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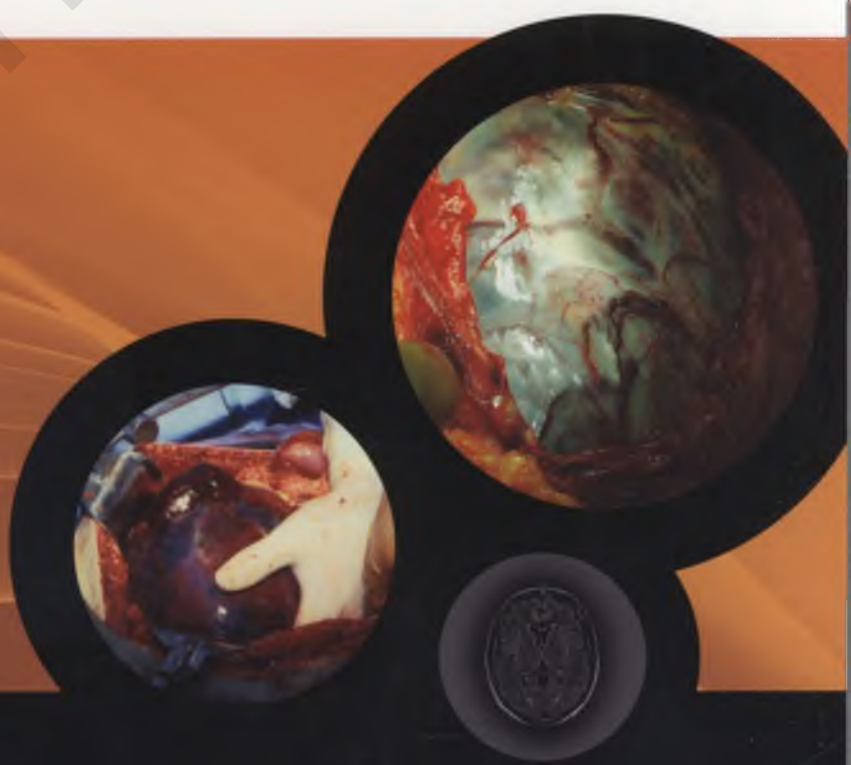
Management of Acute Obstetric Emergencies

Baha M. Sibai



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Mickey Karram, MD
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Baha M. Sibai, MD
Professor
Department of Obstetrics and Gynecology
University of Cincinnati College of Medicine
Cincinnati, Ohio

Illustrated by **Joe Chovan, Milford, Ohio**

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1600 John F. Kennedy Blvd.
Ste 1800
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Contributors

Baha M. Sibai, MD
Professor
Department of Obstetrics and Gynecology
University of Cincinnati College of Medicine
Cincinnati, Ohio
Epidemiology of Acute Obstetric Emergencies; Acute Changes in Fetal Heart Rate Tracing: When It Becomes an Emergency; Evaluation and Management of Antepartum and Intrapartum Hemorrhage; Evaluation and Management of Postpartum Hemorrhage; Amniotic Fluid Embolism; Management of Severe Sepsis and Septic Shock; Management of Hypertensive Crisis Including Stroke; Management of Eclampsia; Subcapsular Liver Hematoma; Diabetic Ketoacidosis; Thyroid Storm in Pregnancy; Cardiorespiratory Arrest and Cardiorespiratory Resuscitation in Pregnancy; Techniques and Complications of Planned and Emergency Cesarean Section

C. David Adair, MD
Professor
Division of Maternal-Fetal Medicine
Department of Obstetrics and Gynecology
University of Tennessee College of Medicine
Chattanooga, Tennessee
Indications and Techniques for Transcervical and Abdominal Cerclage

John R. Barton, MD
Perinatal Diagnostic Center
Central Baptist Hospital
Lexington, Kentucky
Acute Changes in Fetal Heart Rate Tracing: When It Becomes an Emergency; Management of Severe Sepsis and Septic Shock; Management of Hypertensive Crisis Including Stroke; Subcapsular Liver Hematoma

Annette Bombrys, DO
Perinatal Associates of Southwest Ohio
Kettering Medical Center
Kettering, Ohio
Thyroid Storm in Pregnancy

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Stephen DePasquale, MD
Associate Professor
Division of Gynecologic Oncology
Department of Obstetrics and Gynecology
University of Tennessee College of Medicine
Chattanooga, Tennessee
Indications and Techniques for Transcervical and Abdominal Cerclage

Robert Egerman, MD
Associate Professor
Division of Maternal-Fetal Medicine
Department of Obstetrics and Gynecology
University of Tennessee College of Medicine
Memphis, Tennessee
Pulmonary Embolism in Pregnancy

Michael Karram, MD
Gynecology and Urogynecology
Seven Hills Women's Health Centers
The Christ Hospital
Volunteer Associate Professor of Obstetrics and Gynecology
University of Cincinnati
Cincinnati, OH
Techniques and Complications of Planned and Emergency Cesarean Section

Mickey Karram, MD
Director of Urogynecology
The Christ Hospital
Cincinnati, Ohio
Avoiding and Managing Lower Urinary Tract Injury During Vaginal and Abdominal Deliveries

Ryan Loftin, MD
Maternal-Fetal Medicine Fellow
Department of Obstetrics and Gynecology
University of Cincinnati College of Medicine
Cincinnati, Ohio
Peripartum Cardiomyopathy and Pulmonary Edema

John O'Brien, MD
Perinatal Diagnostic Center
Central Baptist Hospital
Lexington, Kentucky
Indications and Techniques for Transcervical and Abdominal Cerclage

Candice Snyder, MD
Department of Obstetrics and Gynecology
University of Cincinnati College of Medicine
Cincinnati, Ohio
Cardiorespiratory Arrest and Cardiorespiratory Resuscitation in Pregnancy

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James Van Hook, MD
Professor and Director
Maternal-Fetal Medicine Division
Department of Obstetrics and Gynecology
University of Cincinnati College of Medicine
Cincinnati, Ohio
Peripartum Cardiomyopathy and Pulmonary Edema

Katherine Wolfe, DO
Maternal-Fetal Medicine Fellow
Department of Obstetrics and Gynecology
University of Cincinnati College of Medicine
Cincinnati, Ohio
Cardiorespiratory Arrest and Cardiorespiratory Resuscitation in Pregnancy

Lynlee Wolfe, MD
Medical Fellow
Maternal-Fetal Medicine
Department of Reproductive Medicine
UCSD Medical Center
San Diego, California
Indications and Techniques for Transcervical and Abdominal Cerclage

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Preface

Acute medical and surgical emergencies in pregnancy and postpartum are the leading cause of maternal mortality and morbidity worldwide. During the past decade there has been a substantial increase in the incidence of these emergencies. Secondary to the change in demographics of women considering pregnancy as well as a change in obstetric practice, these emergencies are expected to continue to increase. Specifically many women are delaying pregnancy until they are in their 40s. Couple this with an epidemic of obesity as well as rising cesarean section rates (with a tremendous increase in repeat cesarean section) and one can easily understand why these emergencies will continue to commonly occur. Also due to an improvement in medical and surgical care and advances in medical technology, many women with serious preexisting medical and surgical disorders are now surviving to reproductive age and are capable of pregnancy.

Specific emergencies that have resulted from these changes in maternal demographics and obstetric practice include an increased rate of life-threatening hemorrhage (both antepartum and postpartum), cardiovascular complications (pulmonary embolism, edema, cardiomyopathy, amniotic fluid embolism, and cardiorespiratory arrest), severe life-threatening hypertensive emergencies (eclampsia, stroke, liver hemorrhage), sepsis, and septic shock.

Due to an increased likelihood of these life-threatening obstetric emergencies, it is important that obstetricians as well as health care providers in general be prepared to deal with such emergencies.

There are several textbooks on medical complications during pregnancy and critical care obstetrics. However, this book is unique in that it focuses on acute maternal emergencies in labor and delivery, postpartum, emergency room areas, and intensive care.

The goals are to provide a step-by-step approach to the diagnosis and management of these emergencies with emphasis on anticipation and preparation in the form of education and development of protocols. It also emphasizes the need for a multidisciplinary team approach to deal with these emergencies. The format of the book uses case presentation with expert discussion using photographs, illustrations, and algorithms to highlight appropriate management of the various emergencies presented.

For most of the covered topics, video clips are present in the accompanying DVD, in the hope of augmenting the text. The DVD contains numerous PowerPoint presentations, surgical procedures, and instructional videos on a variety of topics. Included is a unique video demonstration of how to appropriately manage maternal cardiac arrest in pregnancy, including techniques of cardiopulmonary resuscitation.

I hope the information contained in this textbook will result in improved pregnancy outcomes for all women worldwide.

Baha M. Sibai, M.D.

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Epidemiology of Acute Obstetric Emergencies

1

Baha M. Sibai M.D.

Video Clips on DVD

1-1 PowerPoint Discussion of Epidemiology of Acute Obstetric Emergencies

The incidence of acute medical and surgical emergencies in pregnancy and postpartum has increased during the past decade, and is expected to continue to increase in the future. This increase has resulted from the change in demographics of women who are pregnant or considering pregnancy (Table 1-1), as well as the change in obstetric practice (Table 1-2). Pregnancies in women 40 years and older (about 3%) are much more common than they were 10 years ago. Indeed, seeing women who are pregnant at age 50 or more is not an infrequent occurrence. This is related to the fact that more women are delaying getting pregnant to a later age (personal choice) or starting a new family (change in paternity). The availability of assisted reproductive technologies also has had an effect. With advanced maternal age there are increased rates of chronic hypertension, obesity, type 2 diabetes mellitus, preeclampsia, placenta previa, and abruptio placentae. In addition, these women are more likely to have multifetal gestation because of the need for assisted reproduction, and more likely to require cesarean delivery. The frequency of multifetal gestation among all pregnancies in the United States is 3.5%.

The percentage of pregnant women who are obese or morbidly obese (20% to 30%) has also increased during the past decade. Obesity is associated with increased incidence of hypertensive disorders of pregnancy, type 2 diabetes mellitus and gestational diabetes, cesarean section, cardiopulmonary complications, anesthetic challenges, and wound infections/sepsis. The percentage of women who are pregnant for the first time is also increasing; these women are at increased risk for all types of hypertensive disorders, and are more likely to have elective induction of labor as well as emergency cesarean section.

A major contributor to medical and surgical emergencies is the increasing number of pregnant women with preexisting serious medical disorders (see Table 1-1). Because of improvements in medical and surgical care and advances in medical technology, pregnancy in women with severe cardiopulmonary disease and end-stage renal disease is more frequent than it was a decade ago.

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Table 1-1 Factors Increasing Incidence of Medical and Surgical Emergencies in Obstetrics

- Change in maternal demographics
- Advanced maternal age ≥40 years
- Obesity (body mass index [BMI] >30 kg/m^2)
- Increased percentage of multifetal gestation (3.5%)
- Increased percentage of nulliparity
- Pregnancy with chronic medical disorders
  - Long-standing chronic hypertension
  - Pregestational diabetes mellitus
  - Complicated cardiac disease
  - Severe renal disease or end-stage disease
  - Severe cystic fibrosis
  - Solid organ transplants
  - Stroke

Table 1-2 Change in Obstetric Practice

- Elective induction of labor
- Elective cesarean section
  - Primary and repeat
- High-order cesarean section (≥3)
  - Placenta previa and abruptio placentae
  - Placenta accreta/percreta
  - Uterine rupture
  - Cesarean hysterectomy
  - Blood transfusion (≥4 units)
- Multifetal gestation
  - Severe preeclampsia
  - Preterm labor, tocolytics
  - Prolonged bed rest
  - Uterine overdistention
  - Invasive procedures
  - Cesarean hysterectomy

The recent changes in obstetric practice have also led to an increased incidence of medical and surgical emergencies. The increased rates of elective cesarean section (primary on maternal request) and repeat cesarean sections have led to increased number of pregnant women with three or more cesarean sections. In addition, the presence of previous cesarean section increases the risks of placenta previa, abruptio placentae, and placenta accreta and percreta. These latter complications are more likely to result in massive blood loss, disseminated intravascular coagulopathy, cesarean hysterectomy, need for ventilatory support, and admission to an intensive care unit (ICU).

Multifetal gestation is associated with increased rates of placental abnormalities, preeclampsia, and preterm labor. In addition, uterine overdistention increases the risks of preterm rupture of membranes, abruptio placentae, and uterine atony. These women require prolonged periods of bed rest, which increases their risk for thromboembolism. Women with multifetal gestation with preterm labor requiring tocolytics and steroids for fetal lung maturity are also at increased risk for pulmonary edema and cardiomyopathy. Moreover, patients with multifetal gestation are more likely to require invasive diagnostic and therapeutic procedures such as cervical cerclage, serial amnioreduction, or fetoplacental surgery, procedures associated with an increased rate of obstetric emergencies.

