetica: etiology, diagnosis, and outcome of surgical decompression. *Ann Plast Surg*, 1995 Dec; 35(6): 590–4.
316. Grossman MG, Ducey SA, Nadler SS, Meralgia Paresthetica: diagnosis and treatment. *J Am Acad Orthopaed Surg*, 2001. 9:336–44.
317. Colachis SC 3rd, Pease WS, Johnson EW, A preventable cause of foot drop during childbirth. *Am J Obstet Gynecol*, 1994. 171:270–2.
318. Adoranto BT, Carlini WC, "Pushing palsy": A case of self-induced bilateral peroneal palsy during natural child birth. *Neurology*, 1992. Apr; 42(4): 936–7.
319. Reif ME, Bilateral common peroneal nerve palsy secondary to prolonged squatting in natural childbirth. *Birth*, 1988. 15:100–2.
320. Babayev M, Bodack MP, Creatura C. Common peroneal neuropathy secondary to squatting during childbirth. *Obstet Gynecol*, 1998. May; 91(5 Pt 2):830–2.
321. Wilbourne AJ, Iatrogenic nerve injuries. *Neurol Clin*, 1998. 16:55–82.
322. Luzzio CC, Meralgia Paresthetica. *eMedicine (imedicine.com) topic = 76*, 2007.
323. Shah S, Miller PR, Meredith JW, Chang MC, Elevated admission white blood cell count in pregnant trauma patients: An indicator of ongoing placental abruption. *Am Surg*, 2002. Jul; 68(7):644–7.
324. Schiff MA, Holt VL, Pregnancy outcomes following hospitalization for motor vehicle crashes in Washington State from 1989 to 2001. *Am J Epidemiol*, 2005. Mar 15; 161(6):503–10.
325. Kady D, Gilbert WM, Anderson J, Danielsen B, Towner D, Smith LH, Trauma during pregnancy: An analysis of maternal and fetal outcomes in a large population. *Am J Obstet Gynecol*, 2004. Jun; 190(6):1661–8.
326. Curet MJ, Schermer CR, Demarest GB, Bieneik EJ 3rd, Curet LB, Predictors of outcome in trauma during pregnancy: Identification of patients who can be monitored for less than 6 hours. *J Trauma*, 2000. Jul; 49(1):18–24.
327. Sekul A, Meralgia Paresthetica *emedicine imedicine.com. topic = 590*, 2007.
328. Kuo LJ, Penn IW, Feng SF, Chen CM, Femoral neuropathy after pelvic surgery. *J Chin Med Assoc*, 2004. Dec; 67(12):644–6.
329. Huang WS, On PY, Yeh CH, Chin CC, Hsieh CC, Wang JY, Iatrogenic femoral neuropathy following pelvic surgery: A rare and often overlooked complication – four case reports and literature review. *Chang Gung Med J*, 2007. Jul–Aug; 30(4): 374–9.
330. Fanning J, Carol T, Miller D, Flora R, Postoperative femoral motor neuropathy: Diagnosis and treatment without neurologic consultation of testing. *J Reprod Med*, 2007. Apr; 52(4):285–8.
331. Celebrezze JP Jr, Pidala MJ, Porter JA, Slezak FA, Femoral neuropathy: An infrequently reported postoperative complication. Report of four cases. *Dis Colon Rectum*, 2000. Mar; 43(3):419–22.
332. Brasch RC, Bufo AJ, Kreienberg PF, Johnson GP, Femoral neuropathy secondary to the use of self-retaining retractor. *Dis Colon Rectum*, 1995. 38:1115–8.
333. Roblee MA, Femoral neuropathy from the lithotomy position: Case report and new leg holder for prevention. *Am J Obstet Gynecol*, 1967. 97: 871–2.
334. Chen SS, Lin AT, Chen KK, Chang LS, Femoral neuropathy after pelvic surgery. *Urology*, 1995. Oct; 46(4):575–6.
335. Dar AQ, Roginson APC, Lyons G, Postpartum neurologic symptoms following regional blockade: A prospective study with case controls. *Int J Obstet Anesth*, 2002. 11:85–90.
336. Simonson C, Barlow P, Dehennin N, Sphel M, Toppet V, Murillo D, Rosenberg S, Neonatal complications of vacuum-assisted delivery. *Obstet Gynecol*. Mar 2007; 109(3):626–33.

## Chapter 24 MIDWIVES AND OPERATIVE OBSTETRICS

### Lisa Summers

*There is nothing more difficult to take in hand, more perilous to conduct, or more uncertain of success, than to take the lead in the introduction of a new order of things.*

Niccolo Machiavelli (1469–1527)

*Il Principe* (The Prince), 1513

W.A. Rebhorn (trans.)

New York: Barnes & Noble Classics, p. 25.

### MIDWIVES IN HIGH-RISK OBSTETRICS

From early on, midwifery care in this country usually has been described as care of “normal, healthy” women. Midwifery education and practice have focused on healthy women and normal birth. The reality, however, is that professional midwifery established itself in this country by providing care to “vulnerable populations” – women who lived in areas where little health care was available: the “hollers” of Appalachia, Indian reservations, the inner cities, the barrios, and border towns. Out of necessity, therefore, midwives have had to develop a degree of expertise in caring for “at-risk” women and gain skill with procedures such as those addressed in this text.

Midwives have taken on procedures such as first assisting and vacuum extraction as a means to expand access to care, particularly in rural and underserved areas, places where there is no doctor.

Colposcopy and ultrasonography are additional good examples of procedures midwives have learned to perform to expand access to needed services: if the midwife can follow up on the abnormal Pap test result or perform a biophysical profile (BPP) without having to book another appointment or make a referral in a low-resource setting, the client is more likely to obtain the necessary care.

This chapter focuses on the practice of midwives with regard to operative obstetrics. It should be noted that the term *midwifery* as used herein refers to the education and practice of *certified nurse-midwives* (CNMs) and *certified midwives* (CMs) who have been certified by the American College of Nurse-Midwives (ACNM) or the American Midwifery Certification Board, Inc. (AMCB), formerly the American College of Nurse-Midwives Certification Council, Inc. (ACC).

## **HISTORICAL BACKGROUND: THE AMERICAN COLLEGE OF NURSE-MIDWIVES**

Just as the American College of Obstetricians and Gynecologists (ACOG) is critical to the development of standards for obstetric practice among physicians, the ACNM is critical to the development of standards in midwifery. ACNM, the oldest women's health care organization in the United States, accredits midwifery education programs, administers and promotes continuing education programs, establishes clinical practice standards, and creates liaisons with state and federal agencies and members of Congress. The mission of ACNM is to promote the health and well-being of women and infants within their families and communities through the development and support of the profession of midwifery as practiced by certified nurse-midwives and certified midwives.

ACNM defines a CNM as "an individual educated in the two disciplines of nursing and midwifery, who possesses evidence of certification according to the requirements of ACNM." In recent years, ACNM has established a mechanism for people to become certified as a midwife without a nursing credential – often referred to as *direct entry*. These graduates must pass the same certifying examination and earn the CM title.

The U.S. Department of Education has recognized the ACNM Division of Accreditation as an accrediting agency for midwifery and nurse-midwifery education programs. All of the 43 accredited programs are associated with an institution of higher learning. Most midwifery education programs are found in academic medical centers having medical schools and obstetric residency programs (e.g., Yale, Emory, Columbia, University of California San Francisco, University of Michigan), providing a valuable opportunity for interdisciplinary teaching and learning [2]. (For a current listing of accredited midwifery education programs see the ACNM website [[www.midwife.org/about.cfm](http://www.midwife.org/about.cfm)].)

CNMs are licensed in all 50 states and the District of Columbia. In most states, CNM practice is regulated by the board of nursing, although the regulatory agency is sometimes a board of medicine, a public health board, or a midwifery board.

Physicians might become aware of other midwives who are neither CNMs nor CMs practicing in their state. Laws and regulations vary greatly,

but a growing number of states have recognized the certified professional midwife (CPM) credential as well as other forms of direct-entry midwifery, or as it is sometimes called, *lay midwifery*. ACOG has published a position statement that "recognizes the educational and professional standards currently used by the American Midwifery Certification Board (AMCB) to evaluate and certify midwives," and goes on to state that "while ACOG supports women having a choice in determining their providers of care, ACOG does not support the provision of care by lay midwives or other midwives who are not certified by the AMCB."

Today there are about 7000 CNMs/CMs practicing in the United States, attending just over 10% of all vaginal births [3]. The majority of CNM/CM-attended births (97% according to the most recent birth certificate data) occur in hospitals, with a small percentage of births occurring in birth centers and the home.

### **Physician Assistants**

According to the American Academy of Physician Assistants, *physician assistants* (PAs) are health professionals licensed to practice medicine with physician supervision. PAs are trained in educational programs accredited by the Accreditation Review Commission on Education for the Physician Assistant, and on graduation PAs take a national certification examination developed by the National Commission on Certification of Physician Assistants in conjunction with the National Board of Medical Examiners. To maintain the national certification, PAs must log 100 hours of continuing medical education every two years and sit for recertification every six years. Graduation from an accredited PA program and passage of the national certifying examination are required for state licensure.

PAs conduct physical examinations, diagnose and treat illnesses, order and interpret tests, counsel on preventive care, assist in surgery, and in most states may write prescriptions. Their training is based on the medical model and designed to complement physician training. Women's health and obstetrics are routine components of PA education, but the degree to which individual PAs provide obstetric care varies greatly, depending on physician preference and the training and expertise of the PA.

State laws also play a role in defining PA scope of practice, although most state rules and regulations do not address PAs and deliveries. Only four states delineate the specific context of PA participation in obstetric care. (See the AAPA's Summary of State Law References to PA Participation in Obstetrical Care and Deliveries.) Hospital regulations are likely to provide a much greater influence over the scope of obstetric practice of PAs and the degree to which they may perform operative obstetric procedures.

### Range of Procedures

Physicians who collaborate with midwives are sometimes puzzled, confused, or surprised by the apparent inconsistencies of practice from one midwife to another. A physician in an academic medical center might train with midwives who limit their case load to essentially healthy women, referring women with seemingly inconsequential risk factors, and then go into practice with midwives who manage diabetic patients and assist births with vacuum devices. A brief overview of midwifery scope of practice makes clear why these variations in practice exist.

### Core Competencies

In 1978, midwifery educators first defined *core competencies*, the fundamental knowledge, skills, and behaviors expected of a newly graduated midwife [4]. The document, revised four times and now titled *Core Competencies for Basic Midwifery Practice*, continues to serve as the template for curricula in midwifery education programs accredited by the ACNM Division of Accreditation (DOA) [5]. Because the document serves to define the scope of basic midwifery practice, it is also a guideline for other health care professionals and policy makers.

Although the language has changed somewhat over the years, the important themes have remained constant. What is called the *midwifery management process* – the systematic collection of data, problem identification, and development of a plan of care – is much the same process taught to physicians and is the underpinning of the specific competencies outlined for each clinical component of care (i.e., antepartum, neonatal, perimenopause, and postmenopause). The concept of collaborative management is the framework for those health care problems that fall out of the range of the “essentially

**TABLE 24.1** Hallmarks of Midwifery

The art and science of midwifery are characterized by these hallmarks:

1. Recognition of pregnancy, birth, and menopause as normal physiologic and developmental processes
2. Advocacy of nonintervention in the absence of complications
3. Incorporation of scientific evidence into clinical practice
4. Promotion of family-centered care
5. Empowerment of women as partners in health care
6. Facilitation of healthy family and interpersonal relationships
7. Promotion of continuity of care
8. Health promotion, disease prevention, and health education
9. Promotion of a public health care perspective
10. Care to vulnerable populations
11. Advocacy for informed choice, shared decision making, and the right to self-determination
12. Cultural competence
13. Familiarity with common complementary and alternative therapies
14. Skillful communication, guidance, and counseling
15. Therapeutic value of human presence
16. Collaboration with other members of the health care team

healthy” and has been described in the core competencies and other standard-setting documents. A set of professional responsibilities for midwives is also articulated.

The initial document was developed in part because educators needed to describe the distinct discipline of midwifery, yet there had long been an acknowledgement of the body of knowledge drawn from medicine, nursing, social science, and public health. ACNM made explicit those distinctions in the 1997 revision of the core competencies by adding the Hallmarks of Midwifery (Table 24.1) [5,6]. Midwives who can clearly articulate the art and science of midwifery as defined by the hallmarks can often help other health care providers to better understand their clinical and administrative decision making.

The evolution of the core competencies reflects the changes over time in women's health care, the health care industry, and the role of CNMs/CMs in the United States. For instance, the 1997 revision

includes a section that explicitly acknowledges the role of midwives in primary care of women and describes the management of common health problems [6]. “Familiarity with practice management and finances” was recognized as an essential requirement for professional survival. The ability to “evaluate, apply, interpret, and collaborate in research” was added, as “evidence-based care” became an increasingly important part of the vocabulary of health care.

The current document can be found on the ACNM Web site [[www.midwife.org/display.cfm?id=484](http://www.midwife.org/display.cfm?id=484)].

### ***Expanded Practice***

The core competencies describe *basic* midwifery practice – the knowledge and skills expected of all CNMs/CMs. Particularly relevant to this chapter is what is known among midwives as “expanded practice,” those procedures or components of practice such as first assisting, use of a vacuum extractor, or performing circumcision, which can be acquired beyond basic midwifery. An understanding of this concept is made easier by a brief review of the history of documents that guide the expansion of practice.

The ACNM first grappled with the question of expanded scope of practice in the late 1960s, with the issue of abortion. Abortion reform legislation had been passed in New York (home of many CNMs), and obstetric services and family planning clinics, anticipating a dramatic increase in the number of abortions, were planning how services would be provided and by whom. Would midwives be abortion providers? When midwives turned to the ACNM for guidance, the question was referred to the Clinical Practice Committee, whose members set out to develop guidelines to address which “extensions” of midwifery practice were appropriate.

The committee considered developing laundry lists of procedures deemed appropriate (or not). Realizing, however, that practice would evolve, the committee chose instead to develop guidelines that would allow midwives to use their judgment, in their particular clinical setting, to determine which expansion of practice might be appropriate. The guidelines were approved in 1972 as the *Standards for the Evaluation of Nurse-Midwifery Procedural Functions* and were later revised slightly to become

**TABLE 24.2 American College of Nurse-Midwives  
Standard VIII\***

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The midwife

1. Identifies the need for a new procedure, taking into consideration consumer demand, standards for safe practice, and availability of other qualified personnel.
  2. Ensures that there are no institutional, state, or federal statutes, regulations, or bylaws that would constrain the midwife from incorporation of the procedure into practice.
  3. Demonstrates knowledge and competency, including
    - a. Knowledge of risks, benefits, and client selection criteria.
    - b. Process for acquisition of required skills.
    - c. Identification and management of complications.
    - d. Process to evaluate outcomes and maintain competency.
  4. Identifies a mechanism for obtaining medical consultation, collaboration, and referral related to this procedure.
  5. Reports the incorporation of this procedure to the ACNM.
- 

\*Midwifery practice may be expanded beyond the ACNM core competencies to incorporate new procedures that improve care for women and their families.

the *Guidelines for the Incorporation of New Procedures into Nurse-Midwifery Practice*. With the 2003 revision of the *ACNM Standards for the Practice of Midwifery*, the guidelines were retired and incorporated into Standard VIII. The current Standards are available on the ACNM Web site; Standard VIII is found in Table 24.2.

As is true with medical education, there are some variations in the clinical experiences provided from one program to another, and there are some gray areas as practice evolves. For instance, midwives educated in the years when intrauterine devices (IUDs) were off the market received didactic training and used clinical models but were unable to develop clinical experience in IUD insertion. The core competencies clearly include the performance of episiotomy and repair with the administration of local anesthesia; however, the use of a vacuum device is clearly an expanded practice procedure.

### **TRAINING AND CREDENTIALING**

To ensure patient safety, the standards mandate a process for acquisition of required skills as well

as a process to evaluate outcomes and maintain competency. They do not specify precisely what that process is for each expanded practice procedure or skill, although handbooks and continuing education programs provided by ACNM do provide guidance, as described here.

### CLINICAL PRIVILEGING

ACNM recommends that institutional guidelines, which govern the initial granting and renewal of clinical privileges for CNMs/CMs, clearly differentiate privileges granted for basic midwifery practice by all CNMs/CMs and those that might be requested by some CNMs/CMs for selected expanded practice procedures [10]. Recently, concern about the need to document current competence has led many institutions to develop criteria for a minimum number of procedures required for renewal of privileges, and CNMs/CMs are being affected by these criteria just as physicians are. To date, the ACNM has not taken a position on a minimum number of births, vacuum-assisted deliveries, or any other procedures that are required to maintain competence.

### IMPORTANCE OF STATE LAWS AND REGULATIONS

The standards direct the midwife to consider relevant statutes and regulations that might constrain the midwife from incorporation of a particular procedure. As a practical matter, state law often looks to national standards developed by the profession when defining scope of practice. Many state laws and regulations governing midwifery refer to ACNM documents, thereby permitting advanced practice procedures consistent with the guidelines. ACNM publishes *Nurse-Midwifery Today: A Handbook of State Laws and Regulations*, a resource that is regularly updated and available on the ACNM website. A policy analyst on the ACNM staff is also available to assist with interpretation of state laws and regulations.

### PROFESSIONAL LIABILITY CONCERNS

ACNM defines midwifery practice as “the independent management of women’s health care, focusing on pregnancy, childbirth, the postpartum period, care of the newborn, and the family planning and

gynecological needs of women.” Although some physicians object to the establishment of an “independent” scope of practice of any provider not a physician, obstetrician/gynecologists who have worked closely and effectively with midwives over the years understand the importance of clearly defining a separate scope of practice. Requirements for supervision of care to healthy women are not only unnecessary and duplicative but they are also not cost-effective. Perhaps the most compelling argument to avoid “supervision and direction” language is today’s litigious environment and concerns about professional liability; such language places the physician in an unfair and undeserved position with regard to liability.

*Independent* should not, however, be interpreted to mean *alone*, because there are clinical situations when any prudent practitioner would seek the assistance of another qualified practitioner. Crucial to the definition of midwifery practice is the further statement that the CNM/CM practices “within a healthcare system that provides for consultation, collaborative management, or referral as indicated by the health status of the client.” When the midwife is not independently managing the care of a client, it is critical – to provide effective care and to avoid liability – that all parties be fully aware (and that the chart clearly reflects) who is responsible for the management of care.

Responsibility begins with the midwife as she or he assesses the patient: does the patient’s health status fall within the scope of his/her midwifery practice? If not, the degree to which the CNM/CM continues to be involved in the care can vary greatly depending on the clinical setting and the skills and expertise of the midwife and of the consulting physician. A patient from a rural birth center practice might be quickly transferred to medical management, whereas the same patient might be collaboratively managed in a tertiary care center. Although ACNM does not provide standard guidelines for client selection, nor specific lists of diagnoses that merit collaborative management or referral, ACNM has published definitions for these patterns of care:

- *Consultation* is the process whereby a CNM or CM who maintains primary management responsibility for the woman’s care seeks the advice or opinion of a physician or another member



of the health care team. Although many of the CNM/CM consultations are with an obstetrician/gynecologist, midwives (just like their physician colleagues) may consult with a dermatologist about a suspicious skin lesion or with a cardiologist to evaluate a murmur, for example.

- *Collaboration* is the process whereby a CNM or CM and physician jointly manage the care of a woman or newborn who has become medically, gynecologically, or obstetrically complicated. The scope of collaboration may encompass the physical care of the client, including delivery, by the CNM or CM, according to a mutually agreed-upon plan of care. When the physician must assume a dominant role in the care of the client due to increased risk status, the CNM or CM may continue to participate in physical care, counseling, guidance, teaching, and support. Effective communication between the CNM or CM and physician is essential for ongoing collaborative management.
- *Referral* is the process by which the CNM or CM directs the client to a physician or another health-care professional for management of a particular problem or aspect of the client's care.

From a risk management perspective, all providers must communicate effectively about their respective responsibilities when they are jointly involved in the care of a patient. The consultant or referral physician should be clear about the precise role he or she is asked to assume. The nurse who is carrying out the plan as ordered should also be clear about who is writing orders and who should be notified of changes in patient status. Last but not least, the client and her family should be clear about who is managing care. Anyone who reviews the chart later (e.g., the quality assurance committee or attorney) should have an equally clear picture of the process of care.

## FIRST ASSISTING

First assisting is one of the most common expanded practice skills of interest to midwives. When a midwife is caring for a patient in labor and a problem arises that necessitates cesarean delivery, the midwife's ability to first assist can significantly shorten "decision-to-incision" time. Although cesarean delivery is the most common operation for

which midwives first assist, many CNMs/CMs also first assist for gynecologic surgery.

In addition to patient care incentives, there are practice management incentives for first assisting by midwives. In some practice settings, the ability of the midwife to first assist avoids the need for a second physician to leave the office or come in for surgery. First assisting is a billable service for the midwife, assuming of course that the midwife is appropriately credentialed.

ACNM publishes *The Midwife as First Assistant*, a handbook that is used as the text for ACNM-sponsored workshops on first assisting. Midwives can also use the handbook to guide themselves through an individualized plan of study to gain the necessary knowledge for first assisting. This document may be found at: [[www.shopacnm.com/clinical](http://www.shopacnm.com/clinical)].

Clinical training is key to becoming a competent first assistant, and physicians very often serve as mentors or preceptors for that clinical training. The physician who mentors the novice CNM first assistant should actively participate in the education, training, and evaluation of the first assistant. The ACNM handbook includes tools that can be used to document training and the attainment of clinical competency.

The basic education and training of the CNM/CM includes many components of preoperative care (i.e., history and physical examination, obtaining informed consent), perioperative care, and postoperative care (i.e., pain relief, postoperative assessment). The degree to which the midwife provides pre- and postoperative care varies with the setting and sometimes with the individual case. Clear communication about these roles is essential.

The obstetrician/gynecologist should train the midwife first assistant in skills that foster active participation in cases; this becomes especially important when a case is unexpectedly challenging or complications arise. The midwife first assistant should be knowledgeable in the entire procedure for cesarean delivery and should be trained in the skills necessary to complete the delivery of the infant and secure hemostasis of the uterus should the surgeon have difficulty or become unable to continue the case. This is especially important in the small community hospital where another surgeon might not be immediately available.

Midwife first assistant training includes the pathophysiology, assessment, treatment, and sequelae

of potential complications related to obstetric/gynecologic surgery. Appropriate collaboration, risk management, and documentation practices must be included. As a part of ongoing quality assurance, the midwife first assistant is expected to maintain a log of all surgical assist cases.

### VACUUM-ASSISTED BIRTH

Any birth attendant who has listened to a prolonged second-stage bradycardia and desperately urged an exhausted mother to maximize her bearing-down efforts can easily understand the appeal of being able to assist delivery with a vacuum device. Some CNMs/CMs, particularly those who practice in rural settings or community hospitals where they might be the only obstetric provider in house, have chosen to expand their practice to include vacuum-assisted birth.

As with first assisting, the ACNM has produced a handbook, *Vacuum-assisted Birth in Midwifery Practice*, and offers a workshop to provide didactic knowledge and training with models. After the workshop, midwives are expected to establish program of supervised practice appropriate to their clinical setting. This document may be found at: [[www.shopacnm.com/clinical](http://www.shopacnm.com/clinical)].

The motive behind offering the handbook and workshop is not to encourage midwives to attain skills in vacuum-assisted birth but to underscore the need for careful training beyond basic midwifery education for those who undertake the acquisition of this skill. The handbook lists prerequisite knowledge and skills, such as “expert skill in abdominal and pelvic exam,” and stresses that, “assisting birth with a vacuum device is an advanced practice skill, and carries with it the risk of serious complications and medicolegal liability.” Midwives are cautioned not to embark on the training program unless they are completely confident that they possess the prerequisite knowledge and skills.

In Great Britain, midwives who are trained in the use of vacuum extraction devices are called *midwife ventouse practitioners* (MVPs). The following quote from a ventouse midwife is useful to midwives as they consider their motivation to take on this skill [8]:

**Locally, prospective MVPs are interviewed by midwifery managers and selected as much for attitude as for knowledge and clinical skill.**

**Hopefully, this selection of midwives by midwives will ensure that the right people are chosen. We do not need midwives who are by nature interventionists . . . . We should admire the ventouse midwife, who, instead of arriving the birthing room in a blaze of glory, enters quietly and observes the situation, maybe suggesting a change of position. I feel a great sense of success when I realize my skills are not going to be needed. I love the buzz of leaving a room knowing a woman has done it herself. If I ever walk out with a bigger buzz because I have performed an assisted delivery, then it will be time to hang up the suction cup. . . .**

In some clinical settings in the United States, an experienced ventouse midwife will train another, but in many settings, it is a physician who serves as the clinical preceptor. The physician must have a thorough understanding of the midwife’s requisite skills and provide appropriate supervision throughout the process.

Physicians called on by the midwife for an assisted delivery have occasionally provided spur-of-the-moment training to the midwife. The importance of avoiding a “see one, do one, teach one” approach to vacuum-assisted delivery cannot be overemphasized. It is equally important to respect the fact that some midwives will not choose to take on this skill, even after many years of experience.

Just as physicians do, the midwife must carefully weigh the benefits of vacuum-assisted birth against the serious risks associated with the device and choose carefully those cases for which its use is appropriate. For example, the ACNM warns, “with the possible exception of rare emergency situations during which preparations for operative delivery are being made, midwives should limit their use of the vacuum device to outlet or low pelvic procedures.” Clinical practice guidelines should clearly outline those clinical situations in which the midwife may perform a vacuum-assisted birth.

Midwives are cautioned to ensure that privileging bodies and insurers understand when a midwife is providing vacuum-assisted birth services. The scope of the privileges in the hospital must clearly include the use of a vacuum device. Ideally, the hospital committees that monitor morbidity and mortality and performance improvement activities should include a midwife or, at a minimum, have a midwife available when cases that include a midwife

are reviewed. For midwives, who might be likely to use a vacuum device less frequently than physicians, monitoring of outcome statistics and periodic reevaluation is particularly important. Because the CPT codes define vacuum extraction as an “integral component” of vaginal delivery, and midwives are credentialed by most payers to provide delivery services, the fact that a vacuum device was used does not typically present a billing problem. With regard to professional liability insurance, it is critical for the midwife to ensure that the carrier understands expanded midwifery practice skills, and specifically that the midwife is credentialed to use a vacuum device.

As stated earlier, the majority of CNM/CM-attended births (97% according to the most recent birth certificate data) occur in hospitals, with a small percentage of births occurring in birth centers and the home. Occasionally, the question of the use of a vacuum device in the out-of-hospital setting is raised. The American Association of Birth Centers (formerly National Association of Childbearing Centers) has developed standards used to accredit birth centers. Vacuum extractors are addressed in the standard regarding quality: “The birth center provides high-quality, family-centered, maternal and newborn services to healthy women anticipating an uncomplicated pregnancy, labor, and birth that reflect applicable professional standards for conduct of the practitioners responsible for services rendered and recognize the basic human rights of the childbearing woman and her family.” Specifically, “drugs for induction or augmentation of labor, vacuum extractors, forceps, recorded electronic fetal monitors, and ultrasound imaging are not recommended during normal labor and are not appropriate for use in birth centers” [11]. Likewise, vacuum extractors are not appropriate for use in the home setting.

The ACNM handbook was written to assist a midwife practicing in the United States. Through its Department of Global Outreach, the ACNM also publishes *Life-Saving Skills (LSS) for Midwives*, a manual designed to help reduce maternal mortality in developing countries. Vacuum extraction is covered in the LSS manual, although the midwives who teach LSS abroad and also teach vacuum extraction workshops in the United States stress that the advice provided to midwives in developing countries might well differ from that provided to mid-

wives in the United States. The website reference is: [[www.midwife.org/global.cfm](http://www.midwife.org/global.cfm)].

### NEWBORN CIRCUMCISION

The medical and ethical issues raised by the practice of circumcision have generated debate among midwives that is similar to the ethics debate that has occurred in other professional groups [12–14]. It is clear, however, that many midwives are interested in attaining this surgical skill, and there are many institutions at which midwives are credentialed to provide newborn circumcision. As with other procedures described above, CNMs/CMs follow the ACNM standards in expanding their practice to include circumcision and can turn to physician colleagues to provide clinical training.

### FUTURE TRENDS

In a 1979 paper entitled, “Nurse-midwife in complicated obstetrics: Trend or treason?” a midwife practicing in a large tertiary care center pointed out that “with advances in perinatal medicine, the ability to identify threats to mother or fetus, risk factors, has led to increasing numbers of women being labeled ‘at risk’” [15]. She urged midwives to “reevaluate ‘normal’ in light of perinatal advances and their own capabilities.” She argued that “complicated obstetric cases are complicated in the area of strength for nurse-midwives,” areas such as counseling, education, nutrition, family involvement, continuity, and close surveillance, and that to limit midwifery practice to normal “is to deny appropriate care to hundreds of at-risk pregnancies.”

In the intervening years, access to technology has expanded, the tendency to use that technology has escalated, and midwives have indeed reevaluated “normal” and their own capabilities.

The use of hospitalist physicians is a trend that might influence the profession of midwifery and midwifery practice in the future. The hospitalist movement began with internal medicine in the 1990s [16] and has been growing in popularity [17]. The field of obstetrics, with its particularly long and erratic hours and rising professional liability concerns, is an obvious specialty for the use of hospitalists. These obstetric providers, who have been dubbed *laborists* [18], are employed by the hospital to care for women who arrive without prenatal

care, or to avoid the disruption of office-based practice that results when a laboring patient must be evaluated and cared for during office hours.

Given their history of caring for the uninsured and their commitment to labor management, midwives are well equipped to service as laborists. Many midwives provide triage services in hospitals, and a growing number now work fulltime in labor and delivery suites as laborists. One might reasonably wonder whether the hospitalist movement will lead to more midwives with skills in first assisting and vacuum extraction.

Midwives have long been involved in medical education and have valued the opportunity to develop partnership models between medicine and midwifery [19–21]. A 1994 survey showed that over one half of U.S. allopathic medical schools were formally using CNMs as educators [22]. Although most midwives are involved in obstetric residency programs, midwives teach obstetrics in family medicine residencies as well [23]. With the institution of limited duty hours for residents, more medical centers are looking to midwives to teach normal obstetrics and to manage labor patients. As medical center faculties and administrators seek to adapt to these new requirements, increased use of midwives not only helps maintain high quality care but also lays the foundation for sound collaborative practices between physicians and midwives.

One author summarized the 2002 national vital statistics data and concluded that “we have entered the new millennium in the midst of a childbirth crisis” [24]. There are indeed ever-increasing rates of intrapartum interventions such as induction of labor and cesarean delivery, with continued poor perinatal outcomes such as preterm delivery and low birth rate [25]. The rising cesarean delivery rate clearly affects how midwives view their role, and it is reasonable to assume that the rising cesarean rate will lead to more midwives first assisting. A better understanding of the practice of midwifery and a system that moves further toward embracing the midwifery model of care could truly begin to reverse these trends, however. Of course, physicians and midwives cannot improve the maternity care system on their own; they must work with public health professionals and health care policy makers to advocate for the needs of mothers and families in addressing the significant barriers to health care in this country.

## CONCLUSION

This chapter focuses on the practice of midwives in operative obstetrics and addresses relatively obvious questions about “using technology appropriately,” that is, appropriate training and skills. It also highlights the importance of the less obvious and more difficult questions of “the appropriate use of technology.”

Debate about the appropriate role of technology is found in all medical specialties but becomes even more complex in the field of obstetrics. With 4 million babies born in this country each year and childbirth being the second-most common hospital discharge diagnosis, the sheer volume of cases means that the decision-making choices of physicians and midwives have a significant impact on the health care system. Unlike clients in most other specialties, most clients of midwives present not suffering from an illness but as essentially healthy women. The current society does, however, have remarkably high expectations for a good outcome, and the increasing trend to seek legal recourse for anything less has had a significant impact on decision making. “Wrongful life” suits and those brought on behalf of young children highlight another complexity unique to the obstetric field—the need to weigh the risks and benefits of any intervention not for one patient but for two. Technologic advances and the aptly named specialty of “maternal-fetal” medicine have fundamentally changed the nature of obstetric decision making. Finally, midwives and obstetricians struggle to care for women and families in a health care system that was generally agreed to be in genuine crisis a decade ago. System-wide problems have overwhelmed the incremental measures meant to alleviate them and are now larger than ever and continue to grow.

Society must continue to view childbirth as an essentially normal process, while recognizing that some problems do require intervention. Midwives and obstetricians must understand and communicate to families that appropriate intervention has remarkable promise to improve health, but not every problem can be solved with intervention, and intervention always carries risks as well as benefits. The decision to intervene must be based on identified need and evidence-based knowledge, rather than on pressures brought to bear by the market, competition, or the prevailing medical culture.

When health care providers debate the evidence – and there will never be enough data to answer all the questions – they must not fall victim to turf battles or defensive rhetoric. Collectively and collaboratively, midwives and physicians must provide women with the safest and most effective care for childbirth by doing what should be done, not what can be done, or what always has been done.

## REFERENCES

1. Rooks J. *Midwifery and Childbirth in America*. Philadelphia: Temple University Press, 1997.
2. Harman PJ, Summers L, King T, Harman TF. Interdisciplinary teaching: A survey of CNM participation in medical education in the United States. *J Nurse Midwifery* 1998;43(1):27–35.
3. Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Menacker F, Munson ML. Births: Final data for 2003. *National vital statistics reports; Natl Vital Stat Rep* 2005;54(2):1–116.
4. Education Committee, ACNM. Core competencies in nurse-midwifery: Expected outcomes of nurse-midwifery education. *J Nurse Midwifery* 1979;24(1):32–36.
5. Avery MD. The history and evolution of the Core Competencies for basic midwifery practice. *J Midwifery Women's Health* 2005;50(2):102–107.
6. Roberts J, Sedler KD. The Core Competencies for Basic Midwifery Practice: Critical ACNM document revised. *J Nurse Midwifery* 1997;42(5):371–372.
7. Summers L. The genesis of the ACNM 1971 Statement on Abortion. *J Nurse Midwifery* 1992;37(3):168–74.
8. Charles C. Practising as a midwife ventouse practitioner in an isolated midwife-led unit setting. *MIDIRS Midwifery Digest* 2002;12(1):75–77.
9. ACNM. *Clinical Privileges & Credentialing Handbook*, ed 2. Silver Spring, MD: ACNM, 2005.
10. Summers L, Williams DR. Credentialing certified nurse-midwives and certified midwives. *Synergy* 2003;30–34.
11. American Association of Birth Centers (formerly National Association of Childbearing Centers). *Perkiomenville, PA: Standards for Birth Centers*, 2003.
12. Milos MF, Macris D. Circumcision: A medical or a human rights issue? [comments]. *J Nurse Midwifery* 1992;37(2 Suppl):87S–96S.
13. Gelbaum I. Circumcision: To educate, not indoctrinate – a mandate for certified nurse-midwives. *J Nurse Midwifery* 1992;37(2 Suppl):97S–113S.
14. Gelbaum I. Circumcision: Refining a traditional surgical technique [comments]. *J Nurse Midwifery* 1993;38(2 Suppl):18S–30S.
15. Reedy, NJ. Midwife in high risk obstetrics: Trend or treason? *J Nurse Midwifery* 1979;24(1):11–17.
16. Wachter RM, Goldman L. The emerging role of "hospitalists" in the American health care system. *N Engl J Med* 1996;335:514–517.
17. Wachter RM, Goldman L. The hospitalist movement 5 years later. *JAMA* 2002;287:487–94.
18. Weinstein L. The laborist: A new focus of practice for the obstetrician. *Am J Obstet Gynecol* 2003;188:310–312.
19. Afriat CI. Nurse-midwives as faculty preceptors in medical student education. *J Nurse Midwifery* 1993;38(6):349–352.
20. Angelini DJ, Afriat CI, Hodgman DE, Closson SP, Rhodes JR, Holdredge A. Development of an academic nurse-midwifery service program: A partnership model between medicine and midwifery. *J Nurse-Midwifery* 1996;41(3):236–242.
21. Sedler KD, Lydon-Rochelle M, Castillo YM, Craig EC, Clark N, Albers L. Nurse-midwifery service model in an academic environment. *J Nurse Midwifery* 1993;38(4):241–245.
22. Harman PJ, Summers L, King T, Harman TF. Interdisciplinary teaching: A survey of CNM participation in medical education in the United States. *J Nurse Midwifery* 1998;43(1):27–35.
23. Payne PA, King VJ. A model of nurse-midwife and family physician collaborative care in a combined academic and community setting. *J Nurse Midwifery* 1998;43(1):19–26.
24. Lydon-Rochelle MT. Minimal intervention – nurse-midwives in the United States. *N Engl J Med* 2004;351(19):1929–1931.
25. Strong TH. *Expecting trouble: The myth of prenatal care in America*. New York: New York University Press, 2000.

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*Be not the first by whom the new are tried,  
Nor yet the last to lay the old aside.*

Alexander Pope (1688–1744)  
*An Essay on Criticism*, 1711, l.135

Medical simulations attempt to recreate events or scenes in clinical practice that are considered important to know or understand. Such simulations are a representation of reality used with the intent to plan, teach, or even entertain. *Simulator* refers to all the technologies used to imitate various specific tasks. Medical simulation probably predates recorded history. There is evidence that ancestors to the Siberian Mansai people built scaled leather dolls of women as birthing models [1]. Plastic, rubber, and cloth dolls were and are in common use in labor and delivery units to teach medical students and house staff the cardinal movements of labor, techniques to manage the second stage, and instrumental or breech delivery. Although the first medical simulation might have been related to childbirth, more recent research and high technologic simulations have been in the field of anesthesia. The continued development of simulation technologies and products has stemmed from fields seemingly remote from medicine. Major contributions have been made by the U.S. military, the Hollywood film industry, and the computer gaming industry.

Aviation simulation and war games were already an integral part of military training before World War II. Simulators have been credited with reducing aviation accidents and improving performance of fighter pilots [2]. The U.S. Air Force has a simulator realistic enough to exert g-forces on trainees. Currently, commercial airline pilots use simulators to maintain competence in performance of routine duties and to train for rare or potentially catastrophic contingencies.

Modern medical simulation began in the late 1960s, when a patient simulator named Sim One was developed at the University of Southern California [3]. This simulator had a heartbeat, as well as a measurable blood pressure and spontaneous breathing, and could respond to specific intravenous medications. Sim One was used to train anesthesia residents in basic intubation skills. During the decades that followed, more complex and sophisticated simulators have been developed and became available. Today, various human patient simulators

are in use at a growing number of medical schools in the United States.

The growing interest in simulations for obstetrics has been prompted by contemporary changes in medical education and concerns for patient safety. Medical schools and residency training programs have been forced to develop strategies to combat decreased patient availability for teaching. Although many think that the 80-hour work-week restriction for residents in obstetrics and gynecology is mainly responsible for a decrease in training opportunities, this restriction is only one of several factors. The combination of shortened hospital stays, increased patient complexity, more home and ambulatory care, declining reimbursements, and reports of medical errors have all led to an increased interest in simulation scenarios. The unique aspect of obstetrics, involving the occasional and unpredictable life-threatening emergency with a conscious patient, can make bedside teaching awkward or counterproductive. Simulation exercises can introduce students to clinical situations, train senior physicians in new procedural tasks, and demonstrate how to manage crises. Dr. David Leach, Executive Director of the Accreditation Council for Graduate Medical Education (ACGME), asserts that simulation enhances both safety and predictability [4]. Leach has stated that a patient who is to undergo the procedure demands that the residents who have not performed a given procedure do it for the first time away from the patient whenever possible. Hence, the current interest and development of simulations and simulators in obstetrics stem from respect for patients and the concept that physicians should obtain a level of competence in a procedure *before* patient contact.

Low-fidelity simulators have been used for teaching in obstetrics and gynecology for years. What has recently began to change is research in medical simulation and evidence for the use of simulation in the specialty. Between 1998 and 2000, there were 210 published reports that related to some form of computer-assisted medical instruction [5]. Of these reports, however, only 11% included any form of assessment of the training and its effectiveness. In addition, there were only 13 studies that related to the field of obstetrics and gynecology. Since the publication of Letterie's 2003 article, an increasing volume of evidence-based research has been con-

ducted in this field, a situation that will continue to expand [5].

Various simulation scenarios for residents and students have been published, including those for vaginal delivery, operative vaginal delivery, vaginal breech delivery, shoulder dystocia, and postpartum hemorrhage [1,6]. At the 2006 Council on Resident Education in Obstetrics and Gynecology (CREOG) and the Association of Professors in Gynecology and Obstetrics (APGO) Annual Meeting, more than ten abstracts were presented that included the use of simulators for training in areas as diverse as circumcision and preeclampsia. Furthermore, in 2006, Michael Mennuti, President of the American College of Obstetricians and Gynecologists (ACOG), formed a task force on reentry into practice. Central to this concept of physicians returning to practice after taking time off (e.g., to care for young children) is the use of simulators for continuing medical education. Excellence in obstetrics requires manual dexterity, quick emergency management skills, the ability to make complex decisions, and effective communication skills. It therefore stands to reason that the role of simulation and simulators will increase in the training of obstetricians in the twenty-first century.

### SIMULATION PRODUCTS

*Medical simulators* attempt to recreate scenes in clinical practice that are considered important to know or understand. Simulators are the technologic tool to initiate tasks for clinical scenes. Simulators can be immersive, like a data cave with multiple imaging screens and a virtual reality suit, or an aviation simulator. In contrast, simulators can also be as simple as a computer game or a manikin. Some simulators use live actors. *Hybrid simulators* are combinations of the different types of simulators. *High-fidelity* simulators suggest close reproduction of the actual clinical environment, whereas *low-fidelity simulators* supply a rudimentary or incomplete clinical environment. *Haptics* is the term used for a simulator that provides forced feedback to the user. An example is a simulator in which the surgeon actually feels the needle penetrating tissue when in reality it is moving through thin air. *Virtual reality* immerses participants into theoretical three-dimensional environments. Not all

**TABLE 25.1 Commercial and Academic Simulator Resources**

Adam-Rouilly	<a href="http://www.adam-rouilly.co.uk">www.adam-rouilly.co.uk</a>
Gaumard Scientific	<a href="http://www.gaumard.com">www.gaumard.com</a>
Limbs and Things, Ltd.	<a href="http://www.limbsandthings.com">www.limbsandthings.com</a>
Medical Education Technologies	<a href="http://www.meti.com">www.meti.com</a>
Sim Surgery	<a href="http://www.simsurgery.com">www.simsurgery.com</a>
Simulaids, Inc.	<a href="http://www.simulaids.com">www.simulaids.com</a>
Society for Medical Simulation	<a href="http://www.socmedsim.org">www.socmedsim.org</a>
Stanford University	<a href="http://www.anesthesia.stanford.edu">www.anesthesia.stanford.edu</a>
The Chamberlin Group	<a href="http://www.thecgroup.com">www.thecgroup.com</a>
Uniformed Services University	<a href="http://www.simcen.usuhs.mil">www.simcen.usuhs.mil</a>

simulators need to be extremely sophisticated, however. Macedonia and colleagues coined the ARRON rule (“*as reasonably realistic as objectively needed*”) for guiding the development of simulation scenarios [1]. Simulators can vary by their tasks, capability, sophistication, and cost. Simulators can be purchased from several commercial companies or built or modified by the users. Table 25.1 lists some currently available commercial and academic simulator resources. Whereas extremely sophisticated surgical simulators exist for laparoscopy, hysterectomy, cystoscopy, and robotic surgery, most birthing simulators are less sophisticated. Gaumard Scientific makes a broad range of maternal and neonatal simulators, ranging from a hemipelvis with a birthing doll to an intubational full manikin with an internal piston for simulating delivery. Many simulators incorporate computer graphics for assessment of the mother and the fetal heart rate (FHR). Several companies, including Limbs and Things, The Chamberlain Group, and Adam-Rouilly, continue to develop simulated tissues and models with more realistic feel. As this growing industry evolves, a good general reference source is the Society for Medical Simulation ([www.socmedsim.org](http://www.socmedsim.org)). Several universities, including the Uniformed Services University, develop and modify existing commercial products to meet their individual needs. Perhaps the best way to review the available simulators and simulations for obstetrics is to study published reports from educators in obstetric training programs.

## SIMULATION IN EDUCATING MEDICAL STUDENTS AND RESIDENTS

### Rationale for Simulation in Obstetrics and Gynecology

Obstetrics and gynecology as a field is uniquely suited to the use of simulation training for medical students, residents, and staff physicians. Many common procedures and examinations taught to medical students, such as a spontaneous vaginal delivery or pelvic examination, can be intensely emotional and private issues for the patient. For residents, emergencies are a relatively common occurrence both in the operating suite and in the labor and delivery unit. Residents must learn to handle such events quickly and correctly to prevent poor outcomes. Obstetric procedures can also be difficult to teach in front of patients because they are alert and conscious, often having family members present and sometimes recording the proceedings with a video camera. Furthermore, concerns for professional liability claims might prevent some staff teachers from allowing residents and students to perform procedures during obstetric emergencies. For instance, when a woman experiences a shoulder dystocia, the attending physician almost always moves a resident out of the way and completes the delivery, because the attending physician is more experienced and bears the legal responsibility for the mother and infant. Thus, more and more physicians in training are denied the opportunity to practice emergency procedures. This deficit in training can become a significant problem when the resident becomes a staff physician and must manage complications without assistance in the absence of real experience. Furthermore, after their initial residency training, physicians who practice in an area with low patient volume might not have the regular exposure to common emergencies that occur in larger facilities, and therefore they need some way to keep their skills current so that they can be prepared for obstetric emergencies.

Medical simulation is an ideal solution for trends in contemporary practice and training. Using models and simulators make it possible to supplement the basic didactic learning that residents and students receive with the ability to practice in a situation in which there is absolutely no risk to the patient or infant. As a nonmedical example, no professional



football team would take the field for a game without having spent hours and hours practicing plays and preparing for every contingency expected from their opponent. Data from within obstetric community are also beginning to accumulate. Evaluation of a shoulder dystocia simulation found that all of the residents in the study, both those randomized to simulation training and those randomized to basic didactic lectures, could recite the same number of interventions for this emergency [6]. Those randomized to simulator training exhibited improved performance, however, suggesting that simulator practice enhanced their ability to apply their knowledge. Whereas an oral examination tests recall of knowledge only, simulation incorporates psychomotor skills into training and tests competency in procedures.

Resident work-hour restrictions, which include no increase in the duration of residency training, reduces the trainee's experience in obstetric procedures. These restrictions in hours also have resulted in additional workload for attending physicians. The net result is to limit the time that the more experienced physicians have available for teaching, as well as lowering the number of emergencies that residents are exposed to during their training.

Residents, and even current staff physicians who trained after 2000, have limited or no exposure to breech vaginal deliveries, because cesarean delivery is now commonly performed for singleton fetuses in a breech presentation [7]. This change in practice, however, does not exclude patients from presenting in advanced labor with a breech fetus, and the physician still must manage both mother and baby. This is an area for which simulation training could be beneficial, allowing physicians with experience and prior training to pass their skill along to the next generation of obstetricians [8]. The reality of this new academic environment suggests that simulation training will become even more critical in the future. Simulations allow additional training for basic procedures and emergencies that can be incorporated into the regular academic schedule and complement the basic didactic lectures. Again, although the idea of simulation makes sense, the evidence for this in performance improvement has been slower in coming. Fortunately, this is an issue that is receiving increasing attention in multiple areas of obstetrics.

## SIMULATIONS FOR UNDERGRADUATE MEDICAL EDUCATION

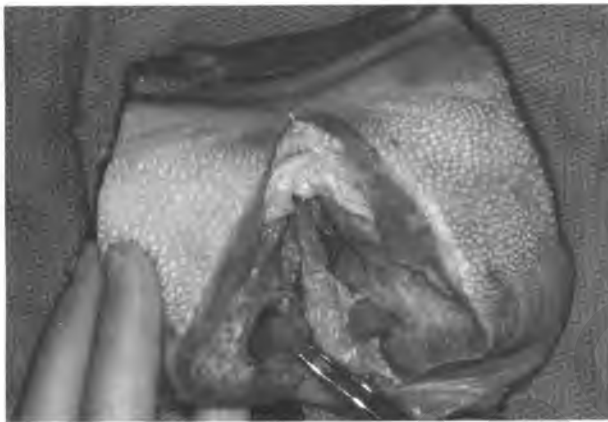
During clinical rotations on the obstetrics and gynecology service, medical students are expected to learn how to perform basic procedures that are intimate and sensitive by their very nature. In fact, there has even been controversy in the lay media whether patients under anesthesia should undergo pelvic examinations by a medical student [9]. Changes in public attitudes and reports such as this have made the use of simulators for teaching these basic procedures increasingly important.

In 2003, a simulation curriculum was incorporated in the medical student curriculum at Georgetown University. The simulation program included a CD-ROM with a Web-based curriculum of the procedures and an instructional video of procedures, and a scheduled training session with a full-sized female birthing simulator. Procedures taught in the simulation training included Leopold maneuvers, fetal scalp electrode placement, intrauterine pressure catheter placement, artificial rupture of membranes, cervical examination, and spontaneous vaginal delivery [10]. Surveys were given to all medical students who rotated through the hospital during their scheduled obstetric and gynecologic rotation to evaluate their comfort level with the different procedures. The group of students who completed their rotation just before implementation of the simulation training was compared with those students who underwent training afterwards. A total of 78 students completed their third-year obstetric and gynecologic rotation during the study period. Of these, 18 students underwent training with the simulator, and 60 did not. Compared with students who did not receive simulator training, trained students reported that they were significantly more comfortable with performing fundal height measurements, Leopold maneuvers, and artificial rupture of the membranes. The simulator-trained students also reported a better understanding of the indications for placing a fetal scalp electrode and intrauterine pressure catheter.

Medical students at the Uniformed Services University and a growing number of medical schools use the Noelle anthropomorphic birth simulator (Gau-mard Scientific, Coral Gables, FL) for their initial training in the conduct of vaginal deliveries. A simulated birthing suite provides an environment for



**FIGURE 25.1.**  
A simulated delivery used for medical student education.  
For color reproduction, see Color Plate 10.



**FIGURE 25.2.**  
A simulated perianal laceration is created with a beef tongue and turkey leg tendons. The tendons are inverted into the tongue to simulate the anal sphincter. For color reproduction, see Color Plate 11.

students to learn basic skills, including how to prepare and drape a woman for delivery, use of instruments involved with birth and laceration repair, management of the third stage of labor, and newborn resuscitation (Figure 25.1). Figure 25.2 shows a model for practicing laceration and episiotomy repair made from a beef tongue and turkey leg. A representative list of obstetric simulations for medical students is included in Table 25.2.

### **SIMULATIONS IN GRADUATE MEDICAL EDUCATION**

With the implementation of the 80-hour work-week restrictions, program directors and academic

**TABLE 25.2 Obstetric Simulations for Medical Student Education**

Pelvic examination
Fundal height
Leopold maneuvers
Artificial rupture of membranes
Fetal scalp electrode and intrauterine pressure catheter placement
Vaginal delivery
Laceration repair
Breast examination

physicians have turned to simulation to supplement their residents' educational experience by focusing on important tasks and targeted simulation training. Whereas it was not uncommon for residency training programs to use a crude birthing fetus within a bony pelvis to demonstrate different procedures and delivery techniques, only recently has there been an emphasis on simulation for graduate medical education. Irwin and colleagues, with the support of ACOG and CREOG, compiled a Web site of simulations and teaching models used throughout the United States for resident education. The stated purpose of this compilation is to provide educators with a resource of models for teaching particular skills ([www.acog.org/creogskills/](http://www.acog.org/creogskills/)). This continually updated reference includes models published in the literature in English or a presented at a national APGO or CREOG meeting. Table 25.3 shows some of the models reported as part of the resident's training curriculum. To create simulations, educators must identify their learners and the critical tasks they are expected to learn. Several simulations for education have been studied and reported in major journals, and representative examples are described later.

### **Shoulder Dystocia**

A *shoulder dystocia* occurs when the fetal shoulder becomes impacted on the maternal symphysis and does not spontaneously deliver after the fetal head. This is a relatively common complication and has been estimated to occur in approximately 2% of vaginal deliveries (depending on definition). In addition, although there are known risk factors for this complication, such as diabetes, fetal macrosomia, and maternal obesity, none of these is sensitive

**TABLE 25.3 Simulation and Teaching Models for Graduate Medical Education**

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Abdominal wall closure
Amniocentesis
Breech delivery
Cerclage placement
Cervical examination
Cesarean delivery
Circumcision
Cord prolapse
Episiotomy/laceration repair
Surgical skills
Operative vaginal delivery
Pudendal block
Shoulder dystocia
Sonography

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enough to predict the occurrence of shoulder dystocia.

In a recent article, Deering and colleagues described a multicenter randomized trial involving residents and training with a shoulder dystocia simulation [11]. Residents were randomized by institution and training level (by year group) to either a training session on shoulder dystocia with a full-sized female simulator or their regular didactic lectures. Several weeks after the training, all residents then underwent testing with a standardized shoulder dystocia simulation, and their performance was analyzed with both subjective and objective grading scales. The residents who underwent simulation training remembered more of the defined critical tasks and had better scores in all subjective categories, including overall performance (Figure 25.3).

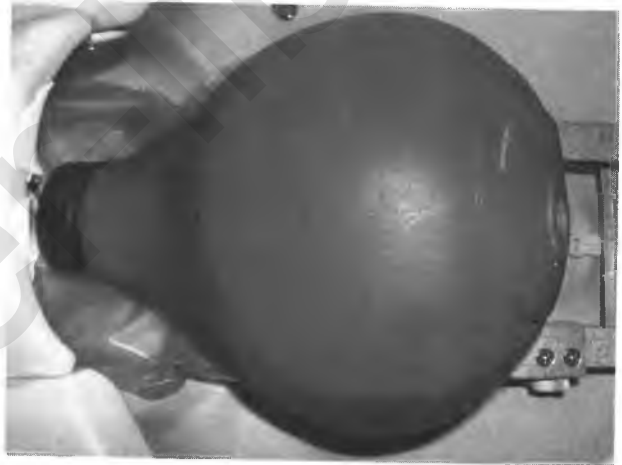
### Postpartum Hemorrhage

*Postpartum hemorrhage* is a relatively common obstetric emergency, occurring in 4% to 6% of all deliveries. The increased blood supply to the term uterus allows for the loss of up to 500 ml of blood per minute, which can result in the rapid decompensation of even a young and healthy parturient. The standard approach to the patient with postpartum hemorrhage includes a physical examination to determine the etiology, which is most commonly uterine atony, the performance of fundal massage,



**FIGURE 25.3.**

*The goal of this shoulder dystocia simulator was to teach residents to deliver the posterior arm. For color reproduction, see Color Plate 12.*



**FIGURE 25.4.**

*An inflatable uterus used to simulate uterine atony. For color reproduction, see Color Plate 13.*

and the administration of standard uterotonic medications. A stepwise approach to this emergency is very important, as well as knowing the appropriate medications to give, the proper dosages, the route of administration, and how often they should be repeated, because medication errors can result in significant morbidity. Residents from three programs underwent training with a postpartum hemorrhage simulation that used a standard obstetric birthing model equipped with an inflatable uterus (to simulate uterine atony) that was able to bleed from the model's cervical os and out of the vagina (Figure 25.4) [12].

All simulations were graded by at least two different staff physicians with a standardized grading sheet constructed from current literature on the topic. The residents were expected to recognize the postpartum hemorrhage and take appropriate steps to correct the problem. The scenario ended when the trainee administered two medications correctly and performed uterine massage or after 5 minutes. The residents were then given immediate feedback on their performance. Both objective and subjective performance was measured with standardized grading sheets, and these results were then placed into a database and analyzed. A total of 40 residents from three institutions underwent simulation training. Findings were significant in that only 22 of 40 (55%) were able to correct the hemorrhage within 5 minutes, and the same number (55%) made at least one error, either the dose or route, in the medications they requested. This study demonstrated that a simulated postpartum hemorrhage scenario can identify important deficiencies in resident knowledge and performance, with no risk to patients. The standardized grading form worked well for the purpose of evaluation and was reliable in this study as well.

### Eclampsia

The rarity of eclampsia, with an incidence of 1 in 2000 women in developed countries, and its potential for maternal and fetal morbidity and mortality, makes it an important emergency to simulate. Eclampsia drills can be used to teach and test house staff about pharmacologic intervention and monitoring. A report by Thompson and coworkers described the use of eclampsia drills that were conducted without warning in different areas of the hospital with a simulated patient [13]. After the simulated eclamptic event, members of the team that responded were briefed on their performance. Thompson's group identified several system deficiencies that led them to initiate changes in practice solutions. Thus, simulations might benefit not only the individual providers but also healthcare teams.

### Operative Vaginal Delivery

Macedonia and colleagues developed, refined, and validated a simulation for teaching operative vaginal delivery [14]. The model included a birthing



FIGURE 25.5.  
*Forceps or vacuum delivery can be simulated [17,18]. For color reproduction, see Color Plate 14.*

manikin for small-group training in the use of Simpson, Kielland, and Piper forceps (Figure 25.5). Participants improved their efficiency with training. Furthermore, the model accurately discriminated subjects by level of experience. The author also incorporated digital recording into simulation sessions. Review of recordings with participants provided excellent feedback and further enhanced learning.

### Ultrasound Scanning and Procedures

Lee and colleagues described an ultrasound image library combining an interactive multimedia with a three-dimensional heart model for prenatal ultrasound training [15]. Their prototype provided residents with a broad exposure to ultrasound anomalies and offered an improved opportunity to understand how a series of two-dimensional ultrasound images can actually represent complex three-dimensional biologic structures like the heart.

Amniocentesis is an obstetric procedure that is frequently performed to obtain genetic information or to seek evidence of intraamniotic infection and fetal lung maturity samples for testing. Amniocentesis requires excellent hand-eye coordination and the ability to conceptualize a three-dimensional space with the two-dimensional ultrasound images. When performed in the midtrimester, this procedure does carry a significant risk to the unborn child, however, with a fetal loss rate of approximately 1 in 200. Because of its difficulty and potential risk,

amniocentesis lends itself well to simulation. Pittini and coworkers reported on the use of an amniocentesis simulator in the training of junior and senior residents as well as maternal-fetal medicine fellows [16]. They found that a hands-on training session with the amniocentesis simulator, in addition to a standardized curriculum, resulted in a significant improvement in the trainee's performance of the procedure even after controlling for level of training. Several groups have described the construction and utility of amniocentesis trainers.

Maier and colleagues built an amniocentesis trainer from a commercially available storage box [17]. Sonodense spherical targets 2.3 cm in diameter were taped to the bottom, and the box was filled with a gelatin mixture. The trainer improved the ability of the operator to perform a free-hand amniocentesis, orient the ultrasound transducer, follow a needle with continuous sonographic guidance, and hit a 2-cm target. Although this group created their own simulator for this study, an amniocentesis trainer is commercially available from the company Limbs and Things (Bristol, United Kingdom).

### Vaginal Breech Delivery

Breech vaginal delivery of a singleton pregnancy is no longer encouraged by ACOG since the publication of the randomized term vaginal breech trial in 2000 [7]. (See Chapter 12, Breech Presentation.) Training opportunities with actual patients are virtually nonexistent because the most women with a persistent breech presentation are scheduled for a cesarean. These recommendations, however, do not prevent the occasional occurrence of emergent and sometimes precipitous breech deliveries. As a result, simulation training has been introduced in an attempt to provide the opportunity to teach safe breech delivery. Using a birthing manikin, residents from two separate institutions were tested for their breech vaginal delivery skills (Figure 25.6) [19]. Residents participated in a breech delivery simulation, after which they were instructed in the proper techniques and corrections were made to their initial performance. At a later date, the same residents again performed a simulated breech vaginal delivery, and their performance was then compared with their before-and-after training simulations. Compared with their pretraining scores, trained residents had significantly higher scores in 9 of 13 critical



FIGURE 25.6.  
*A simulated vaginal breech delivery. For color reproduction, see Color Plate 15.*

delivery components and overall performance and safety of the delivery.

### Innovative Approaches

Not all obstetric simulations require expensive manikins or sophisticated simulators. Erickson described the construction and use of a model designed to introduce trainees to neonatal circumcision using the Gomco clamp [20]. The model was constructed from a clipboard, an infant pacifier, fingers cut from examination gloves, a rubber band, a 3-ml syringe, tape, one or two folded surgical towels, and a Gomco clamp with 1.3-cm bell. A circumcision kit containing three hemostats, one pair of surgical scissors, and a scalpel blade was provided. With each trainee using separate models, one instructor was able to guide four interns through three simulations in a one-hour teaching period.

In another setting, using a number of inexpensive and easily obtained materials, Macedonia successfully developed a simulation model for resident education in cervical cerclage. The components of this model included a birth manikin, an operating table, a balloon, sponges, and surgical gloves. This experience and that of Erickson emphasize the importance of innovative leadership in the development of education programs using medical simulations (Figure 25.7) [14].

### Other Uses

Another area related to patient care, safety, and professional liability is the documentation of deliveries, especially if they are other than normal,

**TABLE 25.4 Key Delivery Documentation Components for Shoulder Dystocia**


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Date of delivery
Time of delivery
All providers present at delivery
Classifies complication as shoulder dystocia
Notes which shoulder was anterior
Notes how long it took to deliver the shoulder
Notes infant birthweight
Notes Apgar scores
Notes if cord gases sent
Mentions that infant is moving all extremities after delivery
Notes pediatrician called for delivery
Includes estimated blood loss
Includes all maneuvers used
Includes correct order of maneuvers used
Notes patient had epidural anesthesia

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**FIGURE 25.7.**  
*A simulation for cerclage placement. For color reproduction, see Color Plate 16.*

spontaneous, and uncomplicated. A total of 33 residents who underwent training on a shoulder dystocia simulation were asked to write delivery notes after they completed the delivery [11]. These delivery notes were then analyzed for 15 key components (Table 25.4). Fully 67% of the residents recorded fewer than 10 of the 15 key components, and only 18% noted which shoulder was anterior during the delivery. Furthermore, only 45% listed the head-to-body delivery interval in the note. Although this study did not evaluate whether simulation training could improve documentation, it did demonstrate that simulation can be used to identify

deficiencies in documentation after obstetric emergencies. The investigators subsequently used these data to emphasize this topic to residents, and it spurred their interest in curriculum development.

Simulations can be used to address the issue of physician professionalism. Gisondi and colleagues identified professionalism issues encountered during their training in the emergency department, including patient confidentiality, informed consent, withdrawal of care, practicing procedures on the recently deceased, and use of do-not-attempt-resuscitation orders [21]. They then created patient-physician scenarios to address each of these and used high-fidelity patient simulators. They found that performance improved significantly as the level of training increased and thought that the exercise was helpful, especially for junior residents. All interns across specialties at Walter Reed Army Medical Center complete an exercise in which they must break bad news to standardized patients who represent a couple with a critically ill child. The sessions are recorded and subsequently reviewed with the interns, in an effort to improve their communication skills.

Colletti and coworkers reported on two groups of medical students who were given either instruction in communicating bad news to patients with a standardized patient instructor (SPI), or no additional training [22]. They all subsequently underwent testing with a clinical performance examination. They found that students who received SPI training performed significantly better than those without such training.

An integrated program of forceps simulation and lectures can be used to improve residents' counseling of patients during a simulated operative vaginal delivery [23]. Residents underwent testing with a simulation in which they were expected to deliver a fetus with forceps. If they did not counsel the patient's husband, a live human actor, or the manikin prior to beginning to place the forceps, the "husband" would ask the resident if the forceps were dangerous, which prompted the resident to respond and counsel the simulated patient. Residents then underwent training with the risk management department and an obstetrician who gave lectures on the importance of appropriate counseling for operative vaginal deliveries. They were then retested on the forceps scenario several weeks later. Sixteen residents completed the initial forceps testing, and then eleven residents attended a lecture and

had simulation testing afterwards. Lecture attendance prior to the simulation scenario was associated with a higher likelihood of offering the patient the option of a cesarean and addressing more maternal and fetal complications.

An increasing number of simulations thus are being used for medical education. Numerous programs have been developed not only to teach but also to assess surgical skills. The concept of objective structured assessment of technical skills was suggested by Reznick from the University of Toronto [24,25]. Goff and colleagues have also shown that an objective structured assessment of technical skills can assess obstetric and gynecologic residents' surgical skills with a high degree of reliability and validity [26].

### CONTINUING MEDICAL EDUCATION

In contrast to medicine, other vocations that involve life-threatening emergencies on a daily basis, such as commercial and military aviators, must complete hands-on tests to maintain their certification. These generally include several hours in high-fidelity aviation simulators that evaluate their ability to make decisions when several common complications and emergencies occur.

There is a precedent for the use of simulation in medical education, beginning with medical students. Commencing in June 2004, Step 2 of the U.S. Medical Licensing Examination (USMLE) included a clinical skills (CS) examination. This examination consists of a live physician-standardized patient encounter with the scenarios planned to reflect the environment in which physicians are expected to apply their knowledge and skills [27]. With this relatively recent change in this national examination, medical and surgical specialties probably will follow suit with their certification examinations. What is still lacking in the field of obstetrics and gynecology, however, are sufficient data on the validity and reproductivity of simulations, as well as the ability to distinguish between those practitioners who are merely competent versus the masters of the art. Before simulations can be incorporated into licensing and certification examinations, the scenarios will require this higher degree of sophistication. Nevertheless, just as simulation is achieving greater roles in undergraduate and graduate education, its implementation into continuing medical

education grows steadily. There are several university, professional society, and commercial courses that incorporate animals, cadavers, inanimate models, and task trainers to expose the learner to hysteroscopy, laparoscopy, urogynecologic procedures, laser therapy, and robotic surgery. Furthermore, a growing number of obstetrician/gynecologists will face scenarios that require them to take time off from practice, such as having children. Hospitals and departments chairs may choose not to grant privileges for procedures not recently performed. Simulations could provide a way to learn new techniques, document experience, and prove competency in procedures previously mastered but not recently performed.

### PATIENT SAFETY AND TEAM DRILLS

#### **Patient Safety**

The ultimate goal of all medical simulation training is to improve patient safety and outcomes. A recent article concerning all pregnancy-related deaths in North Carolina over a four-year period reported that 40% were preventable [28]. Improvement in medical care was considered the most important factor in preventing these deaths. Another landmark publication on this topic was the report *To Err Is Human* [29]. This study estimated that between 45,000 and 98,000 patients die in the United States each year as a result of preventable medical errors, at a cost of around 29 billion dollars. Although it makes sense and logically follows that outcomes will improve as physicians practice for the procedures and emergencies they encounter, this is an easy and difficult goal to both achieve and demonstrate, depending on the scenario being simulated. For instance, the task might be to improve laparoscopic skills. Laparoscopy is a common procedure for which the patient is asleep, and sophisticated models are available. It is relatively simple to design an appropriate training program for the physician to complete and then reassess them in the actual operating suite. As demonstrated in a large study by Birkmeyer and coworkers, surgical experience accounts for an extremely large amount of operative mortality [30]. This lends additional credence to the basis for simulation, and to the fact that increased exposure could improve outcomes.

Conversely, medical emergencies are much more difficult to teach and assess in a real environment. For example, when a complication such as a shoulder dystocia occurs, the patient is generally alert and oriented, and the infant is at significant risk. During this emergency, the senior physician does not have the luxury of stepping back and explaining to the junior physician what the exact maneuvers are and how to do them. Instead, he or she is focused on delivering the shoulder and resolving the emergency. In addition, to assess the effectiveness of simulation training for an emergency such as a shoulder dystocia, it is not possible to have an observer at every delivery in the hopes of having a shoulder dystocia and evaluating the junior physician's performance. For many obstetric emergencies, other markers, such as long-term outcomes from an institution after training is implemented or performance on the simulators after training, are the surrogate markers that can be used.

Physicians must not give up on the important task of attempting to demonstrate improvement in patient safety and outcomes, because although difficult, it is not impossible. As evidence-based results are gathered that show the benefits of simulation in obstetrics and gynecology, it seems reasonable that more investment can be made in scenario development, and liability risk can be reduced.

### Team Drills

Numerous issues can prevent teams from working together. Currently, several programs across the United States are being run in an attempt to promote teamwork and improve communication and performance during medical emergencies.

Another recent focus in the arena of patient safety has been the implementation of both *team training* and *involving the entire core team in simulating training of various procedures and emergencies*. This training is a logical step to take simulation to the next level, because these events rarely occur in a vacuum, and even if the physician is completely confident and competent, he or she can rarely correct an emergency alone. For instance, when a postpartum hemorrhage occurs and the patient requires a transfusion, the physician is not typically the one who goes to the blood bank for blood products. Similarly, during a shoulder dystocia, the physician cannot apply gentle downward traction on at the fetal

head and perform the McRoberts maneuver and suprapubic pressure at the same time. Because of this, every member of the team must not only know what is required of them in an emergency but also be able to communicate this effectively. Thompson and coworkers evaluated an eclampsia drill [13]. They identified several problems, including difficulty summoning senior staff urgently, multiple protocols for managing eclampsia, wasted time fetching individual interns for management of seizures, and confusion about staff roles that resulted in inefficient activity. Solutions developed as a result of these team drills included rapid activation of the team through one call from the switchboard, development and dissemination of an evidence-based protocol for eclampsia management, strategically placed "eclampsia boxes" with appropriate medication doses in the hospital, and clear division of tasks in the management protocol.

Crew resource management (CRM) was instituted in the aviation industry in the 1970s after a report from the military Inspector General noted that 70% of aircraft-related fatalities were the result of poor teamwork and human error [31]. Medicine in recent years has begun to apply the tenets of this teamwork training from the aviation sector to the current practice of medicine.

In an attempt to apply CRM to the field of obstetrics, the Department of Defense and the Risk Management Foundation of the Harvard Medical Institutions recently funded a study for labor and delivery units [31]. Teams of instructors were created to teach the physician, nursing, and ancillary staff how to implement basic teamwork concepts. This new direction emphasizes the importance of training not only the physicians but also the entire team, in an attempt to improve outcomes. The weighted adverse outcome index (WAOI) measure was developed to determine whether the teamwork training had been effective. The WAOI provides a weighted score depending on the poor outcome, with the highest score for a maternal death and the lowest for a third- or fourth-degree laceration. Although this score was used in the multicenter trial for teamwork training, because outcomes such as maternal and neonatal deaths occur very rarely, simulation training might be used as a surrogate marker to evaluate teamwork performance.

In the United States, the captive insurer for the Harvard Medical Institutions now offers a 10%



discount in malpractice premiums for physicians who participate in the teamwork training course. In the United Kingdom, the 1999 Confidential Inquiry into Maternal Deaths and Towards Safer Childbirth called for the use of drills in anticipation of obstetric emergencies. Implementation of these drills is necessary for Level Two accreditation by the clinical negligence scheme for trusts, which conveys a 20% discount on liability premiums for U.K. Trusts [13].

### FUTURE APPLICATIONS AND SUMMARY

Many factors have spurred interest in the development of simulations for physicians, healthcare providers, and teams in obstetrics. Challenges in medical education, including work-hour restrictions, professional liability concerns, generational differences in practice patterns, shrinking availability of patients for teaching and reimbursements, and the unique practice of obstetrics (with conscious patients), all foster the growth of simulation. Concern for patient safety, evidence of efficacy of team drills in other fields, the ability to learn new techniques, reentry into practice for senior physicians, and a potential reduction in liability premiums might lead to development and refinement of obstetric simulations. It seems ironic that it has never been safer for a woman to deliver a baby in the United States but never more dangerous for her obstetrician or midwife as far as liability concerns. As the enthusiasm for simulations and simulators grows and the simulators themselves become more realistic, it is reasonable to anticipate that this methodology will be incorporated into every level of medical training. Evidence of simulation's becoming more accepted in clinical practice can be seen in a recent decision by the Food and Drug Administration, which requires physicians who desire to perform a carotid artery stenting procedure to first demonstrate their competency on a simulator. Similarly, physicians performing first-trimester aneuploidy screening with serum analogs and sonographic evaluation of nuchal thickening must prove competence in obtaining sonographic images prior to submitting serum samples for evaluation. All of these factors and scenarios suggest that simulations will be a growing part of obstetrics training and practice. Ultimately, respect for patients and their right to have procedures done by competent

physicians will be responsible for the incorporation of simulation into current practice.

### REFERENCES

1. Macedonia CR, Gherman RB, Satin AJ. Simulation laboratories for training in obstetrics and gynecology. *Obstet Gynecol* 2003;102:388-92.
2. Brown JL. Visual elements in flight simulation. *Aviat Space Environ Med* 1976;47:913-4.
3. Good ML. Patient simulation for training basic and advanced clinical skills. *Medical Education* 2003; 37(Suppl 1):14-21.
4. Leach DC. Simulation: It's about respect. *ACGME Bulletin*, Chicago, IL. Dec 2005.
5. Letterie GS. Medical education as science: The quality of evidence for computer-assisted instruction. *Am J Obstet Gynecol* 2003;188:849-53.
6. Deering SH, Poggi S, Macedonia C, Gherman RB, Satin AJ. Resident competency in the management of shoulder dystocia improves with simulation training. *Obstet Gynecol* 2004 Jun;103(6):1224-8.
7. Hannah ME, Hannah WJ, Hewson SA, Hodnett ED, Sagai S, William AR. Planned caesarean section versus planned vaginal birth for breech presentation at term: A randomized multicenter trial. *Lancet* 2000;356:1375-83.
8. Queenan JT. Teaching infrequently used skills: Vaginal breech delivery. *Obstet Gynecol* 2004 Mar; 103(3):405-6.
9. Goldstein A. Practice vs. privacy on pelvic exams. *Washington Post*, May 10, 2003, Page A01.
10. Deering SH, Hodor JG, Wylen M, Poggi S, Nielsen PE, Satin AJ. Additional training with an obstetric simulator improves medical student comfort with basic procedures. *Simulation in Healthcare: The Journal for the Society for Simulation in Healthcare* 2006;1(1):32-4.
11. Deering SH, Poggi S, Hodor JG, Macedonia C, Satin AJ. Evaluation of residents' delivery notes following a simulated shoulder dystocia. *Obstet Gynecol* 2004 Oct;104:667-70.
12. Deering SH, Chinn M, Hodor JG, Nielsen, Benedetto T, Mandel L, Goff B. Validation and testing of a postpartum hemorrhage simulator for instruction and evaluation of residents. *APGO/CREOG Annual Meeting*, Orlando, FL, March 2006.
13. Thompson S, Neal S, Clark V. Clinical risk management in obstetrics: Eclampsia drills. *Qual Saf Health Care* 2004;12:127-9.

14. Macedonia CR, Kopelman J, Sarno A, Satin AJ. Development and validation of a model teaching operative vaginal delivery utilizing an anthropomorphic simulator. Poster 167. *Am J Obstet Gynecol* 2002 Dec;187(6, pt. 2) Suppl:S106.
15. Lee W, Ault H, Kirk JS, Comstock CH. Interactive multimedia for prenatal ultrasound training. *Obstet Gynecol* 1995 Jan;85(1):135–40.
16. Pittini R, Oepkes D, Macrury K, Reznick R, Beyene J, Windrim R. Teaching invasive perinatal procedures: Assessment of a high-fidelity simulator-based curriculum. *Ultrasound Obstet Gynecol* 2002 May;19(5):478–83.
17. Maher JE, Kleinman GE, Lile W, Tolaymat L, Steele D, Bernard J. The construction and utility of an amniocentesis trainer. *Am J Obstet Gynecol* 1998 Nov;179(5):1225–7.
18. Smith JF Jr, Bergmann M, Gildersleeve R, Allen R. A simple model for learning stereotactic skills in ultrasound-guided amniocentesis. *Obstet Gynecol* 1998 Aug;92(2): 303–5.
19. Deering SH, Brown J, Hodor J, Satin AJ. Simulation training and resident performance of singleton vaginal breech delivery. *Obstet Gynecol* 2006 Jan; 107(1):86–9.
20. Erickson SS. A model for teaching newborn circumcision. *Obstet Gynecol* 1999 May; 93(5 Pt 1):783–4.
21. Gisondi MA, Smith-Coggins R, Harter PM, Soltysik RC, Yarnold PR. Assessment of resident professionalism using high-fidelity simulation of ethical dilemmas. *Acad Emerg Med* 2004 Sept;11(9): 931–7.
22. Colletti L, Gruppen L, Barclay M, Stern D. Teaching students to break bad news. *Am J Surg* 2001 Jul; 182(1):20–3.
23. Deering SH, Hodor J, Chinn M, Bertram K, Altman S, Satin AJ. Counseling patients for forceps deliveries: Use of simulation to assess and improve competency. APGO/CREOG Meeting, Salt Lake City, Utah, March 2–5, 2005.
24. Reznick RK. Teaching and testing technical skills. *Am J Surg* 1993 Mar;165(3):358–61.
25. Reznick R, Regehr G, MacRae H, Martin J, McCulloch W. Testing technical skill via an innovative “bench station” examination. *Am J Surg* 1997 Mar; 173(3):226–30.
26. Goff BA, Nielsen PE, Lentz GM, Chow GE, Chalmers RW, Fenner D, Mandel LS. Surgical skills assessment: A blinded examination of obstetrics and gynecology residents. *Am J Obstet Gynecol* 2002 Apr;186(4):613–17.
27. Dillon GF, Scoles PV. An examination of clinical skills in the United States Medical Licensing Examination (USMLE). *ACGME Bulletin* Dec; 2005: 16.
28. Berg CJ, Harper MA, Atkinson SM, Bell EA, Brown HL, Hage ML, Mitra AG, Moise KJ Jr, Callaghan WM. Preventability of pregnancy-related deaths: Results of a state-wide review. *Obstet Gynecol* 2005 Dec;106(6):1228–34.
29. Kohn LT, Corrigan JM, Donaldson MS [editors], Committee on Quality of Health Care in America. Institute of Medicine. *To Err Is Human: Building a Safer Health System*. Washington, DC: National Academy Press; 2000, p. 287.
30. Birkmeyer JD, Stukel TA, Siewers AE, Goodney PP, Wennberg DE, Lucas FL. Surgeon volume and operative mortality in the United States. *N Engl J Med* 2003 Nov 27;349(22):2117–27.
31. Mann S, Marcus R, Sachs B. Lessons from the cockpit: How team training can reduce errors on L&D. *Contemp OB/GYN* 2006 Jan;51(2):34–45.

## Joanna M. Cain

*Whenever cases occur, attended with circumstances not heretofore observed, or in which the ordinary modes of practice have been attempted without success, it is for the public good . . . that new remedies and new methods of chirurgical treatment should be devised. But in the accomplishment of this salutary purpose . . . the faculty should be scrupulously and conscientiously governed by sound reason, just analogy, or well authenticated facts.*

Thomas Percival (1780–1804)

*Medical Ethics: or, a Code of Institutes and Precepts*

*Adapted to the Professional Conduct of Physicians and Surgeons*

Manchester: S. Russell, 1803, pp. 14–15

The sometimes-conflicting moral obligations that physicians hold to both mother and fetus in obstetrics (termed *maternal-fetal conflict*) capture the most poignant of ethical quandaries. Fortunately, circumstances in which the pregnant woman makes choices that are not in the best interest of the developing fetus are actually quite rare. Most pregnant women accept remarkable levels of bodily intrusion or invasion, increased costs, and time-consuming medical interventions to ensure the greatest likelihood of successful outcome. For few other situations would it be anticipated that any person should submit to the level of personal discomfort, potential life-threatening intervention, and self-denial that is routinely expected of pregnant women for an entity that is not yet and might never be a person. In this society, an expectation of extraordinary self-sacrifice practiced by most pregnant women is the norm, and pregnant mothers who question this behavior become immediately suspect. While this group of conflicting obligations is discussed, recognition of the extraordinary nature of this “normal” behavior should be a constant companion.

### THE MATERNAL-FETAL RELATIONSHIP

#### Maxims

The moral conflicts that surround abortion, prenatal diagnosis, invasive fetal therapy, and maternal refusal of recommended care all carry an implied concern for the dependent variable in the choices made – the fetus. Virtually every aspect of the fetus in relationship to society, to biologic reality, and to its mother has been explored by philosophers, theologians, and the learned societies [1–4,6,26,41]. Physicians and their patients are left with few maxims and many gray areas, but it can be agreed that

1. The fetus is a biologic reality [5]. Its continued existence depends on an intimate connection with the maternal circulation.
2. The mother is the first patient. No argument can be made concerning the fetal-maternal relationship that ignores the mother as the only

participant capable of autonomous judgment and choice. Furthermore, only women can get pregnant, and they can have ways of thinking about moral problems that differ from the principle-based ethical structures common to medical ethics training [7].

3. The social context is important in understanding the maternal-fetal relationship; however, genetic linkages that have traditionally been called on in this context are being challenged by surrogacy and other reproductive technologies. These changes complicate this social context [8–10].
4. Because medicine interacts at any level with the maternal-fetal relationship, the goals of medicine, as defined by Jonsen, Siegler, and Winslade, must be revisited [10]. Intervention should accomplish one of the goals of medicine: a) promotion of health; b) relief of symptoms, pain, and suffering; c) cure of disease; d) prevention of untimely death; e) improvement of functional status or maintenance of compromised status; f) patient education and counseling about the condition and its prognosis; and g) avoidance of patient harm in the course of care.

The arguments made by Chervanek and McCullough are consistent with these statements and suggest conceptualizing of the fetus as a *potential* patient when viability becomes possible [1]. As discussed in detail earlier in this text, fetal viability is not a defined rigid marker but a working definition that varies among institutions and reflects periodic improvements in medical practice [42]. As a practical matter, fetal survival is highly unlikely if the pregnancy is less than 22 completed weeks and the estimated fetal weight is less than 350 g. Fetal survival is occasionally possible with gestational ages beyond 23 completed weeks and an estimated fetal weight of 350 g to 400 g, but many if not most of these very small survivors have serious morbidity and permanent injury. Reasonably likely and intact neonatal survival (defined as >50% survival and >50% normal) is not anticipated unless the period of gestation is at least 24 (and preferably 25) completed weeks with an estimated fetal weight of 450 g to 500 g. As always, it should be remembered that the statistics for individual institutions will vary and that these guidelines are based on antepartum *estimates* of both weight and gestational age. Thus, these parameters

serve as general indicators of possible outcomes as evaluated prior to delivery for purposes of counseling. Within these parameters and for the purpose of this discussion, a *viable fetus* is defined as an infant that has the potential to survive delivery, the neonatal period, and infancy. This requires an appropriate biologic potential and the availability of various interventions that are applied after the test of reasonableness.

The tests of reasonableness that used here include meeting the goals of medicine for the mother (who is the first patient) and the fetus (who might be a patient). In doing so, physicians must always guard against the risk of “reproducing existing conditions of social and racial inequality” [11]. This situation can occur if court orders for interventions in favor of the fetus and against the wishes of the mother are sought more often with poor women of color, or if the reporting of positive drug testing results is more common based on race. The obligations of healthcare providers to the mother are invariable; to the fetus, they can vary widely. There are also obligations of healthcare providers and mothers to society as a whole to ensure a just and equitable life for all its members [12,14–15]. Although the mother is the first patient and therefore carries more moral weight, it does not follow that all autonomous maternal choices meet criteria that are in the best ethical interest of a fetus or of society. To further explore this basis for maternal-fetal obligations, the potential medical interventions that diagnose or treat the fetus require critical review [2].

### The First Trimester

As Table 26.1 illustrates, the benefits of first-trimester procedures focus on weighing maternal and parental burdens, namely either to bear a child with predictable congenital defects or not to bear a child at all. The institution of first-trimester risk assessment for trisomy 21 and the choices to be made even before testing bring these issues into sharp focus [12–15]. Predictable ethical quandaries arise for both physicians and parents while weighing such decisions when the only potential intervention is induced abortion.

The initial issue for clinicians attempting to fulfill their obligations to patients is the choice whether to continue a pregnancy, regardless of the

TABLE 26.1 Risks and Benefits from Medical Interventions for Fetal Diagnosis or Therapy

Gestational Age	Studies/Procedures/Surgery	Fetal Risks	Maternal Risks	Fetal Benefits	Maternal Benefits	Comments
1st trimester 10–12 weeks	CVS	9 wks <sup>†</sup> Fetal losses exceeds background rate by 0.6–0.8% <sup>‡</sup>	Infection Hemorrhage <1% Mild discomfort	+/-	Ability to choose burdens; reassurance	
	Ultrasound scan	–	–	+/-	Potential identification of maternal risk with placenta previa	Could successfully identify an at-risk pregnancy or congenital anomalies.
	Abortion	–	Variable	–	+/-	No psychological long-term detriment demonstrated.
	Selective reduction	Fetal loss with reduction of other pregnancy	Limited	+ (for remaining)	–	Reduction could increase the possibility of survival children and reduce maternal risks.
16–20 weeks	Amniocentesis	Fetal loss <0.3–1%	Infection Hemorrhage <.10%	+/-	Ability to choose burdens; reassurance	
	PUBS (18 weeks +) Fetal transfusion	Fetal loss 1–2.3% Premature labor 5–9%	Infection, rare complications 0.6%	++	–*	Fetal death is a rare outcome.
24+ weeks	Maternal Rx for fetal cardiac defects	+/-	+ Reaction to cardiac drugs	++	–*	
	Fetal surgery	++	+ Bleeding hemorrhage	+/-	–*	See Chapter 20.
	Cesarean delivery	–	++ serious <1% death – rare	+/-	–*	See Chapter 18.
	Preterm labor management	–	1% risk pulmonary edema	+/-	–*	Risks dependent on GA <33 weeks
	PROM observation	–	Risk of fatal infection	+/-	–*	

\*Increased chance of survival of child.