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Table 1-3 Steps for Successful Outcome for Management of Obstetric Emergencies

- Identify patients at risk (red alert)
  - Antepartum
  - Intrapartum
  - Postpartum
- Develop mandatory policies and procedures
  - Nurses
  - Physicians
- Mandatory training in obstetric emergencies
  - Advanced life support in obstetrics
  - Management of obstetric emergencies
  - Fire drills for infrequent emergencies
- Identify an obstetric emergency response team
- Provide and maintain in labor and delivery/recovery area
  - Adequate staff
  - Adequate equipment and supplies

In view of the above changes in maternal demographics and obstetric practice, it is prudent that all health professionals and obstetric units providing care for such patients be prepared to manage the expected increase in the number and percentage of medical and surgical emergencies in their obstetric population. Some of the steps that need to be taken to ensure patient safety and improve pregnancy outcome are listed in Table 1-3.

These steps should include development of a system to identify and flag all patients who are considered at risk for obstetric emergencies. Once identified, all units should have in place policies and procedures on how to prevent or reduce the risks of these emergencies as well as how to respond to them if they develop. This should consist of a multidisciplinary team that includes nursing staff, anesthesia, senior obstetrician, operating room team, blood bank, and other physicians as needed. These procedures should detail what to do under elective conditions as well as if a patient presents as an emergency.

There are several conditions in obstetrics that are more likely to be associated with emergencies or that may lead to adverse outcome. Thus all units should develop mandatory protocols to address some of these conditions (Table 1-4). This list may be modified according to the specific obstetric unit.

In most obstetric units in the United States, the majority of medical and surgical emergencies are infrequent or rare, unpredictable, and can develop very rapidly. This implies that health care professionals providing care for these patients will have minimal to limited experience in handling these emergencies. Thus it is prudent that all obstetric units develop drills for rehearsal and testing of response and skills of these individuals in the presence of some of these expected emergencies (Table 1-5). This should also include evaluation of the response time as well as effectiveness of the obstetric emergency response team.

Finally, obstetric units providing care for high-risk pregnancies should have a designated area in labor and delivery and/or recovery that is adequately staffed and equipped to handle obstetric emergencies. In addition, they should have well-defined protocols regarding which patients to transfer to other ICUs (medicine or surgery), and how these patients will be promptly transferred as well as who will manage these patients once transferred.

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Table 1-4 Mandatory Protocols to Prevent or Reduce Obstetric Emergencies

- Elective inductions and delivery
  - Accurate dating criteria
  - Cervical Bishop score
  - Induction and ripening agents
  - Availability of staff
- Tocolytic agents
  - When to start and when to stop
  - Methods and intensity of monitoring
  - Drugs to use in women with preexisting conditions
    - Cardiac disease
    - Hyperthyroidism, hypertension
    - Pregestational diabetes
    - Placenta previa, abruptio placentae
    - Pulmonary or urinary tract infections
- Magnesium sulfate for preeclampsia or eclampsia
  - When to start and when to stop
  - Monitoring for signs or symptoms of toxicity
  - Management of toxicity
- Antepartum, intrapartum, and postpartum hemorrhage
- Thromboembolism prophylaxis
  - Antepartum, intrapartum, postpartum
  - Method and duration of prophylaxis
- Pregnant patients seen in emergency department area
  - Initial evaluation
  - Method of fetal monitoring
  - When to send to labor and delivery unit
  - Need for immediate delivery
- Pregnant or postpartum patients in intensive care unit
  - Indications for transfer
  - Physician in charge
  - Methods for fetal monitoring
  - Need for immediate delivery
- Goal-directed sepsis protocol

Table 1-5 Fire Drills to Recognize and Respond to Obstetric Emergencies

- Pulmonary embolism
- Pulmonary edema
- Amniotic fluid embolism
- Eclampsia or hypertensive crisis
- Intrapartum or postpartum hemorrhage
  - Shock
  - Disseminated intravascular coagulopathy
  - Blood components and fluid replacement
  - Medical-surgical interventions
    - Medications
    - Packing
    - Ligation of vessels
    - Cesarean hysterectomy
    - Bladder or ureteral injury
- Severe sepsis/septic shock
- Diabetic ketoacidosis
- Thyroid storm
- Cardiopulmonary arrest

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Suggested Readings

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2

Acute Changes in Fetal Heart Rate Tracing: When It Becomes an Emergency

Baha M. Sibai M.D.
John R. Barton M.D.

Video Clips on DVD

2-1 PowerPoint Discussion of a Variety of Fetal Heart Rate Tracings

Continuous electronic fetal heart rate (FHR) monitoring is widely used to monitor all pregnant women with high-risk medical or obstetric conditions, as well as most pregnant women undergoing labor and delivery. The objectives of FHR monitoring during labor are early detection of changes in FHR baseline and patterns in order to identify certain categories that are predictive of fetal hypoxia and acidosis (Table 2-1). Once these changes are identified, the next step is for the medical provider to decide on which ones require careful observation and which FHR require immediate delivery.

Guidelines have been published by the National Institute of Child Health and Human Development (NICHD) working group for definitions, interpretation, and management recommendations for various categories of FHR tracings. The research group defined three categories as either normal (Category I), indeterminate (Category II), and abnormal (Category III). These definitions are listed in Table 2-2 (Category I) and Table 2-3 (Category III). Category II is defined as any pattern not included in Category I or III. An example of Category I FHR patterns is seen in Figure 2-1, of Category II in Figure 2-2, and of Category III in Figure 2-3. The same group also recommended abolishing the term *hyperstimulation* for uterine activity and suggested using the term *uterine tachysystole* (Table 2-4). The definition of baseline variability is described in Table 2-5. Minimal or absent variability can be due to medications that depress the fetal central nervous system, hypoxia, or acidosis (maternal or fetal). The presence or absence of FHR accelerations was not considered important to define the three categories.

The NICHD criteria define normal FHR baseline as a rate of 100 to 160 bpm. Fetal bradycardia is defined as a baseline of <100 bpm for at least 10 minutes, whereas fetal tachycardia is defined as a baseline of >160 bpm for at least 10 minutes. Fetal tachycardia can be related to various etiologies (Table 2-6). Therefore, management should be individualized based on the etiology, the persistence of the pattern, and response to corrective factors.

It is important to emphasize that a category II FHR pattern will be seen in almost all labors, and most of these are reassuring, whereas in others they can be ominous requiring immediate delivery. Therefore, there are potential pitfalls with the new NICHD classification (Table 2-7).

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Table 2-1 Objectives of Fetal Monitoring in Labor

- Identify fetal heart rate patterns associated with asphyxia
  - Ischemia (▼) tissue perfusion
  - Hypoxemia (▼ $[O_2]$ in blood)
  - Hypoxia (▼ $[O_2]$ in tissue) → acidosis
  - Asphyxia (hypoxia and metabolic acidosis)
    - Organ injury
    - Cerebral insufficiency
    - Cerebral palsy
    - Fetal death
- Allow obstetric interventions to avert adverse outcome
  - Medical
  - Instrumental vaginal delivery
  - Cesarean section

Table 2-2 Category I FHR Pattern (Normal)

- Baseline rate, 100-160 bpm
- Moderate variability (6-25 bpm)
- Absent variable and late decelerations
- Absence or presence of early accelerations/decelerations

Table 2-3 Category III FHR Pattern (Abnormal)

- Definition
  - Absent variability (zero) plus either
    - Recurrent late decelerations (20 min)
    - Recurrent variable decelerations (20 min)
    - Bradycardia (<100 bpm for ≥10 min)
  - Sinusoidal pattern
    - Smooth, undulating, sine wave
    - Cycle frequency 3-5/min
    - Persists ≥20 minutes
- Treatment
  - Immediate delivery

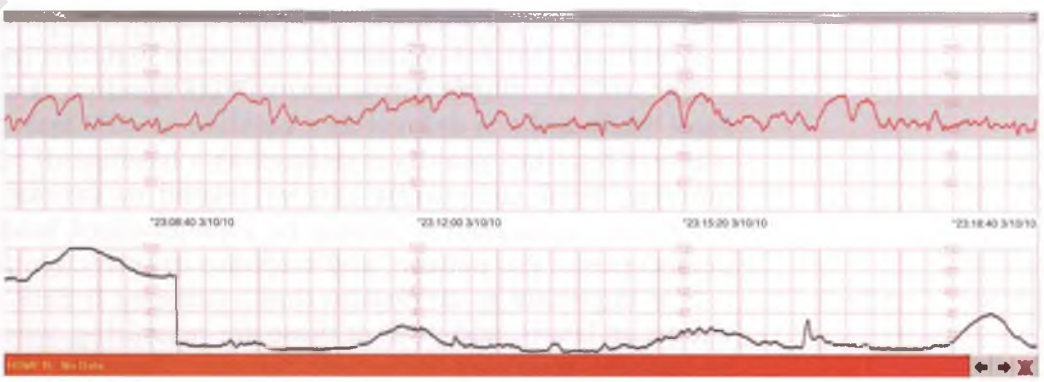

Figure 2-1 Fetal heart rate tracing showing normal baseline and moderate variability (Category I).

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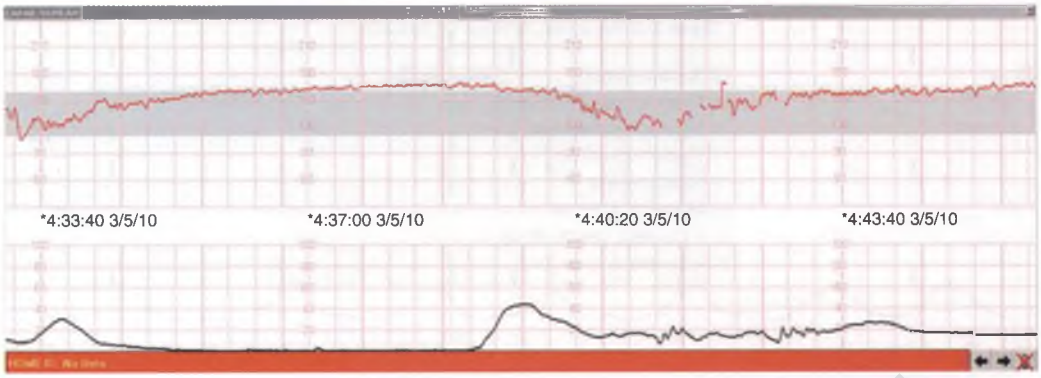

Figure 2-2 Fetal heart rate tracing revealing moderate variability and late deceleration (Category II).

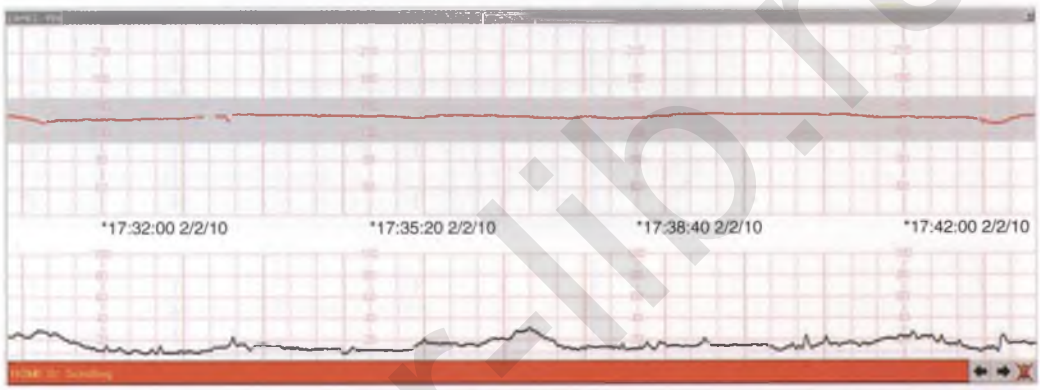

Figure 2-3 Absent variability and recurrent late decelerations (Category III).

Table 2-4 Uterine Tachysystole

- Definition
  - More than 5 contractions in 10 minutes
  - Averaged over 30-minute window
  - With/without FHR decelerations
- Treatment
  - Discontinue oxytocin or prostaglandins
  - Give oxygen by mask
  - Give terbutaline 0.25 mg IV
  - Consider delivery if associated with abnormal FHR pattern and no response to therapy

Table 2-5 Baseline FHR Variability

Defined as fluctuations that are irregular in amplitude and frequency (visually quantitated as the amplitude of the peak-to-trough in bpm).

Amplitude Range	Classification
• Undetectable	• Absent
• Undetectable to <5 bpm	• Minimal
• 6 to 25 bpm	• Moderate
• More than 25 bpm	• Marked

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Table 2-6 Etiologies of Fetal Tachycardia

- Betamimetics
- Fever/chorioamnionitis
- Fetal stress/hypoxia
- Hyperthyroidism
- Fetal anemia
- Maternal acidosis
  - Diabetic ketoacidosis
  - Severe dehydration

Table 2-7 Potential Problems with the New NICHD Classification

- Recurrent variable or late decelerations with minimal variability is Category II
- Bradycardia and minimal variability is Category II
  - Abruption
  - Prolapsed cord
  - Uterine rupture
- Tachycardia and minimal variability is Category II
  - Vasa previa with fetal bleeding
  - Diabetic ketoacidosis
- Any variable is Category II
  - Virtually every woman in labor
  - Contraindication to oxytocin?

Case 1: Cord Compression

A 24-year-old G1 presented in labor at 40 weeks' gestation, 70% effacement, with vertex at S-2 station. FHR tracing revealed normal baseline, moderate variability, and presence of accelerations (see Fig. 2-1). This is considered a Category I tracing. The patient was observed over several hours with minimal cervical dilation. Oxytocin was started for labor stimulation. After spontaneous rupture of membranes, the rate of oxytocin infusion was increased every 20 minutes. About 2 hours later, FHR and uterine contraction tracings revealed uterine tachysystole with repetitive variable decelerations and moderate baseline variability (Fig. 2-4).

Discussion

This FHR tracing is consistent with a Category II pattern in association with uterine tachysystole. Appropriate management should include stopping the oxytocin to relieve tachysystole and pelvic examination to check for the presence of occult cord prolapse. The pathophysiology and management of variable decelerations are listed in Table 2-8. In this case, pelvic examination revealed no cord between the presenting part and maternal pelvic structures. In addition, oxytocin was discontinued, and maternal position was changed to the side to relieve any potential cord compression. The potential causes of cord compression during labor include oligohydramnios, tight cord around the neck, shoulder or body cord prolapse, or placental abruption. In patients attempting a trial of vaginal delivery after previous cesarean section, repetitive variable decelerations can be an early sign of uterine rupture with the cord protruding through the window in the scar.

In this patient, uterine tachysystole resolved after stopping the oxytocin, however, repetitive variables continued. Consequently, amnioinfusion was started in an attempt to correct the repetitive variable decelerations with the assumption that they were related to reduced amniotic fluid. Following amnioinfusion, the variable decelerations improved, and 2 hours later pelvic examination revealed the cervix to be complete with vertex at zero station. The patient was instructed to start pushing. During pushing, variable decelerations resumed and became deeper with a late component in association with minimal variability (Fig. 2-5). The patient was asked to stop pushing and oxygen was administered by face mask at 10 L/min. Despite this management, the FHR pattern did not improve, and the decision was made for emergency cesarean section. At time of delivery, there was a loop of cord around the fetal shoulder. The infant had an Apgar score of 1 and 6 at 1 and 5 minutes, respectively. Cord arterial blood gas revealed a pH of 7.08, Pco_2 of 80 mm Hg, and base excess of –8.6 mEq/L. The arterial gas results were consistent with respiratory acidosis.

This case illustrates the risk of cord compression during descent of the fetal head in the second stage of labor. The changes in FHR pattern and their timely detection led to prompt delivery prior to the development of significant metabolic acidosis. It was also appropriate not to wait until the FHR pattern changed to Category III to proceed with cesarean delivery.

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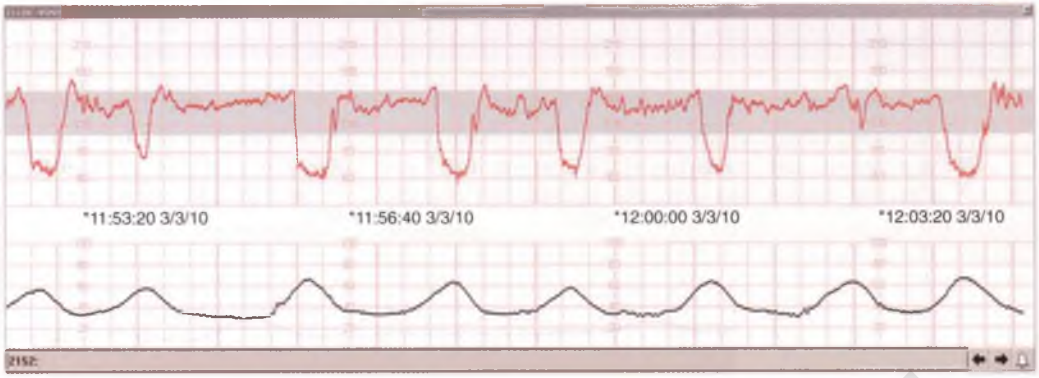

Figure 2-4 Tachysystole; repetitive variables (severe by old criteria); moderate variability.

Table 2-8 Pathophysiology and Management of Variable Decelerations

- Pathophysiology
  - First vessel compressed: vein
    - ▼ Venous return
    - ▼ Cardiac output
  - Reflex ▲ FHR to maintain blood pressure (shoulder)
  - Next vessels compressed: arteries
    - Obstruct low-resistance placental vascular bed
    - Slowing of FHR to maintain blood pressure (drop in rate from baseline)
  - Remove pressure, process is reversed
    - Increase in FHR (V- or U-shaped deceleration)
    - Overshoot (increase in FHR above baseline)
- Management
  - Stop oxytocin if in use
  - Vaginal exam
  - Change in maternal position
  - If pushing, stop and reassess
  - Amnioinfusion if oligohydramnios

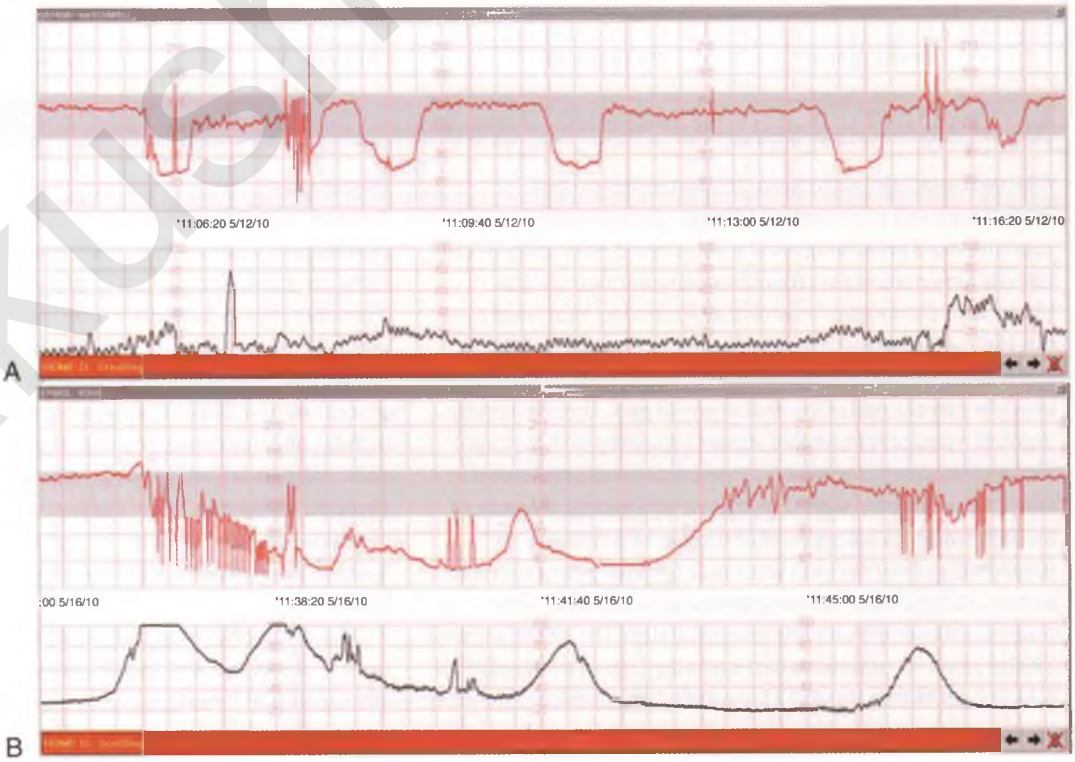

Figure 2-5 **A,** Fetal heart rate tracing revealing variable decelerations with minimal variability during maternal pushing. **B,** Fetal heart rate tracing revealing variables with prolonged decelerations during pushing.

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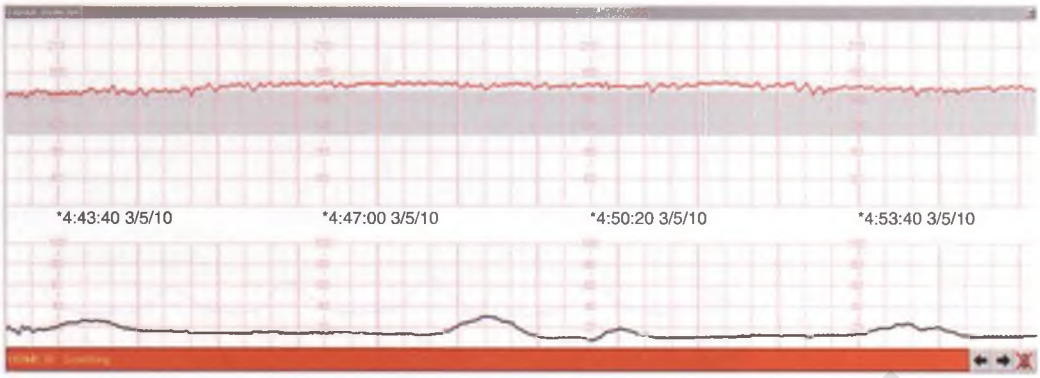

Figure 2-6 Fetal tachycardia; minimal variability; dysfunctional labor.

Case 2: Chorioamnionitis

A 26-year-old G2 presents at 36 weeks' gestation because of rupture of membranes 10 hours prior to admission. Cervical evaluation revealed cervix 2 cm dilated, 50% effaced, and vertex presentation. Because she had no uterine contractions for 2 hours, oxytocin was initiated for induction of labor. After 6 hours of oxytocin use, her contractions were irregular and mild. The patient was noted to have a temperature of 101° F and a pulse of 110 bpm. The FHR and uterine contraction patterns revealed fetal tachycardia with minimal variability with dysfunctional labor pattern (Fig. 2-6). A diagnosis of chorioamnionitis was made and the patient received intravenous clindamycin and gentamicin, and rectal Tylenol for fever. Oxytocin was continued and 2 hours later maternal fever subsided, but FHR pattern did not change. Pelvic examination revealed no change in cervical dilation, and the decision was made for cesarean delivery. The infant had a weight of 2860 g with Apgar scores of 4 and 8 at 1 and 5 minutes. Arterial cord gases were normal.

Discussion

This patient developed acute chorioamnionitis during labor. As a result she developed a fever and there was fetal tachycardia. In addition, patients with chorioamnionitis are at increased risk for dysfunctional labor patterns and for postpartum hemorrhage. Management includes the use of broad-spectrum antibiotic coverage, reduction of maternal temperature, and timely delivery. In this case, fetal tachycardia and minimal variability continued despite treatment of maternal fever. In addition, there was inadequate uterine activity despite increased doses of oxytocin over an 8-hour period, and no cervical change. Because the patient was remote from delivery, appropriate management required timely delivery by cesarean section because of the concern about possible fetal infection and increased oxygen requirements by the fetus. The FHR pattern is Category II, but does not require emergency delivery.

Case 3: Abruptio Placentae

A 38-year-old G3P0 with chronic hypertension on oral methyldopa was admitted at 33 weeks' gestation with superimposed preeclampsia. She was started on corticosteroids for fetal lung maturity and continued on oral methyldopa. Ultrasound examination revealed an estimated fetal weight at the 15th percentile with reduced amniotic fluid (AFI = 8.4 cm), and slightly elevated umbilical artery Doppler ratio. Three days later she complained of lower abdominal pain. Uterine activity and FHR monitoring revealed absent variability with recurrent late decelerations (see Fig. 2-3). Pelvic examination revealed a cervix to be fingertip with no evidence of bleeding. Because of the FHR pattern, a diagnosis of abruptio placentae was suspected and the decision was made for emergency cesarean section. At the time of delivery there was evidence of 25% placental abruption with retroplacental clots. The infant weighed 1400 g (small for gestational age), and cord gases revealed pH of 7.14, Pco_2 of 46 mm Hg, and a base deficit of −12 mEq/L. The cord gases were consistent with metabolic acidosis.

Discussion

Patients with chronic hypertension and superimposed preeclampsia are at increased risk of fetal growth restriction (20% to 25%), and abruptio placentae (2% to 3%). Therefore, these patients require close observation of fetal growth and well-being. Because of the findings on ultrasound examination and potential risk of abruptio placentae, the patient received daily monitoring of uterine activity and nonstress testing. The FHR pattern was consistent with Category III, and because of suspected abruptio placentae, such pattern requires emergency cesarean delivery. A delay in delivery leads to progressive hypoxia and acidosis with ultimate fetal death or neonatal injury.

The pathophysiology of recurrent late decelerations is usually uteroplacental insufficiency. This could be transient after maternal hypotension (supine position or epidural administration) or as a result of excessive use of oxytocin or

Case 3: Abruptio Placentae—cont'd

prostaglandins with associated prolonged tachysystole. In such cases, recurrent decelerations will resolve after correction of the etiology, and labor can then be continued for possible vaginal delivery. In contrast, in patients with recurrent decelerations secondary to chronic uteroplacental insufficiency (fetal growth restriction or post-term pregnancy) or acute insufficiency (abruptio placentae or ruptured uterine scar), management requires prompt delivery by cesarean section unless vaginal delivery is imminent.