<sup>†</sup>Oromandibular-limb hypogenesis likely increased in procedures at <9 wks; not present at later gestational ages [44,45].

<sup>‡</sup>See refs. 43,45,46.

CVS, chorionic villus sampling; PUBS, percutaneous umbilical cord sampling; PROM, premature rupture of membrane; GA, gestational age.

reason. For this discussion we accept some level of a physician's ethical obligations to an individual fetus, starting with the fetus's entry into viability and a period during which beneficial clinical intervention can be applied. Some fetuses might never become viable; for example, those with severe congenital abnormalities incompatible with survival. In the first trimester, neither invasive fetal studies nor interventions directly benefit the fetus. Furthermore, spontaneous abortion can be likely, regardless of interventions performed.

The ethical arguments regarding the basis of ethical obligations toward the viable or nonviable fetus follow many routes. As Dworkin points out, "Most people accept the sanctity of human life but disagree in complex ways about the implication for abortion" [14]. He would detach this concern for sanctity from a discussion of legal or ethical rights, thus characterizing arguments as attached or detached. Steinbock prefers characterizing these arguments as continuous or discontinuous [15], seeking either to prove or disprove continuity to adult values or personhood to imply status for rights. As she points out, "... only the assumption that the unborn is a human being like any other, entitled by law's protection, could justify the prohibition of abortion." Steinbock's "interest" argument reasons that the fetus has no interest in survival because it is preconscious and presentient, and she steps away from continuity or detached arguments. Regardless of the basis of the ethical argument and despite the controversy, society generally agrees that the mother's life and health must prevail over fetal life and health. The choice to terminate a pregnancy thus has focused instead on certain burdens of mothers: protecting mental or physical health; relieving economic, social, or demographic problems; and supporting a view of liberty rights of the penumbra of the Fourteenth Amendment that "... recognizes rights to bodily integrity and a person's most basic decisions about family and parenthood" [2,15,16].

There could, however, be ethical limits to this freedom to terminate pregnancy at will in the first trimester that derive from issues of justice and not autonomy. Consider prenatal diagnosis that results in intended abortion for a nondesired sex. It is generally thought that abortion for a fatal or seriously disabling sex-linked disease would prevent maternal and fetal/infant suffering if the infant were born alive, and thus abortion meets a medical goal. Abor-

tion merely because of maternal (or paternal) valuing of one sex over another, however, raises entirely different issues about the benefits of prenatal information. The element of relieving suffering based on knowing the sex of the fetus breaks down when there is no disease, and the only issue is the cultural bias of the parents. Preconceptual sex selection fails morally for the same reasons that selective abortion for sex does. The arguments are well outlined by multiple authors [17–19] and can be categorized as follows:

1. Gender, race, economic status, or phenotype (e.g., height) are not diseases but social distinctions. As such they are not a morally relevant basis for medical decisions. As Wertz and Fletcher point out, "Prenatal diagnosis for nonmedical reasons makes a mockery of medical ethics" [19].
2. Societal harms and the obligation to avoid them are the responsibility of all citizens, including medical personnel and patients. Inequality and neglect of socially unwanted children are supported by compliance with morally unacceptable underlying beliefs.

Blank suggests further harms of preconceptual selection techniques as they become less intrusive and allow control of progeny's characteristics (height, sex, appearance, etc.) [20]. Given these changes, such technology is more likely to become a marketable service. Allowing this to occur risks commercializing children. Children, in other words, become consumer goods that are evaluated for various measures of "quality." Children born with genetic or congenital abnormalities – or the "wrong" sex – might face greater discrimination as society reinforces a pervasive sense of right to discriminate with the ability and hence the right to make even earlier distinctions. Because the appearance of "unacceptable" characteristics in the culture could have been "prevented," tolerance for differences and valuing potential benefits that people with differences bring to a culture could diminish. This fact is particularly true in a cultural ethic that has fostered personal destiny, choice, control, and an entrepreneurial approach to medicine. The long-term cost of uncontrolled genetic choice to society could be significant – an ethical harm that bears consideration on both extremes of a scale. There could be moral limits to a patient's right to choose an

abortion to discriminate against progeny based on nonmedically relevant genetic or phenotypic characteristics. By the same outcome, requiring termination for characteristics that are less well regarded socially is also morally repugnant.

There have been extensive reviews of another area of elective termination, the issue of selective reduction of multiple gestation [21,30,40]. The issue revolves around the balance between harm and benefit that depends on adequate outcome data for multiple gestations, and on outcome data that result from the use of new reproductive technology. The ethical principle proposed by Evans and coworkers for use in this setting – proportionality, a variant of consequentialism – is one that also serves to govern interventions in the second and third trimester [21]. This is “. . . to balance risks and benefits so that actions have the greatest chance to cause the least harm and the most benefit to persons directly involved. . . .” This principle leads these authors to a recommendation that only pregnancies with three or more fetuses have outcomes sufficiently serious to warrant reduction. Selective reduction of multiple gestations is a controversial area in which each case requires individual consideration, weighing risks, patient values, and benefits.

This tension between patient choice (autonomy) based on the patient's intrinsic value structure and the greater ethical good of a society or the personal value structures of physicians has led to a variety of approaches in prenatal diagnosis and abortion counseling. The physician-patient counseling relationship is the last arena for consideration. The burdens and benefits of individual choices and the implications of particular choices are at issue. Although these concerns require continuous public debate to form a societal consensus of values, pressure from various segments of society to promote commercialization of prenatal selection of the “fittest” (and the implied eventual requirement to terminate undesirable fetuses, regardless of parental wishes) while restricting reproductive choice to “protect” the fetus clearly illustrates the extremes of potential harms that could intervene in the decision-making process [23]. Although authors argue the need for public policy or a “legal approach. . . to offer to our society a way to deal democratically with the societal choices related to the use of genetics,” they generally agree that in this democratic society there is no forum for this discussion as yet [24]. Given these

facts and recognizing the ethical harms that extreme legal measures could cause, the role of physician and patient counseling for responsible choices remains our principal means of addressing these issues.

The decision to undergo prenatal testing requires considered consultation between patient and physician. Clarity about the purpose and outcomes of prenatal diagnosis should be achieved before such testing is performed [15,25]. Parents seek prenatal diagnosis because of a natural human desire to have healthy offspring, but the screening tests available often suffer from low predictive value, and all involve some risk. Few well-constructed outcome studies include realistic cost comparisons. Furthermore, genetic problems identified in the future through new investigations could identify potential risks for the development of medical problems of varying severity. The choices to introduce such tests into standard prenatal “screening” present challenges as the benefits and burdens of these tests are weighed in the overlapping domains of parent and physician concerns, social demands, and financial constraints. (See Chapter 2, Prenatal Genetic Assessment, for additional discussion.)

### Second and Third Trimesters

The issues of maternal and healthcare professional obligations outlined for the first trimester change as the possibility of beneficial interventions for fetal outcome become more possible. Following the logic that the mother is the first patient, the risks, discomfort, and wishes of the mother form the strongest element in decision making. Offering interventions requires assessing risks as perceived by the patient. Often these risks are weighed in the context of the greater good for the family unit. As with all ethical questions, solutions must be based on sound medical data, with accurately known risks and success rates. This discussion considers three circumstances in which these issues might come up, with varying degrees of maternal and fetal risk, fetal benefit, and outcome information.

Consider the difficult but now progressively rare situation of severe Rh isoimmunization, in which fetal anemia is known to exist. The physician recommends percutaneous umbilical venous transfusion. The risk to the mother, according to Table 26.1, is approximately 1% to 2% per procedure depending upon the severity of the fetal condition. Without

treatment, there is a risk of fetal injury or death. The benefit for the fetus is a function of several variables but might range from 10% to 90%. The gestational age is 26 to 27 weeks. The mother refuses to undergo the procedure. She clearly has a legal right to refuse, but what are the physician and patient obligations in this case?

The physician has an obligation to offer therapy to benefit the fetus in light of the mother as patient. It is a reasonable assumption that mothers who have chosen to continue their pregnancies want a viable child and would suffer if preventable harm occurred to the fetus. The fact that one can intervene beneficially gives the fetus some standing as a patient, but the fetus has no ability to consent to or override the mother's decision. In this case, viability assumes borderline significance but is not compelling. The decision that the mother makes clearly involves considerations for her health that her healthcare givers cannot know – consideration of benefit for her overall circumstances or that of the family, for example. Given the fact that there are such clear benefits for fetal survival in this case, the American College of Obstetricians and Gynecologists (ACOG) have outlined the physician's obligations [26–30].

The ACOG Committee analysis of maternal-fetal conflicts charges the responsible clinician to be certain that the mother (as a patient) has a realistic understanding of the proposed procedure and its benefits and risks. A further requirement is to explore any potential unspoken fears, concerns, or outside pressures on her to correct any errors or assumptions that might have led her to refuse. In this sense, the ACOG Bulletin emphasizes the need for the medical caregivers to be sure that all elements of autonomy are present. Specifically, they must ensure that the patient can correlate, consider, communicate, and evaluate the quality-of-life differences in the absence of undue outside interference [11]. The interaction between patient and physician has multiple dimensions, which affect the framework from which the physician or caregiver presents the information, as well as the ability of the patient to choose rationally rather than from intuition [31]. Caregivers must recognize these issues. Recruiting a second opinion for another point of view is often helpful. Although the mother's decision holds primacy, the physician is obligated to clarify erroneous or eccentric views. Informed consent does not necessarily imply that the ultimate choice that the patient

reaches is the one that the physician views as either "good" or "reasonable." Appeal to the courts for a court order, which seems on the surface to be a "fair" resolution to a difficult situation, usually provides a vehicle for approval of the physician's choice [13]. This is not necessarily the most ethical or legally acceptable outcome. The use of the legal system to force maternal compliance is therefore discouraged.

This discussion of obligations of the physician is also pertinent to the varieties of fetal interventions available, such as prenatal treatment of a twin-twin transfusion syndrome, relief of a urinary tract obstruction, or repair of a neural tube defect [32–35,39]. (See Chapter 20, Fetal Surgery, for additional discussion.) These and other rare or investigational research in prenatal fetal surgical procedures commonly lack detailed outcome data about success, failure, or the range of possible deleterious results from the intervention. Patients often hear outcomes only as complete resolution or death, not as a potential range of outcomes. Similarly, the substantial maternal risks in such surgery are not commonly appreciated. In such a vacuum of data about outcomes, parental interest in the best outcome for the pregnancy and their children can interfere with good decision making. Much of this experimental work remains under study or protocol guidelines with institutional review board guidance of the informed consent process. Again, stringency in informed decision making is critical. Specific attention must be paid to overcoming the tendency toward an emotional basis for judgment. Researchers must be cautious because of the culturally heavy weighting with which most parents view "saving" their child [14]. Approaches to this quandary include suggestions to include pediatricians, ethicists, and other members of the healthcare team in the decision-making process [35].

What if a patient routinely behaves in a fashion that is detrimental to a pregnancy, for example, uses drugs, alcohol, or smokes [26,29,35–37]? Should physicians act to protect the fetus, even to imprison a mother to prevent potentially toxic fetal exposure? The potential harms of such physician action could extend beyond the circumstances of this one patient. Such restrictions might cause this particular patient, as well as others, to avoid care when some beneficial intervention might be negotiated otherwise. The greater harm of discouraging use of medical care has more potential harm for



greater numbers of fetuses than the benefit of incarceration for a few. Indeed, the present economic facts of the lack of adequate drug treatment programs for those who use them also argue against such an approach [36]. In a utilitarian sense, the harms outweigh the benefits. The mother remains the first patient, and her greater benefit and other potential mothers' benefits, the physician's duty.

In viewing the medical indications for benefit, the ability to predict fetal outcomes accurately based on various fetal monitoring techniques has been argued without clear resolution; therefore, the predictive value of medical recommendations and the limitations of present medical knowledge must be examined critically. The choices that a mother makes, however, are just as open to careful scrutiny. The capacity to choose responsibly in labor can be diminished. As an example, the refusal for a medical intervention after hours of labor, treatment with  $MgSO_4$ , and recent injections of a narcotic in a woman who "wanted everything done" might require using surrogate decision makers if it is clear that she has lost elements of her capability to choose.

Another set of issues about consent is raised if the patient were 14 years old. Although she might be legally an "emancipated minor," the maturity of her ability to consider complex outcomes can be tenuous. Information presented to an adolescent requires more effort and sensitivity from medical caregivers to avoid rejection and promote consideration. Two additional aspects are worthy of consideration. First, some adolescent patients have greater maturity of judgment than other chronologically older patients. Second, the consequences of choice a person makes while she is an adolescent continue into her later life, when a more mature ability to make decisions has developed. The harm of merely accepting a reactive decision by an adolescent might be not only immediate but could also affect future feelings about her own worth. In counterpoint, pressuring adolescents to acquiesce with medical decisions can also have harmful long-term psychological sequelae. The coercion might not be direct but rather a consequence of a desire to please caregivers, and a fear of abandonment if she does not.

The mother might consider the information presented to her and make an informed choice that conflicts with the recommendations of her caregivers, which could cause great anguish in the medical staff who are invested in maximizing fetal survival

while respecting maternal choice. In the author's view, however, the mother's choice still has primacy because the mother is the first patient. In the case of operative intervention, potential viability lends greater stringency to the need to be certain first about the medical indication for cesarean delivery and the diagnosis of distress. The next concern is to ascertain that the decision is being made by a patient who is capable of choosing, or by a surrogate who can represent the choices a patient would have made if she were capable.

What if the problem occurs in the last trimester of pregnancy, with a mother refusing a cesarean delivery for fetal distress? Are the concerns different because it is a third-trimester event? The court-ordered cesarean delivery is the extreme end of coercion in the obstetric setting, and the issues it raises are ethically and legally complex. The preceding discussion illustrates the possible multiplicity of observed levels of coercion by staff and diminution of capacity to choose already impeding the respect for patients' wishes or autonomy in that setting. The requirements of informed consent are intended to protect this ethical right of patients to self-determination. The legal principle often tied to these deliberations is the right to privacy, bodily integrity, and a protected liberty to refuse unwanted treatment [37]. Curtailment of this right, as in other medical situations, might be based on the ethical principles of beneficence and proportionality. As noted by Mahowald, these conflicts can be "...maternal wishes versus foetal interests where maternal welfare is not at stake..." as illustrated previously [38]. They also could be "...maternal welfare versus foetal welfare where maternal wishes give priority to the foetus..." when maternal wishes are unknown or when both maternal and fetal welfare are opposed by maternal wishes.

Adding to the confusion is the uncertain status of the fetus. The status of the fetus as a biologic reality was stated previously. As the trimesters advance, the increasing access to the fetus for beneficial medical intervention allows the fetus to assume an aspect of patienthood. This situation is still proposed as falling under maternal benefit, because an improved outcome could potentially prevent maternal suffering; however, the assessment of the proportional risk and benefit included elements of maternal values and needs that would be unknowable to her healthcare givers and that would require maternal assessment

in the informed consent process. This concern continues into the third trimester. The issue of viability has been cited as a point of compelling state's interest in potential life since *Roe v. Wade*. The state cannot trade its interest in the health and well-being of the mother in favor of benefits to the health of the fetus, however. The problems lie in the fact that fetal life is in continuity with born infant human life, and complete dissociation of the two is impossible. Furthermore, fetal life cannot be completely dissociated from maternal life, so that valuing one cannot become independent of the other.

There is no direct analogous relationship to that of mother and third-trimester fetus from which to draw legal or ethical parallels. The closest analogy proposed for legal requirements for medical care is often that of parental bone marrow donation for a dying child [1,37–38]. In both situations, a potential born child's life will be affected by a parental choice. Indeed, legally society does not force parents to donate bone marrow or kidneys to save the life of their child. This precept follows a line of thought that attaches no legal duty to a person to render needed assistance even if a life might be saved, and especially where rendering aid would put the person at some risk.

Involvement of legal processes is often evoked because of fear of liability; however, the imposition of a legal order to accept cesarean delivery (or bone marrow donation) for a patient also requires physicians to act as agents of the state rather than as patient advocates. The maintenance of the physician-patient relationship is critical to medical care. A role for the caregiver in policing a patient's behavior or choices is antithetical to that relationship and is potentially harmful if such behavior further distrust of physicians by pregnant women. Often the very women who present these issues are those who have previously had deleterious judgmental care by the medical system. As an example, physicians often believe and usually act as if maternal addiction is the product of failure of individual will power. In their judgment, physicians and other caregivers often might ignore medical facts concerning the complex hereditary, environment, and social issues that impair competency related to drug or alcohol use. Another example of the fragility of this relationship is a patient who has been sexually, psychologically, or socially abused by a spouse, family member, or physician. The further limitation of her

self-determination by a court order might cause her to become nonfunctional or even suicidal.

These comments would argue in favor of avoiding the retention of legalistic solutions to these problems and support the guidelines set by the ACOG Ethics Committee [29,30]. The benefits or harms and legal or psychological coercion for cesarean delivery must be framed by the patient's values and setting. Physicians must retain the role of patient's advocate for the patient's immediate and long-term medical benefit. For both patient and fetus, a physician obligation remains to be sure that all elements of informed consent are present. If informed consent is not possible, a reasonable attempt to find a surrogate decision maker who respects the values and choices of the patient must be initiated. In the absence of such a surrogate, information about patient values and an assumption that favors maternal benefits and the best possible maternal and fetal survival are reasonable guides to decision making. It will be a rare case that falls outside this paradigm. These rare cases, although they are of interest to philosophers and lawyers, make both "bad" law and equally suspect ethical rule when they are applied to society in general.

Strong offers two guidelines to use for exceptions to a strong prohibition against forced cesarean delivery or medical care, after careful consideration of the particular patient circumstances [37]. First, the treatment must pose no significant health risk to the woman or promote her interest in life or health. Second, compelling and usually multiple reasons to override her autonomy exist. For example, abandonment of dependent children by her death, protection of her life, and protection of the fetus's life are reasons to override autonomy, thereby promoting the well-being of the community.

## CONCLUSION

Although the weight of the original maxims proposed in the beginning of this chapter vary through different gestational ages, they remain the basis for understanding the complex nature of the maternal-fetal relationship and the ethical problems encountered in medical care. The fetus is a biologic reality that can form a continuum to a born human child. Continued fetal existence depends on an intact maternal circulation. Access to the fetus is possible only by accepting varying levels of

maternal risk. The mother is the first patient and the only one capable of autonomous decision making for herself and the fetus. Society's understanding of this relationship and of the fetus as a potential patient is being challenged and changed by technologic advances.

At all times, physicians must benefit the first patient – the mother – by offering care that advocates for the mother. Physicians can also be, secondarily, advocates for fetal well-being, but not at the expense of their obligation to the mother. If physicians can maintain this ethical focus, it is likely to encourage use of the medical care system by those wary of implied “police” activities of health-care givers. The benefits of increased access and exposure to medical care through renewal of psychological or financial barriers for the well-being of mothers, born children, and society as a whole are unquestionable. This broader societal benefit is also the physician's obligation.

## REFERENCES

1. Chervenak F, McCullough L: Does obstetrics ethics have any role in the obstetrician's response to the abortion controversy? *Am J Obstet Gynecol* 1990; 163:1425–1429.
2. Ryan KJ: Abortion or motherhood, suicide and madness. *Am J Obstet Gynecol* 1992;Apr;166(4): 1029–1036.
3. Curran C: Contemporary debate in philosophy and religious ethics. In: Reich W (ed): *Encyclopedia of Bioethics*. New York: Macmillan, 1978, pp. 17–26.
4. Thompson JJ: A defense of abortion. *Philos Public Aff* 1971;1:217–234.
5. Engelhardt HT Jr: The ontology of abortion. *Ethics* 1974;84:217–234.
6. Engelhardt HT Jr: Viability, abortion, and the difference between a fetus and an infant. *Am J Obstet Gynecol* 1973 Jun 1;116(3):429–434.
7. Harris LH: Rethinking maternal fetal conflict: Gender and equality in perinatal ethics. *Obstet Gynecol* 2000 Nov; 96(5 Pt 1):786–791.
8. Byk C: The Human Genome Project and the social contract: a law policy approach. *J Med Philos* 1992 Aug;17(4):371–380.
9. Jonsen AR: Ethics of reproductive technology: the deconstruction of a paradigm. *Logos* (Santa Clara) 1988;9:3–9.
10. Jonsen A, Siegler M, Winslade W: *Clinical Ethics*, 3rd ed. New York: McGraw-Hill, 1992.
11. Meyer M: Patients' duties. *J Med Philos* 1992 Oct; 17(5):541–555.
12. Chervenak FA, McCullough LB: Implementation of first-trimester risk assessment for trisomy 21: Ethical considerations. *Am J Obstet Gynecol* 2005 Jun; 192(6):1777–1781.
13. Sharma G, McCullough LB, Chervenak FA: Ethical considerations of early (first vs. second trimester) risk assessment disclosure for trisomy 21 and patient choice in screening versus diagnostic testing. *Am J Med Genet C Semin Med Genet* 2007 Feb 15; 145(1):99–104.
14. Dworkin R: *Life's Dominion: An Argument About Abortion, Euthanasia and Individual Freedom*. New York: Alfred Knopf, 1993.
15. Steinbock B: *Life Before Birth: The Moral and Legal Status of Embryos and Fetuses*. Boston: Oxford University Press, 1992.
16. Robertson JA: Casey and the resuscitation of Roe v. Wade. *Hastings Cent Rep* 1992 Sep–Oct;22(5):24–28.
17. Zarutskie PW, Muller CH, Magone M, Soules MR: The clinical relevance of sex selection techniques. *Fertil Steril* 1989 Dec;52(6):891–905. Review.
18. Johnson SR, Elkins TE: Ethical issues in prenatal diagnosis. *Clin Obstet Gynecol* 1988 Jun;31(2): 408–417.
19. Wertz D, Fletcher J: Fatal knowledge? Prenatal diagnosis and sex selection. *Hastings Cent Rep* 1989;19: 21–27.
20. Blank RH: *Regulating Reproduction*, 1st ed. New York: Columbia University Press, 1990. pp. 45–91.
21. Evans MI, Fletcher JC, Zador IE, Newton BW, Quigg MH, Struyk CD: Selective first-trimester termination in octuplet and quadruplet pregnancies: Clinical and ethical issues. *Obstet Gynecol* 1988 Mar;71(3 Pt 1):289–296.
22. American College of Obstetricians and Gynecologists: *Multifetal pregnancy reduction in Ethics in Obstetrics and Gynecology*, Second Edition. Washington, DC. American College of Obstetricians and Gynecologists Epub www.acog.org 2007.
23. Gates E: Maternal choice, will it work both ways? *Women's Health Issues* 1990;1:25–27.
24. Chescheir N, Cefalo R: Prenatal diagnosis and caring. *Women's Health Issues* 1992;2:123–132.
25. Nolan K: First fruits: genetic screening. *Hastings Cent Rep* 1992;22(suppl):S2–S4.

26. American College of Obstetricians and Gynecologists. Maternal Decision Making, Ethics, and the Law: Committee Opinion. Washington, DC: American College of Obstetricians and Gynecologists, 321, November 2005.
27. American College of Obstetricians and Gynecologists: Using Preimplantation Embryos for Research. Committee Opinion. Washington, DC: American College of Obstetricians and Gynecologists, 347, November 2006.
28. American College of Obstetricians and Gynecologists: Code of Professional Ethics. Washington, DC: American College of Obstetricians and Gynecologists. January, 2004.
29. American College of Obstetricians and Gynecologists: Ethical Decision Making in Obstetrics and Gynecology Committee Opinion, 390, 2007. Washington, DC: American College of Obstetricians and Gynecologists.
30. American College of Obstetricians and Gynecologists: Ethics in Obstetrics and Gynecology, Second Edition., Washington, DC: American College of Obstetricians and Gynecologists. (www.acog.org), Epub, 2007.
31. Redelmeier DA, Rozin P, Kahneman D: Understanding patients' decisions. Cognitive and emotional perspectives. *JAMA* 1993 Jul 7;270(1):72–76. Review.
32. Elias S, Annas GJ: Perspectives on fetal surgery. *Am J Obstet Gynecol* 1983 Apr 1;45(7):807–812. No abstract available.
33. Ruddick W, Wilcox W: Operating on the letus. *Hastings Cent Rep* 1982 Oct 12;(5):10–14.
34. American College of Obstetricians and Gynecologists: Neural Tube Defects, Number 44, July 2003. Washington, DC: American College of Obstetricians and Gynecologists.
35. American Academy of Pediatrics Committee on Bioethics: Fetal therapy – ethical considerations. *Pediatrics* 1999 May;103(5 Pt 1):1061–1063.
36. Chavkin W: Drug addiction and pregnancy: Policy crossroads. *Am J Public Health* 1990 Apr;80(4):483–487. Review.
37. Strong C: Court-ordered treatment in obstetrics: The ethical views and the legal framework. *Obstet Gynecol* 1991 Nov;78(5 Pt 1):861–868.
38. Mahowald M: Beyond abortion: Refusal of caesarean section. *Bioethics* 1989 Apr;3(2):106–121.
39. Chervenak FA, McCullough LB. Ethics of maternal-fetal surgery. *Semin Fetal Neonatal Med* 2007 Dec; 12(6):426–431. Epub 2007 Aug 2.
40. Committee on Ethics, ACOG Committee Opinion Number 369, June 2007. Multifetal pregnancy reduction. *Obstet Gynecol* 2007 Jun;109(6):1511–1515.
41. Chervenak FA, McCullough LB, Levene MI: An ethically justified, clinically comprehensive approach to peri-viability: Gynaecological, obstetric, perinatal and neonatal dimensions. *J Obstet Gynaecol* 2007 Jan;27(1):3–7. Review.
42. Vavasseur C, Foran A, Murphy JF: Consensus statements on the borderlands of neonatal viability: From uncertainty to grey areas. *Ir Med J* 2007 Sep;100(8):561–564.
43. Philip J, Silver RK, Wilson RD, Thom EA, Zachary JM, Mohide P, Mahoney MJ, Simpson JL, Platt LD, Pergament E, Hershey D, Filkins K, Johnson A, Shulman LP, Bang J, MacGregor S, Smith JR, Shaw D, Wapner RJ, Jackson LG, NICHD EATA Trial Group: Late first-trimester invasive prenatal diagnosis: Results of an international randomized trial. *Obstet Gynecol* 2004 Jun;103(6):1164–1173.
44. Kuliev A, Jackson L, Froster U, Brambati B, Simpson JL, Verlinsky Y, Ginsberg N, Smidt-Jensen S, Zakut H: Chorionic villus sampling safety. Report of World Health Organization/EURO meeting in association with the Seventh International Conference on Early Prenatal Diagnosis of Genetic Diseases, Tel-Aviv, Israel, May 21, 1994. *Am J Obstet Gynecol* 1996 Mar;174(3):807–811.
45. Jackson LG, Zachary JM, Fowler SE, Desnick RJ, Golbus MS, Ledbetter DH, Mahoney MJ, Pergament E, Simpson JL, Black S, et al: A randomized comparison of transcervical and transabdominal chorionic-villus sampling. The U.S. National Institute of Child Health and Human Development Chorionic-Villus Sampling and Amniocentesis Study Group. *N Engl J Med* 1992 Aug 27;327(9):594–598. PMID: 1640952.
46. Natarajan G, Klein MD: The fetus as a patient: Prenatal diagnosis and fetal therapy. *emedicine*, 2006 (www.imeicine.com topic = 2953).

## Shanna L. Burke

*Is it nothing to you, all ye that pass by?  
Behold and see if there be any sorrow  
like unto my sorrow, which is done unto me,  
Wherewith the Lord hath afflicted me in the  
day of His fierce anger.*

Lamentations: 1:12  
The Holy Bible

Perinatal loss is a traumatic and often life-altering experience for women and their families [1–4,8,12,14,15,17,18]. This is in part because there is a unique set of emotions that is attached to losing a fetus or infant and because this loss greatly disrupts the family narrative [3]. Unfortunately, the effects of the loss of a fetus, a stillbirth, or the death of an infant are neither fully recognized nor socially legitimized, and because of this, support by other family members, friends, and even health professionals is often limited. An important reason for this is the general lack of understanding that establishing a relationship to a child begins long before birth [2].

Our response to perinatal loss reflects concepts of death in American society and culture [5–7]. In our society, death remains a proscribed subject. In 1968, Lifton suggested reasons why death is a taboo subject [7]:

- *Urbanization* – A growing number of Americans have moved to urban environments, away from more natural locales where the normal cycle of life and death is witnessed.
- *Exclusion of the aged and dying* – People of advanced ages are segregated into hospitals, hospice centers, or nursing homes and places where the general public is not required to see them.
- *Movement away from multigenerational homes* – Extended families are now less likely to live close by or under the same roof. Thus, when elderly family members die, these events are largely relegated to hospitals, nursing homes, and other skilled nursing facilities.
- *Secularization in modern society* – Religion is a way to deemphasize death and focus on the afterlife and immortality of the soul. Although for some religion still functions in this way, for many Americans this coping mechanism is either weak or nonexistent.
- *Advances in medical technology* – Advances in medical technology provide parents with the potential for control over reproductive decisions. Given the

change in laws (i.e., *Roe v. Wade* in 1973), parents can now act to control the fate of a pregnancy. As Lifton argues, "... these advances have compromised the ability to understand death as a natural part of human life".

Clinicians frequently have difficulty with perinatal deaths. Little in their training prepares them to face this issue. Fetal or neonatal loss makes even professional health practitioners feel uncomfortable and uncertain. Work in recent decades has emphasized the importance of grieving rituals and, in the response to perinatal loss, recent experience favors proactive involvement with affected families, despite the difficulties that this presents for the birth attendants. Recognizing the importance of death and the historical limitations of medical specialists in facing these issues, this chapter explores the literature concerning perinatal losses, grief, and bereavement. In this process, the role of the professional caregiver in family counseling and support is discussed in detail. Recommendations for practice are made, and, finally, a bibliography for women and their families is provided.

## DEFINITION OF TERMS

*Grief* is best understood as the emotional, mental, and physiologic reactions to a loss [5]. Because perinatal and neonatal losses are often unexpected, the grief reaction is complicated, often prolonging the distress. *Mourning*, which differs from grief, is the outward expression of the person's internal feelings. Many factors, including culture, societal norms, gender, and forms of personal expression, influence the mourning process.

### History: The Stages of Grief

It was not until the 1970s that grief and death became a subject for public discussion in the United States and that bookstores began to carry texts for bereaved parents as well as for professionals concerning these issues. This development accompanied a shift from religious discussions of grief to more academic, candid, and nondenominational approaches.

Freud was the first to attempt to outline task-oriented steps in grieving. He thought that it was vital to "... disengage from the relationship with the deceased in order to place libidinal energy into a new

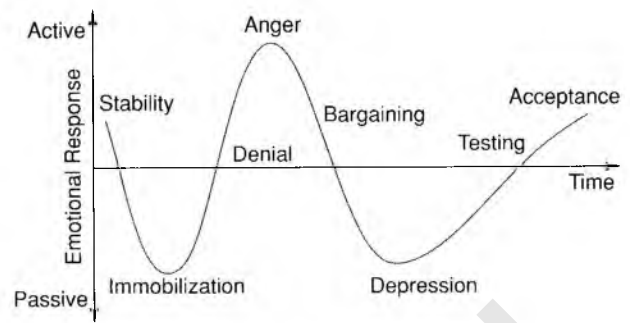


FIGURE 27.1.

*The grief cycle. (From Straker D. The grief cycle [updated 2006; cited 2006 Nov 28]; [http://changingminds.org/disciplines/change\\_management/kubler\\_ross/kubler\\_ross.htm](http://changingminds.org/disciplines/change_management/kubler_ross/kubler_ross.htm); with permission.)*

relationship . . ." [8]. This disengagement was characterized as a *task*, whereas separation and abandonment were the ultimate objectives in Freud's version of *grief work*. Of note, however, is that Freud did not study mourning empirically, and that the loss of his daughter, Anna Freud, occurred after he had finished his bereavement writings.

A pivotal event in opening the issues of death and grieving to the general public was the influential book, *On Death and Dying: What the Dying Have to Teach Doctors, Nurses, Clergy, and Their Own Families*, by Elisabeth Kübler-Ross. This popular text evoked a flurry of interest and prompted other critiques and commentaries [6].

In her initial original formulation, Elisabeth Kübler-Ross identified five stages of grief: 1) denial and isolation, 2) anger, 3) bargaining, 4) depression, and 5) acceptance (Figure 27.1). These stages were developed originally as the result of work with terminally ill patients and their families. Despite limitations, they do have universal application and can be creatively applied to fetal and neonatal loss and the grief process as experienced by the surviving family members.

### Denial

*Denial* is the "No, not me" thought: the lack of acceptance that death has occurred. Denial is the result of an initial shock to the psyche and is a refusal to accept a change in one's world. In the case of perinatal loss, the death could have occurred from a spontaneous miscarriage, an ectopic pregnancy, or an induced abortion. Apart from cases of pregnancy termination, perinatal losses are often unexpected

and the fetus might have already been dead for some time by the time of diagnosis.

### **Anger**

*Anger* is the “Why me?” step. A bad outcome is usually accompanied by a sense of resentment because “. . . other people’s pregnancies have turned out well, but mine did not. . .” Often this anger is displaced at God or whatever deity or spiritual figure the couple embraces in their lives. The couple might view this spiritual deity as an “all-powerful” figure and the “enforcer of the death sentence” on their child. Anger is inevitable at one point or another. Couples often need permission to feel the full force of their anger at this difficult time.

In the hospital setting, this anger is often displaced toward nurses and other medical staff, including the perinatologist or obstetrician. This anger could include accusations of misdiagnosing or mishandling the problem: in sum, not doing enough to save the fetus or child.

### **Bargaining**

*Bargaining* is the “Yes, this is my situation, but. . .” step. In this stage, a couple might accept that their fetus/child is dead but try to strike bargains with God about future children, children that have already been born, or for more time with the deceased child. Parents might also find themselves asking that their lives be taken instead of that of their child.

### **Depression**

*Depression* is the “I-don’t-care-anymore” phase, when the parents begin the actual mourning process, when the grief is internalized. After anger and bargaining, the depressed person becomes despondent and might push others away. This depression is noticed in passive behavior and in actions or somatic complaints.

### **Acceptance**

*Acceptance* is the “Yes, me” stage of grief, in which the couple have mourned their loss and can move on with their lives. To reach this point of partial resolution, the parents must have acknowledged that

the loss has occurred. Although their loss still lingers in memory, it is no longer a constant thought. Once this stage is reached, people begin to move on with their lives, take ownership for their actions, and heal.

## **RETHINKING THE STAGES OF GRIEF**

In their 2005 book, Elisabeth Kübler-Ross and David Kessler [9] responded to the critics of their original compartmentalized hierarchy of grieving, stating:

**The stages have evolved since their introduction, and they have been very misunderstood over the past three decades. They were never meant to help tuck messy emotions into neat packages. They are responses to loss that many people have, but there is not a typical response to loss, as there is no typical loss. Our grief is as individual as our lives.**

The five stages – denial, anger, bargaining, depression, and acceptance – are a part of the framework that makes up learning to live with the fact that there is one who has been lost. These stages are best considered as tools to help people frame and identify what they are feeling and how these emotions relate both to the lost object as well as to the future. The proposed stages are not stops on some linear timeline in grief, however. Not everyone with a loss goes through each or all of these stages in a specific order.

In an analysis of the process of loss and resolution, Worden [10,11] argues that the completion of certain tasks is necessary for the grieving process to be completed. These tasks include:

- Acceptance of the loss
- Experiencing the pain of the grief
- Adjusting to the new environment without the deceased
- Commemorating and memorializing the loved one and integrating the loss into one’s life, while reinvesting the energy into something else in one’s life

Unfortunately, the original writings of Kübler-Ross [6], the revised statements made by Kübler-Ross and Kessler [9], and the task-oriented process

outlined by Worden [10,11] do not reflect the “lived experiences” as reported by many parents experiencing perinatal losses. These writings still indicate that after the grief work is complete, one should be able to “get over their loss.” The unfortunate reality for most families with a perinatal loss is that active grieving never entirely disappears nor does the distress completely heal [5].

### EFFECTS ON HEALTH PRACTITIONERS

Perinatal loss also has a profound effect on the members of the obstetric team. The delivering obstetrician or midwife often faces a complex and emotionally stressful task when an acute loss occurs. Personal distress, self-doubt, and anger are common reactions. Knowing what to say or do is often difficult, especially when the parturient is not well known to the clinician, when there is a language barrier, or when the family is from another culture, with very different norms and practices.

Cultural factors are important in how grief is experienced. As an example, as Chan and coworkers report, death is a taboo subject in the Chinese culture [1]. There is the prevailing notion that one should not discuss death in front of the sick and dying or their family. Confucianism and Buddhism play a large role in the daily living of many Chinese people, including bereavement and end-of-life care. Nurses and other staffers that come in contact with the family must be able to sense subtle emotional innuendos. It is common within the Chinese culture for emotions to be masked and not outwardly expressed; emotional problems and issues are often disguised as somatic complaints. The important features of each person’s culture and religion must be understood. Sample questions include the following [25]:

- Can you help me to better understand your beliefs that could affect funerals, burials, or a preference for cremations?
- Does your family prefer to observe these practices, or has your family developed their own rituals?
- Is there anyone else to whom you think I should speak to learn more about these practices?
- What are your beliefs about death and the afterlife?

### THE GRIEVING MOTHER

Perinatal losses and the birth of premature or precarious infants are all-too-common events. More than one in four pregnancies end before term in either a spontaneous miscarriage or are purposely ended by physician intervention as a result of fetal abnormalities, perceived fetal jeopardy, maternal jeopardy, or other obstetric complications [12,13]. While current technology often leads to intervention, it also provides new windows into fetal life, directly involving women with their pregnancies at a much earlier time than was possible in a prior generation. At present, women commonly view their fetuses by means of real-time ultrasound scanning at one or more times during their pregnancies. This fosters an additional layer of attachment that complicates grief when an unanticipated loss occurs. Although a substantial percentage of pregnancies end with embryonic or fetal loss, little attention is given to the grieving process in early pregnancies for the mother or the family that she imagined the fetus would one day join. This is unfortunate because maternal grief over a miscarriage or induced abortion is emotionally equivalent to the loss of a stillborn child or the death of an infant [2,14,15].

Although the embryo might not have fully developed at the time of a miscarriage, the mother has already created expectations for motherhood and plans for the child. In addition, if the mother had previous feelings of ambivalence about the pregnancy, her guilt might increase, as will feelings of fury, umbrage, and sorrow. Psychologically, the mother views the developing embryo as a child. As a result, in a miscarriage she feels a loss comparable to that of a mother losing a full-term infant. The grieving mother might have a response triggered by thoughts about what the child might have been, might have done, or how he or she could have contributed to the world. Last, guilt feelings often prevail. The mother might feel like a failure because of her incapacity to carry a child successfully. In a study conducted by Seibel and Graves involving women with pregnancy loss, one fourth fully thought that they were responsible in some way for their miscarriage [17].

Following a loss, it is often difficult for women to return home or to their work lives [2]. They feel incomplete or like a failure, with “nothing to show” for their many weeks of pregnancy. As with



situations of women who miscarry, women experiencing a stillborn might feel that they are defective or are failures because of their inability to have a healthy surviving child. Couples and their families often feel isolated because others might not understand the need or existence of grief. Because the child never existed to those outside the close family relationship, it is difficult for outsiders to fully grasp the impact of the loss. An often unresolved issue is the family unit. Siblings must be involved in the mourning process. If other children felt envy or angry feelings towards the new baby, this might make them feel responsible and guilty now that the infant has been lost [2]. Despite pregnancy loss and often prior to complete resolution of the grieving process, many women go on to conceive again. Over one half of the women who experience perinatal loss will become pregnant again within 22 months [26]. Most of those who do not become pregnant within the first two years appear to do so by choice rather than due to a biologic inability to reproduce.

### ***Stillbirth***

Because perinatal loss is often sudden and contrary to the expectations of motherhood, it can have a particularly devastating and traumatic affect on the mother. Whether a loss results from either an induced or spontaneous delivery, women with stillborns suffer substantial psychological morbidity, with feelings of fear, anxiety, and perceived helplessness [1].

Grout and Romanoff [3], focusing on the changing perception of the mother's world, state:

**Parents, particularly mothers, spend the pregnancy preparing for the new baby. This psychological preparation includes constructing a representation of the new family member, based on prior experiences with babies, experiences from the family of origin, and indirect experience with the baby-to-be. . . . It may also include daydreaming through the expectations for the new baby, imagining oneself in a new social role (especially if the baby is a first-born), and using stories with siblings to help them to create a representation of the anticipated baby. . . . When the baby dies before or soon after birth, this process is abruptly**

**halted. Parents have fewer mental representations of their dead baby with which to do the work of grief.**

Important elements in predicting a woman's reaction to a perinatal demise include: [26]

- The extent of the attachment to the baby
- The degree of investment in the pregnancy
- The gestational age

Stillbirths intensify the emotional issues around childbearing. In more advanced pregnancies an elaborate bond has formed between mother and fetus. By the time of the loss, baby showers and other culturally bound celebrations of birth have often occurred. Furthermore, the entire pregnancy might have been problem free until the loss. With the mother and family so heavily invested in the pregnancy psychologically, an unanticipated loss is perceived as particularly traumatic and can be emotionally devastating.

### ***Twins and Higher-order Multiples***

The loss of an infant in a multiple gestation raises special issues. The grief of these parents is sometimes underestimated, especially if a singleton survives, and their grief also can be different from that of mothers who experience the loss of one child [27]. Parents might essentially be discouraged from grieving openly when there is a loss of one of a multiple gestation. The strong bias is to focus instead on the surviving infant (or infants) or other children in the home. The discomfort of others also motivates the parents to keep silent and mourn quietly and alone [27–30]. Parents who lose multiples might be grieving not only the loss of the infants themselves but also the loss of their unique status as parents of twins.