Case 4: Tachysystole

Oxytocin is commonly used for augmentation and/or induction of labor. The use of excessive doses of oxytocin may lead to tachysystole with or without FHR changes (Fig. 2-7). If tachysystole develops, the rate of oxytocin should be reduced or discontinued. Oxytocin use should be discontinued in the presence of nonreassuring FHR tracing (Category III). If fetal tachycardia or recurrent variable decelerations continue with the use of oxytocin (Fig. 2-8), the drug should be discontinued, the patient should be given oxygen by mask, and fetal tracing reassessed.

In summary, the NICHD criteria for interpretation of FHR tracings are very useful in evaluating patients in labor. The interpretation of these criteria and their management should be used in conjunction with other clinical criteria. Management of the various FHR categories should be individualized.

Figure 2-7 Uterine tachysystole with fetal tachycardia.

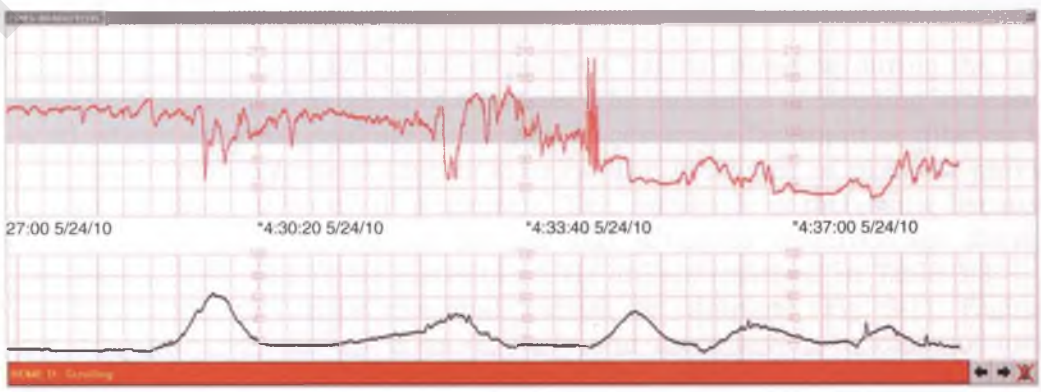

Figure 2-8 Fetal heart rate with prolonged deceleration.

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Suggested Readings

ACOG Practice Bulletin: Intrapartum fetal heart rate monitoring: nomenclature interpretation, and general management principles. Number 106, July 2009.

Blix E, Sviggum O, Koss KS: Inter-observer variation in assessment of 845 labour admission tests: comparisons between midwives and obstetricians in the clinical setting and two experts. BJOG 2003;110:1–5.

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Macones GA, Hawkins GDV, Spong CY, et al: The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring. Update on definitions, interpretation and research guidelines. Obstet Gynecol 2008;112:661–666.

Parer JT, Ikeda T, King TL: The 2008 National Institute of Child Health and Human development report on fetal heart rate monitoring. Obstet Gynecol 2009;114:136–138.

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3

Evaluation and Management of Antepartum and Intrapartum Hemorrhage

Baha M. Sibai M.D.

Video Clips on DVD

3-1 PowerPoint Discussion of How to Evaluate and Manage Intrapartum and Antepartum Hemorrhage

Severe obstetric hemorrhage is an important cause of maternal and perinatal mortality and morbidity worldwide. The incidence of obstetric hemorrhage is increasing because of recent changes in obstetric demographics. Obstetric hemorrhage can develop before delivery, in labor, or immediately postpartum. A list of important causes of obstetric hemorrhage is summarized in Table 3-1. In order to be prepared to deal with obstetric hemorrhage, it is important that all obstetric units develop mechanisms and action plans that identify patients at risk by color coding of medical records during the antepartum period, in labor, and postpartum (Table 3-2). This chapter discusses evaluation and management of obstetric hemorrhage associated with placenta previa, abruptio placentae, uterine rupture, and placenta accreta/percreta.

Placenta Previa

Placenta previa is a major cause of painless vaginal bleeding during the late second trimester and in the third trimester. The incidence of bleeding increases with advanced gestational age secondary to cervical effacement or dilation in association with increased uterine activity. The incidence of placenta previa is approximately 0.5% of all gestations. This incidence is increasing because of increased rates of cesarean section and advanced maternal age during pregnancy. Risk factors for placenta previa are summarized in Table 3-3.

Placenta previa can be marginal, partial, or complete (Fig. 3-1). Vaginal bleeding prior to delivery is more likely and more severe in cases of total previa.

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Table 3-1 Causes of Obstetric Hemorrhage

Antepartum/Intrapartum

• Abnormal placentation
  • Placenta previa
  • Severe abruptio
  • Accreta/increta/percreta
• Uterine rupture
• Existing coagulopathies

Postpartum

• Severe cervicovaginal, vulvar, and perineal lacerations
  • Operative vaginal delivery
  • Precipitate labor
• Retained products of conception
• Uterine atony
• Uterine prolapse
• Vascular lacerations during cesarean section

Table 3-2 Action Plans to Identify Patients at Risk for Obstetric Hemorrhage Using Color Codes

Antepartum
◦ Code red • Placenta accreta/percreta • Complete previa • Abruptio plus dead fetus • Preexisting coagulopathy ◦ Code orange • Multiple cesarean section ≥3 • Preexisting vertical/classical scar • Uterine over distension • Preexisting rare antibodies • Severe anemia • Thrombocytopenia • Jehovah's Witness ◦ Code yellow • Prophylactic anticoagulation • Partial previa with bleeding • Prophylactic tocolytics • Morbid obesity
Intrapartum
◦ Code red • Acute fatty liver of pregnancy/HELLP syndrome • Previous cesarean section and placenta previa with bleeding • Uterine rupture • Abruptio with bleeding • Previous severe postpartum hemorrhage ◦ Code orange • Twins in labor • Abnormal presentation • Induction of labor • Prolonged, protracted, or precipitate labor • Preterm labor/preeclampsia on magnesium sulfate or nifedipine • VBAC/obesity • Chorioamnionitis

Table 3-3 Risk Factors for Placentae Previa

• Prior history of previa • Prior cesarean section • Multiparity • Advanced maternal age • Previous suction curettage for abortion • Smoking

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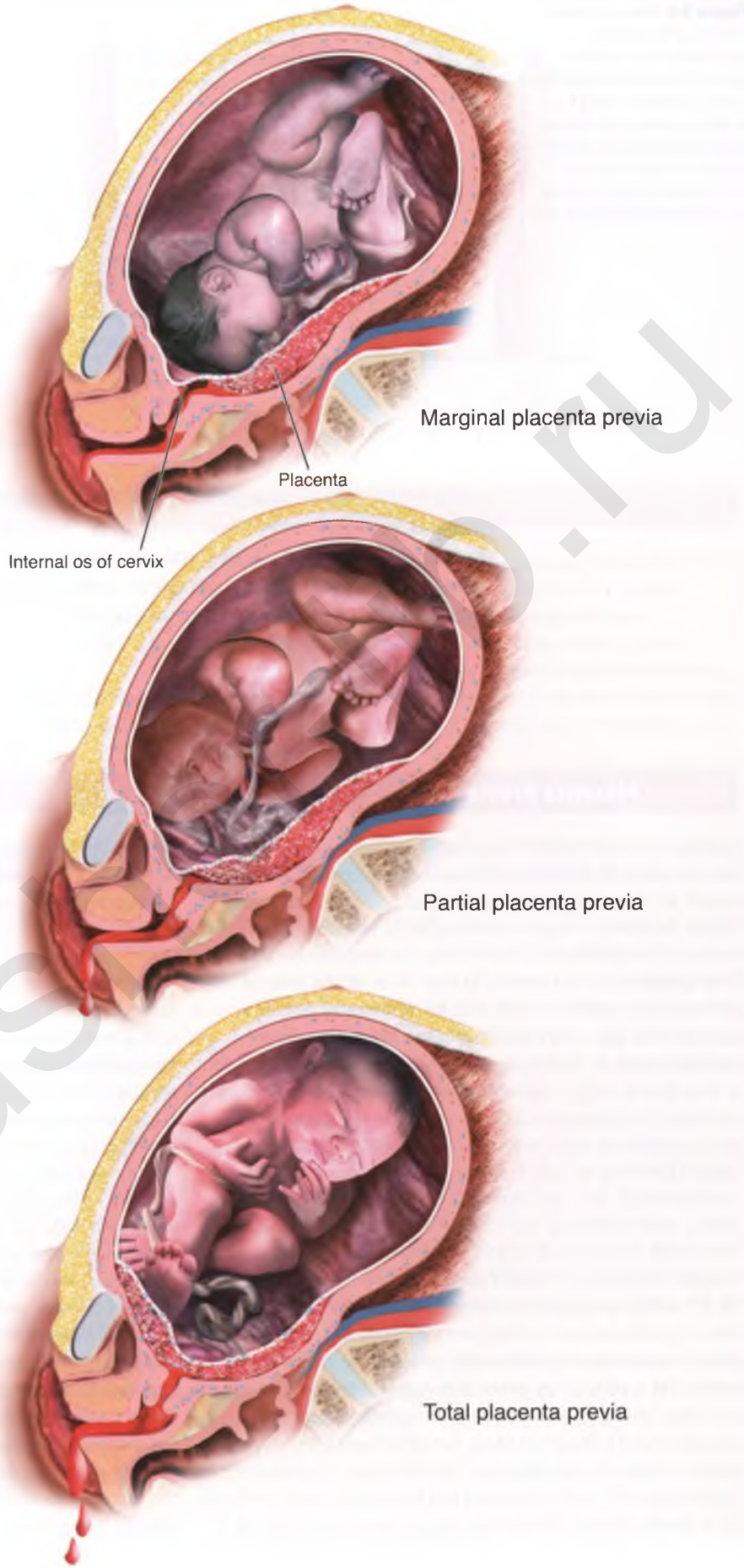

Figure 3-1 Different types of placenta previa.

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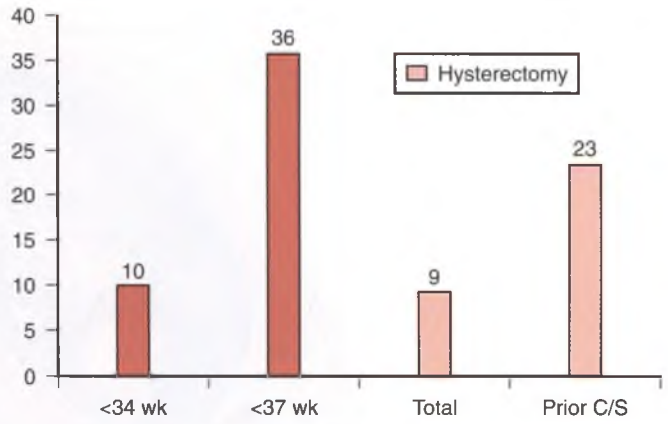

Figure 3-2 Rates of preterm delivery and cesarean hysterectomy in placenta previa (n = 346). (Adapted from Choi S-J, Song SU, Jung K-L, et al: Antepartum risk factors associated with peripartum cesarean hysterectomy in women with placenta previa. Am J Perinatol 2008;25:37–42.)

Placenta previa is associated with increased maternal and perinatal morbidities particularly in those with total previa, previous cesarean section, and multifetal gestation. Maternal morbidity includes the need for blood transfusions and hysterectomy, whereas both perinatal mortality and morbidities are increased secondary to premature delivery (Figs. 3-2 and 3-3).

Case 1: Placenta Previa

A 38-year-old G2P2 with two previous cesarean sections (one was for arrest of dilation at term and the second was elective repeat at 39 weeks' gestation). She had normal prenatal care except for painless vaginal bleeding at 30 weeks' gestation, requiring hospitalization. Ultrasound examination revealed total placenta previa covering at least 4 cm of the internal cervical os. In addition, there was no evidence of placenta accreta. Vital signs were stable, and the patient received corticosteroids for fetal lung maturation. She was observed for 3 days during which vaginal bleeding stopped; her hematocrit at time of discharge was 29%. At 33 4/7 weeks' gestation, she was hospitalized again with a second episode of painless vaginal bleeding for which she received another course of corticosteroids. She was observed in the hospital for 4 days during which bleeding subsided. She was again discharged home with a hematocrit of 26%. The plan was for elective cesarean section at 37 weeks' gestation. Two weeks later (at 36 1/7 weeks' gestation) she presented with profuse vaginal bleeding with uterine contractions for which she required blood transfusions and emergency cesarean section with delivery of a 2600-g live infant with Apgar scores of 3 and 6 at 1 and 5 minutes, respectively. During cesarean section she continued to have bleeding from the lower uterine segment with ultimate cesarean hysterectomy. She required transfusions of 5 units of packed red blood cells and 2 units of fresh frozen plasma. Postoperatively, she was monitored for 2 days in the intensive care unit, and was discharged home with her infant 7 days after admission. The pathology report of the hysterectomy specimen revealed no evidence of placenta accreta.

Discussion

Early detection of total placenta previa, particularly in patients with previous cesarean section is critical because it allows for interventions that will lessen the risks of adverse maternal and fetal outcomes as was evident in this case. Common errors in management include failure to appreciate the clinical significance of repetitive vaginal bleeding, the significant drop in maternal hematocrit with the need for blood transfusions, and failure for continued hospitalization for close monitoring and timely delivery. This patient with previous cesarean section with total and central previa (covering 4 cm of internal os) was at very high risk for antepartum bleeding, preterm delivery, and the need for hysterectomy (see Figs. 3-2 and 3-3). As expected, she had two episodes of painless vaginal bleeding prior to 34 weeks' gestation. It was appropriate to administer corticosteroids because of the concern about the need for preterm delivery. Of concern was the fact that she was discharged home after the second episode of vaginal bleeding with a hematocrit of 26%. During the second hospitalization, the patient should have received 2 units of packed red blood cells (PRBCs) to keep her

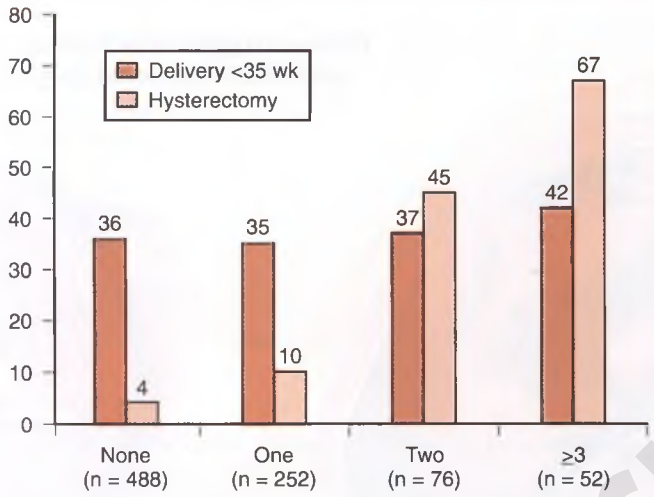

Figure 3-3 Gestational age at delivery and need for hysterectomy in placenta previa. (Adapted from Grobman WA, Gersnoviez R, Landon MB, et al: Pregnancy outcomes for women with placenta previa in relation to number of prior cesarean deliveries. Obstet Gynecol 2007;110:1249–1255.)

Case 1: Placenta Previa—cont'd

hematocrit at least 30%. In addition, she should have remained hospitalized for close monitoring of vaginal bleeding and uterine activity for timely delivery. During hospitalization, the patient should have been counseled about the need for preterm delivery and potential hysterectomy. In addition, the blood bank should have been notified to have 2 to 4 units of crossmatched PRBCs available in case she required urgent delivery.

Because the physician did not appreciate the significance of repetitive vaginal bleeding in a patient with previous cesarean section and central previa, the patient was discharged home with a plan for elective section at term, which led to a life-threatening emergency to both fetus and mother. This case highlights the need to develop protocols for surveillance and management of pregnant women with total placenta previa.

Abruptio Placentae

Abruptio placentae is defined as premature separation of the placenta after 20 weeks' gestation. Placental separation can be clinically revealed (Fig. 3-4A and B), in which case the patient presents with painful vaginal bleeding with or without contractions or it could be concealed (occult) separation (see Fig. 3-4C and D), in which case blood accumulates behind the placenta, and there is no vaginal bleeding. Placental abruption is revealed in approximately 75% of cases and concealed in 25%. The abruption could also be partial or complete. The exact incidence of abruptio is unknown; however, there are several risk factors for abruptio placentae (Table 3-4). In general, the majority of patients with abruptio present with moderate to severe pain that can be intermittent or constant. In case of anterior placenta, the pain is usually abdominal (mid to fundal), whereas with posterior placental location, the pain is usually in the back. In moderate to severe placental abruption, uterine activity and fetal heart rate monitoring demonstrate one or more of the findings listed in Table 3-5.

Pregnancies complicated by moderate to severe abruptio placentae are associated with substantial maternal and perinatal complications (Table 3-6). These complications are more frequent in those with placental separation affecting more than 50% of the placenta, and in those with concealed abruptio. In these cases, the blood loss can be at least 1000 mL at the time of presentation. Therefore, moderate to severe placental abruption should be considered a life-threatening emergency to both mother and fetus (Table 3-7).

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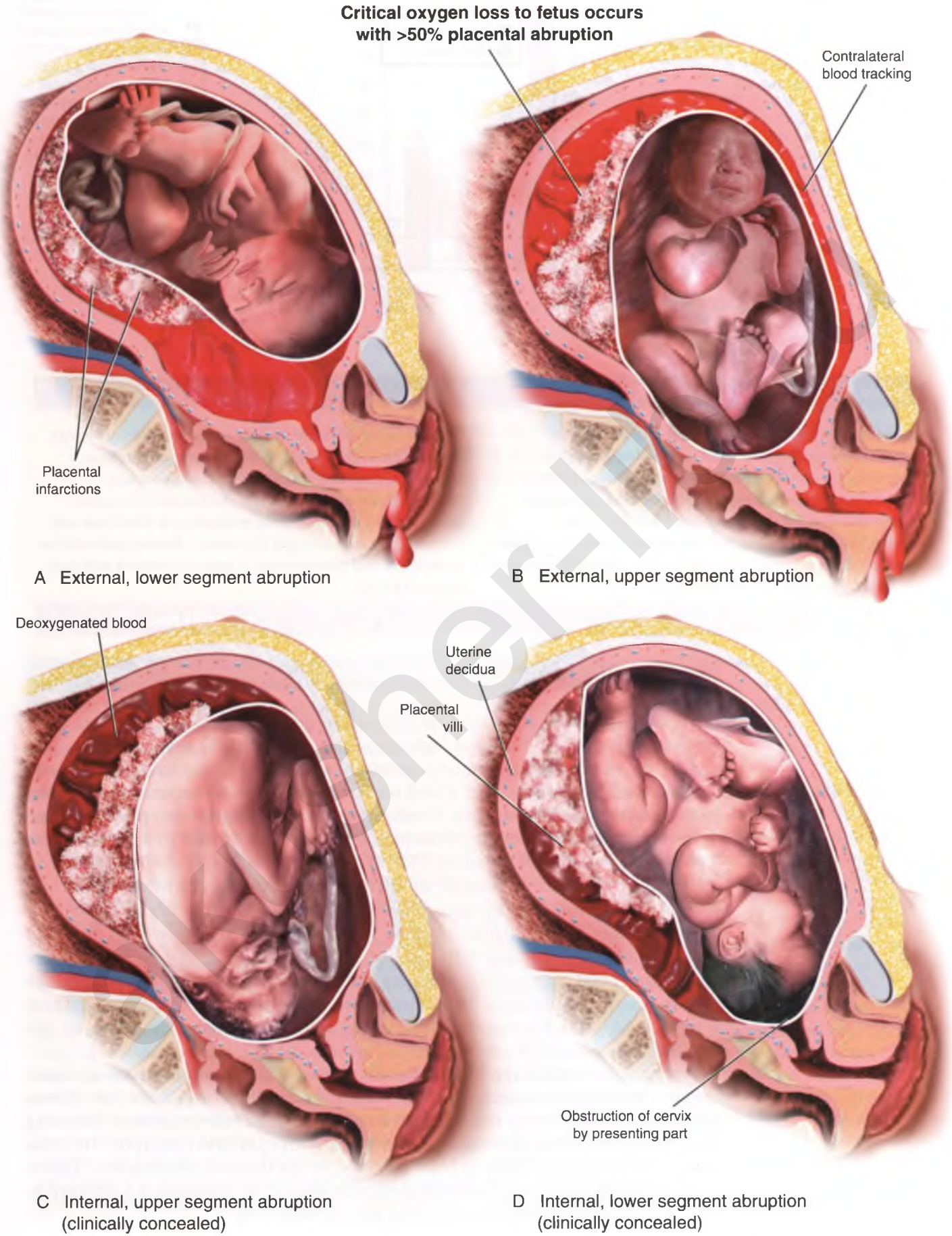

Figure 3-4 **A** through **D,** Different types of placenta separation (both occult and revealed).

Table 3-4 Risk Factors for Abruptio Placentae

- Previous abruptio placentae
- Preterm premature rupture of membranes
- Severe hypertension/preeclampsia
- Substance abuse, smoking
- Major abdominal trauma
- Uterine rupture
- Advanced maternal age
- Multifetal gestation
- Two or more previous cesarean sections
- Oligohydramnios

Table 3-5 Uterine Activity and Fetal Heart Rate Findings in Moderate to Severe Abruption

Uterine Activity	Fetal Heart Rate
• High frequency–low amplitude	• Absent accelerations
• Increased resting tone	• Decreased variability
• Uterine irritability	• Variable/late decelerations
• Tetanic contractions	• Bradycardia

Table 3-6 Peripartum Complications of Moderate to Severe Abruptio Placentae

- Fetal
  - Nonreassuring tracing
  - Hypoxia and acidosis
  - Death
- Neonatal brain injury/death
- Maternal
  - Hypovolemic shock (intrapartum/postpartum)
  - DIC
  - Acute renal failure
  - Acute respiratory distress syndrome
  - Amniotic fluid embolism
  - Death

Table 3-7 Risk Factors for Adverse Pregnancy Outcome in Abruptio Placentae

- Previous abruptio associated with fetal death
- Current abruptio in association with fetal death
- Concealed abruptio or >50% separation
- Onset of signs and symptoms at <34 weeks
- Uterine rupture
- Severe hypertension/preeclampsia, acute cocaine overdose
- Delivery >20 minutes after nonreassuring FHR tracing

Case 2: Abruptio Placentae

The patient is a 22-year-old G2P0 with normal prenatal care except for history of smoking cigarettes (one pack/day). At 34 weeks' gestation she presented to triage area because of abdominal pain and pink vaginal discharge. Physical examination revealed a blood pressure of 115/70 mm Hg, and pulse of 94 to 98. Vaginal examination revealed cervix 2 cm dilated, 40% effaced, vertex presentation at −2 station with no evidence of vaginal bleeding. Uterine activity monitoring revealed irritable contractions, and the fetal heart rate (FHR) tracing demonstrated a normal baseline with moderate variability, and occasional decelerations (Fig. 3-5A). She was given a fluid bolus for hydration and discharged home after 1

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Case 2: Abruptio Placentae—cont'd

hour of observation despite the fact that she was still having uterine irritability and occasional decelerations. Four hours later she presented back to triage area because of continued abdominal pain and mild vaginal bleeding. On arrival, her BP was 115/76 mm Hg, and the heart rate was 106 to 115 beats per minute (bpm).

A cervical exam revealed cervix 3 to 4 cm dilated, 70% effaced with mild vaginal bleeding. There was again uterine irritability with recurrent decelerations (see Fig. 3-5B) for which she had position change and maternal oxygen (10 L/min). Approximately 50 minutes later, she was transferred to the labor and delivery area for close monitoring. Cervical exam was 4 cm dilated, 70% effaced, with vertex at zero station. Internal fetal scalp electrode was placed and the amniotic fluid was blood tinged. Subsequent monitoring revealed uterine tachysystole with repetitive decelerations (see Fig. 3-5C and D). Abruptio placentae was suspected, and blood was sent for complete blood count (CBC), coagulation studies, and for crossmatch. Because of progressive cervical change and concern about coagulopathy, the plan was conservative. The FHR tracing was getting worse with continued decelerations and absent beat-to-beat variability with drop of baseline to a rate of 120 (see Fig. 3-5E and F) and finally to 100 when emergency cesarean was done with delivery of a 2190-g infant with Apgar scores of 0, 0, and 1 at 1, 5, and 10 minutes, respectively (see Fig. 3-5G). Cord arterial gases revealed a pH of 6.80, Pco_2 of 100, HCO_3 of 16, and a base deficit of 19.8. The infant subsequently died 2 days later. At the time of cesarean section there was 60% abruption.

Initial blood tests results on admission revealed a hematocrit of 28%, platelet count of 144,000, prolonged prothrombin time, and fibrinogen of 114 mg/dL. Subsequent blood tests revealed the presence of disseminated intravascular coagulopathy for which she received 4 units of PRBCs and 4 units of fresh frozen plasma (FFP). The patient was discharged home 4 days after surgery.