In the experience of the author, health practitioners have difficulty relating to families who have lost one or more in a multiple gestation. This avoidance has several roots. Inadequate training and supervision, as well as a general discomfort with death are part of the problem. Also, practitioners commonly experience a greater sense of guilt in such cases because of the known increased risk for these pregnancies, which were supposed to be under close surveillance by medical personnel. Thus the loss

implies either a failure of proper diligence on the part of the caregivers or a failure of the methods used for fetal evaluation. Even when bereaved parents of multiples are pleased with services provided in the prenatal and labor and delivery departments, the parents who have lost a fetus or an infant from a multiple gestation were still more dissatisfied with the support they received compared with parents who had lost a singleton [31].

### THE GRIEVING FATHER

There are distinct differences in the grief suffered by fathers. These differences are only partially attributable to the differences in the physical experiences with the pregnancy [46,47]. The bereavement of fathers is strongly affected by prevailing American sociocultural expectations of masculinity.

Fathers' experiences are quite different from mothers' with a pregnancy, especially with infants who are live born but not expected to live. In fact, men are outwardly less affected by a perinatal death, partly because they are experienced at hiding their emotions and their reactions to all tragic events, including a perinatal loss. As reported by the fathers themselves, their grief is often shorter and less intense than that experienced by the mother. This grief is often described by the fathers as intermittent, because the pregnancy itself, to them at least, was intermittent. Staudacher describes the difference for the mother and the father [19]. She writes

**The intensity of that experience and the time spent with the child allow the mother to see that the body the child is in is just not going to last; it's not worth trying to hang on to the dying child. The father, on the other hand, who spends less time with the child, may be removed enough from the situation that he'll try to hang on longer. He'll take a longer time to accept the inevitable before he goes through the detachment process.**

Although Staudacher is clearly describing an anticipated death in this case, the opposite reaction can be expected from fathers in the case of a sudden death, such as a miscarriage or stillbirth.

When there is a miscarriage or a stillborn child, fathers are often overlooked during mourning, even

more so than if the death involved an older child. In addition, because of their apparently muted response, fathers often dismiss or underestimate their own grief. Because of this they can receive little social support. The focus of concern is frequently the mother's physical health. Reflecting the fact that outward mourning is not socially acceptable and that men are expected to be protectors and grief managers, the father is expected to protect his family. In times of grief this translates to accepting a major role in family decision making and in protecting his family from the deep emotional sadness and stress of the situation. The father therefore might be required to make decisions and in fact might seek decisions to make about his wife's care, the child's burial, and many other matters. Although this frenetic activity occupies his time and mind, it can also serve to increase his stress.

As part of his management tasks, the father is also expected to somehow handle his family's grief, even though he is also coming to grips with his own bereavement [19]. Fathers commonly attempt to help comfort and control the grief of others to shorten the grieving period and "make it easier."

Many fathers report a need to keep busy during their own grieving period. One father reported doing projects that had a very distinct beginning and ending as he struggled to seek a way of achieving a sense of both satisfaction and control [16].

In the case of a miscarriage or stillbirth, many fathers report feeling guilty, at least partially because of having impregnated their wives. This adds to the emotional tumult the couple is experiencing. The father might also feel guilty because he wanted to have sex during the pregnancy, or that he did not spend enough time at home, or was "not otherwise there enough" for the woman emotionally.

### BEST PRACTICES FOR HOSPITAL STAFF

It is usually the task of the attending physician to be the first to notify the family that a perinatal death has occurred. The attending physician should relay this terrible news to the family with as much empathy as possible. For the family, it is as if "... the door to dreams and opportunities briefly opened and had been cruelly and [un]accountably shut..." [23]. Hospital personnel have a special and important role in perinatal grief and mourning. In 1983, Gardner surveyed 100 couples that had experienced a

stillbirth [20]. Couples identified as most helpful the nurses who tended to their needs after the child's death; however, many other hospital personnel also play a role in bereavement.

When stillborns occur, there are institutional and usually state requirements for reporting and the handling of remains. In the Commonwealth of Massachusetts, a fetal death is defined as, "... death prior to the complete expulsion or extraction from its mother of a fetus, irrespective of the duration of pregnancy... that... does not breathe or show any other evidence of life such as beating of the heart, pulsation of the umbilical cord or definite movement of voluntary muscles" [21]. Other states have regulations that are different in wording. Depending upon the circumstances at the time of delivery, the gestational age and whether the legal definition of a stillborn is met, specific legal forms may need to be prepared. Institutional policies may require the collection of samples and usually define how the fetus and placenta are to be handled. Clinicians should be aware of these requirements and protocols not only to be in compliance with local law but also to avoid potentially avoidable subsequent problems with documentation. Beyond the issues of grief, it is well to recall that fetal loss can have medical-legal importance, emphasizing the need for full and accurate recording at the time of delivery.

Following local requirements, the attending clinician present at the birth may be required to complete a fetal death report, which is filed with the state public health department. In the author's hospital in the Commonwealth of Massachusetts, depending on the clinical classification of the perinatal death, there are different requirements for reporting. Because necessary procedures are state and institution specific, clinicians must remain aware of the legal and protocol requirements in their area.

### ***Bereavement Protocol***

Virtually all delivery services have developed formalized bereavement policies, specifying which steps should be taken when stillbirths occur. The recommendations made by most institutions include many of the elements in the following list, derived from the author's hospital policy book [22]:

- All infants are weighed, measured, and given an estimation of gestational age.
- Pictures are taken of the infant and either given to the parent(s) at the time of delivery or kept on file for 6 months with the name and medical record number attached. If and when the parents decide to ask for these photos, they will be available.
- The nurse manager, clinical supervisor, and medical director are notified of the loss.
- A spiritual care or a bereavement counselor is notified.
- The remains are prepared in the following manner, in case an autopsy is to be performed:
  - The infant has a light coating of baby oil applied so that the blankets do not stick to the skin.
  - The infant is diapered, swaddled in a blanket, identified with a toe tag, and also another tag on the outside of its blanket, and taken to the morgue.
  - An autopsy permit is obtained and documented on the chart with notification of the medical examiner, if applicable.
- A death certificate is made available for the attending physician to fill out, with the autopsy permit (if appropriate) in accordance with the family's decision and state statutes.
- Documentation of the parents' wish for either burial or hospital disposition is included with the forms.
- The name of the funeral director is obtained, if applicable.

### **How to Talk to the Grieving Family**

Many studies reiterate the importance of health practitioners' sensitivity and honesty when delivering potentially devastating information, such as a perinatal death [32–45]. In talking to a grieving family, health practitioners should always be honest, but they must combine this with empathy. Expressing empathy is often difficult, especially when perinatal and neonatal deaths occur, often because the practitioner and the attending physician or care provider have experienced burnout from this issue. It is always appropriate and recommended to

- Parents are allowed to view, hold, and bond with their infant.

express one's condolences, but it is not advisable to say anything such as, "You can always have another child," "It was fate [God's will]," or "Things are better this way." To the grieving family, these statements convey a lack of empathy and might not always ring true, because the mother might be unable to have another child or might not believe in fate or God.

A few simple questions posed to the grieving family can usually begin a conversation and help the professional healthcare worker gain knowledge and insight about the grieving mother or family's personal grieving experience. Some examples of questions include [25]:

- Was this death expected?
- Did you have any warning?
- (If the infant died in the hospital) Were you there at the time of death? What was that like for you? If you were not there, how do you feel about that?
- Do you have any plans for a wake, memorial service, or a funeral?
- Have you decided to see or hold the baby?

If possible, a follow-up assessment should be done a few weeks after the infant's death. In this post-death period, the following information should be assessed [25]:

- What has been happening since the death?
- How have things been between you and your family? How have things been for you and your partner [if applicable]?
- How have people been reacting to you since the loss?
- How have things been at school, at work, at \_\_\_\_\_?

It is also advisable to gather information related to the mother's physical health, because her grieving could affect this as well. Suggested questions include [25]:

- Have you been able to return to your usual activities?
- How have you been sleeping?
- How has your appetite been?
- How would you describe your energy level?

- Does anything seem to help you to feel better?
- When is it hardest for you?

These questions convey empathy and serve as an excellent tool. They also permit the grieving woman to feel that her concerns and problems have at least been heard, even if they cannot be easily resolved.

## CONCLUSION

Perinatal loss is always difficult. At such times, families need their doctor, midwife, or nurse to be closer to them and provide support. Clinicians find it all too easy to retreat from this difficult task, to make themselves busy, and to believe that "leaving them alone is best." This is a time when the work of grieving must be shared, however. The family long remembers all the events of the birth or miscarriage, and the psychological scars are deep. As professional people, if a physician or other health practitioner can treat to save life or reduce injury, he or she must do so. Similarly, if a life is lost, it remains the health practitioner's responsibility and privilege to support the living and provide them with both strength and guidance.

## RESOURCES FOR PATIENTS

### National Support Groups

Bereaved Parents of the USA (BPUSA)  
National Headquarters  
P.O. Box 95  
Park Forest, IL 60466  
Ph: 708-748-7672

Center for Infant and Child Loss  
639 West Fayette Street, Room 5-684  
Baltimore, Maryland 21201-1585  
Ph: 410-706-5062

CLIMB: Center for Loss in Multiple Births  
P.O. Box 91377  
Palmer, Alaska 99509  
Ph: 907-222-5321  
Website: [www.Climb-support.org](http://www.Climb-support.org)

HAND: Helping After Neonatal Death  
P.O. Box 341  
Los Gatos, CA 95031  
Ph: 800-963-7070

National Council of Jewish Women  
New York Section  
Pregnancy Loss Support Program  
820 Second Avenue  
New York, NY 10017-4504  
Ph: 212-687-5030

Pregnancy and Infant Loss Center, Inc.  
2070 Chain Bridge Road, Suite 450  
Vienna, VA 22182  
Ph: 612-473-9372

Resolve, Inc. (Infertility)  
1310 Broadway  
Somerville, MA 02144-1731  
Ph: 617-623-0744

SHARE: Pregnancy and Infant Loss Support, Inc.  
St. Joseph Health Center  
300 First Capital Drive  
St. Charles, MO 63301-2893  
Ph: 800-821-6819

Sidelines National Support Network  
(for complicated pregnancies, pregnancy after loss)  
P.O. Box 1808  
Laguna Beach, CA 92652  
Ph: 714-497-2265  
Fax: 714-497-5598

Sudden Infant Death Syndrome Alliance  
1314 Bedford Avenue, Suite 210  
Baltimore, MD 21208  
Ph: 800-221-SIDS

The Tenderhearts Support-Triplet Connection  
P.O. Box 99571  
Stockton, CA 95209  
Ph: 209-474-0885

### Books

Canfield, Jack, Hansen, Mark V. *Chicken Soup for the Grieving Soul: Stories about Life, Death, and Overcoming the Loss of a Loved One.* Deerfield Beach, FL: Health Communications, 2003.

Davis, Deborah. *Empty Cradle, Broken Heart.* Golden CO: Fulcrum, 1996.

Finkbeiner, Ann. *After the Death of a Child: Living with Loss through the Years.* Baltimore, MD: John Hopkins University Press, 1998.

Fumia, Molly. *A Piece of My Heart: Living through the Grief of Miscarriage, Stillbirth, or Infant Death.* Berkley, CA: Conari Press, 2000.

Isle, Sherokee. *Empty Arms: Coping with Miscarriage, Stillbirth, and Infant Death.* Itasca, IL: Wintergreen Press, 1992.

Kluge-Bell, Kim. *Unspeakable Losses: Understanding the Experience of Pregnancy Loss, Miscarriage, and Abortion.* New York: W. W. Norton & Co., 1998.

Kohn, Ingrid, Moffit, Perry-Lynn. *A Silent Sorrow.* New York, NY: Routledge, 2000.

Lafser, Christine O'Keefe. *An Empty Cradle, A Full Heart: Reflections for Mothers and Fathers after Miscarriage, Stillbirth, or Infant Death.* Chicago: Loyola, 1998.

Lanham, Carol Cirulli. *Pregnancy after Loss: A Guide to Pregnancy after Miscarriage, Stillbirth, or Infant Death.* New York: Berkley Publishing Group, 1999.

Nelson, James D. (ed). *The Rocking Horse is Lonely – and Other Stories of Father's Grief.* Wayzata, MN: Pregnancy and Infant Loss Center, 1994.

Schweibert P, DeKlyen C, Bills T. *Tear Soup: A Recipe for Healing after Loss* (3rd rev ed). Portland, OR: Grief Watch, 2005. <http://www.griefwatch.com/detail.aspx?ID=60>.

Strommen, Merton P., Strommen, A. Irene. *Five Cries of Grief: One Family's Journey to Healing after the Tragic Death of a Son.* San Francisco: Harper, 1993.

### VIDEO

Paraclete Video Productions. *Footprints on Our Hearts: How to Cope After a Miscarriage, Stillbirth, or Newborn Death [DVD].* Orleans, MA: Paraclete Press and Paraclete Video Productions.

### INTERNET RESOURCES<sup>1</sup>

Hygeia Foundation, Inc. and the Institute for Perinatal Loss and Bereavement. [[www.hygeiafoundation.org](http://www.hygeiafoundation.org)]

The Forgotten Grief. [[www.forgottengrief.com](http://www.forgottengrief.com)]

Grief Watch Perinatal Loss: Infant Loss Recourse for Bereaved Families and Professional Caregivers. [[www.griefwatch.com](http://www.griefwatch.com)]

<sup>1</sup>Please note that these are suggested Web sites, and viewer discretion and caution must be used, because the author has no connections to the webmasters of these suggested web sites. Please view at your own risk.

National Council of Jewish Women – Pregnancy Loss Support Program (PLSP) for Miscarriage, Stillbirth, and Newborn Death. (provides nationwide telephone counseling as well). [www.ncjwny.org/services.plsp.htm]

A Place to Remember: Uplifting support materials and resources for those who have been touched by a crisis in pregnancy or the death of a baby. [www.aplacetoremember.com]

The International Council on Infertility Information Dissemination, Inc.: Miscarriage and Pregnancy Loss. [www.inciid.org/index.php?page=miscarriage]

Storknet: Pregnancy ~ Infant Loss Cubby. [www.storknet.com/cubbies/pil/]

The Ectopic Pregnancy Trust – [www.ectopic.org.uk]

Hannah's Prayer – Christian support for fertility challenges, including infertility or the death of a baby at any time from conception through early infancy. [www.hannah.org]

EriChad Grief Support: home of loving support for bereaved parents. [www.erichad.com]

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## REFERENCES

1. Chan MF, Wu LH, Day MC, Chan SH. Attitudes of nurses toward perinatal bereavement: Findings from a study in Hong Kong. *J Perinat Neonat Nurs* 2005 Jul-Sep;19(3):240–252.
2. Rando TA. *Grief, Dying, and Death: Clinical Interventions for Caregivers*. Champaign, IL: Research Press Company, 1984.
3. Grout LA, Romanoff BD. The myth of the replacement child: Parents' stories and practices after perinatal death. *Death Studies* 2000 Mar;24(2):93–113.
4. Toedter LJ, Lasker JN, Janssen HJE. International comparison of studies using the perinatal grief scale: A decade of research on pregnancy loss. *Death Studies* 2001 Apr–May;25(3):208–228.
5. Walsh-Burke K. *Grief and Loss: Theories and Skills for Helping Professionals*. Boston: Pearson, 2006.
6. Kübler-Ross E. *On Death and Dying: What the Dying Have to Teach Doctors, Nurses, Clergy and Their Own Families*. New York: Macmillan Publishing Co., 1969.
7. Lifton RJ. *Death in Life: Survivors of Hiroshima*. New York: Random House, 1968.
8. O'Leary J. Grief and its impact on prenatal attachment in the subsequent pregnancy. *Arch Womens Ment Health* 2004 Feb;7(1):7–18.
9. Kübler-Ross E, Kessler D. *On Grief and Grieving: Finding the Meaning of Grief through the Five Stages of Loss*. New York: Scribner, 2005.
10. Worden JW. *Grief Counseling and Grief Therapy: A Handbook for the Mental Health Practitioner*. New York: Springer Publishing Company, 1982.
11. Worden W. *Grief Counseling and Grief Therapy: A Handbook for the Mental Health Practitioner* (3rd ed). New York: Springer Publishing Company, 2002.
12. Shaw CT. Grief over fetal loss. *Fam Pract Recert* 1983;5:129–145.
13. Professional Desk Reference. *PDR Family Guide to Women's Health and Prescription Drugs* (vol. 4). Montvale, NJ: Medical Economics Data, 1994.
14. Peppers LG, Knapp RJ. Maternal reactions to involuntary fetal/infant death. *Psychiatry* 1980 May;43(2):155–159.
15. Stack JM. Spontaneous abortion and grieving. *Am Fam Physician* 1980 May;21(5):99–102.
16. Paraclete Video Productions. *Footprints on Our Hearts: How to Cope After a Miscarriage, Stillbirth, or Newborn Death [DVD]*. Orleans, MA: Paraclete Press and Paraclete Video Productions, 1997.
17. Seibel M, Graves WL. The psychological implications of spontaneous abortions. *Reprod Med* 1980 Oct;24(4):161–165.
18. Moulder C. Towards a preliminary framework for understanding pregnancy loss. *J Reprod Infant Psychol* 1994;12:65–67.
19. Staudacher C. *Men & Grief: A Guide for Men Surviving the Death of a Loved One, A Resource for Caregivers and Mental Health Professionals*. Oakland, CA: New Harbinger Publications, 1991.
20. Gardener JM. *Nursing Interventions Perceived as Helpful by Parents of Stillborns*. [unpublished

- master's thesis]. Minneapolis: University of Minnesota, 1983.
21. Commonwealth of Massachusetts, General Laws, Chapter 111, Section 202.
  22. Mercy Medical Center, Department of Obstetrical Services/Family Life Center. Fetal Death/Care of Stillborn. Springfield, MA: Mercy Medical Center, 6 June, 2006.
  23. Johnson MP, Puddifoot JE. The grief response in the partners of women who miscarry. *Br J Med Psych* 1996 Dec;63(Pt. 4):313–327.
  24. Straker D. The grief cycle [updated 2006; cited 2006 Nov 28]. [http://changingminds.org/disciplines/change-management/kubler\\_ross/kubler\\_ross.htm](http://changingminds.org/disciplines/change-management/kubler_ross/kubler_ross.htm).
  25. Jeffreys JS. *Helping Grieving People When Tears Are Not Enough: A Handbook for Care Providers*. New York: Brunner-Routledge, 2005.
  26. Franche RL. Psychologic and obstetric predictors of couples' grief during pregnancy after miscarriage or perinatal death. *Obstet Gynecol* 2001 Apr;97(4):597–602.
  27. Swanson PB, Pearsall-Jones JG, Hay DA. How mothers cope with the death of a twin or higher multiple. *Twin Research* 2002 Jun;5(3):156–164.
  28. Bryan E. The death of a twin. In Sandbank A (ed): *Twin and Triplet Psychology: A Professional Guide to Working with Multiples*. London: Routledge, 1999; pp. 186–200.
  29. Read B, Bryan E, Hallett F. *When a Twin or Triplet Dies*. London: The Multiple Births Foundation, 1997.
  30. Simpson L, Paviour A. *More Than One*. Taroona, Australia: Do Not Press, 2001.
  31. de Kleine M, Cuisinier M, Kollee L, Bethlehem G, de Graauw K. Guidance after singleton and neonatal death. *Arch Dis Child* 1995 Mar;36(2):F125–F126.
  32. Widger KA, Wilkins K. What are the key components of quality perinatal and pediatric end-of-life care? A literature review. *J Palliat Care* 2004 Summer;20(2):105–112.
  33. Wolfe J, Grier HE, Klar N, Levin SB, Ellenbogen JM, Salem-Schatz S, Emmanuel EJ, Weeks JC. Symptoms and suffering at the end of life in children. *N Engl J Med* 2000 Feb;342(5):326–333.
  34. Sexton PR, Stephen SB. Postpartum mothers' perceptions of nursing interventions for perinatal grief. *Neonatal Network* 1991 Feb;9(5):47–51.
  35. Calhoun LK. Parents' perceptions of nursing support following neonatal loss. *J Perinatal Neonat Nurs* 1994 Sep;8(2):57–66.
  36. Malacrida CA. Perinatal death: Helping parents find their way. *J Fam Nurs* 1997;3:130–148.
  37. Finlay I, Dallimore D. Your child is dead. *Br Med J*. 1991 Jun 22;302(6791):1524–1525.
  38. Stewart ES. Family-centered care for the bereaved. *Pediatr Nurs* 1995 Mar–Apr;21(2):181–187.
  39. Papadatou D, Yfantopoulos J, Kosmidis HV. Death of a child at home or in hospital: Experiences of Greek mothers. *Death Studies* 1996 May–Jun;20(3):215–235.
  40. While A, Citrone C, Cornish J. *Bereaved parents' views of caring for a child with a life-limiting disorder*. London: King's College, 1996.
  41. Contro N, Larson J, Scofield S, Sourkes B, Cohen H. Family perspectives on the quality of palliative pediatric care. *Arch Pediatr Adolesc Med* 2002 Jan;156(1):14–19.
  42. American Academy of Pediatrics, Committee on Pediatric Emergency Medicine. Death of a child in the emergency department. *Pediatrics* 1994;93:861–862.
  43. Primeau MR, Lamb JM. When a baby dies: Rights of the baby and parents. *J Obstet Gynecol Neonatal Nurs* 1995 Mar–Apr;24(3):206–208.
  44. Masera G, Spinetta JJ, Jankovic M, Ablin AR, D'Angio GJ, Van Dongen-Melman J, Eden T, Martins AG, Mulhern RK, Oppenheim D, Topf R, Chesler MA. Guidelines for assistance to terminally ill children with cancer: A report of the SIOP working committee on psychosocial issues in pediatric oncology. *Med Pediatr Oncol* 1999 Oct;32(4):44–48.
  45. American Academy of Pediatrics, Committee on Bioethics and Committee on Hospital Care. Palliative care for children. *Pediatrics* 2000;106:351–357.
  46. Armstrong D. Exploring fathers' experiences of pregnancy after a prior perinatal loss. *MCN Am J Matern Child Nurs* 2001 May–Jun;26(3):147–153.
  47. Wing DG, Clance PR, Burge-Callaway K, Armistead L. Understanding gender differences in bereavement following the death of an infant: Implications for treatment. *Psychotherapy* 2001;38:60–73.

## Chapter 28 BIRTH INJURY: LEGAL COMMENTARY IV

Kevin Giordano

*Predictions in acute diseases, whether favorable or unfavorable, are not absolutely certain.*

Hippocrates (c. 460–377 B.C.E.)  
*Aphorisms of Hippocrates*, trans. T. Coar  
London: A. J. Valpy, 1822, xix, p. 26.

Of all medical specialties, obstetrics possesses the greatest potential for disastrous adverse outcomes. This is true in the legal forum as well as within medicine. The patient condition that results in the most malpractice cases against physicians is pregnancy, with the largest number of claims brought on behalf of the birth-injured infant [1]. In any study that investigates the source of the largest indemnity payouts, whether by settlement or jury verdict, it is the so-called brain-damaged baby case that universally tops the charts as the source of the largest indemnity payouts by medical malpractice insurance companies, whether such payouts occur as a result of settlement or jury verdict. In a study performed by the Physician Insurers Association of America, an association for more than 40 malpractice insurers, the average indemnity payment on cases involving birth injury was \$549,481, whereas the average indemnity payout for all patient conditions in claims involving obstetrics and gynecology was \$251,948, and there was a larger number of claims in which the payout exceeded \$1 million than in earlier studies. Other conditions for which there was a prevalence of claims involved improper management of pregnancy or of fetal distress, and injury during normal delivery of a single gestation [2].

Because of both the emotional and monetary stakes involved, and the impact that birth injury cases have on both sides, these cases require considerable resources and are litigated vigorously. The patient's prenatal and intrapartum periods are scrutinized to the highest degree. The fetal heart monitoring strips are analyzed and reanalyzed by the minute, with the hindsight that the baby's condition at birth was compromised. Of considerable importance, because of extraordinary progress of the science of medicine, both parties place a major emphasis on developing evidence to establish legal causation and whether the injury to the baby was indeed caused by the labor process. The issue becomes whether the injury in utero would have been avoided or significantly lessened if the



physician had acted differently (see Appendix I for a detailed discussion of causation). Usually the allegations are of *omission, either the failure perform a cesarean delivery or the failure to effectuate the delivery in a more timely fashion*. The question to be answered by the jury, should it determine that the physician was negligent, is whether the failure to perform a cesarean, or in cases of delay, a more timely cesarean, significantly contributed to the baby's outcome.

It is true that it is the plaintiff's obligation to produce evidence that the injury resulted from the physician's negligence. This element of the case obviously requires the testimony or one or more experts. Although it is the plaintiff's burden to establish the causal relationship, from the defense's perspective, the importance of not just refuting the plaintiff's allegations but also being able to identify an alternative explanation for the outcome cannot be understated. For instance, if the defense can show that the baby's condition is congenital, then earlier intervention during labor would most likely not have altered the outcome. This fact would be a critical blow to the plaintiff's case. Similarly, if the defense can establish that the injury most likely occurred during the prenatal and not during the perinatal period, and that intervention with a cesarean would not have avoided insult to the baby, the plaintiffs would be unable to prove a causal link and thus would be incapable of establishing their burden. This is true even in cases in which the physician's conduct might have deviated from the standard of care.

Antepartum and postpartum assessments have always played a distinct role in determining the onset of injury; however, advances in intrauterine assessment have increased the significance of prenatal findings. Present ultrasonographic techniques can provide a detailed assessment of the baby's condition and possibly identify features indicating neurologic impairment or injury [3]. Other testing, such as fetal growth studies, fetal movement reports, biophysical profile, and heart rate stress and fetal non-stress tests, all contribute to determining the well-being of the fetus before the onset of labor.

Lack of fetal heart rate (FHR) variability at the commencement of labor, in conjunction with clinical indications of depression in the prenatal period, can help to establish that the child's condition was chronic and not the result of acute intrapartum problems [4]. Passage of meconium, fetal acid-base

abnormalities, and Doppler ultrasound flow studies all can aid in the identification of a compromised baby. See Chapter 22, Fetal Assessment.

Important developments in diagnostic tools and neurologic assessment of the newborn have improved the ability to establish that the child's outcome was unrelated to the birth process but was instead the result of some congenital condition or a chronic in utero event. As discussed in the legal commentary that follows Part I of this text, genetics can also be used as a sword against the obstetrician, establishing that proper genetic counseling did not occur and deprived the parents of the right to choose termination. Genetic science, in combination with other developments, can also be a useful shield in defending against claims of alleged birth trauma, however. Genetic testing, antepartum and intrapartum ultrasound scans or monitoring data, postpartum neurologic assessments, and specialized radiographic studies can identify vital pieces of the puzzle that when placed together, establish whether the cause of an infant's defects was something other than a perinatal event. The identification of increasing numbers of birth defects that are congenital and the development of other testing that can establish chronicity have been important tools in successfully defending many "bad-baby" claims that previously were deemed to be from birth trauma. By working with a wide array of expert consultants, defendants are better able to establish that the child's handicapped condition did not result from an hypoxic perinatal event that was negligently caused or misdiagnosed but instead was due to a specific prenatal event or exposure, or to a defect coded within that child's chromosomes.

Neurologic assessment following delivery also provides findings contributing the determination of causal relationships between clinical events and outcome. Evaluation of serial imaging studies, including magnetic resonance imaging (MRI), computed tomography (CT), ultrasound scans, and EEG combined with findings on physical examination (e.g., mental status, cranial nerves, and motor function), and history (including onset of any seizures), as well as abnormalities in liver, kidney, and cardiac function, can assist in the assessment of brain injury and in elucidating the timing and mode of the injury [4]. Consequently, in the attempt to find the cause of a neonate's poor condition, specialists from neuroradiology, neonatology, and pediatric infectious

diseases are often consulted, in addition to pediatric neurologists.

Given advancements in medical knowledge and improvements in diagnostic technique, it is now understood that approximately 80% or more of the serious or permanent neurologic abnormalities that occur in newborns are sustained prior to birth [5]. Chromosomal and hereditary abnormalities, infection, toxic exposures, and in-utero inflammation are considered to be the most common causes. Unfortunately, in any one particular case, the etiology of the infant's injury is sometimes unknown or the case is complex, involving several potentially damaging clinical events, thus raising the potential for an argument established by expert witnesses that the event occurred during labor and delivery because of a management error. Consequently, *the primary issue in birth trauma cases remains whether the treating obstetrician complied with standard of care in identifying potential risk factors and managing the labor and delivery.*

### **ELECTRONIC FETAL MONITORING**

*Fetal monitoring* can be understood as the gathering and interpretation of information from the fetus about its well-being. Thus defined, fetal monitoring includes ultrasonographic evaluation of the fetus, direct observation of amniotic fluid, fetal capillary blood sampling, monitoring of the FHR by auscultation or electronic means, and even registration of the mother's perception of fetal movements. These modalities have developed because of the inability to effectively evaluate the fetus on the basis of risks conferred by maternal disease. The clinician cannot be certain of fetal well-being merely because the mother lacks obvious risk factors.

The premise of fetal surveillance is anticipation and prevention of fetal hypoxia and asphyxia and timely intervention before injury occurs. Intrapartum assessment, principally by the limited technique of EFM, remains a key feature in determining whether intervention is indicated. FHR patterns such as early and late decelerations have been associated respectively with head compression and uteroplacental insufficiency, whereas variable decelerations are thought to be related to intermittent cord compression. This is not what these techniques do best, however. They are best at providing evidence of fetal well-being, and as such are tools of remark-

able value. Normal FHR patterns during labor, for example, give powerful reassurance that the fetus is normally oxygenated and neurologically intact. These findings obviate the need for intervention for fetal indication, regardless of the mother's condition.

Complete reliance on EFM is most likely misguided, however. The introduction of intrapartum EFM and its emergence as the standard for intrapartum management came about based on the results of early, uncontrolled and nonrandomized studies that appeared to show that implementation of EFM reduced perinatal neurologic morbidity and mortality [6]. Despite nearly universal acceptance in both high- and low-risk patients, EFM has not been shown to improve neonatal and perinatal mortality rates. Furthermore, EFM has not been shown to reduce the rates of intrapartum stillbirth, neonatal intensive care unit (NICU) admissions, or Apgar scores. Whereas the primary rationale for instituting EFM testing was to prevent fetal asphyxia during labor, its efficacy over intermittent auscultation has not been established, at least as practiced in the studies performed to date [7]. There have been no major changes in the prevalence of cerebral palsy in the term infant since the introduction of EFM, and it has been found that a nonreassuring FHR pattern is not a specific and reliable predictor of fetal problems such as decreased oxygenation or acidosis [8]. Because EFM demonstrates a low specificity, a significant number of infants found to be normal at birth have had demonstrated FHR patterns considered abnormal during labor.

Despite the lack of predictability, the EFM has remained the dominant factor in clinical decision making that has resulted in increased rates of operative delivery, primarily through increasing the incidence of cesarean and forceps or vacuum extraction delivery, each with its specific maternal and fetal risks [9]. Given this predominance, in cases involving a claim of birth asphyxia, the EFM strips are "Exhibit A" in any malpractice case. Plaintiffs' attorneys and experts attempt to prove that the EFM established that the baby was in a hostile environment and that therefore prompt delivery should have occurred. Defense attorneys and experts attempt to prove the absence of recognized patterns that are indications for expediting delivery. It thus becomes the "tale of the tape" whether surgical delivery was indicated.

As the Court concluded in that matter of *Baglio v. St. John's Queens Hospital* "... [t]he fetal monitoring strips are the most critical evidence to determine fetal well-being at the time of treatment, and in evaluating the conduct of healthcare providers with regard to obstetrical management thereafter. Further, under the facts of this case, the fetal monitoring strips would give fairly conclusive evidence as to the presence or absence of fetal distress" [10]. In *Baglio*, the infant plaintiff's mother had initiated a medical malpractice action against St. John's Queens Hospital and others, alleging that, among other things, the infant plaintiff had suffered brain damage because of a deprivation of oxygen during his delivery. Just prior to commencing the lawsuit, the plaintiffs' attorney requested that the hospital provide her with the infant plaintiff's fetal heart monitoring (FHM) strips. Initially, in response, the hospital sent incorrect FHM strips (i.e., monitoring strips from another pregnancy). After the plaintiffs' attorney demanded the correct FHM strips, the hospital stated that it was unable to locate the correct strips. The court, confronted with a situation in which the hospital had lost the FHM strips, concluded that a judgment should be entered against the hospital because it had deprived the plaintiff of the means of proving her medical malpractice claim against the hospital.

The *Baglio* case emphasizes the evidentiary importance that the FHM strips play in the litigation of a birth trauma case. In addition to the FHR pattern, frequently there are often critically important clinical notations made on the FHM strips by nurses and doctors. The only mention of intrauterine resuscitation or other actions by the caregivers may be included in the handwritten notations on the FHM tapes. As an example, an increase or decrease of oxytocin infusion is commonly recorded on the tapes by hospital personnel or the attending physician. Efforts at intrauterine resuscitation, such as turning the mother to her left side or giving a bolus of intravenous fluid, are also often noted on the FHM tapes.

As discussed in Appendix I, errors in judgment do not constitute medical malpractice, provided the error is reasonable and has occurred after a full and complete analysis of the situation. This defense is most effectively used in instances when the physician demonstrates sound judgment and diligent monitoring, but a depressed baby is deliv-

ered. Even in the best of hands, in the absence of an indication of negligence or misconduct, there are times when babies are born depressed. Typically, in the assessment of whether sound judgment and diligent monitoring have occurred, two principles are consistently at issue. The first is *whether the obstetrician has identified any notable trends in the fetal condition on the FHM strips*. Second, *if any worrisome trends are seen, were they remediable*. In other words, is the cause of the problem correctable and is the maternal-placental unit capable of sustaining adequate fetal oxygen delivery. If patterns of fetal distress are found not to be remediable, the issue in such cases is whether surgical intervention was indicated, and if so, was it undertaken as quickly as was consistent with maternal and fetal safety.

An effective defense must demonstrate that the medical records adequately reflect the physician's ongoing thought process in providing obstetric care. EFM studies can be carried out for many hours, and therefore the physician must periodically assess the entire tracing and look for subtle signs of deteriorating fetal condition. This type of frequently performed review and analysis can help to protect the physician by confirming his or her presence and observations.

Given the current role that EFM has in clinical decision making and thus its importance during the defense of a malpractice case, the obstetrician should consider the following:

- Suspicious or even ominous FHR patterns are common even in normal healthy babies, as well as those with fetal hypoxia/asphyxia. Determining the timing of intervention can be difficult, and the interpretation of such patterns is unfortunately subjective.
- The initial management of a nonreassuring FHR pattern requires determining the cause of the FHR pattern and undertaking corrective efforts. Discontinuance of any oxytocic agent, changing the maternal position, increasing fluid infusion to improve placental perfusion, and administration of oxygen are among standard conservative management measures.
- If conservative measures are not effective, expediting delivery is sometimes necessary. This can be accomplished by operative vaginal delivery or by a cesarean. If the tracing is interpreted as abnormal,

and the decision to intervene is made, the delivery should be accomplished with deliberate haste.

- Knowledge of and compliance with the American College of Obstetricians and Gynecologist (ACOG) bulletins on FHM is imperative. ACOG bulletins do not attempt to establish the standard of care; however, compliance with or departure from any of their recommendations can play a significant role during any trial.
- The patient must be included in the decision-making process once material risks arise. Keeping the patient informed of issues that could change the plan for labor management and obtaining informed consent are essential. The obstetrician should always document these discussions in the medical records.
- In the presence of a serious abnormality, efforts should be undertaken to establish the cause. Although often the cause is unidentifiable, a complete evaluation can establish that the inability to determine a cause is due to the nature of the infant's insult and not to the lack of clinical review.
- In the event of a bad outcome, the obstetrician must make him- or herself available and show concern for both needs of the family and the health of the baby. If there is any apprehension about the substance of what should be conveyed to a patient after an adverse event, the obstetrician might want to seek advice from the hospital's risk management team as soon as possible.

### PLACENTAL PATHOLOGY

Histologic examination of the placenta and cord can provide valuable insight in explaining abnormal neonatal outcomes.

In 1892, Ballentyne [11] wrote:

**A diseased foetus without its placenta is an imperfect specimen, and a description of a foetal malady, unless accompanied by a notice of the placental condition, is incomplete. Deductions drawn from such a case cannot be considered as conclusive, for in the missing placenta or cord, may have existed the cause of the disease or death. During intrauterine life of the foetus, the membranes, the cord and the**

**placenta form an organic whole and disease of any part must react upon and affect the others.**

Ongoing advancements made in placental pathology have given specialists the increased capability to correlate certain pathologic findings with specific causes. Placental villous abnormalities, identification of infarcts, and the presence of nucleated red blood cells in fetal vessels, among other findings, can assist in establishing the onset of an injury. In cases of alleged malpractice, pathologic examination of the placenta by a trained pathologist can establish that the neurologic or motor impairment suffered by a child is traceable to a specific intrauterine event, or to disordered intrauterine growth, thus removing perinatal events from consideration in etiology. It is now commonplace for the histologic examination of the placenta to be central in determining whether the child was harmed as a result of intrapartum mismanagement or misdiagnosis. Using objective findings to establish that the outcome was an event unrelated to the physician's care and therefore beyond his or her control can be critical to the outcome of the case.

Unfortunately, in most instances, despite the availability of significant scientific advancements, neither the etiology of a neurologic insult nor its timing can be scientifically established [12]. Markers have not been identified for many hereditary conditions. Clinical findings used to assess the intrauterine, antepartum, and postpartum status of the infant could have features of a particular disease process, but often these data are either nonspecific or can be present even in normal babies [13]. For instance, although meconium staining of the amniotic fluid is associated with increased risk of quadriplegic cerebral palsy, the majority of children born with meconium in the amniotic fluid are normal [14].

Similarly, the ability of pathologists to make a conclusive diagnosis pinpointing the cause and timing of any event leading to an infant's irreversible brain damage or death is limited. Abnormal pathologic findings such as a two-vessel cord, meconium staining of the membranes, or signs of chorioamnionitis can occur even in deliveries resulting in a healthy baby [15,16]. Given this, even if certain pathologic findings exist suggesting a chronic process, the plaintiff's experts can and will argue that such findings are coincidental and often seen in instances when there is no harm. Thus it will be

claimed that these data do not provide evidence that the findings are related to the actual cause of the infant's injury.

The complexity of causation for fetal injury and the limitations of current knowledge are serious barriers to the accurate assignment of etiology. The point at which a common clinical event becomes a causative factor in fetal injury is often difficult to ascertain. Infection (chorioamnionitis) is recognized as an important factor in the development of cerebral palsy. In fact, it is believed that many cases of cerebral palsy are erroneously attributed to birth asphyxia, when infection/inflammation is the actual cause [17,18]. Some histologic evidence of chorioamnionitis is often present in the placenta in otherwise normal deliveries, however. Consequently, the finding of chorioamnionitis alone does not necessarily exclude intrapartum birth asphyxia as a potential etiology for a birth injury [19]. On contrast, the finding of funisitis may have more significance. Conversely, in other cases, because the pathologic findings appear to fall within the range of normal but the presentation mirrors that of asphyxia, it might be impossible to establish that chorioamnionitis was the specific cause of the bad outcome. At times, some conditions can occur in the placenta without leaving any recognizable traces. For example, B19 parvovirus, a recognized cause of fetal hydrops, can occur without any demonstrable placental structural pathology [20].

Because in most situations the cause and timing of onset of the baby's condition cannot be scientifically established, the issue ultimately becomes a battle of expert opinion. This is illustrated in the following case [21]. The plaintiff was a high-risk patient admitted to a labor and delivery service after rupture of membranes. She claimed that there was a delay in placing her on the fetal heart monitor and a further delay in the diagnosis of fetal distress. Because of this, a cesarean delivery was not performed in a timely manner. At birth, the baby was cyanotic, with no spontaneous respirations. The 1-minute Apgar score was 1. After resuscitation, the baby had the return of good color, heart rate, and tone, and was breathing on his own. The 5-minute Apgar score was 8. At the time of admission to the nursery, he was noted to be alert and exhibited no neurologic abnormalities. He ate and urinated well. Approximately 12 hours after birth, however, the baby became cyanotic and then exhibited right-

sided seizure activity lasting several minutes. He was ultimately diagnosed with hypoxic ischemic encephalopathy and was subsequently noted to have permanent neurologic injuries. At the time of trial, despite being 13 years old, the child functioned at the level of 2 to 3 years old.

It was the defendants' position that although the length of time it took for the placement of a fetal heart monitor was longer than the usual, the delay did not represent negligence. The defense also claimed that there was no evidence that earlier placement would have affected care. As to the timing of the cesarean, it was averred that although the tracings became nonreassuring, they did not indicate fetal distress. Thus, although concerning, the FHR monitoring did not suggest "... an extreme urgency to do the [cesarean] section." Consequently, the defendants claimed that although in the presence of fetal distress the standard of care required delivery in no more than 30 minutes, this "situation was a bit different," because there was no true distress. Thus, it was their claim that although the duration between decision to perform a cesarean and delivery was approximately 45 minutes, the delivery in this clinical setting was timely.

The evidence on causation was contested. One defense expert went so far as to conclude that there was "absolutely no way" that the injury occurred at or about the time of delivery, but rather days before. Thus, neither the time at which the monitor was placed nor the interval between decision to deliver and the cesarean was relevant to the causation of the injury. Conversely, the plaintiffs' expert testified that had the infant been delivered earlier, there would have been little or no brain damage.

The jury verdict was in favor of the plaintiff. The defendants sought to overturn the judgment on grounds that the plaintiffs failed to present "anything beyond pure speculation" with respect to the cause of the baby's mental retardation. It was their contention that the testimony of the plaintiff's experts was not accompanied by necessary reasoned analysis, therefore reducing it to mere conclusions that were incapable of supporting the verdict in this cause.

As the court noted, the determination of the legal cause of the baby's condition boiled down to credibility – a battle of the experts. It was further noted that the disagreement between the medical experts as to cause and effect did not preclude a verdict

for the plaintiff; rather, an expert opinion, held to a reasonable degree of medical certainty, provides an adequate basis for a jury finding that a concept of causation was proved. The jury was free to believe the plaintiff's expert testimony about the time-placement of the fetal monitor, the timing of the cesarean, and whether either had any effect on outcome. Conversely, the jury was also free to disbelieve the plaintiff's experts and instead rely on the testimony of the defendant's experts. Making the determination is totally within the province of the jury and not the role of the court to decide.

### EDUCATION AND TRAINING

Preventable errors in the provision of care are a constant source of patient morbidity, excess cost, and litigation. The landmark publication, *To Err Is Human* [22], estimates that between 45,000 and 98,000 patients die each year as a consequence of preventable errors, with the economic burden of errors estimated at 29 million dollars.

The common perception is that adverse events are the product of "human error;" that is, some individual deficiency that occurs by the failure of someone to meet performance requirements or through sheer bad luck. Close evaluation of many adverse outcomes, however, indicates that rather than the errors of people, problems in the organization of care and communication between caregivers are frequently the root cause of the problem. In a study analyzing closed-claim files involving obstetrics and gynecology, the investigators concluded that 78% of the cases appeared to have at least one potentially preventable cause [23]. Analysis of adverse events is typically through quality assurance programs and peer review, emphasizing the individual care provided. Occasionally, an adverse event is studied at morbidity and mortality grand rounds. The process of comprehensive evaluation of adverse events to identify system errors and to address broad changes in clinical procedures, to prevent errors in daily practice, is in its infancy in hospital practice, however.

The unequivocal institutional goal must be the improvement of the quality of care and the reduction in error rates and patient injuries. The hospital culture must be one of patient safety and good outcomes. A key component to a culture of safety is designing systems that encourage health-care providers to report not just adverse events

but averted mishaps, or "near misses." In the study of closed claims discussed earlier, the authors concluded that a case-by-case analysis of adverse outcomes might lead to a conclusion that each event was idiosyncratic; however, the researcher stated, "...our aggregated data revealed clusters of common issues that suggested opportunities for system-level improvement" [23]. The ability to analyze not only adverse events but also near misses that currently go unrecognized provides a greater opportunity to identify conditions that could lead to adverse events. The reporting system must clearly be one that fosters reporting without the potential for adverse consequences to the reporter.

Once identified, strategies must be developed to improve systems, as well as individual training if necessary. In some instances, this involves implementation of technology. The improved technology could be directly for purpose of diagnosis or treatment that has been developed through scientific advancement. Other technology might be developed as a substitute for the human element, or as a method for cross checking choices such as medications doses and timing, thus decreasing the potential for error.

Furthermore, simulations have become progressively more important components in educational systems. Simulation can be through the use of drills, to ensure that all the members of the team are fully aware of their roles and can function together when confronted with a real-life emergency. Computer programs can also be effective tools. Newer advancements are providing simulators so that training of residents and attending physicians can be undertaken in a safe environment and allow hands-on training for the implementation of new techniques or for the review of situations that involve uncommon but potentially serious occurrences. An example is the development by Johns Hopkins of a simulator that trains physicians confronted with shoulder dystocia. Among other things, the simulator measures the force that is applied by the physician in performing downward traction, to provide an understanding that gentle steady force is all that is needed during the delivery [24]. For additional discussion, see Chapter 25, Education and Certification.

The following considerations are important:

- Any program that is designed to create an atmosphere of patient safety must have multiple components. Conferences and courses are of great

assistance in the on-going education of physicians. Computer programs and drills are also of great benefit. Educational tools that allow hands-on training through the use of simulation provide a dimension that cannot be reproduced through any other modality.

- Any system with a goal of truly reducing preventable injuries must change the internal culture that assumes that the human factor is responsible for most adverse errors, and therefore improvement in current practices and protocols cannot dramatically affect outcomes. It is true that human error is inevitable; however, systems that quantitatively analyze bad outcomes and near misses can identify causes of human error and improve systems that screen for error, or undertake additional individual training to reduce the likelihood of error that results from insufficient training or lack of experience.
- Create a culture within the obstetric unit that enhances patient safety instead of fostering low expectations. Communication is a critical component of healthcare delivery, and efforts should improve communication methods between providers at every level. Obstetricians should take the lead in encouraging nursing staff and subordinate care providers to raise concerns about the decision-making process. Often nursing staff and subordinates recognize risk but are fearful of raising the issue with a particular obstetrician.
- Incorporate medicolegal education as a means for physicians to better understand the process and eliminate any mindset that lawsuits are inevitable and instituting good risk management tools are ineffective.
- Support root cause analysis when near miss or actual patient injury occurs, to analyze how both individuals and the system have failed.

### **NURSE MIDWIVES AND OPERATIVE OBSTETRICS**

Nurse-midwifery in the United States dates back to 1925. At that time, Mary Breckenridge developed the Frontier Nursing Service in Kentucky, a program that used public health registered nurses, who had received additional nurse-midwifery edu-

cation in England, to staff nursing centers in the Appalachian mountains. The centers offered family healthcare services, including childbearing and delivery care, to underserved residents within this remote region [25]. The first nurse-midwifery education program in the United States began in 1932, at the Maternity Center Association of New York City. This program enrolled public health nurses and awarded a certificate in nurse-midwifery on completion of program requirements. Presently, there are more than 40 programs accredited by the U.S. Department of Education, primarily associated with academic medical centers. Furthermore, all 50 states have promulgated regulations providing for licensure of midwifery practice.

Certified nurse-midwives (CNM) are regulated at two levels. Similar to the role of ACOG, midwifery certification is uniformly standardized and provided through a national organization, the American Midwifery Certification Board (AMCB). Certification is granted on completion of certain established requirement and satisfactory completion of an examination. Licensure is a process that takes place at the state level in accordance with specific state laws and regulations can vary from state to state.

Recently, some states have begun recognizing "direct-entry" midwives. The direct-entry midwife enters the profession not through nursing but by other routes that can include clinical training or apprenticeship with a senior midwife or obstetrician. Most commonly accepted is the Certified Midwife (CM). CMs have not completed a nursing program but are required to complete an accredited educational program and must satisfactorily complete the same certifying examination as the CNM, which is administered by the AMCB. Additionally, an increasing number of states have recognized other forms of direct-entry midwives, not certified by the AMCB. The legality and regulation of direct-entry midwifery varies from state to state. The states that allow other direct-entry midwives to manage labor and delivery typically require some form of registration or permit; however, in many states direct-entry midwifery is prohibited.

As the incorporation of CNMs/CMs has become more prevalent, they have been a valuable resource in many obstetric practice settings. The midwife philosophy includes an emphasis on individualized care and support during pregnancy and delivery, with as little obstetric intervention as possible [26]. The use

of midwives in attending the labor and delivery of mothers who are at low risk for obstetric complications has been shown to be a cost-effective, safe alternative to delivery by physicians [27]. Midwife-attended births have birth outcomes comparable to those by physicians and are less likely to have obstetric interventions (e.g., EFM and epidural analgesia use), factors contributing to decreased costs. Furthermore, national and state malpractice data do not indicate significant increased liability risk for physicians who employ or supervise nonphysician clinicians [28]. ACOG has published a position statement that supports the role of CNMs and CMs of midwives in the management of women's health issues [29]. ACOG restricts its supportive position to only those midwives who have been certified by the AMCB, however.

In the obstetric practice setting, however, defining the relationship between obstetrician and CNM/CM is very important and can have potential impact on legal liability. The ACNM defines midwifery practice as the "independent management of women's healthcare, focusing on pregnancy, childbirth, the postpartum period, care of the newborn, and the family planning and gynecological needs of women" [30]. In its broadest sense, the definition gives considerable autonomy to a midwife in the management of the patient during clinical evaluation (i.e., history taking, physical assessment, ordering appropriate laboratory tests and procedures), therapeutic management (i.e., outlining care, providing prescriptions, coordinating consultations and referrals), labor and delivery (non-operative), as well as in general women's health maintenance and risk-reduction issues. Consultation and collaboration are as required in the clinical circumstance. To midwives, the term *collaboration* implies co-management of a patient who requires some level of care or evaluation beyond their scope of practice. In these circumstances, the role of each is determined by the particular clinical situation.

ACOG has weighed in on the relationship between obstetrician/gynecologists and midwives. The joint statement between ACOG and the American College of Nurse-Midwives (ACNM) includes a statement that "... the appropriate practice of the certified nurse-midwife includes the participation and involvement of the obstetrician/gynecologist as mutually agreed upon in written medical guidelines/protocols . . . . This does not necessarily imply

the physical presence of the physician when care is being given by the certified nurse-midwife" [31].

Most important are the applicable statutory regulations, however. Existing law defining the role of each can vary from state to state. Some state regulatory schemes require that a nurse-midwife be "supervised" or "directed" by a physician with obstetric privileges and also limits the number of midwives any particular physician may supervise. The terms *supervised* and *directed*, as used in the context of the regulation, often do not necessarily imply the physical presence of an obstetrician/gynecologist while care is being given by a nurse-midwife. The requirement of most states is that a nurse-midwife must have a "clinical practice relationship" with an obstetrician/gynecologist that "shall be based on mutually agreed upon medical guidelines and protocols" [31].

Consequently, the interpretation of statutory authority in most states allows for nurse practitioners to practice independently, requiring no direct supervision under the law. In these situations, the physician's liability exposure is much more limited and is for the most part confined to his/her own negligence, as in any situation of caring jointly for a patient.\* Conversely, the potential for individual liability of the CNM/CM is increased as it becomes incumbent on him/her provide care within their scope of practice and seek consultation or collaboration in the appropriate circumstances. In fact, one of the leading reasons nonphysician clinicians are involved in litigation is based on a claim that they rendered a service beyond their capabilities.

The following considerations are important:

- Review state laws on licensure, scope of practice, and supervision of CNMs/CMs. The clinical practice relationship between the obstetrician/gynecologist and the CNM/CM should provide for mutually agreed-on written medical guidelines/protocols for clinical practice that define the individual and shared responsibilities of the CNM/CM and the obstetrician/gynecologist in the delivery of healthcare services. Guidelines must be periodically reviewed and updated

\*This is, however, separate and distinct from the concept of employer liability. In cases in which the obstetrician is the *employer* of the CNM/CM, there remains *vicarious liability*, because any employer can be held responsible for the actions of its agent.



to ensure appropriate care and consistency with statutory regulations.

- Liability risks for physicians who employ CNMs/CMs can be minimized by developing and following reasonable guidelines for their performance, supervision, and review. Prior to employment, verify the CNMs/CMs credentials and prior experience thoroughly.
- It is critical that obstetrician/gynecologists and CNMs/CMs have a clear understanding of their individual, collaborative, and interdependent responsibilities. As agreed on by ACNM and ACOG, the maternity care team must include either an obstetrician/gynecologist with hospital privileges or another physician with hospital privileges, to provide complete obstetric care.
- Guidelines/protocols must be established for ongoing communication, which provide for and define appropriate consultation between the obstetrician/gynecologist and the CNM/CM. For optimal quality of care and reduced liability risk, there must be an interdependent practice of the obstetrician/gynecologist and the CNM/CM working in a relationship of mutual respect, trust, and professional responsibility. There should be clear "open lines " of communication to discuss issues or concerns about patient treatment.
- Informed consent about the involvement of the obstetrician/gynecologist and the CNM/CM should be obtained from the patient.

## REFERENCES

1. Hospital Professional Liability Benchmarking Report MMC (Marsh & McLennan Companies) 2007.
2. Physician's Insurers Association of America (PIAA) Closed Claims Study, 2005.
3. Edwards AD. New approaches to brain injury in preterm infants. *Dev Neurosci* 2002;24:352–354.
4. Blumenthal J. Cerebral palsy – medical legal aspects. *J R Soc Med* 2001;94:624–627.
5. Nelson K, Leviton A. How much of encephalopathy is due to birth asphyxia? *Am J Dis Child* 1991; 145:1325–1331.
6. Benson RC, Schubeck F, Deutschberger J, et al. Fetal heart rate as a predictor of fetal distress: A report from the collaborative project. *Obstet Gynecol* 1968;32:259–266.
7. Vintzileos AM, Nochimson DJ, Guzman ER, et al. Intrapartum electronic fetal heart rate monitoring versus intermittent auscultation: A meta-analysis. *Obstet Gynecol* 1995; 85:149–155.
8. Freeman RK. Intrapartum electronic fetal monitoring – a disappointing story. *N Engl J Med.* 1990; 322:624–626.
9. Vintzileos AM, Nochimson DJ, Guzman ER, et al. Intrapartum electronic fetal heart rate monitoring versus intermittent auscultation: A meta-analysis. *Obstet Gynecol* 1995;85:149–155.
10. *Baglio v. St. John's Queens Hospital* 303 A.D.2d 341 (NY 2003).
11. Ballentyne JW. *The Diseases and Deformities of the Foetus*, Vol. 1 Edinburg: Oliver and Boyd, 1892.
12. Cowan R, Rutherford M, Groenendaal F, et al. Origin and timing of brain lesions in term infants with neonatal encephalopathy. *Lancet* 2003;361:736–742.
13. Blumenthal I. Cerebral palsy – medicolegal aspects *J R Soc Med* 2001;94:624–627.
14. Naeye R. Can meconium in the amniotic fluid injure the fetal brain? *Obstet Gynecol* 1995;86:720–724.
15. Nelson K. The neurologically impaired child and alleged malpractice at birth. *Neurol Clin* 1999;17: 283–293.
16. Blumenthal I. Cerebral palsy – medicolegal aspects. *J R Soc Med.* 2001;94:624–627.
17. Blumenthal I. Cerebral palsy – medicolegal aspects *J R Soc Med* 2001;94:624–627.
18. Yoon B, Romero R, Park J. Fetal exposure to an intra-amniotic inflammation and the development of cerebral palsy at the age of three years. *Am J Obstet Gynecol* 2000;182:675–681.
19. Redline RW, Heller D, Keating S, Kingdom J. Placental diagnostic criteria and clinical correlation. *J Placenta* 2005;19(Suppl):26.
20. Petersson K, Norbeck O, Westgren M, Broliden K. Detection of parvovirus B19, cytomegalovirus and enterovirus infection in cases of intrauterine fetal death. *J Perinat Med* 2004;32(6):516–521.
21. *Wareing v. United States* 943 F Supp 1504 (1996).
22. Institute of Medicine. *To Err is Human: Building a Safer Health System*. Washington, DC: The National Academies Press, 2000, p. 287.

23. White AA, Pichert JW, Bledsoe SH, Irwin C, Entman SS. Cause and effect analysis of closed claims in obstetrics and gynecology. *Obstet Gynecol.* 2005 May;105(5 Pt 1):1031–1038.
24. March D. Simulator helps ID least forceful way to manage problem deliveries. *The JHU Gazette*, March 2005;34:27.
25. A brief history of the frontier nursing service. <http://www.frontiernursing.org>.
26. Clark S, Martin JA, Taffel SM, Metropolitan Life Insurance Co. Trends and characteristics of births attended by midwives. *Statistical Bulletin* Jan–Feb 1997;78:1.
27. Keleher KC, Mann LI. Nurse midwifery care in the academic health center. *J Obstet Gynecol Neonatal Nursing* 1986;15(5):369–372.
28. The use of nonphysician clinicians in a medical practice. MIEC Claims Alert No. 25, Aug 1999.
29. ACOG/ACNM. Joint statement of practice relations between obstetrician-gynecologists and certified nurse-midwives/certified midwives, Oct 2002.
30. American College of Nurse Midwives, Definition of midwifery practice, 2004.
31. ACOG ACNM Joint statement of practice relations between obstetrician-gynecologists and certified nurse-midwives/certified midwives, Oct 2002.

# APPENDIX

## Appendix I APPENDIX OF LEGAL PRINCIPLES

### Kevin Giordano

*When you have assembled what you call your "facts" in logical order, it is like an oil lamp . . . filled and trimmed; but which will shed no illumination unless first you light it.*

Antoine de Saint Exupéry (1900–1944)

*The Wisdom of the Sands (Citadelle)* 1948 (posthumous)

This appendix includes a more technical discussion of selected legal principles as they apply to medical practice in obstetrics and gynecology. Important common law elements of medical negligence are discussed, and in many instances, specific case law is referenced. These comments are not to be construed as legal advice; however, they provide a general review of selected concepts and principles that underlie the legal approach to specific issues involving common problems of consent, expert testimony, duty, and causation. Application of these precepts to a specific case requires a review of the facts of a specific case and expert opinion by a qualified legal practitioner.

Medical malpractice is simply a form of negligence. The distinction between ordinary negligence and malpractice turns on the fact that the acts or omissions cited involve matters of special medical knowledge not ordinarily possessed by laypersons. Because of the complexity of the issues, these cases cannot be determined solely on the basis of the common everyday experience of the jury [1]. The analytic line that separates medical malpractice cases from other negligence cases is that typically a jury can determine the outcome only if expert medical testimony has been introduced.

The essential elements of proof in a medical malpractice action are 1) a deviation or departure from accepted [medical] practice, and 2) evidence that such departure was a proximate cause of injury or damage to the plaintiff.

The law recognizes that obstetricians are not guarantors of the success of any medical treatment provided to a patient. Consequently, an obstetrician is not automatically deemed liable for the occurrence of an undesirable result. Instead, malpractice arises when there is a departure from the standards practiced in the same or similar circumstances by a reasonably competent obstetrician. The duty owed to any patient is to provide care consistent

with the accepted standard of care at the time and under the circumstances of the case. Implicit in this definition of medical negligence is that the clinician has departed from the duty that is owed to a patient.

#### DUTY: PATIENT–PHYSICIAN RELATIONSHIP

In the absence of a patient–physician relationship, a medical malpractice case cannot be maintained. Historically, the relationship between physician and patient was viewed as solely arising out of an implied contract in which the patient knowingly seeks medical attention from the physician, and the physician knowingly accepts the person as a patient [2]. From this mutual understanding, the physician’s duty to provide care consistent with good and accepted practice arises. In most cases, relying upon the principles of contract law, the existence of a relationship is clear, yet there are many cases in which determining whether a physician–patient relationship existed is often difficult, and an application of contract law is unjust. In such circumstances, the courts must consider the totality of the circumstances.

Case law has made it clear that the mere fact that a physician has had contact with a patient, or a physician treating any patient, does not in itself impose a duty. If, in fact, a patient has only had *de minimis* consultative contact with a treating physician, that contact might be considered insufficient to support a physician–patient relationship. An example of *de minimis* contact is the case of *Childs v. Weiss* [3]. In *Childs*, an ED nurse telephoned an obstetrician on call for the emergency department and advised him that an obstetric patient from a different locality was in the emergency department having labor pains. In response, the physician told the nurse to “have the girl call her doctor and see what he wanted her to do.” The physician was not involved in the incident beyond that communication. The issue before the court was whether the obstetrician was obligated to see every patient who arrived for treatment at the emergency department and whether he could be held liable for refusing to respond to the call [4]. The Texas court found it particularly significant that the only action taken by the doctor was to instruct the nurse to have the patient call her own doctor. On this limited interaction, the court thus held that no physician–patient relationship was formed [5].

Other examples exist where minimal contacts between the patient and the physician have led courts to conclude that the interaction was insufficient to establish relationship. In *Sullenger v. Setco Northwest*, the defendant doctor, on entering patient’s room, was asked whether he would like to manage the case and declined [6]. Similarly, no relationship was found when a consulting physician gave the treating physician his informal opinion of the patient’s condition, without seeing the patient, and this opinion was relayed to the patient’s mother [7]. The court held that there was no evidence “...from which it could be concluded that [consulting physician] has consented to treat the child, or any from which it could be inferred that he consented to act in a consulting capacity” [8].

An important policy consideration underlying the reluctance of courts to impose a duty when there is only minimal contact between a physician and a patient is the identified need for access to information between professionals. Courts have equated the consulted doctors with source material, such as a treatise or textbook, noting that the consultant contributed to the body of information available to the treating physician when treating a patient. Consultations between professional people facilitate the free flow of information between colleagues and work to the benefit of the physician and the patient. To assess liability against “...doctors with whom a treating physician has merely conferred... would unacceptably inhibit the exchange of information and expertise among physicians” [9].

Given the evolving medical environment, however, courts have expanded the definition of the physician–patient relationship beyond the need for the mutuality of an implied agreement between the patient and the physician. With the complexity of modern medicine and its dependence on subspecialists and complicated medical technologic advances, care for patients has increasingly been undertaken by teams of professionals, some of whom never actually come in contact with the patient to whom care is being provided. As one commentator has stated [10],

**The health care environment requires cooperation and teamwork. Physicians are dependent upon many other health care professionals in a health care institution to ensure good patient care. . . . The health care professional is**

**obligated to take actions to protect the interest of patients, who are innocent parties in the health care environment. A failure to act in the interest of good patient care or in the protection of the public welfare creates liability.**

Consequently, analysis of this relationship through the lens of contract law is useful but often is not the dominant factor when additional, overriding circumstances are present. *Medical professionals are now being held accountable when they participate in the care of a patient if their actions do not meet the standard of care, even though the physician and the patient have never met.* Recent case law makes it clear that physicians are not shielded from liability merely because they have not had any actual contact with a patient. In *Wheeler v. Yettie* [11], the plaintiff was a pregnant woman who was brought to the emergency department, and a decision was required to determine whether she could be transferred to another, more distant medical facility to give birth. She was examined by two nurses. During a single telephone call, one of the nurses discussed the plaintiff's status with an on-call physician. The physician, who had privileges at the hospital, had no direct contact or connection with the plaintiff. In fact, the plaintiff was completely unaware that the nurse had telephoned the physician. The on-call physician evaluated the information communicated to him and made a medical decision that the plaintiff could be transported. Until the time of this decision, prior case law had held that no such relationship was deemed to exist when "... the rules and custom of the particular hospital required only that the 'on-call' physician consult with the attending physicians." In distinguishing the case for these prior decisions, the *Wheeler* court noted the "... unique circumstances presented in a transfer situation," and determined that the role of the on-call physician was not merely to be available for consultation, but that he actually had rendered medical decisions that foreseeably would have an impact on decision making about the plaintiff, thus establishing a physician-patient relationship [12]. The court found that the traditional rule was no longer applicable, and that a physician-patient relationship can be deemed to exist between an "on-call" attending physician and a patient when treatment was either required by hospital rules or in fact was undertaken by the physician. The *Wheeler* court reasoned that

the best approach to determining the existence of a legal relationship is not to use a bright line rule such as whether a mutual agreement to provide care existed between the parties, but instead to undertake a qualitative analysis of the physician's actions. This type of analysis undertakes to determine the extent to which the consultative physician has exercised independent professional judgment and the extent to which that judgment was relied upon by the careproviders [13].

It is the qualitative analysis approach that is generally employed currently when one is attempting to evaluate the evidence of any physician-patient relationship. For instance, such an approach was used in *Gilinsky v. Indelicato* [14]. In *Gilinsky*, the plaintiff introduced evidence to show that, during a 5-hour period, the defendant physician was consulted on seven different occasions about the physical symptoms of a specific patient, who ostensibly needed emergent medical treatment. The telephone calls were placed by the physician consultant, and the duration of the calls was approximately 38 minutes total. The case went to trial, and the consultant was a named defendant. The jury held the defendants liable and awarded damages to the plaintiff. On appeal, the defendant consultant argued that the jury's decision was in error because there was no physician-patient relationship that existed at the time. In framing the issue, the court concluded that the question was whether sufficient evidence existed that a reasonable jury could find that the physician's involvement crossed the boundary that divides mere curbside advice from actual direction. The court concluded that there was evidence that the nature of the consultation was "continuous and substantial" rather than "fleeting and informal." Thus it was determined by the appellate court that a jury, drawing on their common everyday experiences, could conclude that the defendant failed to act as a reasonably prudent physician under like circumstances by attempting to diagnose and direct the treatment of the plaintiff over the telephone.

The *Gilinsky* court identified the following circumstances to be highly probative in evaluating whether a physician-patient relationship came into existence: 1) the extent to which the consultative physician exercised his professional judgment in a matter bearing directly on the plaintiff, and 2) the ability the consulting physician to foresee that his exercise of judgment ultimately would determine

the precise nature of the medical services rendered to the plaintiff. Where the consultative physician is merely providing informal advice to another physician as to how to proceed with respect to a patient whose identity is unknown to him, and the treating physician exercises his or her own independent judgment in determining whether to accept or reject such advice, the consultative physician probably will not be regarded as a joint provider of medical services nor as a party to a physician–patient relationship; however, the determination will be made by evaluating the circumstances of each case.

Courts have noted that by eroding the historical requirement of a mutual agreement between the patient and physician, they do not wish their decisions to be construed to hamper the free flow of information between medical professionals. This does not detract from the distinction that occurs when a physician exercises professional judgment while in consultation with another physician, when the latter subordinates his/her independent professional judgment to that of the consultant, specifically when this subordination of judgment was reasonably foreseeable. Assuming that such a relationship existed, a physician can be properly regarded as a provider of medical treatment to the plaintiff, with the consultant functioning with the primary physician and not simply as a source of information.

If a duty should arise during a consultation, the important point is whether the consultant could foresee that physician attending the patient would rely on his/her advice and perform the exact procedures that were given. The issue is whether there was actual direction that the consulting physician provided to the primary treating doctor and whether the consultant should have been aware of the likelihood of his/her advice being followed directly. There need not be evidence to suggest that either the consulting physician or the plaintiff knew each other or had in any way contemplated a physician–patient relationship before the consultant's involvement in the case. In fact, the patient need not even be cognizant of the consultant's involvement in her care.

Medicine still has a need for the curbside consultation. Particularly in light of the rapidity of scientific advancements being made in medicine, informal consultations are of utmost importance and can be a more efficient means of assisting physicians

in keeping current with medical information. The law remains conscious of this benefit, and the mere discussion between professional people of hypothetical situations cannot be viewed as a basis for liability. To hold otherwise would tend to adversely affect the quality of the services that these physicians offer to the public. Consultations of this manner are evaluated on the particular circumstances attendant to the communication(s) of each case. A specialist should make it a practice to state clearly that without a formal consultation, any discussion is general and does not constitute a diagnosis or treatment plan. The more in-depth any particular discussion becomes, especially when it is clear that it involves a real patient or the specialist is called on more than once, then the consulting physician should consider recommending that a comprehensive clinical evaluation be performed before rendering any further advice.

### Supervision of Resident Physicians

Although it does not conform to traditional notions of a doctor–patient relationship, a physician's duty of reasonable care has been extended to the supervision of residents. Courts in most jurisdictions have recognized a duty of care owed by a hospital to provide medically acceptable rules and regulations that would ensure appropriate supervision of ill patients, and the failure to do so is a reasonable basis on which to find a breach of the standards of medical care owed to that person [15,16]. In the case of *Maxwell v. Cole* [16], a department chairman was held legally responsible for failing to implement acceptable rules and regulations in the administration of a residency program sufficient to provide reasonable supervision of hospitalized patients. In *Maxwell*, the patient suffered a bladder perforation during an elective tubal ligation. The resident physicians caring for the patient failed to diagnose the condition postoperatively in a timely manner, and the patient developed septic shock. As the court determined, the hospital's role as a provider of healthcare requires some obligation of supervisory responsibilities over those who practice medicine within the institution. The court stated that "if the chief of service fails to provide medically acceptable rules and regulations which insure appropriate supervision of ill patients, then it is reasonable to find that a breach of the standard of medical care by the individual has occurred."

Supervisory responsibility also trickles down to attending physicians who exercise improper supervisory oversight. In *Moeller v. Hauser* [17], the defendant had performed surgery on the plaintiff, and postoperative complications developed. The lawsuit was brought against the physician in his capacity as surgeon but also in his capacity as the supervising physician of the residents who provided care to the patient during her postoperative course. The jury found against the attending in this secondary capacity but not as surgeon. In affirming the jury verdict against the surgeon, the court held that, in addition to the duty that arose out of the doctor–patient relationship, the appellate doctor would also have liability for his patients’ injuries caused by the negligent postoperative care rendered by the resident physicians. In *McCullough v. Hutzel Hosp.* [18], the appellate court concluded that a supervising physician owed the plaintiff a duty of care in supervising the residents actually caring for the plaintiff [19]. The plaintiff in that case claimed that the defendants improperly performed a tubal ligation on her. Because the surgery was performed in a teaching hospital, a resident physician supervised by the defendant actually performed the operation. A few months after the operation, the plaintiff became pregnant and subsequently underwent a therapeutic abortion and a hysterectomy. The jury awarded damages to the plaintiff. In upholding the verdict the court stated, “Even though the surgical procedure was actually performed by a resident, defendants were under a duty to see that it was performed properly. . . . [The defendants’] failure to take reasonable care in ascertaining that the surgery was competently performed renders them liable for the resulting damages” [19].

Supervisory responsibility has also arisen despite little or no ties to establish a physician–patient relationship between the attending and the particular patient. In *Mozingo v. Pitt Cty. Mem. Hosp.* [20], the defendant obstetrician had on-call duty as a supervisor for the obstetric resident physicians who were caring for hospitalized patients. During that duty, the attending physician remained at home, available to take telephone calls from the residents. During the evening, the obstetrician received a telephone call from a resident physician informing him that she had encountered a problem with a delivery, specifically, a shoulder dystocia. The obstetrician stated that he would be there immediately and left his

home for the hospital, which was located approximately two miles away. Not surprisingly, by the time the attending obstetrician arrived at the hospital, delivery had been accomplished. Unfortunately, the child had suffered neurologic impairment.

An action was brought against the senior obstetrician as the on-call supervising physician when the plaintiff child was born. The plaintiffs alleged that the physician “. . . failed to make reasonable effort to monitor and oversee the treatment administered by the second-year OB resident physician, and the agents of the Defendant, [the] Hospital.” The attending obstetrician sought dismissal of the action, claiming that he had no physician–patient relationship and was not vicariously liable for the residents because they were not his agents. If the court decided that according to the law, the plaintiff could not prove the existence of a duty under any set of circumstances, then the case would be dismissed. In weighing the facts, the court considered the plaintiffs’ expert’s sworn affidavit, which stated that the defendant “should have called in at the beginning of his on-call coverage and periodically thereafter to check on the status of the patients” being treated and managed by the residents. In addition, the affidavits submitted on behalf of the defendant stated that an “on-call physician may take calls at home ‘unless a problem is specifically anticipated.’” The court agreed that the resident physicians were not the agents of the attending physician. As noted by the court, however, according to defendant’s own experts, “simply remaining at home and available to take telephone calls is not always an acceptable standard of care for supervision of residents.” It therefore concluded that the a jury could find that the defendant’s failure to call in and periodically check on the status of the patients being treated by the residents was a breach of duty, therefore “a genuine issue of material fact was established as to whether the defendant doctor breached the applicable standard of care and thereby caused the plaintiffs’ injuries.”

The *Mozingo* case did not establish liability against the physician; instead, it determined that in light of the evidence, the jury had to decide whether the attending physician acted reasonably even though he did not call in to the hospital to determine the condition of the patients for whom he was covering. One of the important lessons from *Mozingo* is that the risk of exposure to liability for nonpatients

is undeniably greater for those physicians who practice in an academic setting and are involved in the education and training of residents. Understanding all hospital rules and regulations regarding supervision of residents is essential. Similarly, any on-call agreements and contracts with the hospital that include provisions regarding resident supervision must be understood. Providing reasonable oversight in accordance with hospital policy or contractual obligations is the key to reducing liability.

### The Physician As Good Samaritan

Is the duty that arises any different when the physician–patient relationship arises out of a moral or ethical decision to assist, rather than the usual manner in which physicians within their specialty are called on to provide care? It is a well-accepted legal proposition regarding rescuing a person from harm that “no one is obliged by law to assist a stranger, even though he can do so by a mere word, and without the slightest danger to himself” [21,22]. This is true for laypersons as well as physicians [21,22]. Illustrating the point is the case of *Hurley v. Eddingfield* [22]. In *Hurley*, the plaintiff’s child was dangerously ill, and the plaintiff requested medical attention from the defendant. The defendant had no prior physician–patient relationship with the family or child, nor was he under any other independent duty to provide care; however, he was the only physician available to provide care. Nonetheless, he refused to offer care to the plaintiff’s child. The child died, and the plaintiff sued. The trial court held in favor of the physician, ruling that there was no duty imposed by law, nor any duty that was imposed by contract or otherwise, that required the physician to render care.

What is the liability of a physician who chooses to act in such situations, however? Does holding a physician responsible for causing injury when happening on the scene of an emergency make sense, when failing to act would create no liability? A noted legal scholar, Dean William Prosser, summarized this conflict by concluding: “The result of all this is [would be] that the Good Samaritan who tries to help may find himself mulcted in damages, while the priest and the Levite who pass by on the other side go on their cheerful way rejoicing” [23].

Nonetheless, the common law imposes a duty that once a bystander endeavors to provide assis-

tance, that person has a duty to do so reasonably. A volunteer could thus be held liable for injuries caused by his or her negligent assistance. In *United States v. DeVane* [24], the court was confronted with a nonmedical case in which the Coast Guard undertook to rescue a boat that was in peril. This decision is considered a discretionary function of the Coast Guard, is not a legal duty. The court noted though, that although the decision to conduct a search and rescue operation is discretionary, “. . . having undertaken the rescue and engendering reliance thereon, the obligation arose to use reasonable care in carrying out the rescue.”

Given the potential liability in these situations, Good Samaritan legislation has been passed in every jurisdiction. The objective of the legislation is to encourage the rendering of medical care to those who need it but otherwise might not receive it by persons who come upon such victims by chance, without the accoutrements provided in a medical facility, including expertise, assistance, sanitation, or special equipment. In 1959, California became the first state to immunize physicians from tort liability who render assistance at the scene of an emergency. The law was enacted despite the fact that “. . . there could be found no instance, in California or any other state, of a physician being sued for negligence in rendering aid at the scene of an emergency” [25]. A California Court of Appeals explained the rationale for the legislation as follows [26]:

**[The statutes] were enacted to aid the class of individuals though requiring immediate medical care were not receiving it. Typically, it was the roadside accident victim who, as a result of the strictures of the common law malpractice doctrines, was left uncared for. However, hospital patients . . . have historically enjoyed the benefits of full medical attention. There is no need for special legislation to encourage physicians to treat this class of individuals. The Good Samaritan sections were directed towards physicians who, by chance and on an irregular basis, come upon or are called to render emergency medical care. Often, under these circumstances, the medical needs of the individual would not be matched by the expertise of the physician and facilities could be severely limited.**



Typically, Good Samaritan statutes extend immunity to care provided to victim(s) at the scene of an accident or emergency; however, the legislation is not uniform, and various disparities exist among the statutes. The most significant difference is the definition of “scene of an accident or emergency.” Legislative enactments in some states contain specific provisions to include emergency care provided in a hospital setting exclusive of care provided in the emergency department. In other states, however, Good Samaritan statutes unequivocally exclude immunizing healthcare providers from emergency care rendered to patients in a hospital or other healthcare facility. Still another group of states have promulgated statutes that do not explicitly address whether in-hospital care is shielded from liability.

In those jurisdictions that fall in the latter category, a further disparity exists in that there is no universal court interpretation for statutory language such as “scene of an emergency”; judicial decisions interpreting statutes that neither expressly exclude nor expressly include in-hospital emergency medical care are split. The difference in outcome between the cases is based, in great measure, on the court’s interpretation of the intent of the legislature when enacting the statute, and whether there is evidence favoring immunity in that setting. One of the most comprehensive discussions of this debate is incorporated in the decision of *Velazquez v. Jiminez* [27].

In *Velazquez*, the patient was hospitalized for the purpose of delivering her baby. Complications occurred during the delivery owing to a severe shoulder dystocia. After delivering the baby’s head, the treating obstetrician was unable to deliver the rest of the baby’s body, and she rang for assistance. The defendant physician responded and provided help in the delivery. After responding, the defendant first attempted to complete the delivery vaginally. When those efforts proved unsuccessful, the defendant assisted in making preparations for an emergency cesarean. The baby, who was born severely brain damaged, spent his life in a dependent state and died of pneumonia before reaching his third birthday.

The defendant physician had no prior relationship with or connection to the plaintiff. As a specialist in maternal-fetal medicine, the physician was responsible both for attending to high-risk patients and for supervising resident physicians who cared

for the academic center’s clinic patients. The plaintiff was neither a high-risk nor a clinic patient, however; rather, she was the patient of a private attending physician with staff privileges at the medical center.

It was the defendant’s contention that the evidence supported that the physician had no obligation to respond to the physician’s call, and thus her involvement was voluntary. Thus the court was confronted with whether the legislature intended the state’s Good Samaritan Act to intercede and immunize the assisting obstetrician from suit for care she provided while providing care within the hospital setting. The issue was not addressed by the legislation in the statute. The court’s pursuit was to attempt to determine the goal of the legislature at the time that statute was enacted. In ruling against applying immunity to physicians who respond to emergencies in the hospital, the court stated that “. . . [the Defendant’s] suggestion that she qualifies as a Good Samaritan because she had no prior duty to [the plaintiff] misconceives the Good Samaritan Act entirely. Although the absence of a preexisting duty is one element that volunteers must establish to qualify for Good Samaritan immunity . . . standing alone it does not satisfy the statute.” The court further ruled that immunity would inure to those volunteers providing care at the “. . . scene of an accident or emergency . . .” and that the reasonable interpretation of such language should be understood to incorporate only locales at which the provision of adequate and necessary medical care is compromised by the existing conditions. Their assessment determined that physicians who care for patients in hospitals are not volunteers in the same sense as are persons who by chance come upon the scene of an accident. Moreover, physicians who provide emergency care in hospitals have at their disposal all the modern diagnostic and therapeutic equipment of the institution. In essence, as the court recognized that the choice of whether to extend immunity to the hospital setting was for the legislature to determine. Given that, the *Velazquez* court was unconvinced that the state statute, as worded, reflected a legislative choice in favor of immunity in those circumstances.

It should be recognized that the *Velazquez* case addressed a legal issue and did not establish liability. Under consideration was whether the physician was immunized from potential liability. Although the

court determined that she did not have immunity, it was ultimately for a jury to determine whether the care she provided under the circumstance was reasonable. Thus, Good Samaritan Acts render a very circumscribed population of emergency volunteers immune from suit. In all other situations, people are subject to the ordinary common law rules governing conduct and negligence. That is, if a party has a duty to act and provides care in a negligent manner, the breach is potentially actionable [28]. In the absence of a preexisting legal duty, if a party undertakes to act and does so in an unreasonable manner, that conduct is also potentially actionable [29,30].

Standard of care depends on the facts of each particular case and the clinical setting in which the physician finds him- or herself. In an emergency setting, a physician responding to an emergency might not be familiar with the patient's medical history or any coexisting disease and thus is at a disadvantage when compared with the patient's personal physician. Although the existence of an in-hospital medical emergency might not create the application of the Good Samaritan statute, the limitations faced by the responding physician must be considered by the jury in determining whether the volunteer practitioner complied with the standard of care.

### Standard of Care

Once a physician-patient relationship exists, the obstetrician's duty to a patient is to employ the care and skill of the ordinary physician practicing in the specialty of obstetrics. The obligation that arises out of this relationship does not require the obstetrician to be the most skillful or careful physician. Instead, the obligation requires the obstetrician to meet a minimal standard, that being to use the care of the average or ordinary practitioner. If a physician possesses reasonable and ordinary learning and uses care such as that used in like or similar situations by practicing physicians of reasonable and average skill, then they are not negligent, even though the judgment employed might subsequently be proved incorrect. Thus, a mistake in judgment on the part of a physician is neither a breach of the duty owed, nor is it evidence that there has been a departure from the skill that would have been provided by the average competent physician. That is true even if there were alternative procedures in the treatment

of the patient that might have lead to an improved outcome.

Consequently, to maintain an action for medical malpractice, after establishing that an obligation or duty to the patient existed, the plaintiff has the burden of proving 1) the standard of acceptable professional practice in the profession that the defendant practices at the time of the alleged wrongful action, and 2) that the doctor acted with less than, or failed to act with, ordinary and reasonable care in accordance with such standard(s). Specifically, in reference to obstetric cases, the plaintiff must establish through expert testimony what the standard of care required. Furthermore, the patient must prove that the obstetrician failed to act within the recognized standard of practice or care within the specialty of obstetrics and gynecology for the condition(s) in question at the time of the alleged malpractice.

In the past, the applicable standard of care was established by the locality in which the obstetrician practiced. Most jurisdictions have repudiated the "same or similar" community test in favor of a national standard, however. Thus it is generally held that a physician is under a duty to use the degree of care and skill that is expected of a reasonably competent practitioner in the same class of practitioners to which he or she belongs, acting in the same or similar circumstances. Whatever geographic impediments might have existed previously that justified the need for a "similar locality" rule are no longer applicable in view of the present-day realities of the medical profession. As the Shilkret court observed [31]:

*The modern physician bears little resemblance to his predecessors. As we have indicated at length, the medical schools of yesterday could not possibly compare with the accredited institutions of today, many of which are associated with teaching hospitals. But the contrast merely begins at that point in the medical career: vastly superior postgraduate training, the dynamic impact of modern communications and transportation, the proliferation of medical literature, frequent seminars and conferences on a variety of professional subjects, and the growing availability of modern clinical facilities are but some of the developments in the medical profession which combine to produce contemporary standards that*

**are not only much higher than they were just a few short years ago, but are also national in scope.**

The abrogation of the same or similar community tests has had the greatest impact in widening the pool of potential expert witnesses. The existence of a national standard is normally pro forma, but trial judges are required to address the admissibility of national standard-of-care testimony. Certain principles should govern this assessment: 1) it is insufficient for an expert's standard-of-care testimony to merely recite the words "national standard of care"; 2) such testimony may not be based on the expert's personal opinion or what the expert would do under similar circumstances, nor on mere speculation or conjecture; and 3) such testimony, when critiquing a medical course of action or treatment, must reflect some evidence of a national standard, such as information presented at national seminars or meetings or conventions, or reference to published materials.

Ultimately, what is the applicable standard of care required is a question of fact to be determined by the jury, based on the expert testimony presented, and it can involve the evaluation of conflicting testimony and a weighing of credibility of the expert witnesses presented by both sides. In demonstrating that a particular course of action or treatment is followed nationally, reference to published standards is not required but can be important.

## CAUSATION

In accordance with ordinary negligence principles, the plaintiff in a malpractice action has the burden of proving *causation*, that is, proving by the preponderance of the credible evidence not only that the defendant was negligent but also that they incurred damage as a direct and proximate result of the defendant's negligence. The plaintiff must also present evidence that they sustained legally recoverable *damages*. The rule of *reasonable medical probability* relates to the showing that must be made to support an ultimate finding and to which the medical expert must testify regarding the issue of causation [32]. Proof of causation equating to a "possibility," a "might have," "may have," or "could have" is insufficient to establish the nexus between the plaintiff's injury and the defendant's malpractice.

A plaintiff is not required to establish causation in terms of medical certainty, nor is he or she required to exclude every other reasonable hypothesis. Causation in fact is a matter of probability, not possibility, and in a medical malpractice case, such must be shown to a reasonable degree of medical certainty [33,34]. A reasonable degree of medical certainty is interpreted as greater than a 50% likelihood of an association; that is, the alleged association is simply more likely than not.

The recent trend among courts is to describe causation as requiring that the defendant's conduct be a substantial factor in bringing about the plaintiff's harm. The *substantial factor* test has become popular because in the complex litigation of today, courts are often forced to consider claims that might have arisen from multiple causes. In malpractice cases, referring to "the cause" can be misleading because, although an underlying illness or medical condition might be the cause of the patient's harm, it might also be that such harm could have been averted in the absence of the physician's negligence. Thus, the plaintiff must demonstrate that it is more likely than not that the injury, harm, or condition claimed to have resulted from that negligence was a substantial factor in causing the injury, harm, or condition, and without which that injury, harm, or condition would not have occurred [35–37].