Discussion

Early diagnosis, prompt management, and timely delivery are key to improve maternal and perinatal outcomes in patients with abruptio placentae. Maternal and perinatal outcomes in abruptio placentae are usually dependent on one or more of the following: gestational age at onset, degree of placental separation, type of abruptio (revealed or concealed), maternal and/or fetal conditions at time of presentation, and presence or absence of associated conditions such as severe hypertension-preeclampsia, cocaine overdose, or uterine rupture with extrusion of the fetus into the uterine cavity. In the last case, the placental separation can be complete (100%), which results in either fetal-neonatal death and/or significant neonatal hypoxia-acidosis after 10 minutes of placental separation. In addition, maternal and perinatal morbidities and mortality are substantially increased in those with one or more of the factors listed in Table 3-7.

Common errors in the management of abruptio placentae include failure to appreciate the clinical significance of signs and symptoms such as constant abdominal (anterior placenta) or back pain (posterior placenta), in association with either uterine irritability and/or changes in FHR tracing, acute changes in maternal vital signs, and failure to appreciate the amount of blood loss (both revealed or concealed). Other common errors include failure to obtain the appropriate laboratory tests or serial monitoring of blood tests (CBC, fibrinogen, prothrombin time, platelet count), failure to obtain blood and blood products and administer these products in a timely fashion, and failure to accomplish timely delivery.

Abruptio placentae that is severe enough to kill the fetus, and those with concealed abruptio and/or separation of more than 50% are usually associated by more than 1000 mL of blood loss at the time of presentation. In addition, disseminated intravascular coagulation (DIC) is present in approximately 35% of cases. These patients are usually hypovolemic despite the fact their initial blood pressure can be normal and the pulse is slightly elevated. These patients should receive empirically 4 units of PRBCs and FFP as needed in preparation for or during delivery. In patients with severe hypertension-preeclampsia in association with abruptio, a normal blood pressure indicates hypovolemic shock and should be managed by aggressive fluid and blood infusions being guided by heart rate, urine output, and results of CBC and coagulation tests. Vaginal delivery is recommended when the fetus is dead. If labor is not well established, induction of labor should be initiated by artificial rupture of the membranes and oxytocin if needed. During induction, patient should receive serial monitoring of coagulation studies, vital signs, urine output, and amount of bleeding. Hypovolemia and coagulopathy should be treated promptly with crystalloids, blood, and blood products. Emergency cesarean section is recommended when the fetus is viable (≥24 weeks' gestation) in association with nonreassuring FHR tracing (see Table 3-5).

This patient, with a history of cigarette smoking, presented to the triage area with signs and symptoms suggestive of possible abruptio placentae (abdominal pain, uterine irritability, occasional variable decelerations). She was managed as ruled out preterm labor, observed for only 1 hour, and was discharged home despite the presence of uterine irritability and persistent FHR decelerations. During this visit she should have been monitored for a longer time with uterine activity and FHR. Four hours later she came back with similar complaints in association with vaginal bleeding, advanced cervical dilation, maternal tachycardia, and more frequent uterine contractions and recurrent decelerations.

At that time a diagnosis of abruptio placentae should have been made and blood obtained for CBC, crossmatch, and coagulation studies with plan for emergency cesarean section. Even when the diagnosis of abruptio placentae was considered an hour later, and in the presence of nonreassuring FHR

Case 2: Abruptio Placentae—cont'd

tracing, the plan was to attempt vaginal delivery. During the observation period, there was progressive separation of the placenta with increased hypoxia and acidosis as reflected by changes in FHR tracing (staircase to fetal death tracing). This delay in delivery ultimately resulted in neonatal death secondary to severe hypoxia and acidosis. In addition, it resulted in the woman developing DIC and for the need of transfusion of several units of PRBCs and FFP.

This case highlights several errors in diagnosis and management of abruptio placentae by both the managing physician and the nursing staff. The physician did not appreciate the importance of the changes in uterine activity and FHR tracing, and the nurses failed to communicate to the physician their concern about these changes. In the setting of severe placental abruption in association with nonreassuring FHR tracing, both neonatal survival and intact neonatal survival are progressively reduced if delivery was achieved beyond 20 minutes (Fig. 3-6). This case underscores the importance of developing protocols for surveillance and management of patients with suspected abruptio placentae including mechanisms to improve education, training, and methods of communication among all obstetric providers working in emergency department, obstetric triage, and labor and delivery units.

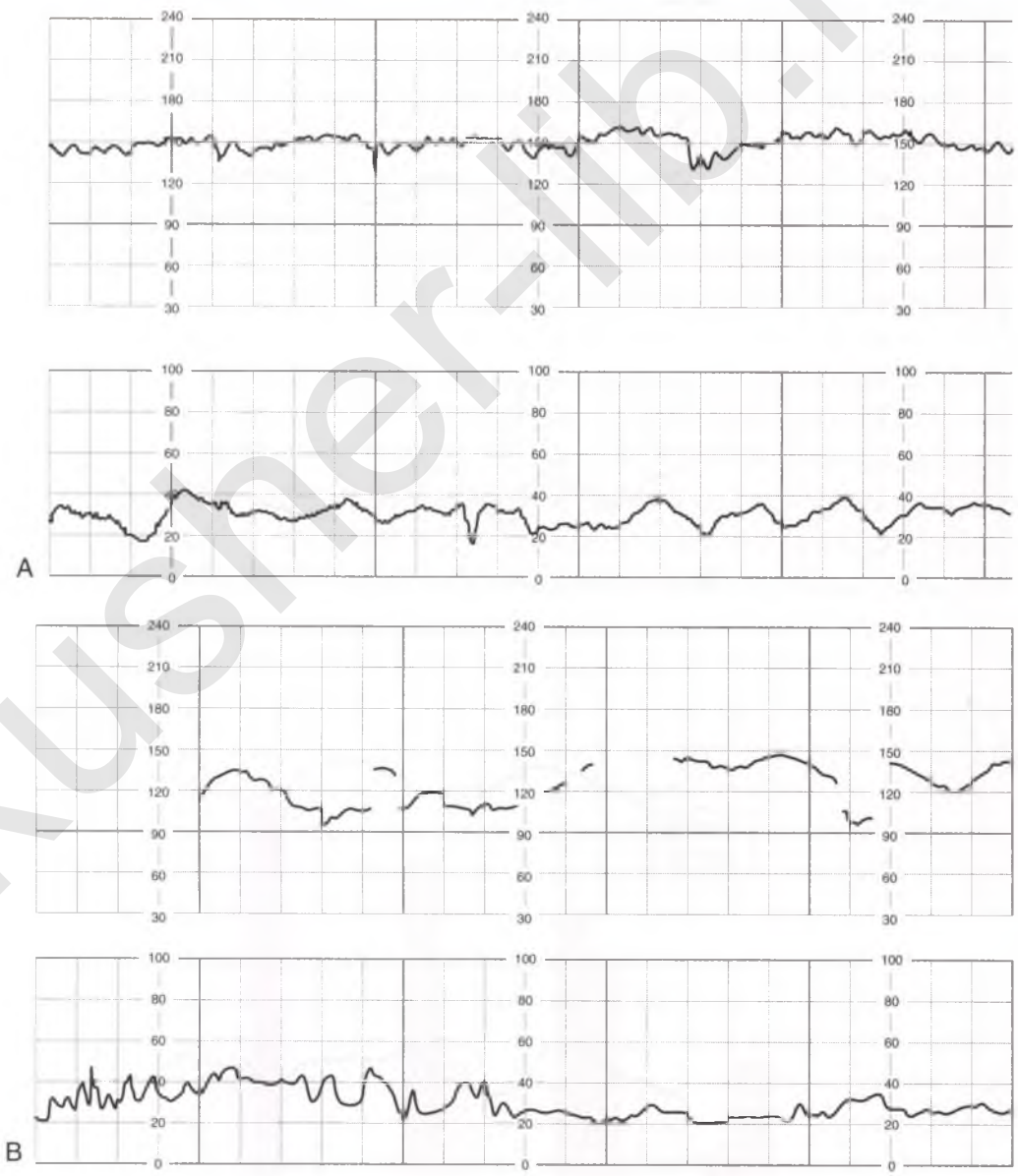

Figure 3-5 A, Demonstrating uterine irritability, normal FHR baseline, moderate variability, and occasional accelerations. **B,** There is evidence of uterine irritability with recurrent decelerations.

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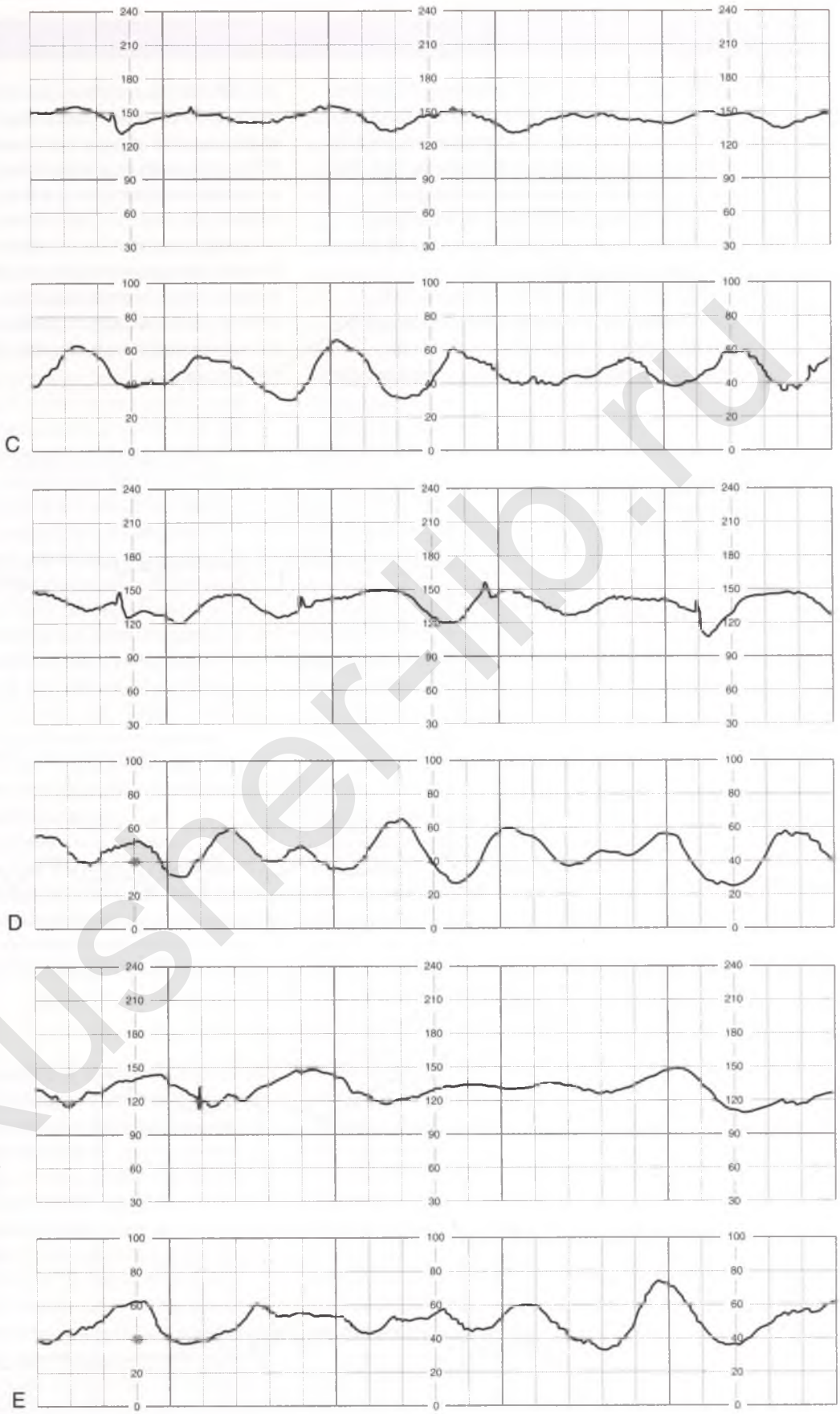

Figure 3-5, cont'd C, Uterine tachysystole with recurrent late decelerations and minimal variability. **D,** Tetantic uterine contractions with repetitive decelerations. **E,** Uterine tachysystole and repetitive decelerations.