Once the substantial contributing factor standard is established, the physician is liable for the reasonable and probable consequence of his or her negligent conduct. Most courts do not apply a pure *foreseeability* test. Thus, it is not a defense in most jurisdictions to claim that damages are not causal because they were either unexpected or surprising. *It is sufficient for liability if a reasonable defendant could foresee that a person could be injured, as opposed to the nature of the injury.* As one court has stated, "... It is of course unnecessary that the party charged should have anticipated the very injury complained of or anticipated that it would have happened in the exact manner that it did. All that is necessary is that he knew or ought to have known that there was an appreciable chance some injury would result" [38].

Prosser stated it this way: "It is as if a magic circle were drawn about the person, and one who breaks it, even by so much as a cut on the finger, becomes liable for all resulting harm to the person, although it may be death" [39].

Consequently, legal causation principles do not require that the obstetrician foresee that his or her negligence would produce the specific harm or damage that resulted. *It is merely required that the clinician could foresee that the conduct could produce harm to the mother or fetus.*

### LOSS OF CHANCE

The traditional standard of the sufficiency of the evidence for establishing a medical malpractice case required a plaintiff to prove, to a reasonable medical probability, that the harm suffered was caused by the negligence of one or more physicians. Many malpractice cases, however, involve a preexisting condition that can lead to serious morbidity, or even mortality, with the outcome being the inexorable result of the medical condition. Delay in diagnosis cases often fall within this category. Under a traditional standard for proving causation, where a preexisting condition is involved, the plaintiff must establish to a reasonable degree of medical certainty that absent the physician's negligence, either survival or a better outcome would have been the result. In other words, that with proper care the plaintiff's chances of survival or a significant improvement were better than 50/50. This created a hardship in many types of cases, particularly in cancer-related cases and obstetrics, as the plaintiff's expert witnesses were called upon to establish that timely treatment would have prevented the outcome, rather than merely have improved the odds.

In recent years, several states have changed the traditional causation standard by adopting some version of the *loss-of-chance doctrine* [40–43]. In adopting to apply the doctrine, one court explained it as follows [44]:

**... the loss of chance theory is, essentially, one that allows an injured plaintiff to recover damages based upon a reduced standard of causation rather than the traditional one which requires the plaintiff to prove that it is more probable than not that the damage suffered was caused by the negligence of the defendant... [The relaxed causation approach] requires [the] plaintiff to present evidence that a substantial or significant chance of survival or better recovery was lost. If [the] plaintiff meets this initial threshold, the causation**

**issue is submitted to the jury, using the traditional proximate cause standard to ascertain whether, in fact, the alleged malpractice resulted in the loss of a substantial or significant chance. Thus, the jury must find by a preponderance of the evidence that the alleged negligence was the proximate cause of the lost chance, but the lost chance itself need only be a substantial or significant chance, for a better result, absent any malpractice, rather than a greater than 50% chance of a better result.**

Initially, this approach was used most often in cases involving allegations of wrongful death. The standard applied that would be applied by the court would be "... if a defendant physician, by action or inaction, has destroyed any substantial possibility of the patient's survival, such conduct becomes a proximate cause of the patient's death" [33,45,46].

The loss-of-chance doctrine was developed in response to the often-harsh results of the traditional "all-or-nothing" rule [47]. Under the traditional formulation, the plaintiff must prove within a reasonable probability that defendant's breach of the standard of care was a substantial factor in causing the underlying injury. Because the compensable injury is viewed as the underlying injury, the plaintiff must prove within a reasonable probability that she would have recovered or survived without the defendant's negligent conduct. If the plaintiff is unable to prove a reasonable probability of recovery/survival, she would recover nothing. It is in these narrow cases that the loss-of-chance doctrine comes into play.

Although the traditional all-or-nothing rule and the loss-of-chance doctrine are similar, the right of recovery is vastly different. For example, a patient suffering from a potentially terminal illness might allege that the defendant physician failed to timely diagnose and treat that illness. Expert medical testimony incontrovertibly established that the physician's failure to diagnose and treat the illness quickly was a breach of the standard of care. The medical evidence also established that it was more probable than not that the breach of the standard of care caused a lost chance of survival. Specifically, it might be determined that the patient had a 45% chance of survival at the time the physician failed to diagnose the illness. Later, when the illness was properly diagnosed, the patient's chance of survival diminished to only 15%. The patient eventually died of the illness.

Under the traditional all-or-nothing rule, the plaintiff would recover nothing because it could not be shown within a reasonable probability that the physician's failure to diagnose caused the patient's death. Conversely, under the loss-of-chance doctrine, the patient could recover for the lost chance of survival on a proportional basis. In this example, if the medical evidence were interpreted as proving within a reasonable probability that the physician's failure to diagnose caused a 30% diminished chance of survival, the patient could recover damages proportional to that 30% loss. Thus, if the total damages were assessed \$100,000.00, the patient would recover 30% of that amount, or \$30,000.00.

The background of this approach is in King's influential article in the *Yale Law Journal*, in which the loss of chance of survival or improved health is conceptualized as a separate cause of action, meriting recovery [48]. In advancing his theory, King postulated that the "... failure [of courts] to distinguish between the functions of causation and valuation, or to identify and value rationally the true interests lost, has created a serious gap in the remedial structure. ..." He also stated that "... the loss of a chance of achieving a favorable outcome or of avoiding an adverse consequence should be compensable and should be valued appropriately, rather than treated as an all-or-nothing proposition." King's argument is that the loss-of-chance recovery in cases involving a preexisting condition merit compensation when it can be proved that except for the negligence of the defendant, the plaintiff would have attained a better result or avoided an adverse consequence. He agreed that courts should apply the traditional reasonable medical probability test, but that in recognizing a chance as compensable in its own right, unless the preexisting conditions of the plaintiff "... have not absolutely preordained an adverse outcome, [then] the chance of avoiding it should be appropriately compensated even if that chance is not better than even."

One commentator has further explained the rationale behind this loss-of-chance doctrine as follows:

**The [traditional] causation approach requires the finder of fact to determine whether the decedent's chances to live or to achieve a more favorable result were more probable than not. Once the evidence shows that a probability**

**did or did not exist, the inquiry ends. As a result, chances of less than 51% are treated as if they were nonexistent. A more sensible approach would be to redefine the victim's injury as the loss of a chance. Instead of attempting to determine whether the physical harm was caused by negligence, a court could examine the extent of the victim's lost chances for cure or improvement and grant a recovery that mirrors the extent of those chances. When viewing the question in the negligence setting, the harm suffered would be the loss of the chance. The relevant inquiry would be whether the defendant "probably" caused a reduction in the victim's chances. If causation were found, the court would provide compensation for the lost chance in direct proportion to the extent of the lost chance [49].**

As an Ohio court put it:

**The loss of a speculative chance of recovery alone is not an injury from which damages flow. We are convinced that the better rule to follow is the one which requires the plaintiff to prove that the defendants' negligence in all probability proximately caused the injury. Only by such proof will the plaintiff have proven proximate cause. . . [50].**

The important feature is that the loss of chance in a specific instance cannot be only speculative. After again stressing that the chain of causation in establishing liability is concerned only "with reasonable probabilities and not possibilities." The court in *LaBieniec v. Baker* opined that "... [in] this case, the evidence failed to remove the decreased chance of successful treatment from the realm of speculation" [51]. The court then clearly stated that "... [s]ince there was no evidence which could have led the jury to a reasonable inference that the defendants' acts of malpractice were the direct and proximate cause of a decrease in the chance of successful treatment, it was proper for the trial court to direct a verdict on this issue."

Under the interpretation of the loss-of-chance doctrine discussed here, the plaintiff is required to prove that defendant's breach of the standard of care was a substantial factor in causing a diminished chance of recovery/survival from the

underlying injury [52–54]. Most often, the loss-of-chance doctrine is employed where the breach of the standard of care involved a failure or delay in diagnosis or treatment.

Under the loss-of-chance doctrine, the *substantial factor test*, as set forth in Restatement (Second) of Torts [55], is applied to determine causation. The substantial factor test is also used in traditional malpractice actions, coming under the all-or-nothing rule; thus, causation remains congruous under the loss-of-chance doctrine. The degree of certainty required to establish causation, likewise, remains the same – reasonable probability [56]. *Reasonable probability* is defined in the usual manner as “more probable than not” or “more likely than not” [47,57,58]. Again, from a statistical viewpoint, reasonable probability means with a greater than 50% chance [59–61].

At the case, the sole distinction between the traditional all-or-nothing rule and the loss-of-chance doctrine is the compensable injury. Under the all-or-nothing rule, the compensable injury is the underlying injury. In contrast, under the loss-of-chance doctrine, the compensable injury is viewed as the lost opportunity of recovery/survival from the underlying injury.

To maintain an action for a lost chance of recovery/survival, the plaintiff must still prove that defendant breached the applicable standard of care and that this breach was a substantial factor in causing a diminished chance of recovery/survival from the underlying disease or injury. The plaintiff must also present evidence proving causation by a reasonable probability, establishing that the chance of recovery/survival was 50% or greater before the negligent act or omission. In the presentation, however, evidence establishing causation or plaintiff’s chance of recovery/survival need not be expressed in terms of percentages; this is a question for the jury. An example of a probable jury instruction in such a case is set forth here.

**Special Jury Questions: Lost chance of recovery/survival case**

Questions for the jury to consider:

1. Do you believe from the evidence that the obstetrician breached the standard of care in the treatment provided to patient? \_\_\_  
Yes \_\_\_ No

If you, the jury, answered “yes,” then you must answer the following question:

2. Do you believe from the evidence that the obstetrician’s failure to exercise reasonable care was a substantial factor in causing the patient’s injury or death?

\_\_\_ Yes \_\_\_ No

If you, the jury, answered the above in the affirmative, you shall find in favor of the plaintiff proceed to the question of damages. However, if you answer the above question “no,” then you must answer the following:

3. Do you believe from the evidence that the physician’s failure to exercise reasonable care in the care and treatment of the plaintiff was a substantial factor in causing her to suffer a lost chance of recovery or survival from the underlying medical disease or injury? For purposes of this instruction, a lost chance of recovery or survival is defined as a 50% or less chance of recovery or survival at the time the defendant physician failed to exercise reasonable care in his treatment of the plaintiff. \_\_\_ Yes \_\_\_ No

If you answer the above instruction in the affirmative, proceed to the next question. If you answer the above instruction in the negative, then you shall find in favor of the defendant physician.

4. What do you find, in terms of a percentage, represents the plaintiff’s chance of recovery or survival, at the time of the defendant physician’s failure to exercise reasonable care in his treatment of the plaintiff. The percentage you find cannot be greater than 50%. \_\_\_% (50% or less). Proceed to Instruction 5.
5. What do you find, in terms of a percentage, represents the plaintiff’s chance of recovery or survival at the time she was properly diagnosed and treated? \_\_\_%
6. The plaintiff’s lost chance of recovery or survival will be determined by subtracting the percentage you find under Instruction 5 from the percentage you find under Instruction 4. The Judge then will determine the

**award by multiplying the total amount you determine as damages by the percentage previously determined to represent the lost chance of recovery or survival (subtracting the percentage under Instruction 5 from the percentage under Instruction 4).**

In the example provided, to recover under the loss-of-chance doctrine, the jury must find that the defendant physician breached the standard of care under Instruction 1, but that such breach was not a substantial factor in causing the underlying injury under Instruction 2. If the jury finds that the physician's breach of the standard of care caused the underlying injury, the plaintiff would be entitled to damages ordinarily recoverable in a traditional malpractice action. If the jury finds that such breach was not a substantial factor *in causing the underlying injury*, the jury may then consider whether such breach caused a lost chance of recovery/survival (Instruction 3). The jury can then fix the exact percentage representing that lost chance (Instructions 4 and 5). Finally, the judge determines the final amount of damages by multiplying the total damages by the percentage representing the lost chance of survival.

## INFORMED CONSENT

The duty to disclose the risks and alternatives of a proposed treatment adequately has been well established in law for many decades. The basis of informed consent is the simple principle that every person of adult years and sound mind has a right to determine what shall be done with his or her own body. Thus, the duty that exists is for a physician to disclose sufficient information to enable the patient to make an informed judgment whether to give or withhold consent to a medical or surgical procedure. Failure to do so constitutes medical malpractice. As the court articulated in *Dingle v. Belin*, "Unlike the traditional action of negligence, a claim for lack of informed consent focuses not on the level of skill exercised in the performance of the procedure itself but on the adequacy of the explanation given by the physician in obtaining the patient's consent" [62].

Prior to 1972, most courts dealing with the problem of consent made the physician's duty depend on the custom of physicians practicing in the community. This rule required a physician to disclose

whichever information a reasonable physician in similar circumstances would customarily disclose. It was held that a physician has a duty in the exercise of ordinary care to inform a patient of the dangers of, possible negative consequences of, and alternatives to a proposed medical treatment or procedure [63]. As with many aspects of malpractice litigation, recovery in nondisclosure lawsuits hinged on the patient's ability to prove, through expert testimony, that the physician's performance departed from medical custom. To recover against a physician for failure to provide such information, the patient was generally required to establish by expert testimony whether and to which extent any information should have been disclosed [64–76].

In a trilogy of cases decided in 1972, however, this traditional standard of customary medical practice was abandoned by three jurisdictions as the basic criterion for informed consent. These cases rejected the customary practice standard as providing insufficient protection for the patient's autonomy, which is the very purpose of disclosure. A new standard was designed to provide a patient with information material to his or her decision on a course of therapy. Most notable is the case of *Canterbury v. Spence* [67]. In *Canterbury*, after a myelogram revealed a "filling defect" in the region of the fourth thoracic vertebra, the attending physician advised the plaintiff that he would have to undergo a laminectomy to correct what was suspected to be a ruptured intravertebral disc. The plaintiff did not raise any objection to the proposed operation, nor did he inquire about the specific details of the procedure. At no time prior to the procedure was the patient advised of any particular risks of the procedure. Due to complications that occurred, the plaintiff became paralyzed following surgery. Despite extensive medical care, he never recovered. He was subsequently able to ambulate only with the use of crutches and was plagued with paralysis of his bowels and urinary incontinence.

In their opinion, the *Canterbury* court noted that historically, informed consent was given by a reasonable person who receives sufficient information to make an intelligent choice, and that it was up to reasonable healthcare professionals to determine the sufficiency of such evidence. Furthermore, the court noted that to be successful in an informed consent malpractice suit, a causal relationship must exist between the physician's failure to divulge

information adequately and damage to the patient. In reaching its decision, the court rejected the historical notion that the practice within the medical community established the sufficiency of the disclosure. Instead, the court stated

Some have measured the disclosure by “good medical practice,” others by what a reasonable practitioner would have bared under the circumstances, and still others by what medical custom in the community would demand. We have explored this rather considerable body of law but are unprepared to follow it. The duty to disclose, we have reasoned, arises from phenomena apart from medical custom and practice. The latter, we think, should no more establish the scope of the duty than its existence. Any definition of scope in terms purely of a professional standard is at odds with the patient’s prerogative to decide on projected therapy himself. That prerogative, we have said, is at the very foundation of the duty to disclose, and both the patient’s right to know and the physician’s correlative obligation to tell him are diluted to the extent that its compass is dictated by the medical profession [67].

The court went on to state

In our view, the patient’s right of self-decision shapes the boundaries of the duty to reveal. That right can be effectively exercised only if the patient possesses enough information to enable an intelligent choice. The scope of the physician’s communications to the patient, then, must be measured by the patient’s need, and that need is the information material to the decision. Thus, the test for determining whether a particular peril must be divulged is its materiality to the patient’s decision: all risks potentially affecting the decision must be unmasked. And to safeguard the patient’s interest in achieving his own determination on treatment, the law must itself set the standard for adequate disclosure [67].

The *Canterbury* court thus established *materiality* as the standard for determining whether a physician has an affirmative duty to disclose the information that the patient claims he or she was deprived. Whether a risk of injury is material to a patient

depends on the severity of the potential injury as well as the probability of its occurrence. If the likelihood of an injury occurring is negligible, then the risk is not considered material and is insufficient to trigger the physician’s duty to disclose. As the court noted, the risk of injury “. . . cannot be considered a material factor . . .” if “the probability of its occurrence. . . is so small as to be practically nonexistent.” Similarly, if the severity of the potential injury is “very minor,” the risk is immaterial and need not be disclosed [68].

Additional court opinions are important in evaluating this point. “Materiality may be said to be the significance [that] a reasonable person, in what the physician knows or should know is his patient’s position, would attach to the disclosed risk or risks in deciding whether to submit or not to submit to surgery or treatment” [69]. Material information “. . . may include the nature of the patient’s condition, the nature and probability of risks involved, the benefits to be reasonably expected . . . the likely result of no treatment, and the available alternatives, including their risks and benefits” [70].

The materiality standard for disclosure does weigh the risks of alternative treatments in deciding which information is material to the patient. An obstetrician in the delivery suite is in the unique situation of having to take into account the best interests of two people, mother and child, in rendering medical care. As such, in recommending a course of treatment, the standard of care might require the doctor to consider the risks to the mother, the risks to the child, and the appropriate balance of these risks. The standard of care that governs a conventional medical malpractice case differs from the materiality standard that governs informed consent cases, however. Under informed consent law, if a risk to the baby or a risk to the mother is material to the patient-mother’s decision, the doctor has a duty to disclose that risk. Once these risks and other material information have been disclosed, it is the patient’s prerogative to balance these risks and choose the form of treatment that best meets that patient’s needs. To the physician, whose training and experience suggest a specific course, the “correct” answer might seem clear, but it is the prerogative of the patient, not the physician, to determine the direction in which his or her interests seem to lie.

In considering the question of materiality the case of *Harrison v. United States* is important [71]. In



*Harrison*, the case involved balancing the risks to the child from a vaginal birth against the risks to the mother from a cesarean. The patient had been admitted at 37.5 weeks gestation for induction of labor. During labor, the baby's head crowned, but the shoulders did not deliver. The delivery team followed standard steps to attempt to resolve the shoulder dystocia. After the initial maneuvers were unsuccessful, the obstetrician delivered the posterior arm, which then allowed delivery of the baby. The baby weighed 4,508 grams at birth and was subsequently diagnosed with Erb's palsy resulting in a permanent neurologic injury. The judge determined that, although the risks of vaginal birth for the baby were "something more than negligible," when these risks were balanced against the risks to the mother from a cesarean, "a cesarean section to avoid brachial plexus injury was not a reasonable medical judgment." Thus, because the cesarean presented a greater risk, it was therefore not medically indicated at the time, and the doctor had no duty to disclose the risks of either procedure.

The court of appeal, however, reversed the district court judge's finding in *Harrison* that the physician had no duty to afford an expectant mother the opportunity to have a cesarean. Applying Massachusetts law, the court found that if a risk to the baby or a risk to the mother is material to the patient-mother's decision, the doctor has a duty to disclose that risk. The court noted

**The patient's opportunity to perform this balancing may assume particular importance when the patient is a mother giving birth. In such a case, the mother may purposefully discount risks to herself in order to choose a treatment or procedure that will present the least risk to her newborn child. While the treating physician will undoubtedly feel the need to balance the welfare of mother and child, the mother may consider her baby's health as the paramount concern.**

The appellate court remanded the case for further findings by the trial judge. In guiding the trial judge about the issue at hand, the court determined that if a risk existed either to the mother's or the child's health, such information would have been material to a reasonable patient. In this setting, therefore, the obstetrician had a duty to disclose that risk. More-

over, because there are only two methods of child-birth, if the district court found the data concerning the risk of vaginal birth to be material to the patient, then the obstetrician also had a duty to present the alternative option of a cesarean that might minimize such risk, regardless of his medical opinion on the proper course of treatment.

In jurisdictions that have adopted the materiality standard, there remains an important role that only medical evidence can fill. Unlike the jurisdictions where the standard for determining informed consent is based on the ordinary physician in a similar circumstance, expert testimony is not necessary, because the issue does not revolve around the accepted standard. Instead, experts are to identify and elucidate the risks and benefits of therapy, and the consequences of leaving existing medical conditions untreated. Furthermore, expert testimony is needed on issues about the cause of any injury or disability suffered by the patient and the lack of proper consent.

The guiding principle is that medical facts are for medical experts; however, for jurisdictions that judge nondisclosure cases, the issue of materiality does not entirely reside within the medical domain. The patient's testimony can establish a physician's failure to disclose particular risk information, and the adverse consequences following the treatment. Experts are unnecessary to establish the materiality of a risk to a patient's decision on treatment, or to the patient's response had material risk disclosure occurred. Nonetheless, it is the rare consent case that can be resolved without expert testimony.

## EXPERT WITNESSES

With few exceptions, in an action based on medical malpractice, the only way in which the jury can properly determine whether the defendant physician's conduct deviated from the appropriate standard of care to constitute negligence is through the testimony of expert witnesses. This does not mean, however, that a jury is bound by the opinions of any of the experts who testify. Such opinions may properly be discarded, even when there is no evidence to contradict them; however, a jury cannot discard an opinion as to the standard of care, and adopt a different standard unless there is testimony in favor of the alternate standard of care. In other words, the jury decides to create a standard that has not been

articulated by an expert. In most cases, the standard of care that is applicable is disputed by the parties. When the expert opinions conflict, the jury, as a trier of fact, must determine which is more worthy of credence. In such a case, in determining what is the greater weight of evidence, the jury should not content itself with merely counting the number of witnesses but should consider the relative qualifications and credibility [76–78]. Expert testimony that is purely speculative should be excluded [72].

Before the jury considers how much weight to ascribe to a give opinion proffered by an expert, however, it is the responsibility of the trial court to determine that the expert witness is “. . . qualified as an expert by knowledge, skill, experience, training, or education” before their testimony is presented to the jury [73]. This gate-keeping function requires the trial court to determine, given the proffered expert’s background, whether the scientific, technical, or other specialized knowledge he [or she] offers “. . . will assist the trier better to understand a fact in issue. . . .” [74,75]. To qualify as an expert in a medical malpractice action, a physician is not required to be familiar with all the medical statistics of a particular community. Furthermore, the appropriate standard of care to be used in any given procedure is not necessarily compartmentalized by a physician’s area of professional specialization or certification. Courts consider the focus in any medical malpractice case to be the procedure performed and whether it was executed in conformity with the recognized standard of care, the primary concern being whether the treatment was administered in a reasonable manner.

One court noted that:

**. . . if an expert was required to have similar, if not identical, education, training, and experience the obvious result of such an application, [would be] to reduce the pool of qualified experts to its lowest common denominator. This is a consequence that [the court has] never intended. Therefore, any doctor with knowledge of or familiarity with the procedure, acquired through experience, observation, association, or education, is competent to testify concerning the requisite standard of care and whether the care in any given case deviated from that standard. The resources available to a physician, his or her specific area of practice, or the length of time**

**he or she has been practicing are all issues that should be considered by the trial justice in making his or her decision regarding the qualification of an expert. No one issue, however, should be determinative. Furthermore, except in extreme cases, a witness who has obtained board certification in a particular specialty related to the procedure in question, especially when that board certification reflects a national standard of training and qualification, should be presumptively qualified to render an opinion. [76]**

Thus, the proffered expert physician need not be a specialist in a particular medical discipline to render expert testimony relating to that discipline [77,78]. In fact, it would have been an abuse of discretion for the court to exclude testimony on the sole basis that the medical specialty of the expert was something other than gynecology or obstetrics, for example [79]. The case of *Sheely v. Leslie* illustrates the issue that confronts the court in the exercise of its gate-keeping function [80].

In *Sheely*, the plaintiff delivered a healthy child at a Rhode Island hospital. At the time of the birth, the plaintiff was under the care of a second-year family practice resident. The faculty supervisor during the delivery was a family practice specialist. During the delivery, an episiotomy was performed that was subsequently repaired. After her discharge from the hospital, the plaintiff developed a rectovaginal fistula that required corrective surgery. Notwithstanding an apparently successful repair after her surgery, the plaintiff continued to experience pain and discomfort at the episiotomy site. The patient and her husband then brought suit, alleging negligent performance and repair of the episiotomy incision.

At the trial on the malpractice action, the plaintiff sought to introduce the expert medical testimony of a board-certified obstetrician/gynecologist. The obstetrician was expected to testify about the faculty supervisor’s alleged malpractice and the standard of care related to the performance of an episiotomy. The plaintiff’s obstetrician/gynecologist expert testified that his board certification represents a level of achievement of skill and knowledge as established by a national standard, in which the standard of care is uniform throughout the medical specialty. This expert was eminently qualified and had delivered approximately 4,000 babies

during his career. Although at the time of the trial this expert witness no longer practiced obstetrics, he testified that he had maintained his familiarity with the standards and practices in the field of obstetrics through weekly conferences, active obstetric work, professorial responsibilities, and continuing education. The defendants objected and filed a pretrial motion to exclude this testimony, arguing that a trained obstetrician/gynecologist was not qualified to testify against a family practice resident who was providing obstetric and gynecologic care.

At trial, the judge excluded the plaintiff expert's testimony, stating: "I fail to see where this case is distinguishable from [prior case law]. I don't quarrel with the doctor's background and qualifications. I think he's the inappropriate expert to testify in this case." The plaintiff did not have any other experts prepared to testify, nor was she able to procure one within the 2-day period allowed by the trial justice. Consequently, the plaintiff's case was dismissed.

On appeal, the plaintiff argued that the trial justice's ruling constituted an abuse of discretion in exercising the gate-keeping function, since the recruited expert was amply qualified to testify concerning the alleged malpractice. The defendant maintained that the plaintiff's expert was not competent to offer expert testimony on the appropriate standard of care, despite the fact that he had the prerequisite knowledge, skill, experience, training, and education in the field of the alleged malpractice, because he had more specialized training than a family practitioner. Furthermore, testimony concerning the standard of care required of a family practitioner practicing obstetrics had to be introduced by an expert in family medicine.

The appellate court refused to reverse the lower court's decision. The court argued that, "The determination of the competency of an expert witness to testify is within the discretion of the trial justice. . . . This court will not disturb that decision in the absence of clear error or abuse. . . ." In similar circumstances, most trial courts would permit the obstetrician/gynecologist to testify against a family practice physician given that the issues involved obstetrics. There were some extenuating circumstances in the *Sheely* case that led to the judge's ruling. It is an example, however, of the role that the trial court is supposed to play in determining the relevance of evidence that the jury should receive. Similarly, even if permitted to testify, the range of

subjects about which an expert is qualified to opine in any particular trial is also a matter within the trial court's discretion.

As the *Sheely* court notes, the gate-keeping function of the trial court requires use of judicial discretion, as such appellate courts will not supplant in judgment an overturn a ruling unless there has been a clear abuse of discretion. Although a trial court's decision on the qualifications of an expert ordinarily is conclusive, an appellate court can come to the opposite conclusion if it determines the trial court reached its decision by applying erroneous legal principles. In *Lake v. Clark*, a lawsuit was brought against a general surgeon for alleged negligence in performing an abdominal hysterectomy [81,82]. In this instance, the appellate court decided that the trial judge erred in refusing to permit a gynecologist to testify about the standard of care.

#### AUTHORITATIVE SOURCES AND LEARNED TREATISES

The rule against hearsay precludes many out-of-court statements, except for statements made by a party to the case or prior inconsistent statements of a witness given under oath. Consequently, if this rule were strictly followed in a medical malpractice case, scientific literature would be precluded. Given the considerable resources available to physicians and the reliability of much of the scientific literature, however, procedural rules provide an exception to the hearsay rule for what are termed *learned treatises*. The Federal Rule states

**To the extent they are called to the attention of an expert witness upon cross-examination or relied upon by the expert witness in direct examination, statements contained in published treatises, periodicals, or pamphlets on a subject of history, medicine, or other science or art, established as a reliable authority by the testimony or admission of the witness or by other expert testimony or by judicial notice. If admitted, the statements may be read into evidence but may not be received as exhibits.**  
[83]

A majority of states have adopted the Federal Learned-Treatise Rule or follow an analogous rule [84–108].

The Federal Rule provides for specific publications to be established as reliable authorities by experts other than an expert under cross-examination, as well as by judicial notice in which a judge accepts the reliability of a particular writing. Expert witnesses may also refer to statements from learned treatises on direct examination, to the extent that they relied on those statements in forming their own opinion.

The use of learned treatises during trial can be a quagmire for trial attorneys. Given the plethora of publications, and the role that controversial research or approaches play in provoking discourse, published statements that are not generally accepted can nonetheless give the appearance of being authoritative and the trial becomes one of journal articles and textbooks. Given that even legitimate authoritative sources are rarely definitive and the application to a particular clinical situation requires interpretation, it is the expert witnesses that are relied upon to provide the medical standards. Literature, when it is used, is most often used to cross-examine an adverse witness, as opposed to bolstering one side or the other with “evidence-based” writings. Interestingly, the Federal Rules of Evidence do not require the cross-examined witness to recognize any treatise as authoritative [109]. This “. . . avoid[s] the possibility that the expert may at the outset block cross-examination by refusing to concede reliance or authoritativeness . . .” [110]. Prior to allowing other means for introduction, if an expert during cross-examination did not legitimize the publication as an authority, the document could not be used. The black-and-white nature of the standard is illustrated in the case of *Swank v. Halivopoulos* [111]. In *Swank*, the plaintiff’s counsel sought to cross-examine the defendant’s medical expert about the standard of care for administration of oxygen to premature infants by reference to the *Standards and Recommendations of the American Academy of Pediatrics*. The defense expert stated that he was unfamiliar with the book, however, and that he did not recognize it as authoritative. The appellate court held that the text had been properly excluded because the defense expert had never acknowledged its authoritativeness.

The promulgation of the Federal Rule and similar state rules of evidence was in response to the perceived danger that an expert can tactically avoid cross-examination with the use of the claim of

authoritative language. As the court in *Gridley v. Johnson* [112] stated:

**An expert need say only that he or she is not acquainted with the book or its author to prevent its use in testing his or her qualifications, no matter how eminent or accepted the author might be. The fewer books and authorities that the witness acknowledges, and the less knowledge he or she has of what has been written in the field, the more difficult it can be to cross-examine this witness along this line. This not acknowledging the source material(s) gives the witness full veto power over the cross-examiner’s efforts.**

In a similar vein, the Illinois Supreme Court observed that “. . . [t]o prevent cross-examination upon the relevant body of knowledge serves only to protect the ignorant or unscrupulous expert witness . . .” [113].

Because of the black-and-white standard, trial cross-examination of expert witnesses often became a dance in semantics, as seen in the case of *Jacober vs. St. Peter’s Medical Center* [114]. In *Jacober*, a trial court prevented the plaintiff’s counsel from cross-examining defense experts about the medical literature. One of the experts testified that a commonly used pediatric textbook (Klaus and Fanaroff: *Care of the High-Risk Neonate*) was “a standard text.” Another expert acknowledged Stanley James as an “eminent neonatologist” who authored a “standard textbook.” Each of the defense experts, however, refused to declare the proffered texts as “authoritative.” The issue on appeal was whether, although not willing to utter the “magic words,” did the testimony of the expert witnesses constitute sufficient acknowledgment that the texts referred to were “recognized and standard authorit[ies] on the subject.” The appellate court overturned the preclusion, arguing that procedural law requires an expert only to recognize a text as a standard authority, not expressly to declare the text “authoritative.” In the eyes of the appellate court, the medical literature that plaintiff’s counsel sought to use on cross-examination strongly supported the plaintiff’s theory and challenged the defense experts’ testimony. The appellate court noted that the trial court’s exclusions precluded the jury from adequately assessing the defense experts’ credibility. In

part, the appellate court was concerned that the trial court's ruling prevented the plaintiff's counsel from referring to any medical articles to rebut the defendant's expert factual assertions despite statements in the literature that might be considered contradictory.

Consequently, the Federal Rule and now several state courts permit cross-examiners to establish the authority of learned treatises through judicial notice or through the testimony of other expert witnesses [115–119]. In addition, the Federal Rule allows experts to refer on direct examination to statements from learned treatises if they relied on those treatises in forming their opinions [120].

The federal courts have explicated how learned treatise statements may be introduced into evidence [121]. An expert witness must lay a proper foundation for the reliability and authoritativeness of a text before its contents can be admitted as a learned treatise [122–123]. Merely because a paper or book is published does not establish the document as an authority, and it is recognized that publication does not automatically render a text a reliable authority [124]. The text's reliability is potentially established by an expert witness acknowledging under testimony that professional people within the field regard the publication as trustworthy.

Duly admitted learned treatises do not independently establish the standard of care in a medical malpractice action, however. They are merely evidence of the standard of care to the extent relied on by the expert witness in direct examination, or called to the attention of the expert witness on cross-examination.

In *Kilpatrick v. Wolfond* [125], the issue of an appeal was the trial court's allowance of the defendant physician's introduction of learned treatises into evidence as proof of the standard of care, rather than merely allowing the jury to derive the reliability of the expert witness's testimony when confronted with literature that appeared to contradict him. Prior to trial, the defendant filed a motion for determination of authoritativeness of certain technical bulletins and committee opinions of ACOG. The motion noted that two of the defendants' experts were present or former members of ACOG and were intricately involved with the organization, including participation on certain committees. An affidavit from one of the experts described ACOG's membership and its purpose and role "to provide

education and to serve as guidance for doctors as to the current state of medicine." The other expert stated by affidavit that he considered the technical bulletins and committee opinions authoritative and that prior to publication the materials are extensively reviewed by committees of doctors chosen for their expertise. Furthermore, affidavit testimony was presented that ACOG materials are authoritative and that "[t]he ACOG publications that are currently in effect are the best available knowledge regarding the issues which they cover and are scientifically agreed upon nationwide." In opposition, the plaintiffs submitted affidavits of experts who stated that the ACOG materials are not "in and of themselves, authoritative" and that "the general obstetric community does not recognize [the materials] per se, as authoritative."

The trial judge framed the legal requirement for introduction as follows: "... the real question is whether they are generally acknowledged, accepted, and utilized in the medical community. . . . [I]f so, then I think that makes them authoritative." The judge went on to state that "I don't think it's supposed to be one hundred percent accurate or per se authoritative. I think it's supposed to be generally acknowledged, accepted and used." In fact, as the judge pointed out, the defendant's attorney acknowledged, "[t]here is not anything in medicine that everybody agrees is absolutely authoritative." The original court accepted the texts in question as authoritative. An appeal was filed and it was argued that the trial court erred in determining that certain medical texts were authoritative for use in cross-examination of their experts. The plaintiff claimed that the preponderance of the evidence did not support a finding of authoritativeness and also asserted that the standard for determining authoritativeness of medical texts should be at least "clear and convincing" evidence.

In this instance, the appellate court determined that the ACOG technical bulletins and committee opinions were authoritative and upheld the introduction of the documents agreeing that the sworn statements from the defendant's own experts met the burden of establishing authoritativeness. The court concluded that even in the absence of acknowledgment of authoritativeness by the cross-examined experts themselves, authoritativeness was properly established by the acknowledgment of other experts. The court noted that one of the

defendant's experts had stated in his affidavit that he considered the ACOG bulletins and committee opinions authoritative, and he also described the extensive procedure by which these bulletins and opinions are formulated, noting that he did not know of other publications that went through such thorough expert review prior to publication. Additionally, other affidavits stated that these ACOG materials were designed to educate and provide guidance for gynecologists and obstetricians in practice.

Ultimately, the ability to rely on medical literature and learned treatises at trial depends on the rules of evidence for the particular jurisdiction. Typically, it does not boil down to a battle of experts, with each party trying to bring in one more expert than the other so that "more weight" favors authoritativeness or nonauthoritativeness. As long as there is some credible evidence to support the conclusion that a text is authoritative, a trial judge is free to act within his or her discretion in deeming a medical text authoritative for purposes of cross-examination. In most instances, literature is not admitted as substantive evidence, but only for purposes of cross-examination. The expert testifying in reference to these data is free to explain his or her reason for disagreement with the text, such as a flawed methodology.

## SPECIAL ISSUES

### **Emergency Medical Treatment and Labor Act Rules**

Amid an increasing number of reports that hospital emergency departments were refusing to accept or treat patients with emergency conditions if the patient did not have medical insurance, Congress enacted the Emergency Medical Treatment and Labor Act (EMTALA) [126]. EMTALA was enacted to address a growing concern that hospitals were "dumping" patients who could not pay, either by discharging them before their emergency condition was stabilized or by transferring such patients to another hospital. It should be recognized that EMTALA is not a malpractice statute; it is not intended to guarantee proper diagnosis or to provide a separate means for bringing a claim based on misdiagnosis or medical negligence. Instead, Congress's manifest intent was that all patients be treated fairly

when they arrive at a hospital's emergency department, and that those patients who are in need of emergent care receive some minimal level. As one court stated, "The avowed purpose of EMTALA was not to guarantee that all patients are properly diagnosed, or even to ensure that they receive adequate care, but instead to provide an 'adequate first response to a medical crisis' for all patients and send a clear signal to the hospital community . . . that all Americans, regardless of wealth or status, should know that a hospital will provide what services it can when they are truly in physical distress" [127].

There are two requirements placed on hospitals by the EMTALA. First, if a patient arrives at the hospital requesting treatment, the hospital must provide an appropriate medical screening examination to determine whether an emergency medical condition exists. Second, if the hospital determines that an emergency medical condition exists, the hospital may not transfer the patient until the medical condition is stabilized, unless certain circumstances exist [128–134].

The determination of whether a given hospital has performed an "appropriate medical screening examination," as defined by EMTALA, varies with the unique capabilities of each specific hospital. The courts have given considerable deference to the screening procedures used by the hospital. Essentially, the court's primary issue in determining whether appropriate screening has been undertaken is to determine whether the hospital adhered to its own procedures, not whether the procedures were adequate if followed. This is of fundamental importance, because a basis for establishing liability for failing to properly screen a patient who presents requesting emergent care can be when the hospital does not follow its own standard procedures [131].

In *Marshall v. East Carroll Parish Hospital*, the U.S. Court of Appeals for the Fifth Circuit held that an appropriate medical screening ". . . is not judged by its proficiency in accurately diagnosing the patient's illness, but rather by whether it was performed equitably in comparison to patients with similar symptoms" [133]; however, a violation of policy or protocol is not a per se violation of EMTALA. The case of *United States v. Rush Foundation Hospital* [134] is directly on point because it considered the issue of appropriate screening examination in the obstetrics setting. During the course of a woman's

prenatal care, the nurse-midwives at her clinic became aware that she had a history of deep-vein thrombosis (DVT) during a previous pregnancy. Because of this risk, in consultation with one of the supervising physicians, the patient was to receive care at a university medical center located in a different city from where she lived. The patient agreed with this plan.

On the day in question, a member of the patient's family called an ambulance service because the patient thought that she was in labor. The paramedic staffing the ambulance, on learning of the patient's medical history, became concerned about transporting her out of the ambulance district without being evaluated locally. Thus, the patient was brought to the hospital at which her prenatal clinic was affiliated.

A registered nurse employed in the labor and delivery department came to the emergency department to examine the patient. The nurse took vital signs, performed a sterile vaginal examination, manually palpated for contractions, and documented the fetal heart tones using a Doppler device. After completing her assessment, the nurse reported her findings to a certified nurse midwife employed at the hospital. The plan was to proceed with transport to the medical center, and therefore the patient was placed back in the ambulance. During transport, however, the patient's membranes ruptured. As time passed, she began to have the urge to push. Based on his experience and training, the paramedic thought that the delivery was progressing, and as a result, the patient was taken to the closest available hospital. A nurse and an emergency physician delivered the baby. There were no complications, and the mother and her baby were ultimately transported to the medical center.

This issue that was presented was whether the nurse's evaluation of this patient represented an appropriate medical screening examination, satisfying the requirements of section 1867 of the EMTALA law. The Inspector General argued that because the patient was not taken to the labor and delivery department and did not receive electronic fetal monitoring, her evaluation was inappropriate. Relying on the hospital's own policy regarding obstetric patients who present to the delivery suite, an attempt was made to establish disparity between the policy and the patient's screening examination. The hospital's policy stated that

**any patient presenting to the Emergency Room for questionable labor from 20 weeks gestation to term will be evaluated by 3 West Nursing staff. After completing identifying data and notifying the floor, the patient will be taken to 3 West observation room to be attached to Electronic Fetal Monitor.**

As the court noted, however, the difficulty with the argument is that the Inspector General presented no evidence from any staff member or concerning any other hospital patient that would tend to prove that other pregnant patients would have received an examination different from the one provided this particular patient. In fact, there was evidence to the contrary. The nurse who performed the evaluation testified that the examination she conducted was the same examination she would have performed on any other pregnant patient presenting to the hospital with similar symptoms. In addition, expert testimony established that the examination performed was the same examination that they would have performed. Moreover, there was testimony to establish that the nurse's evaluation in the emergency department was the functional equivalent to that described in the policy, the only distinction being the change in the point of service for the evaluation. In concluding that the medical screening examination was the same as that which would have been provided to any other pregnant patient presenting to the hospital, the court concluded that there was no violation of the EMTALA.

Thus, for purposes of the EMTALA, the critical point in question is not whether a hospital arrived at a correct diagnosis or determination of the patient's stability, but whether it provided an appropriate medical screening examination. From a practical perspective, as the adopted policy and procedures of a hospital are relevant to the issue, such policies and procedures should be reviewed to ensure that they are consistent with practice. Although in the *Rush Foundation* case the hospital was not found to be in violation of EMTALA, it was their own policy that was being used in an effort to establish that the patient at issue was treated in a disparate fashion.

The other issue of responsibility that the hospital has is to stabilize the patient prior to transferring or discharging him or her. Pursuant to EMTALA, a patient is "stabilized" if his or her condition will not

materially deteriorate during the short time necessary to transfer the patient to another facility [130]. As discussed previously, however, the EMTALA is not a substitute for medical negligence. The hospital's duty under the statute therefore is to stabilize only those emergency medical conditions that its staff detects and that duty does not arise until the hospital first detects an "emergency medical condition" [126,135–137]. Consider *Urban v. King*, in which a plaintiff presented to the obstetrics department of a medical center for electronic fetal monitoring (EFM) [137]. She was pregnant with twins, in a high-risk pregnancy. Her test was nonreactive; however, the fetal heart tones were in the 150s for each twin. The patient's vital signs were normal. The nurse who conducted the test, after consulting with a doctor but without informing the patient of her test results, instructed the patient to come back to the hospital the next morning for a repeat test.