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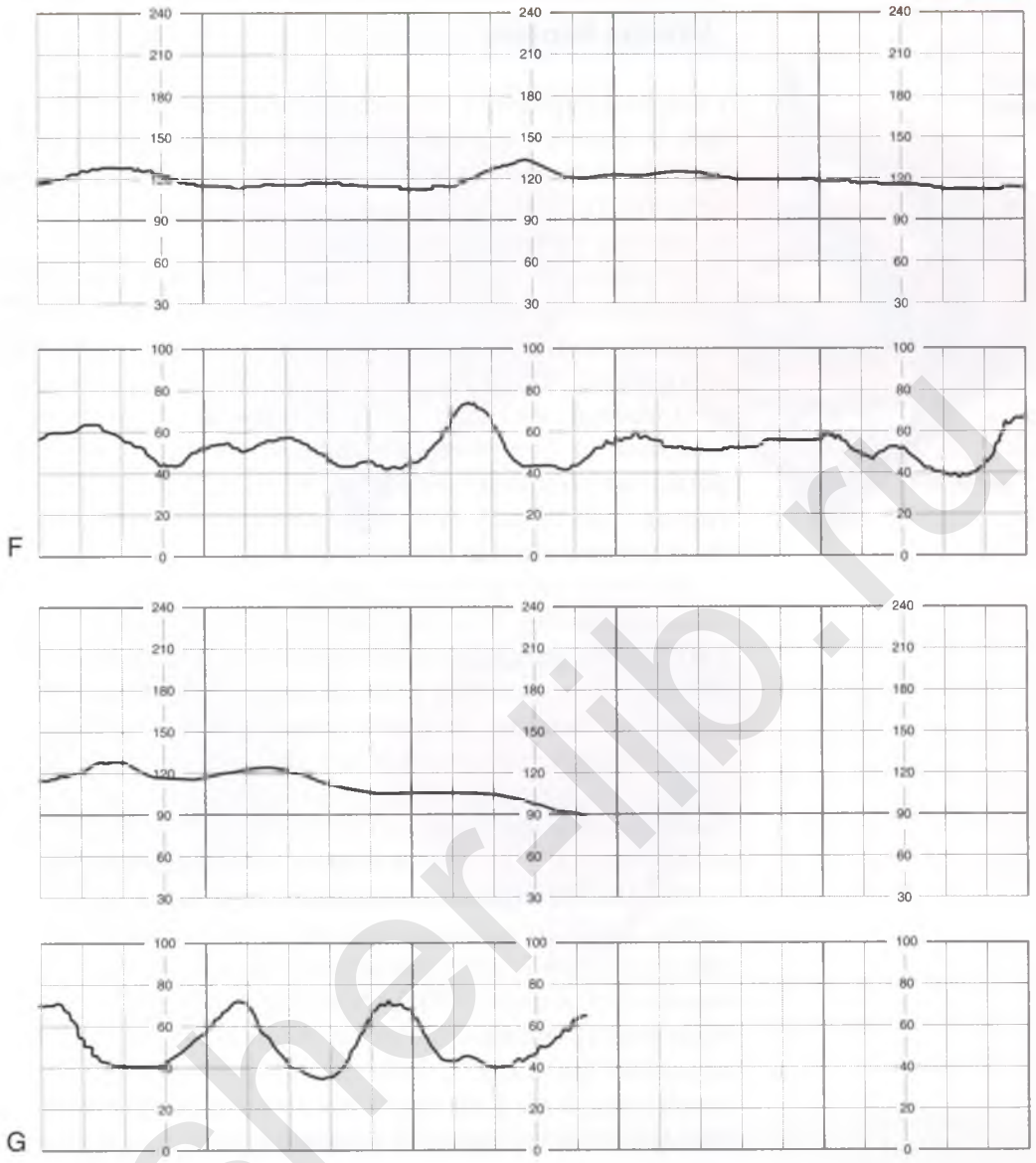

Figure 3-5, cont'd F, Evidence of uterine tachysystole, FHR baseline about 120 bpm with absent variability. **G,** FHR tracing revealing bradycardia. At time of delivery, there was severe metabolic acidosis.

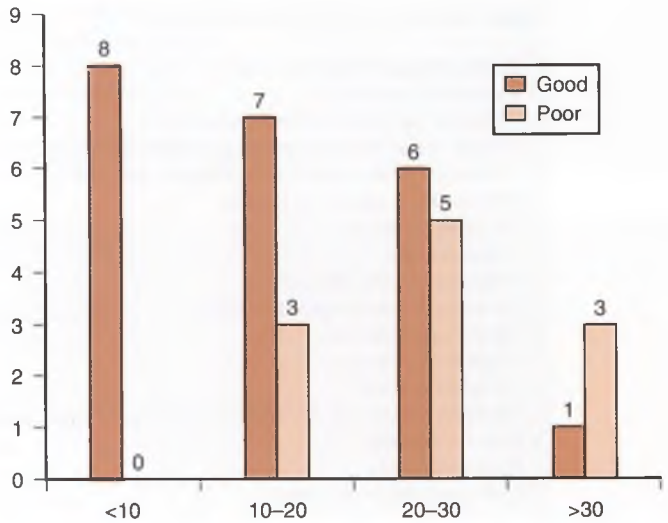

Figure 3-6 Perinatal outcome according to time of delivery in severe abruptio placentae. (Reprinted with permission from Kayani SI, Walkinshaw SA, Preston C: Pregnancy outcome in severe placental abruption. BJOG 2003;110:679–683.)

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Uterine Rupture

Complete rupture of the uterus during pregnancy is a rare obstetric emergency that is usually catastrophic to both mother and fetus. The likelihood of uterine rupture increases toward term due to uterine distention and increased uterine activity. The rate of uterine rupture increases in the presence of one or more of the risk factors listed in Table 3-8.

In patients with previous low uterine segment scar, there may be evidence of uterine dehiscence (the defect is covered by a thin layer of uterine serosa), or there may be a uterine defect (the presenting part is covered by amniotic membrane) (Fig. 3-7). There are also various degrees of uterine rupture: a loop of umbilical cord protruding through the incision with the placenta remains attached, or complete extrusion of the fetus into the peritoneal cavity with partial or complete separation of the placenta (Fig. 3-8). The site of uterine rupture can be at a previous incision (low transverse, low vertical, or fundal), or at various sites in the uterine wall.

Maternal and perinatal outcome following uterine dehiscence or rupture depends on the type and site of rupture, on location of the fetal presenting part (inside or outside the uterine cavity) at time of delivery, and timing of delivery in relation to FHR changes. In general, maternal and perinatal outcomes are usually favorable when there is only uterine dehiscence, and in cases of uterine rupture when delivery is achieved within 20 minutes after onset of recurrent moderate to severe variable or late decelerations. In contrast, both maternal and perinatal mortality and morbidity are significantly increased in the presence of one or more of the factors listed in Table 3-9.

The clinical signs and symptoms of uterine rupture can be gradual or abrupt (Table 3-10). In patients with previous cesarean section attempting a trial of labor, changes in FHR tracing are the most frequent warning signs of uterine rupture. In patients with preexisting fundal scar (classical, T, J), the presenting signs and symptoms of uterine rupture can be fetal death, hypovolemic shock, or vaginal bleeding. In patients with previous cesarean section and fetal demise attempting a trial of labor, the first signs of uterine rupture can be vaginal bleeding and/or maternal hypotension and tachycardia.

Table 3-8 Risk Factors for Uterine Rupture

- Previous history of uterine rupture
- Preexisting uterine scar
  - Previous cesarean section/hysterotomy
  - Previous myomectomy inducing uterine cavity
  - Previous corneal resection from ectopic pregnancy
- Previous silent uterine perforation
  - Suction curettage
  - Hysteroscopy
- Prolonged uterine tachysystole
  - Excessive oxytocin, prostaglandins
- Uterine overdistention
  - Multifetal gestation
  - Polyhydramnios
- Operative vaginal delivery (inappropriate use of forceps, vacuum)
- Grand multiparity
- Placenta percreta
- Prolonged obstructed labor

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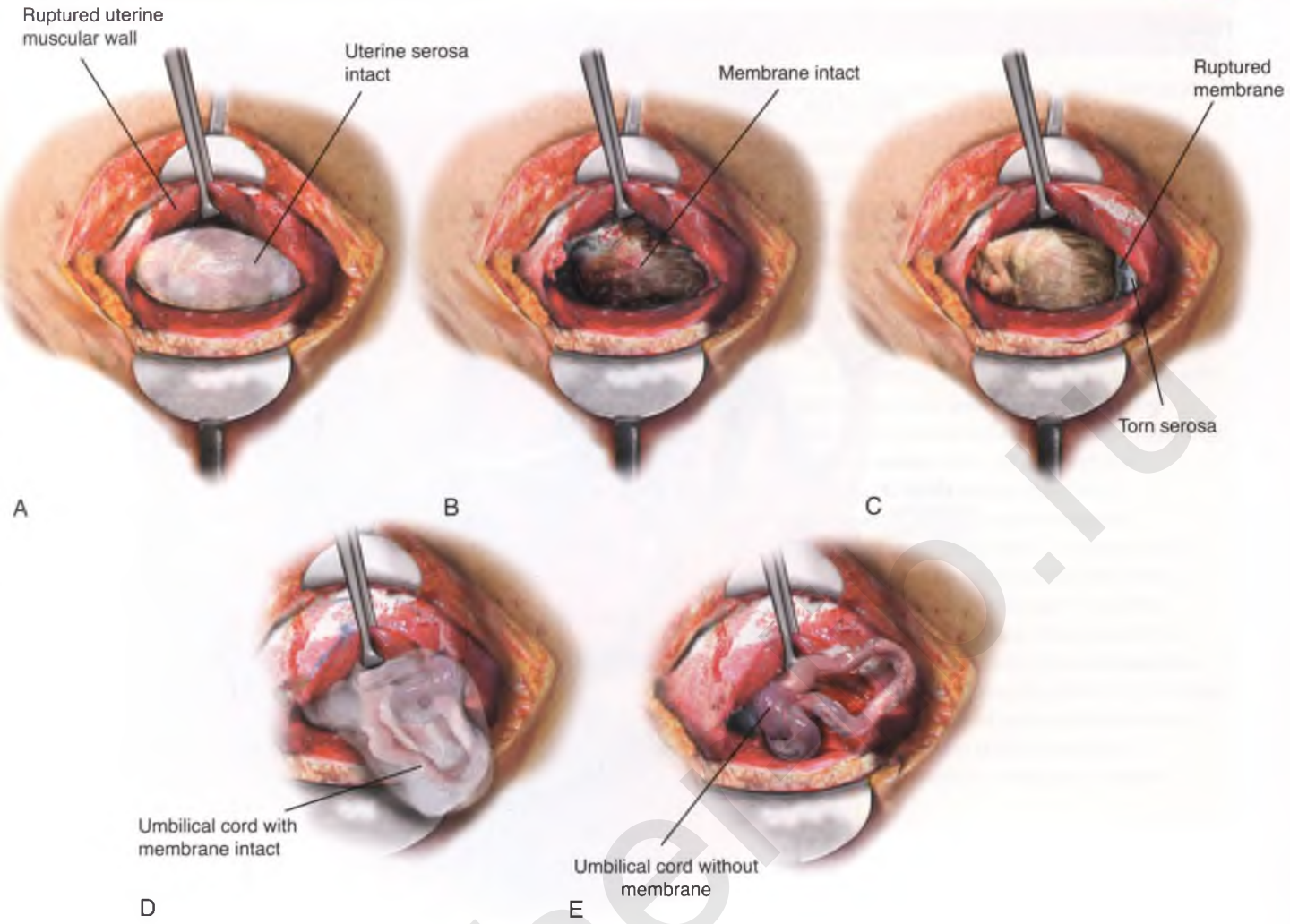

Figure 3-7 Various presentations of uterine dehiscence or rupture.

Table 3-9 Risk Factors for Adverse Maternal/Perinatal Outcome Following Uterine Rupture

- Preexisting multiple cesarean sections (three or more)
- Preexisting classical, T, or J incision
- Lateral or downward extension of the rupture sites
  - Involves uterine or cervical vessels
- Extension of rupture to area of placental implantation
- Extrusion of the fetus into the peritoneal cavity
  - Involves partial or complete abruption
- Delivery beyond 20 minutes in the presence of nonreassuring FHR tracing
- Rupture of uterus without uterine scar
  - Grand multipara
  - Excessive and prolonged tachysystole

Table 3-10 Signs and Symptoms of Uterine Rupture

- Change in FHR
  - Variable/late decelerations
  - Reduced variability
  - Bradycardia
- Vaginal bleeding
- Change in uterine activity
- Loss of fetal station
- Pain at uterine incision
- Hypotension/tachycardia