The patient left the hospital at 20:00 and returned the next day for the repeat test. During the repeat stress test, the morning nurse realized that something was wrong and called in another obstetrician. This second obstetrician ordered a fetal biophysical profile. This study revealed no movements or fetal breathing movements by both fetuses and no fetal heart motion in one. A cesarean was subsequently performed. One baby was delivered stillborn, and the other was born with brain damage. A lawsuit was brought against the medical center for violating the EMTALA by sending this woman home after the first nonreactive stress test. The plaintiffs also initially brought various malpractice claims against the medical center and the center's physicians. The malpractice claims were abandoned by the plaintiffs during the pretrial stage of the case, most likely owing to the inability to prove negligence.

In this case, the plaintiffs did not challenge the hospital's compliance with the first requirement of the Act, that being the duty to perform a screening examination. Instead, they argued that the medical center violated the EMTALA by releasing the plaintiff with an emergency medical condition without first stabilizing that condition. The plaintiffs argued that the statute imposed strict liability such that any failure to stabilize an emergent condition imposes liability. The courts have held, however, that a plain reading of the statute reveals "actual knowledge" of an unstabilized emergency medical condition is a requirement to establish liability. The hospi-

tal therefore cannot be held to stabilize an emergency situation without knowing that an emergency exists.

There are provisions in EMTALA that allow hospitals to transfer patients before stabilizing them. Pursuant to the statute, a hospital may transfer someone who is in active labor or who has an emergency medical condition that has not been stabilized, if either 1) the patient requests a transfer, or 2) a physician determines that based on the information available at the time, the anticipated benefits of transfer outweigh the increased risks inherent during the transfer and so it is in the best interests of the patient. If the transfer occurs because of clinical concern for the patient's condition, before the transfer is accomplished, the physician must certify that, prior to executing the certification, he or she evaluated the patient and weighed the medical risks and the medical benefits of transfer. Similar to the court rulings that a hospital is required to stabilize only the conditions of which it has knowledge, the EMTALA does not require a physician to ascertain correctly all risks and benefits associated with transfer. Thus, a physician's certification ultimately might prove to be incorrect; however, he or she is not responsible if the physicians undertook the appropriate process of actual assessment and weighing of the medical risks and benefits of transfer. The case of *Burditt v. United States* highlights the statute's provisions [138].

In *Burditt*, the patient was in early labor, hypertensive, and thought to be at increased risk of placental abruption and other complications. She presented to the hospital for evaluation, where she remained in the hospital for 2 hours prior to transfer. During this 2-hour period, however, the patient was not reevaluated. The defendant, an obstetrician, made an immediate decision to transfer a pregnant woman without weighing the medical risks and benefits of transfer. Given the patient's high blood pressure and the obstetrician's indifference to the patient's condition during the 2 hours after he conducted his single examination, the court found not that the physician unreasonably weighed the medical risks and benefits of transfer, but simply that he had never made such a judgment. In determining that there was a violation, the court concluded that the facts established that the transfer was so unacceptable that it was likely that the physician had certified that the benefits of transfer outweighed the risks without actually engaging in any meaningful deliberation.



Remarkably, in *Burditt*, the defendant asked the court to interpret the EMTALA statute as requiring an improper, or nonmedical, motive for transfer as an element to be established to prove a violation. To that end, the obstetrician testified that he was completely ignorant of the EMTALA's requirements, and in fact stated that he did not believe that the EMTALA governed his actions. He specifically testified that "I didn't know what I was doing, but I signed her [certification] so I could send her out...because [the nurse] insisted." As the court stated, although it does not assign responsibility for clinical judgment that later turns out to be erroneous, the purpose of the EMTALA is to prevent patient dumping regardless of whether the motive was improper, or for other reasons other than the patient's ability to pay for services.

In addition to certifying the medical need for transferring patients protected by the EMTALA, transfer requires that both qualified personnel and appropriate equipment are used. The statutory definition of *appropriate transfer* requires that "the transfer [be] effected through qualified personnel and transportation equipment, as required, including the use of necessary and medically appropriate life-support measures during the transfer." Courts have interpreted "qualified personnel" to depend on the clinical circumstances. There must be people capable of addressing not just the medical condition, but potential complications that might arise during transport. Similarly, "transportation equipment" has been interpreted to include all physical objects reasonably medically necessary for safe patient transfer. These provisions were also an issue in *Burditt*. The evidence given during trial was that an obstetric nurse and two emergency medical technicians accompanied the patient during transport. Although they were qualified to deliver the patient's baby in the absence of complications, it was undisputed that they were unqualified to perform or treat the other complications from her hypertension. Consequently, the court held that, under the circumstances of this case, only a physician could have fulfilled the "qualified personnel" requirement. Furthermore, despite the increased risk that a hypertensive woman faces for complications, the patient was transferred without a fetal heart monitor in the ambulance, making it difficult if not impossible to diagnose this condition during transport. Given that there was evidence to conclude that a reasonable

physician would have included a fetal heart monitor as equipment to ensure the patient's safe transfer, the failure to do so was also deemed a violation. The fact that the patient was transferred in an ambulance that met state licensing requirements was not sufficient, given the clinical presentation of the patient.

In essence, if the patient has been evaluated in compliance with the hospital's screening procedure and a "good faith" effort has been undertaken to evaluate a patient's condition, it is not a violation of the EMTALA if the hospital fails to diagnose the emergency condition, although there might be grounds for a separate action for medical negligence. Strict liability attaches for any violation of the EMTALA, and there need not be any harm caused to either the mother or the baby. In most instances, when transferring a patient, any emergency medical condition must be stabilized. For a patient in labor, this typically requires admission and delivery. When transferring a patient whose condition has not been stabilized, necessary equipment and qualified personnel must be present to attend not only to the condition but also to potential complications.

### **New Technology and the Standard of Care**

Both medicine and the law have difficulty in establishing what the accepted care standards are. When does emerging technology or progress of knowledge through clinical research become the standard of care? Typically, there is no delineating point in time when a particular new type of evaluation or management becomes "standard." Given this, it must be understood that a physician is not held to a standard of absolute precision, nor is he or she expected to have knowledge of all developing scientific advancements. Rather, his or her conduct and judgment are evaluated in terms of reasonableness under then-existing circumstances, not on the basis of hindsight or in light of subsequent events such as the recognition of diagnostic or testing procedures that did not exist or were in trial at the time that the care was provided. In this context, the legal question remains the same, what should the reasonable practicing obstetrician have known about the existence of the particular procedure, test, or management protocol in question, and the possible indications for such a new approach at that time care was being provided?

As one court put it [139]

**Just when a scientific principle or discovery crosses the line between the experimental and demonstrable stages is difficult to define. Somewhere in this twilight zone, the evidential force of the principle must be recognized, and while courts will go a long way in admitting expert testimony deduced from a well-recognized scientific principle or discovery, the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs.**

In determining whether a novel scientific procedure is generally accepted in the scientific community, the dispositive issue is consensus versus controversy over a particular technique. The standard of care requires that the procedure on which the claim of negligence is premised must be "generally accepted."

Legally, it is for the jury to determine the requisite standard of care, based on the evidence provided during trial. The trial judge, as gate-keeper, must first determine if the evidence surrounding the science of medicine is reliable and therefore adequate for the jury's consideration. A proponent of scientific evidence must prove its reliability and its general acceptance in the community. In attempting to establish its reliability, the proponent primarily relies on the testimony of expert witnesses; however, expert testimony alone might be insufficient. Thus, the party might be required to establish general acceptance of a standard through the use of peer-reviewed publications, clinical trials, or other sources, to demonstrate the attitude of physicians practicing in field or specialty. There is ample room for controversy. Although a particular principle might be established as an "accepted" standard of care, in medicine there is often more than one way to "skin the cat," and it is uncommon for one management scheme to be so overwhelmingly proper that it entirely precludes another approach. Thus, even if a plaintiff can establish that a proposition or scientific advancement had gained general acceptance within the medical community, an obstetrician is not considered negligent if he or she chose a different course, as long as that course is also generally accepted as within the standard of care at the time treatment was provided.

Put another way, if an obstetrician in the exercise of medical judgment undertakes a course of action, he or she is not negligent if the course chosen was recognized as acceptable under the specific clinical circumstances and at the time of treatment. Even if better technology or a better course existed, the physician is nonetheless within the standard of care even if the path chosen, or the failure to employ a particular technology, turns out to be wrong.

In labor and delivery, the standard of care is usually considered to be a national standard, and reference to the particular locality where the physician practices is not relevant. An exception can exist when the issue is one of emerging technology, however. In such cases, it must also be established that the obstetrician knew that the technology was available within his or her community. Should the technology not yet be available to a physician given the particular locality and its surrounding area, then standard of care might be different for that obstetrician versus one who practices in a community where the emerging technology has become available.

### **Abortion and the Law**

There is no topic in obstetrics and gynecology that has created as much public controversy as the issue of abortion. In fact, a search of the internet for the term "abortion," identifies more than 62.6 million "hits" whereas searching the phrase "cesarean section" identifies only 2.2 million "hits." The centerpiece of the tumultuous debate is the landmark case of *Roe v. Wade* [140]. This case is widely misunderstood and, furthermore, the principles set forth in the decision have been affected by subsequent Supreme Court rulings.

Although the Supreme Court's decision in *Roe* provided the basis for a woman's right to choose to have an abortion, the Court did determine that maternal health and potential life are legitimate state interests, and thus, states may regulate abortion practice. Consequently, not every limitation placed on a woman's right to choose abortion can be found to unduly burden that choice. In *Roe*, the Supreme Court observed that the legality of abortion, from the times of ancient Greece to the present day, had its premise in the issue of *viability*. Thus, prior to "quickening," the fetus was considered part of the mother, and destroying a fetus did not fit within the legal definition of homicide. After the American

Civil War, state legislatures began to replace common law concerning abortion, with statutory laws that specifically addressed the issue. Most of these initial statutes dealt severely with abortion after quickening but were lenient on the matter of abortion prior to viability. In *Roe*, the Supreme Court observed that "... throughout the major portion of the nineteenth century, abortion was viewed with less disfavor than under most American statutes currently in effect." In fact, by the end of the 1950s, most jurisdictions had banned abortion entirely unless it was performed to preserve the mother's life.

Ultimately, the historical review provided the underpinnings of Supreme Court's rationale, which recognized that the state's interest in protecting the fetus becomes compelling only after viability; before that time any interest in the unborn fetus must be balanced against concerns for the mother's health, and the state may not protect the fetus at the expense of the mother's health. Thus, in their 1973 decision, the Supreme Court employed the trimester standard for determining the significance of the state's interest in and ability to regulate abortion. In *Planned Parenthood v. Casey* [141], however, the Supreme Court abandoned the trimester framework of *Roe v. Wade*, stating that a state has a profound interest in potential life throughout pregnancy. In this case, the Supreme Court turned the spotlight on the more obscure language in *Roe v. Wade* – that throughout pregnancy, the state retains an "... important and legitimate interest in potential life."

In *Casey*, the challenge was to Pennsylvania's 1989 Abortion Control Act. The 1989 statute required that, except in medical emergencies: 1) a woman wait 24 hours between consenting to and receiving an abortion; 2) the woman be given state-mandated information about abortion and offered state-authored materials on fetal development; 3) a married woman inform her husband of her intent to have an abortion; and 4) minors' abortions be conditioned upon the consent, provided in person at the clinic, of one parent or guardian, or upon a judicial waiver. In addition, physicians and clinics that perform abortions were required to provide to the state annual statistical reports on abortions performed during the year, including the names of referring physicians. The court reaffirmed the validity of a woman's right to choose abortion under *Roe*

*v. Wade* but announced a new standard of review that allows restrictions on abortion prior to fetal viability so long as the restrictions do not constitute an "undue burden" to the woman. A restriction is an *undue burden* when it has the purpose or effect of placing a substantial obstacle in the path of a woman seeking an abortion. Under this standard, only the husband notification provision was considered an undue burden and therefore unconstitutional. All the other provisions were upheld as not unduly burdensome. The *Casey* decision set forth several important points for courts to consider in assessing the constitutionality of laws regulating abortion. Among those points are the following:

- A state may not prohibit any woman from making the ultimate decision to terminate her pregnancy before viability.
- A state may not interfere with a woman's choice to undergo an abortion procedure if continuing her pregnancy would constitute a threat to her health.
- After viability, a state "... may, if it chooses, regulate, and even proscribe, abortion except where it is necessary, in appropriate medical judgment, for the preservation of the life or health of the mother.

Thus, the *Casey* Court allowed the state the right to legislate a requirement that a woman considering an abortion be fully informed of the "... consequences to the fetus, even when those consequences have no direct relation to her health..."

In *Maher v. Roe*, the court let stand a Connecticut statute that limited state Medicaid funding to medically necessary abortions, refusing to fund "elective" abortions [142]. In doing so, it upheld the Hyde Amendment's ban on the use of Federal Medicaid funds for abortion, except those necessary to save the woman's life, determining that the government has no obligation to provide funds for other than medically necessary abortions. Meanwhile, in *Harris v. McRae* [143], the court permitted a Utah law to stand that required the physician to notify a parent of an unemancipated minor before an abortion, except in cases involving mature minors and those whose best interests mandated that the parents not be involved.

Applying the *Casey* standard, however, the Court has deemed a ban on partial-birth abortions as unconstitutional because it lacks an exception for

situations when the procedure is necessary to protect the woman's health. Furthermore, the Court has held that statutes proscribing this type of procedure create an undue burden on a woman's right to abortion because it has the effect of outlawing the dilation and evacuation (D&E) procedure, the safest and most commonly used method for performing second-trimester abortions. In the case of *Stenberg v. Carhart* [144], the Court was concerned with what it perceived to be arbitrary and irrational legislative effort to single out the D&E procedure. The Court stated that despite its profound interest, a state cannot place an "undue burden" on the right of a pregnant woman to seek an abortion. Thus, a state cannot put in effect any statute that has the purpose or effect of placing a substantial obstacle in the path of a woman seeking an abortion of a nonviable fetus. The Court clearly acknowledged that the undue burden standard applies only to previability abortions and that the state's interest in the life of the fetus allows it to regulate or proscribe abortion after viability, except "... where it is necessary, in appropriate medical judgment, for the preservation of the life or health of the mother." Thus, partial-birth abortion laws are unconstitutional, even though those laws were viewed as banning only selected previability abortion methods.

### Wrongful Life and Wrongful Birth

The U.S. Supreme Court decisions in *Griswold v. Connecticut* [145] and *Roe v. Wade* [140] has led the way in creating the legal underpinnings for *wrongful life* and *wrongful birth* cases. In *Griswold*, the Court determined that the decision of a man and woman to procreate or use some form of birth control fell within their constitutional right of privacy. Subsequently, in *Roe*, the Court extended protection for issues surrounding procreation by providing for the right to an abortion in the first trimester. These two cases in particular established that decisions concerning conception, including the right to terminate a pregnancy, are the private and exclusive rights of the parents.

Before the *Roe* decision, an obstetrician practicing in a state where legislation had banned abortion could not be held liable for failing to diagnose any postconception anomaly or condition in which the outcome could not have been altered with treat-

ment. In these jurisdictions, in the absence of the ability to treat the anomaly, an obstetrician's negligence for failure to diagnose the fetus's condition in utero could not be causally connected to harm. The only therapy to avoid the harm, therapeutic abortion, was at that time illegal. This would have been true even if obstetricians then had had access to the today's advanced technology.

Wrongful life and wrongful birth cases do not stem from the parents claiming that their child's defects were caused by her physicians' negligence; rather, they allege that it was the physician's negligence that kept the mother ignorant of the underlying affliction or medical condition and thus precluded her legal right to choose whether to carry the child to term. These legal remedies are not intended to disparage the value of human life. Instead, the remedies are intended to enforce the right of the parents of a genetically unhealthy fetus to have the option of pregnancy termination when preconception or prenatal previability testing could have identified a serious abnormality. Because of these past Supreme Court decisions, most states now recognize in some form a cause of action for wrongful life or wrongful birth.

The evolution of the legal theories behind these types of cases is interesting. Historically, the courts initially refused to recognize wrongful life or birth as a cause for action [146]. Under traditional tort theory, an injured plaintiff is entitled to recover damages only for the harm caused by a negligent party. Claims for wrongful life and birth were considered legally incognizable because it was philosophically impossible to measure damages to a child injured by a hereditary condition as opposed to not being born at all. In declining to recognize a child's claim for wrongful birth, the New York Court of Appeals stated in *Becker v. Schwartz* [147]:

**Whether it is better to have been born at all than to have been born with even gross deficiencies is a mystery more properly to be left to the philosophers and theologians. Surely, the law can assert no competence to resolve the issue, particularly in view of the very nearly uniform high value which the law and mankind has place on human life, rather than its absence.**

The court further concluded

**Simply put, a cause of action brought on behalf of an infant seeking recovery for wrongful life demands a calculation of damages dependent upon a comparison between the Hobson's choice of life in an impaired state and nonexistence. This comparison the law is not equipped to make.**

Another reason that courts were reluctant to recognize the wrongful birth cause of action was that in the past the postconception remedy available – abortion – was illegal. This reasoning is no longer valid after *Roe v. Wade*, which upheld a woman's constitutional right to undergo an abortion during the first two trimesters of pregnancy. As one court noted, "[t]he value of genetic testing programs . . . is based on the opportunity of parents to abort afflicted fetuses, within appropriate time limitations."

In wrongful birth and wrongful life cases, the plaintiff claims to have suffered injury or damage as a result of a lack of information about potential genetic problems of the fetus. The courts have customarily applied principles of tort theory in determining whether a legally valid cause of action can be alleged in such cases; these same courts usually reject such claims when they are based on a theory of informed consent. In obtaining a patient's consent to treatment, the doctor has a duty to disclose all significant medical information that he or she in fact possesses or should possess that is material to a patient's decisions to undergo a proposed therapeutic procedure. The underlying rationale is that before patients can participate in the decision-making process, they must be educated about any risks of such treatment not considered remote. In cases that arise out of wrongful birth or wrongful life, however, the injury does not stem from the nondisclosure of the risks of a proposed course of treatment, but rather from the condition of pregnancy itself.

Consider *Reed v. Campagnolo* [148]. In this case, the plaintiffs' daughter was born with several genetic abnormalities. These included spina bifida, imperforate anus, and ambiguous genitalia. She also had only one kidney, a vesicoenteric fistula, and hydrocephalus. An increasing head circumference ultimately required the placement of a cerebroabdom-

inal drainage shunt. It was the plaintiffs' claim that the treating physicians failed to recommend and perform a blood test for  $\alpha$ -fetoprotein (AFP) on the mother during the pregnancy. Evidence was presented to suggest that had such testing been performed, it would have revealed an elevated level of AFP, an abnormality that would have led to ultrasound scanning and amniocentesis. It was argued that ultimately, through amniotic fluid aspiration and ultrasound scanning, the extent of the fetal defects would have been diagnosed. The family stated that they would have chosen to terminate the pregnancy had they known of the fetal anomalies. The Maryland court rejected the suggestion that the applicable rule regarding the appropriateness of the genetic counseling and testing should be determined by what the reasonable person, similarly situated as the plaintiff, would want to know. Instead, the court stated that the obligation to recommend tests is based on the standard of care, with consideration given to the reasons for or against recommending one or more of the testing options. The case was then remanded for trial to determine whether there had been a breach of the standard of care.

In consideration of this opinion, the physician's liability is therefore not predicated on the patient's subjective state of mind. Instead, liability is determined by the objective standards of the medical profession. This is an important point. To be held liable, a physician must deviate from accepted medical practice. This deviation usually occurs either by the failure to recognize the significance of the genetic history and therefore to not make testing available, or by the physician's failure to appreciate the findings that are revealed by the tests. The following cases exemplify the types of claims possible under these arguments. In *Siemieniec v. Lutheran General Hospital*, a physician failed to follow up with certain aspects of genetic counseling [149]. The plaintiff had sought genetic counseling because she was concerned about an apparently inherited coagulation disorder in her first child. This child was afflicted with what was described as hemophilia. The patient told her treating physician of her desire to terminate the pregnancy if there was a substantial likelihood that a second child would be similarly afflicted. An important fact is that two of the mother's deceased cousins had bleeding disorders. Despite the history, the plaintiff was advised that her risk of being a

carrier of classic hemophilia was very low. She subsequently gave birth to a son. After a bleeding episode, he was subsequently diagnosed with Christmas disease, a coagulation defect involving a deficiency in Factor IX that is transmitted as an X-linked trait. On the basis of these facts, the court upheld her potential claim for recovery if the allegations could be substantiated.

A 1988 case, *Gallagher v. Duke University Hospital*, highlights the difficulties that potentially can result if the treating physicians do not comprehend the significance of the information obtained through genetic testing [150]. The plaintiff gave birth to a daughter, who suffered severe multiple birth defects. Life-sustaining treatment was unsuccessful, and the infant died less than 3 weeks later. A chromosomal analysis was performed on the baby's blood. The cytogeneticist reviewing the karyotype of the affected infant was of the opinion that there were no chromosomal abnormalities. Before conceiving a second child, the plaintiffs were advised by a physician that their chances of having a normal child were the same as any other couple in the general population. They were also advised that neither individual testing nor amniocentesis were indicated because of the reportedly normal test results from the first pregnancy. The couple conceived again, and their second daughter was afflicted with the same severe multiple defects as had occurred in the first pregnancy. A new chromosomal analysis was performed, and on this study, a genetic abnormality was identified. After reexamination of the data from the first child, it was subsequently discovered that the child had also possessed the same genetic defect but that it had not been diagnosed. Further testing revealed a high probability that the husband was the carrier of this genetic abnormality and was likely to transfer it to his issue. The court concluded that the plaintiff had a right to pursue a claim against the treating physician because it had been proved that both children suffered from the same genetic disorder. Furthermore, it was determined that the plaintiff would be entitled to recovery if it could be established that the physician deviated from the accepted standard of care by not properly diagnosing the second child's disorder prior to birth.

It was only until recently that the role genetics played in medical malpractice cases was forensic, specifically as a shield to claims involving perinatal injury. Within the perinatal injury case, a com-

mon plaintiff's claim is that a developmentally disabled child has been damaged by a potentially avoidable birth injury. For example, a litigant might claim that intrapartum asphyxia occurred and the physician failed to properly manage the labor or, more specifically, failed to recognize various clinical signs of fetal stress/distress and intervene appropriately. The advances in genetic science and the recognition of increasing numbers of birth defects have allowed the successful defense of many of these cases. Working with genetic experts established that the child's handicapped condition did not result from an alleged hypoxic perinatal event that was negligently caused or misdiagnosed, but instead was due to a hereditary condition or defect. This type of evidence has been a death knell to many so-called brain-damaged baby cases, even when the physician's conduct may have deviated from the standard of care.

Soon after the introduction of this new genetic technology, the novel theories of physician liability for wrongful birth and wrongful life emerged in medical malpractice litigation. Unlike with the birth of a normal child, courts began to recognize that the birth of a severely deformed baby is necessarily an unpleasant and aversive event and the cause of inordinate financial burden that would not attend the birth of a normal child. An afflicted child requires the expenditure of extraordinary medical, therapeutic, and custodial care expenses by the family, not to mention the additional reserves of physical, mental, and emotional strength that will be required of all concerned. If the diagnosis of abnormality is made sufficiently early in pregnancy, those who do not wish to undertake the many burdens associated with the birth and continued care of such a child have the legal right, under *Roe v. Wade* and subsequent decisions, to terminate their pregnancies.

## REFERENCES

1. *Miller v. Albany Medical Center Hosp.*, 95 A.D.2d 977 (1983).
2. *Findlay v. Board of Supervisors* 230 P.2d 526 (1951).
3. *Childs v. Weiss* 440 S.W.2d at 106, 107.
4. *Ibid.* at 106-107.
5. *Ibid.* at 106.
6. *Sullenger v. Setco Northwest*, 702 P.2d 1139 (Or.Ct.App.1985).
7. *Oliver v. Brock*, 342 So.2d 1 (Ala.1977).

8. 342 So.2d at 4.
9. *Hill by Burston v. Kokosky*, 463 N.W.2d 265 (1990), *ibid.* at 268.
10. Dunn JD, Practice with co-providers. In *Legal Medicine: Legal Dynamics of Medical Encounters* ed 2. St. Louis, Mosby, 1991; pp 434, 438.
11. *Wheeler v. Yettie Kersting Memorial Hospital*, 866 S.W.2d 32 (Tex. Ct. App. 1993).
12. See *ibid.* at 39–40; *Cintron v. New York Medical College Flower and Fifth Ave. Hosps.*, 193 A.D.2d 551 (1st Dept 1993).
13. *Hill by Burston*, *supra* at 267.
14. *Gilinsky v. Indelicato*, D.C., 894 F.Supp. 86 (1995).
15. *McCullough v. Hutzel Hosp.*, 88 Mich. App. 235 (1979).
16. *Maxwell v. Cole*, 482 N.Y.S.2d 1000 (Sup. Ct. 1984).
17. *Moeller v. Hauser*, 54 N.W.2d 639 (1952).
18. *McCullough v. Hutzel Hosp.*, 276 N.W.2d 569 (1979).
19. *Ibid.* at 571.
20. *Mozingo v. Pitt Cty. Mem. Hosp.*, 415 S.E.2d 341 (1992).
21. *Silva v. Providence Hosp.*, 97 P.2d 798, 804 (Cal. 1939).
22. *Hurley v. Eddingfield*, 59 N.E. 1058, 1058 (Ind. 1901).
23. Prosser WL, *Handbook of the Law of Torts* § 56, ed 4. West Publishing Co., 1971: pp. 341–342.
24. Malloy, *supra*, 232 P.2d at 247; cf. *United States v. DeVane*, 306 F.2d 182, 186 (5th Cir. 1962).
25. Frank B. Mapel, III & Charles J. Weigel, II, Good Samaritan laws – who needs them? The current state of Good Samaritan protection in the United States, 21 S. Tex. L. Rev. 327, 330 (1981).
26. *Colby v. Schwartz*, 78 Cal App.3d 885 (Cal Ct. App. 1978).
27. *Velazquez v. Jiminez* 169 N.J. 604 (2001).
28. Restatement (Second) of Torts, Division Two, Ch. 12, Topic 4, Scope Note, at 65–66 (1965).
29. *Dawson v. Bunker Hill Plaza Assocs.*, 289 N.J. Super. 309, 327 (App. Div. 1996).
30. *O'Neill v. Suburban Terrace Apartments, Inc.*, 110 N.J. Super. 541, 545 (App. Div. 1970).
31. *Shilkret v. Annapolis Emergency Hosp. Ass'n*, 349 A.2d. 245 (Md. 1975).
32. *Lenger v. Physican's Gen. Hosp.*, 455 S.W.2d 703, 707 (Tex. 1970).
33. *Hicks v. United States* 368 F.2d 626 (4th Circ. 1996).
34. *Krishnan v. Ramirez*, 42 S.W.3d 212 (Tex. App. 2001).
35. *Connellan v. Coffey*, 187 A.2d 901 (1936).
36. *Gooding v. University Hospital Building, Inc.*, *supra*.
37. *Cooper v. Sisters of Charity of Cincinnati, Inc.*, *supra*.
38. *Callahan v. Cardinal Glennon Hospital* 863 S.W.852 (1993).
39. Prosser WL, *Handbook of the Law of Torts* § 43, ed 4. West Publishing Co., 1971; p. 291.
40. "Proving Causation in "Loss of a Chance" Cases: A Proportional Approach," 34 Cath. U.L. Rev. 747 (1985).
41. *Ibid.* at 749.
42. *Herkovits v. Group Health Co-Op*, 664 P.2d 474 (1983).
43. Recovery for Loss of Chance in a Wrongful Death Action – *Herkovits v. Group Health*, 99 Wash. 2d 609, 664 P.2d 474 (1983), 59 Wash. L. Rev. 981 (1984).
44. *Delaney v. Cade*, *supra*, 255 Kan. 211–12.
45. *Brown v. Koulizakis*, 331 S.E.2d 440 (1985).
46. *McKellips v. Saint Francis Hospital, Inc.*, 741 P.2d 467 (Okla. 1987).
47. Zilich GJ, Cutting through the confusion of the loss-of-chance doctrine under Ohio law: A new cause of action or a new standard of causation? 50 Clev. St. L. Rev. 673 (2002–2003).
48. J. King, 90 Yale L.J., *supra*, p. 1354.
49. 34 Cath. U.L. Rev., *supra*, pp. 766–67.
50. *Kirby v. Spivey*, 307 S.E.2d 538 (1983)
51. *LaBieniec v. Baker*, 526 A.2d 1341 (1987).
52. *Deutsch v. Shein*, 597 S.W.2d 141 (Ky. 1980; adopting the substantial factor test of Restatement (Second) of Torts § 431).
53. *Pathways, Inc. v. Hammons*, 113 S.W.3d 85 (Ky. 2003).
54. *Bailey v. North American Refractories Co.*, 95 S.W.3d 868 (Ky. App. 2001).
55. Restatement (Second) of Torts § 431.
56. *Walden v. Jones*, 439 S.W.2d 571 (Ky. 1968).
57. 61 Am. Jur. 2d Physicians, Surgeons, and Other Healers § 332 (2002).
58. *Miller v. Paulson*, 646 N.E.2d 521 (1994).
59. *Dalebout v. Union Pac. R.R. Co.*, 980 P.2d 1194 (Utah Ct. App. 1999).
60. *Fid. & Guaranty Ins. Underwriters, Inc. v. Gary Douglas Elec., Inc.*, 357 N.E.2d 388 (1974).
61. Nichols SM, *Jorgenson v. Vener*: The South Dakota Supreme Court declares loss-of-chance doctrine as

- part of our common law in medical malpractice torts, 46 S.D. L. Rev. 618 (2000–2001).
62. *Dingle v. Belin*, 749 A.2d 157 (2000).
  63. *Rizzo v. Schiller*, 445 S.E.2d 153, 155 (1994).
  64. *Moates v. Hyslop*, 480 S.E.2d 109, 111 (1997).
  65. *Rizzo v. Schiller*, 445 S.E.2d 153, 154 (1994).
  66. *Bly v. Rhoads*, 216 Va. 645, 650–51, 222 S.E.2d 783, 787 (1976).
  67. *Canterbury v. Spence*, 464 F.2d 772 (1972).
  68. *Ibid.* at 787.
  69. *Precourt v. Frederick*, 481 N.E.2d 1144, 1149 (Mass. 1985) (quoting *LaCaze v. Collier*, 434 So.2d 1039, 1046 (La. 1983)).
  70. *Ibid.* at 243 (quoting *Wilkinson v. Vesey*, 295 A.2d 676 (R.I. 1972)).
  71. *Harrison v. United States* 284 F.3d 293 (2003).
  72. *Banaghan v. Dewey*, 340 Mass. 73, 79 (1959).
  73. *Dodge v. Sawyer*, 288 Mass. 402 (1934).
  74. *Nass v. Duxbury*, 327 Mass. 396 (1951).
  75. *Phillips v. Deihm*, 541 NW2d 566 (1995).
  76. *United States v. Alzanki*, 54 F.3d 994, 1005 (1st Cir. 1995).
  77. *United States v. Sepulveda*, 15 F.3d 1161, 1183 (1st Cir. 1993).
  78. *Mitchell v. United States*, 141 F.3d 8, 15 (1st Cir. 1998).
  79. *Payton v. Abbott Labs*, 780 F.2d 147, 155 (1st Cir. 1985).
  80. *Sheely v. Memorial Hospital* 710 A.2d 161 (1998)
  81. *Lake v. Clark*, 450 So.2d 488 (Fla. 1983).
  82. *Lake v. Clark*, 533 So.2d 797 (Fla. 5th DCA), review.
  83. Fed. R. Evid. 702.
  84. Ariz.R.Evid. 803(18) (1991).
  85. Ark.R.Evid. 803(18) (1991).
  86. Colo.R.Evid. 803(18) (1992).
  87. Del.Unif.R.Evid. 803(18) (1991).
  88. Hawaii R.Evid. 803(b)(18) (1991).
  89. Idaho R.Evid. 803(18) (1991).
  90. Iowa R.Evid. 803(18) (1991).
  91. Kan.Stat. Ann. § 60–460(cc) (1991).
  92. Ky.Rev.Stat. Ann. § 422A.0803(18) (1991).
  93. La.Code Evid. Ann. art. 803(18) (1991).
  94. Me.R.Evid. 803(18) (1991).
  95. Minn.R.Evid. 803(18) (1992).
  96. Mont.R.Evid. 803(18) (1991).
  97. Nev. Rev.Stat. Ann. § 51.255 (Michie 1991).
  98. N.H.R.Evid. 803(18) (1991).
  99. N.M.R.Evid. 11–803 R (1991).
  100. N.C.Gen.Stat. § 8C-1, Rule 803(18) (1991).
  101. Okla.Stat. Ann. tit. 12, § 2803(18) (West 1991).
  102. S.D.Codified Laws Ann. § 19–16–22 (1991).
  103. Tex.R.Civ.Evid. 803(18) (1991).
  104. Utah R.Evid. 803(18) (1991).
  105. Vt.R.Evid. 803(18) (1991).
  106. Wash.R.Evid. 803(a)(18) (1991).
  107. W.Va.R.Evid. 803(18) (1991).
  108. Wis.Stat. Ann. § 908.03(18) (West 1991).
  109. *Cross v. Huttenlocher*, 185 Conn. 390, 440 A.2d 952, 955–56 (1981).
  110. Fed. R.Evid. 803(18).
  111. *Swank v. Halivopoulos*, 260 A.2d 240 (App. Div. 1969).
  112. *Gridley v. Johnson*, 476 S.W.2d 475 (Mo. 1972).
  113. *Darling v. Charleston Community Memorial Hosp.*, 211 N.E.2d 253, 259 (Ill. 1966).
  114. *Jacobson v. St. Peter's Med. Ctr.*, 608 A.2d 304 (1992).
  115. *Darling v. Charleston County Mem'l Hospital* 211 N.E.2d 253 (Ill. 1965)
  116. *Jones v. Bloom*, 388 Mich. 98, 200 N. W. 2d 196 (1972).
  117. *Iverson v. Lancaster*, 158 N. W.2d 507, 517–18 (N.D. 1968).
  118. *Dabroe v. Rhodes Co.*, 392 P.2d 317, 321–23 (1964).
  119. *Thornton v. CAMC*, 305 S.E.2d 316, 321–23 (W.Va. 1983).
  120. FRE 803 (18).
  121. *Schneider v. Revici*, 817 F.2d 987 (2d Cir.1987).
  122. *Tart v. McGann*, 697 F.2d 75, 78 (2nd Cir.1982).
  123. *Hemingway v. Ochsner Clinic*, 608 F.2d 1040, 1047 (5th Cir.1979).
  124. *Meschino v. North Am. Drager, Inc.*, 841 F.2d 429, 434 (1st Cir. 1988).
  125. *Kilpatrick v. Wolfond*, 704 So. 2d 708 (1998).
  126. Strickler J. EMTALA: The basics. JONAS Health Law Ethics Regul. 2006 Jul–Sept; 8(3):77–81.
  127. 42 U.S.C. § 1395dd(a).
  128. *Vickers v. Nash Gen. Hosp., Inc.*, 78 F.3d 139, 142 (4th Cir. 1996).
  129. *Baber v. Hospital Corp. of America*, 977 F.2d 8721, 880 (4th Cir. 1992).
  130. 42 U.S.C. § 1395dd(a), (e) (3) (B).
  131. *C.M. v. Tomball Reg'l Hosp.*, 961 S.W.2d 236, 240 (Tex. App.-Houston [1st Dist.] 1997).
  132. *Repp v. Anadarko Mun. Hosp.* 43 F.3d 529 (10th Cir. 1994)
  133. *Marshall v. East Carroll Parish Hospital*, 134 F.3d 319,322 (5th Cir. 1998).
  134. *United States v. Rush Foundation Hospital*: U.S.



- Dept. Health Hum. Services: No. CR 832 (2001).
135. *Jackson v. East Bay Hosp.*, 246 F.3d 1248 (9th Cir).
  136. *Eberhardt v. City of Los Angeles*, 62 F.3d 1253, 1255 (9th Cir. 1995).
  137. *Urban v. King*, 43 F.3d 523 (1994).
  138. *Burditt v. United States*, 934 F.2d 1362 (1991).
  139. *Frye v. United States* 293 F. 1013 (D.C. Cir. 1923).
  140. *Roe v. Wade* 410 U.S. 113 (1973).
  141. *Planned Parenthood v. Casey* 505 U.S. 833 (1992).
  142. *Maher v. Roe* 432 U.S. 464 (1977).
  143. *Harris v. McRae* 448 U.S. 297 (1980).
  144. *Stenberg v. Carhart* 530 U.S. 914 (2000).
  145. *Griswold v. Connecticut* 381 U. S. 479
  146. *Greco v. U.S.* 893. P2d 345 (1995).
  147. *Becker v. Schwartz* 46 NY 2d 401 (1978).
  148. *Reed v. Compagnolo* 332 Md. 226 (1993).
  149. *Siemieniec v. Lutheran General Hospital* 117 Ill. 2d 230 (1987).
  150. *Gallagher v. Duke University Hospital* 638 F.Supp 979 (M.D.N.C. 1986).

## Appendix II VENOUS THROMBOSIS AND PREGNANCY

John P. O'Grady

*Every improvement in practice must therefore take its rise from the establishment of more just ideas . . . and the causes of the disorders accompanying it, and by a proper attention to these, I am experimentally convinced that not only the method of cure may be much advanced, but, what is still more important, that these mischiefs so distressing and dangerous may be entirely prevented.*

Charles White (1728–1813)

*A Treatise on the Management of Pregnant and Lying-in Women*

London: Dilly, 1773, x.

Venous thrombosis (VT) during pregnancy is a relatively common condition that is potentially dangerous when complicated by venous thromboembolism (VTE) [1,8,12,27]. There are substantial variations among available estimates of incidence for VT and VTE, probably reflecting the different methods of diagnosis and case ascertainment [2–7,9]. A reasonable approximation is 1 to 2 in 1,000 pregnancies. The incidence of VT is generally thought to be threefold higher. In recent decades, while the incidence of VT during pregnancy has remained relatively stable, the VTE incidence has apparently declined significantly [2,9]. The reasons for this change in incidence is unknown. In general, the risk for VT and VTE is greatest in the postpartum period, especially among older women. The risk is also increased among mothers undergoing cesarean deliveries [2]. Smoking, thrombophilias, obesity, and a history of prior thrombosis are other important risk factors for recurrence [3,4,10]. See also Chapter 9, Obstetric Anesthesia, for additional discussion.

### PATHOPHYSIOLOGY

Pregnancy is a high-risk state for both VT and VTE. Pregnancy, and especially the puerperium, increase the risk of deep VT five- to sixfold (OR, 5.7; 95% CI 2.5–12.9), compared with nonpregnant women of the same age [11,12]. This increased risk is attributable to several factors. Most important among these are the existence of underlying thrombophilias (up to 50% of cases) [13], the occurrence of major surgery (primarily cesareans), and the natural increase in several clotting factors (i.e., Factors I, V, VII, VIII, IX, X, and XII) combined with decreases in antithrombin III and in proteins C and S. Most deep VTs occurring during gestation develop postpartum and involve the left leg. This unilateral preference is thought to be due to direct compression of venous return by the right iliac artery as it passes over the left iliac vein.

Although the serious complications of thromboembolism are uncommon, they are still among the leading causes of maternal mortality, especially because maternal losses from the traditional risks of hemorrhage, infection, and hypertension have been progressively reduced by improved clinical management [9,12,27]. Approximately 20% of *untreated* cases of deep VT are complicated by embolism, which has a 15% mortality risk. In contrast, a *treated* deep VT has a likelihood of embolism of <5% and an associated mortal risk of <1%. These data emphasize the importance of diagnosing deep VT promptly and initiating appropriate treatment to reduce the risk of developing VTE.

Tendencies toward a thrombosis or thrombophilia are conditions that are either inherited or acquired [20,23,25,39]. Such thrombophilias are best considered as intrinsic prothrombotic conditions that alone are often insufficient to result in a thrombosis or VTE. These conditions do predispose to thrombotic events in the presence of other specific risk factors or clinical events, such as pregnancy, exogenous estrogen treatment, or surgical trauma. The principal *inherited forms of thrombophilia* include Factor V Leiden mutation, antithrombin III deficiency, the protein C and S deficiencies, and the Factor II (G20210 A) variation. Other important risk factors for thrombosis are either acquired or transient. The most important factors for consideration in this discussion are pregnancy, surgery, or an acutely debilitating orthopedic injury. The most important *acquired thrombotic conditions* include the antiphospholipid syndrome (APLS) and the hyperhomocystinemia syndrome (HHCS). The latter is believed to be both genetic and environmental in cause. These conditions can either be temporary or become permanent. Regardless of whether abnormalities in coagulation are either hereditary or acquired, the various thrombophilias increase the risk of VT and VTE among the affected population by three- to fifteen-fold.

The currently identified inherited forms of thrombophilia and APLS also are variably associated with several obstetric complications, including growth disturbances involving intrauterine growth retardation; abruptio placentae; stillbirth; preeclampsia/pregnancy-induced hypertension; the hemolytic anemia, elevated liver enzymes and low platelet count (HELLP) syndrome; and recurrent

fetal loss [23,25,30,37,39]. The unifying features of these conditions are abnormalities in either the development of normal placentation or in subsequent placental function. Such pathologies are probably due to poorly understood changes in the maternal vascular responsiveness to trophoblastic invasion mediated through immunologic reactions at the cellular level.

## DIAGNOSIS

Unfortunately, the ability to diagnose deep VT and VTE during pregnancy by clinical signs and symptoms is at best limited. In fact, clinical diagnosis is correct only about 50% of the time. The classic findings of peripheral edema and complaints of dyspnea, tachycardia, or tachypnea characteristic of VTE are also common and often normal occurrences during gestation [12,26].

Establishing the correct diagnosis requires a high degree of suspicion, aided by specific testing. Venous duplex ultrasound studies, ventilation/perfusion lung scans, spiral computed tomography (CT) studies, arterial blood gases, and, less frequently, pulmonary angiography or venography are among the common methods used for diagnosis [14–18,27].

In acutely symptomatic pregnant women, neither lung scans nor the various radiographic diagnostic techniques are contraindicated. The magnitude of fetal risk from such investigations is inconsequential compared with the risk of failing to diagnose a VTE. In many cases, however, diagnosis is not easy. As an example, a high-probability ventilation/perfusion scan is nearly specific for an acute embolism, whereas a completely normal scan essentially excludes the diagnosis. Unfortunately, when these studies are performed in suspect cases, low-to-intermediate probability scans are the most common finding, and these have low specificity (~15%–30%) for embolism. In these equivocal cases, additional data such as blood gases, radiography, or invasive tests (e.g., angiography) are required to secure the diagnosis. In recent years, a spiral CT scan has become popular as an ancillary test but might or might not improve overall detection of embolization. These studies claim both high sensitivity (~85%) and specificity (~90%) [17,18].

## TREATMENT

Simple measures to reduce the risk of deep VT, such as early ambulation after surgery, intra- and postoperative use of compression boots or leggings, and perioperative drug prophylaxis, are now part of routine hospital and postsurgical care for most patients, pregnant or not [26]. These measures doubtless avoid some cases of both VT and VTE. Among pregnant women with a *prior VTE history*, or with an *established thrombophilia*, or with certain *obstetric risk factors combined with a thrombophilia*, the risk for thrombosis is higher than that for the general population, and drug prophylaxis with heparin or another agent either during pregnancy or in the puerperium is appropriate to reduce risk.

The principal drugs for use in prophylaxis include the vitamin K antagonists (warfarin [Coumadin]) and various types of heparin. Warfarin crosses the placenta, and, although effective in blocking thrombosis, this drug is potentially teratogenic and its effects are difficult to easily or rapidly modulate. In late pregnancy, warfarin also results in fetal anticoagulation, which places the infant at risk for spontaneous hemorrhage either before or during parturition. There are restricted warfarin protocols that are used during pregnancy for special cases, such as the treatment of women with artificial heart valves or those unable to tolerate heparin [19]. In general, however, the vitamin K antagonists are contraindicated during gestation. Because warfarin is not secreted in breast milk, however, this drug is safe to administer in the puerperium to lactating women who require prophylaxis. Currently, this is the principal use of warfarin in association with pregnancy.

Heparin, which does not cross the placenta, is the drug of choice to inhibit thrombosis in gravid women. Treatment with heparin is not risk free, however; long-term use can result in osteoporosis. Approximately one third of those treated with unfractionated heparin for several months have radiographically demonstrable reductions in bone density. There is also a risk of spontaneous hemorrhage in approximately 2% of cases when heparin is administered in full dose. This risk is the same as in the nonpregnant population. Immune-mediated heparin-induced thrombocytopenia also complicates 5% to 30% of patients treated with unfractionated heparin.

Because of a reduced risk of induced thrombocytopenia (and probably a reduced risk for osteopenia as well) and owing to easier monitoring of effect (anti-factor Xa levels), low-molecular-weight heparin (LMWH) derivatives have progressively replaced unfractionated heparin as the drug of choice for long-term treatment or prophylaxis.

Anticoagulation treatment is generally recommended during pregnancy for several specific medical conditions [21–22,24,27,29,38–39]. These conditions include pregnancies complicated by 1) artificial heart valves; 2) rheumatic heart disease with atrial fibrillation or a history of atrial fibrillation; 3) prior documented VTE, and 4) selected thrombophilias (e.g., homozygous Factor V Leiden, prothrombin G 20210A mutation, antiphospholipid syndrome, and antithrombin III deficiency). Exactly which of this last group to treat and how to order this treatment are the problems; as usual, the devil is in the details.

The concern is not only which women with thrombophilias or other risk factors to treat but also which drug to use and the best dosing schedule. The uncommon cases involving women diagnosed with a VTE during pregnancy are not the issue. An acute embolism is treated essentially the same way for pregnant as for the nonpregnant patients. Supportive care is provided, and full anticoagulation with heparin, or occasionally a vitamin K antagonist, is initiated [12,24,27,36]. Other scenarios for treatment are more controversial.

Alternative and rarely employed treatments for deep VT or VTE during pregnancy include low-dose unfractionated heparin combined with a vena cava filter, or iliofemoral thrombectomy. Direct thrombolytic therapy is hardly ever used and is appropriate only in the rare and extreme situation of maternal hemodynamic instability accompanying acute pulmonary embolization [12]. Postpartum prophylactic treatment should also be considered for all cases when there is a substantially increased VTE risk. Traditionally, the drug of choice in the puerperium has been oral warfarin. The uncertainties in antepartum treatment unrelated to VT and VTE are the main concern. Furthermore, whenever treatment is initiated, it is far from standardized.

The problem cases include the following subsets of patients:

- Women with a history of a prior venous thrombosis but without evidence of embolization, and who are asymptomatic during pregnancy
- Women with an identified thrombophilia who have been symptom free prior to pregnancy
- Women with thrombophilias and an abnormal previous pregnancy history that does not include a demonstrated VT or VTE [13].

There is general concurrence that women with a thrombophilia (including the APS) and a prior history of embolism need continuous antepartum and postpartum prophylaxis to reduce the risk of recurrence [20,28]. In this instance, the risk(s) of treatment are offset by the benefits. The most common therapy is initial LMWH during pregnancy, followed by conversion to a vitamin K antagonist (warfarin) in the puerperium, with treatment continued for 3 to 6 months, and in some cases indefinitely.

Many women with a documented thrombophilia and a prior pregnancy loss or other related obstetric complications have been treated with either unfractionated heparin/LMWH or unfractionated heparin/LMWH combined with low-dose aspirin (81 mg/day) during gestation. The benefits of such treatment as reported in the literature are inconsistent, however. The available studies are all methodologically flawed, usually involving too few treated cases to judge efficacy and risk; thus, the best treatment for many of these cases remains simply unsettled [20,21].

Some data are clear, however. Corticosteroid treatment for the specific group of women with APS (i.e., demonstrated antiphospholipid antibodies plus recurrent fetal loss) is less effective than heparin therapy [30–33]. Steroid therapy alone for these APS cases has largely been abandoned unless there is evidence of a coexisting autoimmune abnormality such as systemic lupus erythematosus or thrombocytopenia. Heparin treatment combined with low-dose aspirin is apparently superior to low-dose aspirin alone, although it is fair to say that there is no agreement on this point in the literature. Recent data suggest that LMWH has equal efficacy with unfractionated heparin when combined with low-dose aspirin in treating APS [34]. Furthermore, the combination of LMWH plus low-dose aspirin in APS is more efficacious than intra-

venous immunoglobulins in achieving live births [38].

Treatment for women who do not have a documented thrombophilia but who do have a prior history of VT without evidence of embolization is also controversial. In some protocols, routine anticoagulation treatment is withheld because of the narrowness of the risk/benefit ratio in light of the low risk of a thrombosis complication. In precisely the same clinical situation, however, other consultants favor treatment [13].

This is a continually evolving field, and recommendations for treatment are under constant revision. Interested readers therefore are referred to other sources for additional information and advised to check specific treatment recommendations from more than one source [26–29,35,39].

## REFERENCES

1. Stein PD, Hull RD, Kayali F, Olson RE, Alshab AK, Meyers FA, Ghali WA, Silbergleit A, Gibson P. Venous thromboembolism in pregnancy: 21-year trends. *Am J Med.* 2004 Jul 15;117(2):121–5.
2. Heit JA, Kobbervig CE, James AH, Petterson TM, Bailey KR, Melton LJ 3rd. Trends in the incidence of venous thromboembolism during pregnancy or postpartum: A 30-year population-based study. *Ann Intern Med.* 2005 Nov 15;143(10):697–706.
3. Simpson EL, Lawrenson RA, Nightingale AL, Farmer RD. Venous thromboembolism in pregnancy and the puerperium: Incidence and additional risk factors from a London perinatal database. *Br J Obstet Gynecol.* 2001 Jan;108(1):56–60.
4. James AH, Jamison MG, Brancazio LR, Myers ER. Venous thromboembolism during pregnancy and the postpartum period: Incidence, risk factors, and mortality. *Am J Obstet Gynecol.* 2006 May;194(5):1311–5.
5. Andersen BS, Steffensen FH, Sørensen HT, Nielsen GL, Olsen J. The cumulative incidence of venous thromboembolism during pregnancy and puerperium – An 11-year Danish population-based study of 63,300 pregnancies. *Acta Obstet Gynecol Scand.* 1998 Feb;77(2):170–3.
6. Soomro RM, Bucur IJ, Noorani S. Cumulative incidence of venous thromboembolism during pregnancy and puerperium: A hospital-based study. *Angiology.* 2002 Jul–Aug;53(4):429–34.

7. Treffers PE, Huidekoper BL, Weenink GH, Kloosterman GJ. Epidemiological observations of thrombo-embolic disease during pregnancy and in the puerperium, in 56,022 women. *Int J Gynaecol Obstet.* 1983 Aug;21(4):327-31.
8. Kierkegaard A. Incidence and diagnosis of deep venous thrombosis associated with pregnancy. *Acta Obstet Gynecol Scand* 1983;62:239-43.
9. Silverstein MD, Heit JA, Mohr DN, Petterson TM, O'Fallon WM, Melton LJ III. Trends in the incidence of deep-vein thrombosis and pulmonary embolism: A 25-year population-based study. *Arch Intern Med.* 1998;158:585-93.
10. Danilenko-Dixon DR, Heit JA, Silverstein MD, Yawn BP, Petterson TM, Lohse CM, Melton LJ 3rd. Risk factors for deep vein thrombosis and pulmonary embolism during pregnancy or post partum: A population-based, case-control study. *Am J Obstet Gynecol.* 2001 Jan;184(2):104-0.
11. Melis F, Vandenbrouke JP, Büller HR, Colly LP, Bloemenkamp, KWM. Estimates of risk of venous thrombosis during pregnancy and puerperium are not influenced by diagnostic suspicion and referral basis. *Am J Obstet Gynecol.* 2004;191:825-29.
12. Sellman JS, Holman RL. Thromboembolism during pregnancy: Risks, challenges, and recommendations. *Postgrad Med.* 2000;108:71-2; 77-8; 81-4.
13. Brill-Edwards P, Ginsberg JS, Gent M, Hirsh J, Burrows R, Kearon C, Geerts W, Kovacs M, Weitz JI, Robinson KS, Whittom R, Couture G. Safety of withholding heparin in pregnant women with a history of venous thromboembolism: Recurrence of clot in this pregnancy study group. *N Engl J Med.* 2000;343:1439-44.
14. Polak JF. Deep venous thrombosis in pregnancy: Noninvasive diagnosis. *Radiology.* 1988;166:377-9.
15. Gill P, Nahum A. Improving detection of venous thromboembolism: New technology holds promise for early, precise diagnosis. *Postgrad Med.* 2000;108: 24-6; 29-32; 38-40.
16. PIOPED Investigators. Value of the ventilation/perfusion scan in acute pulmonary embolism. *JAMA.* 1990;263:2753-9.
17. Mullins MD, Becket DM, Hagspiel KD, Philbrick JT. The role of spiral volumetric computed tomography in the diagnosis of pulmonary embolism. *Arch Intern Med.* 2000;160:293-8.
18. Rathbun SW, Raskob GE, Whitsett TL. Sensitivity and specificity of helical computed tomography in the diagnosis of pulmonary embolism: A systematic review. *Ann Intern Med.* 2000;132, 227-32.
19. Khamooshi AJ, Kashfi F, Hoseini S, Tabatabaei MB, Javadpour H, Noohi F. Anticoagulation for prosthetic heart valves in pregnancy. Is there an answer? *Asian Cardiovasc Thorac Ann.* 2007 Dec;15(6): 493-6.
20. Lim W, Crowther MA, Eikelboom JW. Management of antiphospholipid antibody syndrome: A systematic review. *JAMA.* 2006;295:1050-7.
21. Gates S, Brocklehurst P, Davis LJ. Prophylaxis for venous thromboembolic disease in pregnancy and the early postnatal period. *Cochrane Database.* 2002;2.
22. Walker MC, Ferguson SE, Allen VM. Heparin for pregnant women with acquired or inherited thrombophilias. *Cochrane Database.* 2003;2.
23. Dentali F, Crowther M. Acquired thrombophilia during pregnancy. *Obstet Gynecol Clin North Am.* 2006;33:375-88.
24. Ginsberg JS, Greer I, Hirsh J. Use of antithrombotic agents during pregnancy. *Chest.* 2001;119:122S-131S.
25. Coppens M, Kaandorp SP, Middeldorp S. Inherited thrombophilias. *Obstet Gynecol Clin North Am.* 2006;33:357-74.
26. The American College of Obstetricians and Gynecologists. Practice Bulletin No. 21, October 2000. Prevention of Deep Vein Thrombosis and Pulmonary Embolism. Washington, DC, The American College of Obstetricians and Gynecologists, 2000.
27. The American College of Obstetricians and Gynecologists Practice Bulletin No. 19, August 2000. Thromboembolism in Pregnancy. Washington, DC, The American College of Obstetricians and Gynecologists, 2000.
28. The American College of Obstetricians and Gynecologists Practice Bulletin No. 68, November 2005. Antiphospholipid Syndrome. Washington, DC, The American College of Obstetricians and Gynecologists, 2005.
29. Geerts WH, Pineo GF, Heit JA, Bergqvist D, Lassen MR, Colwell CW, Ray JG. Prevention of venous thromboembolism: Seventh ACCP conference on antithrombotic and thrombolytic therapy. *Chest.* 2004;126:338S-400S.
30. Cowchek FS, Reece EA, Balabam D, Branch DW, Plouffe L. Repeated fetal losses associated with antiphospholipid antibodies: A collaborative randomized trial comparing prednisone with low-dose

- heparin treatment. *Am J Obstet Gynecol.* 1992;166:1318–23.
31. Rai R, Cohen H, Dave M, Regan L. Randomised controlled trial of aspirin and aspirin plus heparin in pregnant women with recurrent miscarriage associated with phospholipid antibodies (or antiphospholipid antibodies). *Br Med J.* 1997;314:253–7.
  32. Kutteh, WH. Antiphospholipid antibody-associated recurrent pregnancy loss: Treatment with heparin and low-dose aspirin is superior to low-dose aspirin alone. *Am J Obstet Gynecol.* 1996;174:1584–90.
  33. Farquharson RG, Quenby S, Greaves M. Antiphospholipid syndrome in pregnancy: A randomized, controlled trial of treatment. *Obstet Gynecol.* 2002;100:408–13.
  34. Noble LS, Kutteh WH, Lashey N, Franklin RD, Herrada J. Antiphospholipid antibodies associated with recurrent pregnancy loss: Prospective, multicenter, controlled pilot study comparing treatment with low-molecular-weight heparin versus unfractionated heparin. *Fertil Steril.* 2005;83:685–90.
  35. Royal College of Obstetricians and Gynaecologists (RCOG). *Thromboprophylaxis during pregnancy, labour, and after vaginal delivery (Guideline No. 37).* London (UK): Royal College of Obstetricians and Gynaecologists, 2004.
  36. Bates SM, Ginsberg JS. How we manage thromboembolism during pregnancy. *Blood.* 2002;100:3470–8.
  37. Empson M, Lassere M, Craig JC, Scott JR. Recurrent pregnancy loss with antiphospholipid antibody: A systemic review of therapeutic trials. *Obstet Gynecol.* 2002;99:135–44.
  38. Triolo G, Ferrante A, Ciccio F, Accardo-Palumbo A, Perino A, Castelli A, Giarratano A, Licata G. Randomized study of subcutaneous low-molecular-weight heparin plus aspirin versus intravenous immunoglobulin in the treatment of recurrent fetal loss associated with antiphospholipid antibodies. *Arthritis Rheum.* 2003;48:728–31.
  39. The American College of Obstetricians and Gynecologists. *Thrombosis, Thrombophilia, and Thromboembolism. Clinical Updates in Women's Health, October, 2007: VI(4).*

## III FETAL HEART RATE MONITORING: SURGICAL PROCEDURES

John P. O'Grady

*Ask, and it shall be given you;  
seek, and ye shall find;  
knock and it shall be opened unto you.*

Matthew: 7:7  
The Holy Bible

It is estimated that 8 of 75,000 pregnant women, or up to 2%, undergo surgical procedures during pregnancy each year [2–4,8,10,14, 17]. This wide range of estimate is because of poor reporting, case exclusion, and differences in case ascertainment. Many of these procedures are directly or indirectly related to pregnancy, such as performance of cerclage. Others are less clearly related, such as ovarian cystectomy, cholecystectomy, or urologic procedures such as stent placement [6]. Finally, there is an important number of surgical cases that are seemingly incidental to pregnancy such as appendectomy, cancer surgery, and various procedures for automobile accidents and other trauma [17]. While in general elective procedures are avoided during gestation, laparoscopy, organ transplantation, cardiopulmonary bypass, and induced hypothermia have all been successfully performed in unique circumstances [4].

The major fetal risks from surgery usually arise from the underlying pathology or from acute problems with maternal hypoxia. Inflammatory processes in the peritoneal cavity, excessive blood loss, and maternal hypotension are the principal clinical causes of fetal loss.

The best perioperative management of these patients is frequently questioned. In determining best practice there is limited literature, because most papers report experience in specific and usually difficult cases or discuss management based on general physiologic or anesthetic principles and no systematic studies are available.

This chapter presents protocols for pre-, peri-, and postoperative fetal evaluation divided by gestational age. In each category, specific recommendations for evaluation are made. Direct fetal monitoring is neither necessary nor technically possible in all cases. In the author's opinion, however, whenever a pregnant woman is taken to surgery, the minimum requirements are the documentation of fetal heart tones either by direct auscultation or by using a Doppler device before and after the surgery. In



most cases, reasonable attention to adequate maternal oxygenation, hydration, and, when the uterus is more than 20 to 22 weeks' size, maternal positioning, is usually all that is required.

The necessity for continuous fetal monitoring during surgery is controversial [7,9,15–17,19]. Major procedures have been successfully conducted without such surveillance. Furthermore, the use of monitoring is by no means universal. In a 1995 hospital survey, only 60% of responding institutions reported routine fetal or uterine monitoring during nonobstetric surgery during pregnancy [8]. As Horigan and coworkers assert [15], neither fetal morbidity nor mortality has been documented to occur without concomitant maternal hypoxia, regardless of whether monitoring occurred or not. In this view, intraoperative monitoring of the fetus is only a distraction because the important abnormalities that lead to fetal harm are reflected in the standard maternal parameters of cardiovascular and respiratory function normally evaluated intraoperatively by the anesthesiologist. Even if this position is correct, it does not release the clinician from pre- and postoperative documentation of fetal condition/status, as well as close attention to the important risk of perioperative and postoperative preterm labor.

The decision to attempt intraoperative surveillance of the fetus and/or record uterine activity is predicated on several assumptions:

- Fetal condition (oxygenation/acid–base balance) is reasonably reflected by FHR patterns and rate.
- There is the technical capability to adequately record the FHR and uterine activity, and more importantly, trained personnel are available to interpret these tracings and suggest appropriate remedial treatment.
- There is plan for intervention if either inappropriate uterine activity or unacceptable or bothersome FHR patterns occur.
- Treatment for perceived fetal problems as reflected in FHR pattern changes is necessary to avoid injury or to provide early treatment for inappropriate uterine activity.

### Gestational Age

To evaluate the appropriateness of electronic fetal monitoring (EFM) and other measures, the clinician

must know the period of gestation. The physiology of uterine perfusion and fetal oxygenation at varying gestational ages is an important consideration. The earlier the gestation, the less critical maternal positioning is, and when the pregnancy is less than 24 weeks, delivery in the case of difficulty is not a reasonable option. Unfortunately, because maternal positioning and routine measurements of maternal brachial artery pressure do not necessarily ensure adequate perfusion at the level of the uterus, monitoring can be required to provide reassurance of the fetal status. In more advanced gestations, both the FHR and the pattern of the fetal heart (i.e., reactive or not, decelerations or not) or direct observation of fetal activity and in-utero breathing movements (i.e., the biophysical profile [BPP]) serve as reasonable, indirect measures of uterine perfusion, adequate fetal oxygenation, and stable condition. See Chapter 9, Obstetric Anesthesia.

### Labor

Labor provides an additional stress to the fetus. Periodic myometrial contractions block uterine perfusion by occluding vessels that traverse the uterine wall. A fetus that is tolerant of a particular set of physiologic circumstances in the absence of labor might show important heart rate changes or develop hypoxia under the recurrent stress of contractions as blood flow is intermittently interrupted. Tocolysis during surgery therefore might be desirable in selected cases, assuming that the drugs administered as tocolytics do not cloud the evaluation of the mother (e.g., by tachycardia) or result in other adverse maternal cardiovascular effects (e.g., hypotension or arrhythmia). Clearly, uterine contractions can be either a harbinger of actual labor or threaten membrane stability. Fortunately, under normal circumstances, the time required for tocolysis is limited, and coverage for the period of surgery or a few hours of postoperative recovery are often all that is required unless there is a persisting inflammatory focus present in the peritoneal cavity [3]. The overall risk of preterm labor in women with pelvic or lower abdominal surgery during pregnancy is 4% to 6%. For nonabdominal procedures, the risk drops to approximately 1% and in these latter cases is related to maternal hypovolemia [12].

Tocolytic agents should not be routinely administered, but if the uterus is irritable or the patient is

perceived to be at increased risk, they should be considered. The risk of uterine activity/threatened labor is greatest when an inflammatory process is present within the peritoneal cavity or when direct manipulation of the uterus or myometrial incisions are performed during the surgical procedure.

### Tocolysis

For acute tocolysis, nitroglycerine administered in boluses of 100  $\mu$ g to 150  $\mu$ g IV every few minutes to effect (to 500  $\mu$ g maximum) is the most rapidly acting tocolytic and the drug of choice for use perioperatively. Although nitroglycerine is efficacious, its effects are brief, and this drug is not suited for other than acute or emergency use. The betamimetics (primarily terbutaline) are also reasonably effective tocolytic drugs but have serious side effects, primarily maternal tachycardia, hyperglycemia, electrolyte abnormalities, and pulmonary congestion, which limit their use. Although parenterally administered terbutaline (100  $\mu$ g–250  $\mu$ g SC or IV) is rapid in onset, it is also relatively brief in duration.

Recently, long-term tocolysis has depended more on other oral agents. The calcium channel blockers (e.g., nifedipine) are popular, as are the prostaglandin inhibitors (e.g., indomethacin). These drugs are best administered prophylactically, with indomethacin having the potential advantage of rectal administration. Indomethacin can be administered for up to 48 hours in doses of 25 mg to 50 mg every 6 to 8 hours. Long-term treatment or more than brief treatment in pregnancies beyond the 32nd to 33rd weeks can lead to important fetal cardiovascular complications and should generally be avoided, unless the pregnancy is followed by real-time ultrasound surveillance. Among the calcium channel blockers, nifedipine, given in titrated doses of 10 mg to 20 mg every 4 to 6 hours, is the most commonly used drug and is generally well tolerated with few maternal side effects of clinical consequence. Magnesium sulfate administered as a continuous infusion, following a loading dose bolus, is no longer favored for tocolysis because of concerns of efficacy and the potential for adverse fetal and maternal side effects [1]. Obviously, if any of the long-term tocolytic agents are administered, the decision to treat must be known by both the surgeon and the anesthesiologist before the surgery.

### POTENTIAL VIABILITY

The various parameters of fetal well-being and the possibility of intervention are necessarily tied to the concept of potential fetal viability. *Potential viability* refers to the likelihood of long-term survival of the infant at a specific gestational age. Important ancillary issues are the inherent normality of the fetus, condition at birth that might be affected by hypoxia, asphyxia, or injury and the availability of neonatal support (neonatal intensive care unit). A related and most important issue for families that is surprisingly little discussed in the literature is that of *intact survival*, a prediction that is often difficult to make. As the period of potential survivability for very small infants has been pushed to progressively earlier gestational ages, the risk for permanent and serious infant injury as a consequence of prematurity has also increased. Difficulty lies in determining how aggressive intervention should be in situations in which the likelihood of fetal survival is low or the fetal condition is thought to be precarious, even if the gestational age is advanced to the point at which there is a reasonable likelihood of survival. This becomes a question of both practicality and ethics.

Based on the experience of the last decade, potential viability is best viewed as a changing measure that depends on several factors, including the extent of local facilities, the experience and skill of the pediatric attendants, the presence or absence of fetal anomalies, and the fetal condition at birth, among other factors. As a practical matter, fetal survival at less than 22 completed weeks is at best uncommon; survival becomes more likely after 23 completed weeks and an estimated weight of approximately 400 g. To reasonably ensure intact survival of approximately 50% or better, the gestational age must be 24 or preferably 25 completed weeks [18]. Because intact survival in very premature infants is so problematic, in pregnancies of less than 24.5 to 25 weeks it is best to attempt to keep these fetuses in utero for additional maturation, while optimizing the mother's condition if possible. Conversely, with gestations advancing beyond the 32nd week, delivery in the face of substantially adverse conditions is a much more compelling strategy owing to the high likelihood of intact survival for these infants. There are no easy answers. At the bitter edge of fetal viability

(22–24 weeks), care must be individualized, balancing both fetal and maternal interests. The family must be involved in the decisions that are reached about intervention if severe fetal jeopardy is possible or suspected, and delivery is among the potential therapies considered by the primary attendants.

The assistance of a pediatrician/neonatologist in counseling is helpful, but continuity of approach is an issue. In the author's experience, obstetricians are generally more pessimistic than pediatricians about infant outcomes in markedly premature pregnancies. To avoid increasing the difficulties in an already problematic situation, all clinicians involved in counseling should present complementary data, reflecting general agreement on major points of potential viability, the risks of long-term disability, and the type of treatment that might be required. It is therefore prudent to review the clinical situation with the pediatric consultant *before patient/family counseling* and reconcile any differences in factual data. Receiving different assessments from otherwise well-meaning physicians is distressing and unnecessarily confusing for the family, especially because this problem is mostly avoidable. Clinicians should be aware of a new website that provides rapid estimates of fetal survivability and permanent injury that can assist in counseling: [www.nichd.nih.gov/about/org/cdbpm/pp/prog\\_epbo/epbo\\_caseestimates.cfm](http://www.nichd.nih.gov/about/org/cdbpm/pp/prog_epbo/epbo_caseestimates.cfm).

## PROTOCOLS

Based on the considerations previously discussed, the author has developed a series of guidelines for perioperative and postoperative management of pregnant women undergoing nonobstetric surgical procedures (Table A.1). The importance of establishing a protocol designed for each institution is emphasized. When the general approach is standardized, appropriate treatment and evaluations are possible without undue confusion and uncertainty, and either overevaluation or underevaluation becomes less likely. There are several important points:

- When surgical procedures are performed on pregnant women, the surgeon, obstetrician, and anesthesiologist should *jointly* decide on measures to be taken for fetal evaluation and actions to avoid or treat presumed fetal jeopardy prior to beginning the surgery. Documentation of the specifics

of the chosen method(s) of surveillance must be noted in the medical record.

- A designated obstetrician must be available if cesarean delivery is considered among the acceptable methods of treatment and if unresolved fetal problems occur or are strongly suspected.
- In all instances, the surgeon should discuss the case preoperatively with the responsible obstetrician and the anesthesiologist.
- If abnormalities in maternal pulse oximetry or EFM or other parameters are observed in pregnancies  $\geq 23$  weeks' gestation during surgery, it is recommended that the responsible obstetrician be promptly notified if standard maneuvers such as maternal positioning, fluid administration, and discontinuation of uterine manipulation or compression do not result in the prompt resumption of more normal findings.
- Anesthesia-induced changes in FHR variability must be distinguished from abnormalities caused by asphyxia/poor perfusion [9].
- Postoperative pregnancy surveillance should continue for 12 to 24 hours, depending on the original pathology and the procedure(s) performed [6].

## **General Management**

It should be remembered that in many instances, even in gestations in the third trimester, continuous monitoring of the fetal heart during surgery is impossible or unsatisfactory because of technical reasons. Either the abdomen is not available for the use of a Doppler monitor, or the maternal position precludes obtaining a good tracing. In most instances, FHR study of the fetus pre- and postinduction of anesthesia and postoperatively by FHR study, non-stress test (NST), or some other method (e.g., a BPP) proves best.

A brief preoperative evaluation of fetal status is reassuring to both patient and surgeon. In addition, if uterine activity is noted, prophylactic tocolysis can be considered. In trauma cases, the immediate preoperative fetal condition can have legal implications, especially if a fetal loss subsequently occurs.

Perioperatively, if the uterus is large enough to become an abdominal organ, supine hypotension is possible, and lateral recumbency positioning is indicated. This is not often an issue of significance,

TABLE A.1 Protocol for Fetal Monitoring in Pregnant Women Undergoing Surgery\*

<p>I. <math>\leq 14</math> weeks (embryonic phase/early fetal life):</p> <ul style="list-style-type: none"> <li>• Pre-/postprocedure, notation, and documentation of fetal heart tones (FHTs) in the medical record by a handheld Doppler device or auscultation by fetoscope</li> <li>• If the clinician is unable to auscultate or locate the FH externally, fetal cardiac motion can be directly visualized by real-time ultrasound scan and subsequently documented.<sup>†</sup></li> <li>• Perioperative pulse oximetry per anesthesia protocol</li> </ul> <p>II. 14–22 Completed Weeks (previable fetus):</p> <ul style="list-style-type: none"> <li>• Maternal LLR or hip wedge positioning is prudent after 20 weeks' gestation, and strongly recommended after 22–23 weeks with attention to possible supine hypotension (BP <math>&lt; 80/50</math>, urine output <math>\leq 30</math> ml/hr). As the uterus is small at gestational ages of 20–22 weeks or less, marked supine positioning is of less importance because the risk of interference with maternal/fetal blood flow is low. More important is the avoidance of unnecessary perioperative pressure to or manipulation of the uterus</li> <li>• At this gestational age, continuous EFM is neither necessary nor usually interpretable. It is recommended that the FHTs be simply auscultated or located and recorded pre-/postprocedure as in I.</li> <li>• Perioperative maternal pulse oximetry is performed by standard anesthesia protocol</li> </ul> <p>III. <math>\geq 23</math> weeks (period of potential extrauterine viability):</p> <ul style="list-style-type: none"> <li>• Preoperative: If the surgery is elective, the patient is stable, and there is no acute maternal decompensation, a normal BPP or NST is performed within the 24 hours preceding the procedure. The study might not be interpretable as reassuring by standard criteria owing to early gestational age, and allowance must be made for this common limitation to avoid unnecessary or inappropriate additional studies. If the maternal condition is acute, a BBP or NST should be performed when clinical circumstances permit. In all cases, at a minimum there should be a FHT obtained and documented in the medical record before surgery. If the recorded FHR is <math>\geq 200</math> beats/min or <math>\leq 100</math> beats/min, the attending obstetrician must be notified.</li> <li>• The charge nurse in the labor and delivery suite should be informed when scheduling cases in the operating suite involving obstetric patients with gestational ages falling within the period of potential viability (<math>\geq 23</math> weeks).</li> <li>• Perioperative LLR or hip wedge positioning should be performed with close attention to possible supine hypotension (BP <math>\leq 80/50</math>; urine output <math>&lt; 30</math> ml/hr).</li> <li>• Continuous external EFM may electively be performed during the procedure (FHR and uterine activity/contraction) if deemed appropriate by the attending obstetrician and technically possible. Often continuous monitoring is not realistic owing to</li> </ul>	<p>difficulties with maintaining a consistent fetal signal or early gestational age. The type of surgery performed also might preclude such monitoring. If the obstetric attending elects not to perform continuous EFM, a pre- and postoperative fetal assessment should be performed in all cases. In nonemergent cases, appropriate maternal positioning and the notation of the FHR following the induction of anesthesia and prior to commencing the surgery is prudent. Intermittent rechecks of the FHR can be electively performed perioperatively by ultrasound or Doppler scan, on a schedule decided on by the attending physician. If intermittent or continuous FHR checks are deemed necessary, however, an obstetrically experienced designated clinician – not the anesthesiologist – must be present to conduct and record these determinations. If the attending physician elects to perform continuous EFM, an obstetrician, an obstetric nurse, or other trained attendant must be present to continuously review the fetal heart tracing.</p> <ul style="list-style-type: none"> <li>• Postoperative: Appropriate postsurgical management includes LLR positioning as is possible, FHR recording, and palpation for uterine contractions every 15 minutes <math>\times 4</math>; then every 30 minutes <math>\times 2</math>. If the FHR is stable (<math>\geq 110</math> beats/min <math>&lt; 180</math> beats/min), and no active contraction pattern is present, the fetal condition is thereafter reevaluated each shift or per physician orders. If the FHR falls outside these heart rate parameters or uterine activity is recurrent or suspected, continuous external EFM should be performed as possible given the constraints of gestational age, maternal positioning, and access to the abdomen. Under these circumstances, the attending obstetrician should be notified for appropriate patient evaluation, possible transfer, further fetal monitoring (e.g., a BPP) or tocolysis.</li> <li>• Tocolysis is not routinely indicated unless uterine surgery is performed, documented uterine irritability is suspected, or an intraperitoneal inflammatory process is diagnosed. Choice of tocolytic agent is a joint decision of obstetrician, surgeon, and anesthesiologist.</li> <li>• FHR findings requiring notification of the attending obstetrician include <ul style="list-style-type: none"> <li>– Recurrent FH bradycardias (FHR <math>\leq 110</math> beats/min) especially if there is a late return to the baseline</li> <li>– Recurrent or fixed FH decelerations (FHR <math>&lt; 110</math> beats/min)</li> <li>– Recurrent uterine activity patterns (contractions <math>\geq 8</math>–10 per hour) or uterine tetany (contraction without apparent relaxation)</li> <li>– Markedly irregular or noninterpretable FHR patterns</li> <li>– Nonreactive EFM tracing in fetuses of <math>\geq 30</math> weeks' gestation</li> </ul> </li> </ul>
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(Continued)

TABLE A.1 Protocol for Fetal Monitoring in Pregnant Women Undergoing Surgery (Continued)\*

- |   |  |
|---|--|
| <ul style="list-style-type: none"> <li>• Perioperative measures to improve an abnormal or possibly abnormal FHR pattern include           <ul style="list-style-type: none"> <li>– L.L.R positioning, or a change to R.L.R positioning</li> </ul> </li> </ul> | <ul style="list-style-type: none"> <li>– Administration of high-concentration oxygen to mother by mask, if she is not intubated or already receiving oxygen</li> <li>– Prompt discontinuation of uterine/abdominal pressure or manipulation</li> </ul> |
|---|--|

\*See text for details.

†If the FHR is recorded as consistently  $\geq 200$  beats/min or  $\leq 100$  beats/min, the attending obstetrician should be notified.

however, until after the 22nd to 23rd week. In all cases, attention to full hydration and the avoidance of even transient maternal hypoxia is important.

Stable pregnant women in good health who lack major medical problems and who are normovolemic can easily tolerate the stress of routine surgical procedures. With reasonable attention to hydration, avoidance of abdominal/uterine pressure or compression, the use of left lateral recumbent (LLR) positioning, and the occasional administration of tocolytic agents, fetal problems should be rare. In most cases, documentation of fetal condition pre- and postoperatively (usually by FHR auscultation or Doppler determination) and reasonable attention to the potential for premature labor are all that is required.

*The suggested precautions and preparations for surgery listed in Table A.1 during pregnancy are intended for nonemergent procedures.* In a true surgical emergency, necessary procedures must be performed for maternal safety under the best possible clinical circumstances that can be established at the time. This is particularly true when maternal hypotension, coagulopathy, sepsis, or hypoxia is present. Urgently required maternal treatment should not be withheld, curtailed, or delayed because of signs suggesting fetal jeopardy.

As a general rule, fetal well-being is best ensured by prompt maternal treatment and cardiopulmonary support. In difficult circumstances, pregnant women should be treated the same as nonpregnant patients, with special attention to the issues of maternal positioning, oxygenation, and hydration. If the maternal condition is precarious or the fetal status becomes either unstable or uncertain, an attending obstetrician must be summoned. One important exception to the dictates of this protocol is perimortem cesarean delivery. How best to act if the mother sustains a cardiopulmonary arrest during surgery is obviously problematic. The best estimate

of gestational age, the time that has elapsed since the arrest, and the adequacy of the maternal resuscitation efforts are critical. The choice concerning prompt delivery is clinical. As a practical matter, in the midst of maternal resuscitation efforts, attempting to locate or visualize the fetal heart is always difficult and frequently impossible. Furthermore, if the heart rate either cannot be determined or the heart is seen to be arrested, it is unclear for how long this has been present. In extreme situations, cesarean delivery can be life saving for both the mother and fetus, because emptying the uterus permits better resuscitation and support for both.

Thus, if the mother is moribund and an adequate trial of resuscitation has been attempted (usually defined as  $\geq 5$  minutes but  $< 15$  minutes), and the gestation is within the period of presumed viability, prompt delivery is indicated [14].

Maternal mortality related to anesthesia is rare. In the review of Cohen-Kerem and coworkers of 12,452 cases of nonobstetric surgeries, the mortal risk was 0.06% [3]. Fetal difficulties related to anesthesia and, more importantly, to the surgical procedure performed and the underlying pathology is the issue. Early pregnancy losses related to anesthesia are uncommon, although difficult to determine due the variable background rate [3]. Because of theoretic risks of anesthetic agents as potential teratogens, the declining incidence of spontaneous losses as pregnancy proceeds, and the potential for patient/family misunderstanding if loss occurs, elective operations are best scheduled in the midtrimester, when possible. Surgery at this time occurs after the majority of spontaneous losses have occurred, the pregnancy is sufficiently advanced to permit both biochemical and ultrasound surveillance to generally ensure normality and that the uterus is not too large as to seriously interfere with observation/exposure. The uterus also is usually relatively nonirritable at this time.

Fetal well-being depends on the adequacy of maternal circulation to the placenta and maternal oxygenation [14,17]. Placental flow reflects both the net perfusion pressure/effective vascular volume/maternal position and the state of myometrial tonus. The presence of contractions is an important observation. At the height of uterine contractions, intervillous flow is essentially occluded due to the multiple physiologic vessel "ligatures" resulting from the interdigitating myometrial fibers. The physiology of uterine blood flow is also of importance. Under normal physiologic conditions, the uteroplacental vascular bed is usually maximally dilated, and autoregulation of flow is minimal or absent. Thus, perfusion highly depends on the effectiveness of the mother's cardiovascular system to provide blood under pressure to the uterus. Important strategies to maintain good placental flow include use of lateral recumbency positioning, rapid and generous fluid loading, Trendelenburg position and, when required, vasopressors. As related issues, it is also extremely important to avoid hyper- or hypocarbia, both of which can have adverse effects on uteroplacental physiology [17].

Based on these concepts, the important principles for pregnant women undergoing surgery include

- Maintenance of a stable intrauterine environment (oxygenation and perfusion)
- Recognition of special features of the physiology of pregnant women (vascular volume, cardiac function, placental circulation, etc.) [5,10,11]
- Attention to the effect(s) of medications/anesthetic agents on the fetus
- Acid aspiration prophylaxis should be used [17]
- There should be clinical attention to the physical aspects of maternal positioning that potentially affect uteroplacental perfusion (lateral recumbency, Trendelenburg position).
- For women facing long procedures and bed rest, prophylactic anticoagulation should be considered. Intraoperative and perioperative compression boots are recommended for all cases.

There is essentially no difference in outcome when procedures performed under laparoscopy are compared with those performed in the usual manner [2,13]. An important retrospective study from the

Swedish Health Registry compared 2,233 laparoscopic with 2,491 standard laparotomy cases. There were no statistically significant differences in outcomes between the groups for a series of major obstetric parameters (birth weight, gestational age, growth disturbances, stillbirths and neonatal deaths, and congenital malformations). An equal distribution of early deliveries (<37 wks) and fetal weights of <2,500 grams were noted for both groups. Thus, despite theoretic risks, laparoscopy in experienced hands has outcomes similar to regular transabdominal surgery. In terms of general recommendations, laparoscopy should be deferred to the second trimester, an open technique for abdominal entry should be used, low intraabdominal pressures are appropriate (<12 mmHg), and intermittent pneumatic boots should be employed on the mother [17].

## REFERENCES

1. Grimes DA, Nanda K. Magnesium sulfate tocolysis: Time to quit. *Obstet Gynecol* 2006;108:986-9.
2. Melnick DM, Wahl WL, Dalton VK. Management of general surgical problems in the pregnant patient. *Am J Surg* 2004;187:170-80.
3. Cohen-Kerem R, Railton C, Oren D, Lishner M, Koren G. Pregnancy outcome following non-obstetric surgical intervention. *Am J Surg* 2005;190:467-73.
4. Goodman S. Anesthesia for nonobstetric surgery in the pregnant patient. *Semin Perinatol* 2002 Apr; 26 (2):136-45.
5. Barron WM. Medical evaluation of the pregnant patient requiring nonobstetric surgery. *Clin Perinatol* 1985;12(3):481-96.
6. Romanoski S. Management of the special needs of the pregnant surgical patient. *Nurs Clin North Am* 2006; 41:299-311.
7. Inturrisi M. Perioperative assessment of fetal heart rate and uterine activity. *JOGNN* May/June 2000; 29:331-36.
8. Kendrick J M, Woodward C B, Cross SB. Surveyed use of fetal and uterine monitoring during maternal surgery. *AORN J* 1995 Sept;62(3):386-92.
9. Liu P, Warren TM, Ostheimer GW, Weiss JB, Liu LMP. Foetal monitoring in parturients undergoing surgery unrelated to pregnancy. *Can Anaesth Soc J* 1995;32(5):525-32.

10. Kuezkowski KM. Nonobstetric surgery during pregnancy: What are the risks of anesthesia? *Obstet Gynecol Surv* 2003;59(1):52–6.
11. Barron WM. The pregnant surgical patient: Medical evaluation and management. *Ann Intern Med* 1984 Nov;101(5):683–91.
12. Gianopoulos JG. Establishing the criterion for anesthesia and other precautions for surgery during pregnancy. *Surg Clin North Am* 1995 Feb;75(1):33–45.
13. Reedy MB, Kallen B, Kuehl TJ. Laparoscopy during pregnancy: A study of five fetal outcome parameters with use of the Swedish Health Registry. *Am J Obstet Gynecol* 1997;177:673–9.
14. Littleford J. Effects on the fetus and newborn of maternal analgesia and anesthesia: A review. *Can J Anesth* 2004; 51[6]: 586–609.
15. Horrigan T, Villareal R, Weinstein L. Are obstetric personnel required for intraoperative fetal monitoring during nonobstetric surgery. *J Perinat* 1999;19(2):124–6.
16. Balki M, Manninen PH. Craniotomy for suprasellar meningioma in a 28-week pregnant woman without fetal heart rate monitoring. *Can J Anesth* 2004; 51(6):573–6.
17. van de Velde M, De Buck F. Anesthesia for non-obstetric surgery in the pregnant patient. *Minerva Anesthesiol* 2007;73:235–40.
18. Varasseur C, Foran A, Murphy J. Concensus statement on the borderlands of neonatal viability: From uncertainty to grey areas. *Ir Med J* 2007 Sept;100(8):561–4.
19. Immer-Bansi A, Immer FF, Hele S, Sporri S, Petersen-Felix S. Unnecessary emergency caesarean section due to silent CTG during anaesthesia? *Br J Anaesth* 2001;87:791–3.

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