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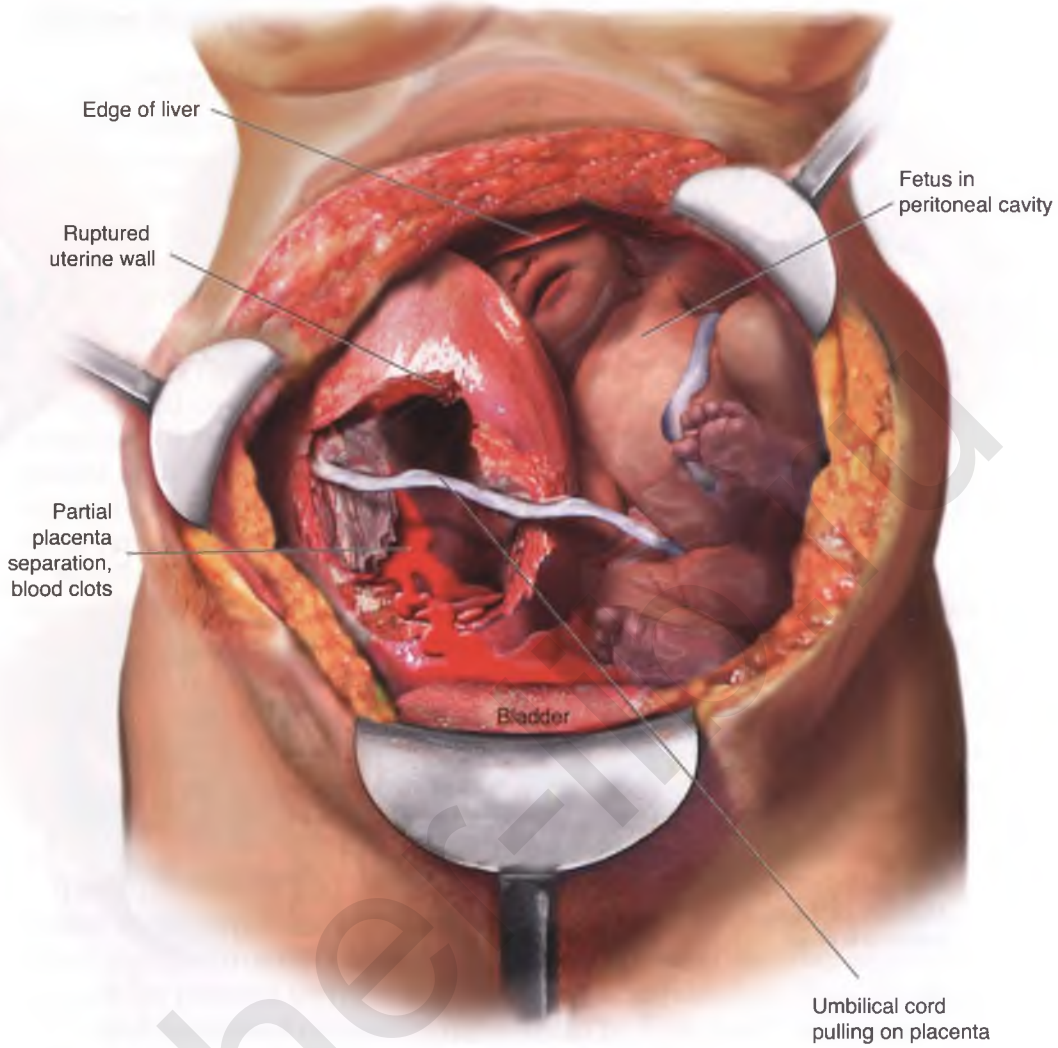

Figure 3-8 Complete uterine rupture with fetus in peritoneal cavity.

Case 3: Uterine Rupture

Patient is a 29-year-old G2P1 with previous low transverse cesarean section for arrest of descent at 40 weeks' gestation of an infant weighing 3860 g (8 lb, 8 oz). During her second pregnancy she desired a trial of labor after counseling about the benefits and risks of a trial of labor. She presented in active labor at 38 3/7 weeks' gestation. Pelvic exam revealed a cervix that was 4 cm dilated, 80% effaced with vertex presentation at −3 station (unengaged vertex). Uterine activity was adequate and the FHR tracing revealed a normal baseline with accelerations and moderate variability (Fig. 3-9A). Two hours later, pelvic exam revealed the cervix to be 6 cm dilated and 90% effaced with presenting part at −2 station. The FHR was reassuring, and she received epidural analgesia. Thirty minutes later she had spontaneous rupture of membranes with clear fluid. A fetal scalp electrode was applied with the FHR demonstrating fetal tachycardia, reduced variability, and recurrent atypical variable decelerations (see Fig. 3-9B and C). Recurrent decelerations continued for almost 1.5 hours in association with absent variability (see Fig. 3-9D). During this period, the patient was treated with IV fluids, amnioinfusion, and oxygen therapy. Pelvic exam revealed cervix to be completely dilated, with vertex at −1 station. At that time, the patient was instructed to push to facilitate delivery. During pushing, the FHR tracing was getting worse and then ended with bradycardia (see Fig. 3-9E). The patient was rushed for an emergency cesarean section with delivery of an infant weighing 3656 g (8 lb) with Apgar scores of 0, 1, and 3 at 1, 5, and 10 minutes, respectively. The infant was delivered within 15 minutes after onset of bradycardia. Cord arterial gases revealed a pH of 6.76, Pco_2 of 120, and base excess of −28. At time of delivery, the infant was in the peritoneal cavity with complete placental detachment. The uterine incision was

Case 3: Uterine Rupture—cont'd

repaired, and the patient discharged home 5 days later. The infant had evidence of hypoxic ischemic encephalopathy with neonatal death at 8 days.

Discussion

A patient with a history of previous cesarean section due to arrest of descent attempting a trial of labor has a lower rate of vaginal delivery as compared to that without such a history. This patient presented in active labor with the presenting part being unengaged. This finding is also associated with a lower chance to have a vaginal delivery. Subsequent cervical examinations continued to show unengaged vertex presentation. Following rupture of the membranes, there was evidence of frequent uterine contractions and recurrent decelerations. At that point, a decision should have been made for repeat cesarean section. Subsequent FHR tracing revealed recurrent atypical variable-late decelerations for which the patient received amnioinfusion. This latter management is inappropriate in patients attempting vaginal birth after cesarean (VBAC) in the presence of recurrent variable decelerations.

When the cervix revealed complete dilation with vertex at −1 station, the patient was encouraged to push despite a progressively worse fetal tracing. These latter pushing efforts resulted in complete uterine scar rupture with expulsion of the fetus into the uterine cavity, pulling the placenta from its attachment site. As a result, the fetus was subjected to both intermittent episodes of hypoxia for almost 2 hours and acute complete asphyxia during the last minutes prior to delivery with resultant severe hypoxia and metabolic acidosis leading to neonatal death (Table 3-11).

Early detection of abnormal progress of labor and/or changes in fetal tracing in patients attempting VBAC is critical because it may allow for prompt interventions that will lessen and/or prevent uterine rupture as it happened in this case. Errors in management include failure to understand the importance of engaged vertex during active labor in a patient with previous cesarean section. Such a finding in a patient with no previous vaginal delivery and with a previous arrest of descent should have been considered a warning sign regarding the potential for repeat arrest pattern. In addition, the managing physicians failed to appreciate the clinical significance of new onset of recurrent severe/atypical decelerations in a patient attempting VBAC. Of concern also was the use of amnioinfusion as a treatment for the ominous fetal tracing described in Figure 3-9C through D, which is highly suggestive of early uterine rupture. Finally, both the nurses and physicians demonstrated lack of knowledge about management of patients in labor attempting VBAC, by allowing the patient to push in the presence of abnormal FHR tracing and unengaged vertex. The patient should have been transferred to the operating room for emergency cesarean delivery at that time.

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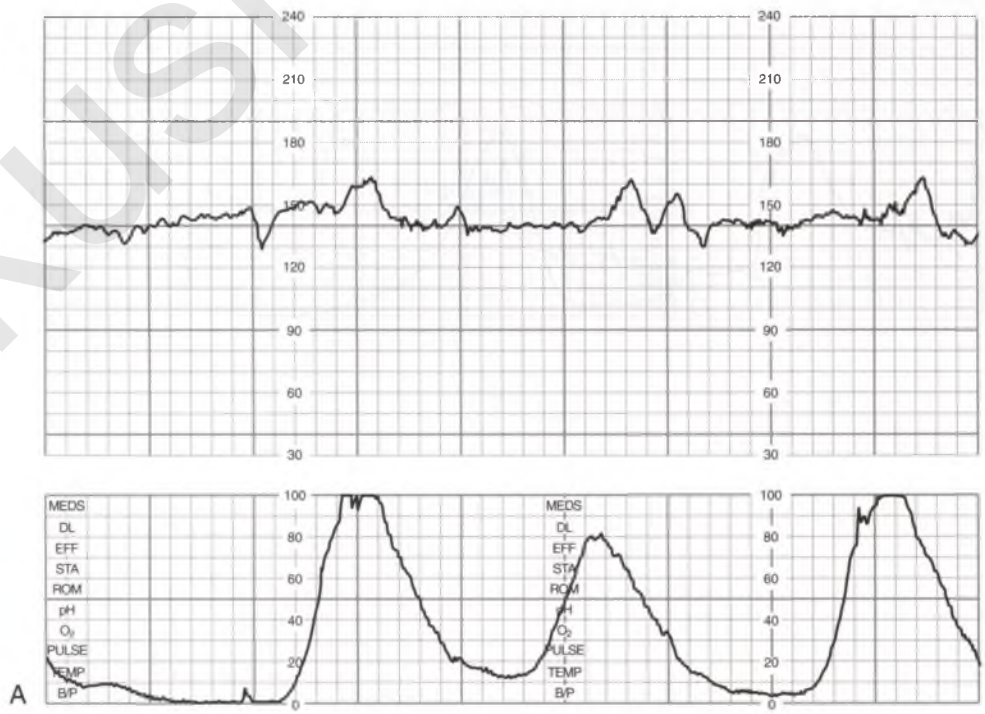

Figure 3-9 A, Demonstrating uterine activity with Category I FHR tracing.

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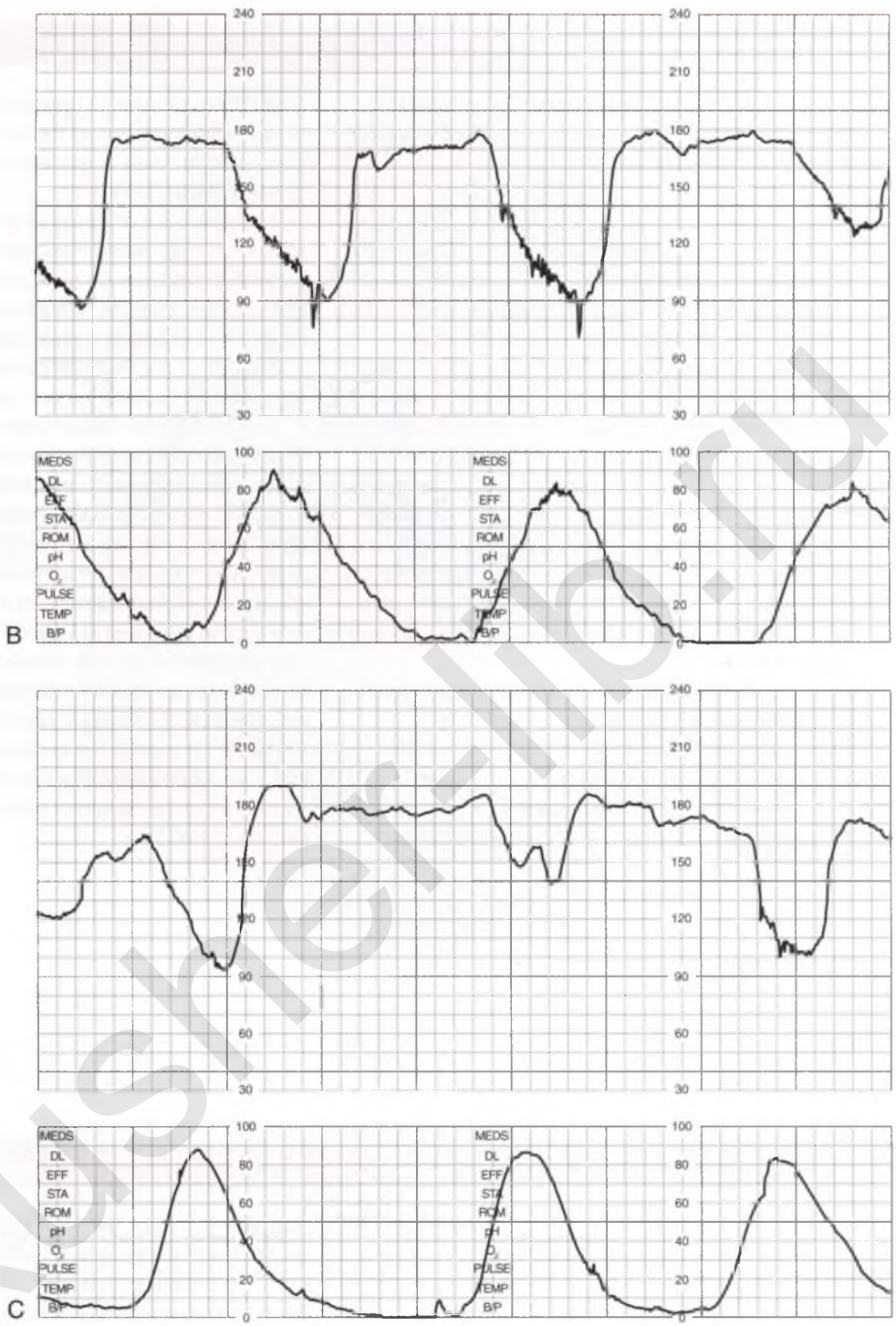

Figure 3-9, cont'd B, Increased uterine activity with recurrent atypical variable decelerations and fetal tachycardia. **C,** Recurrent variables with minimal variability and fetal tachycardia.

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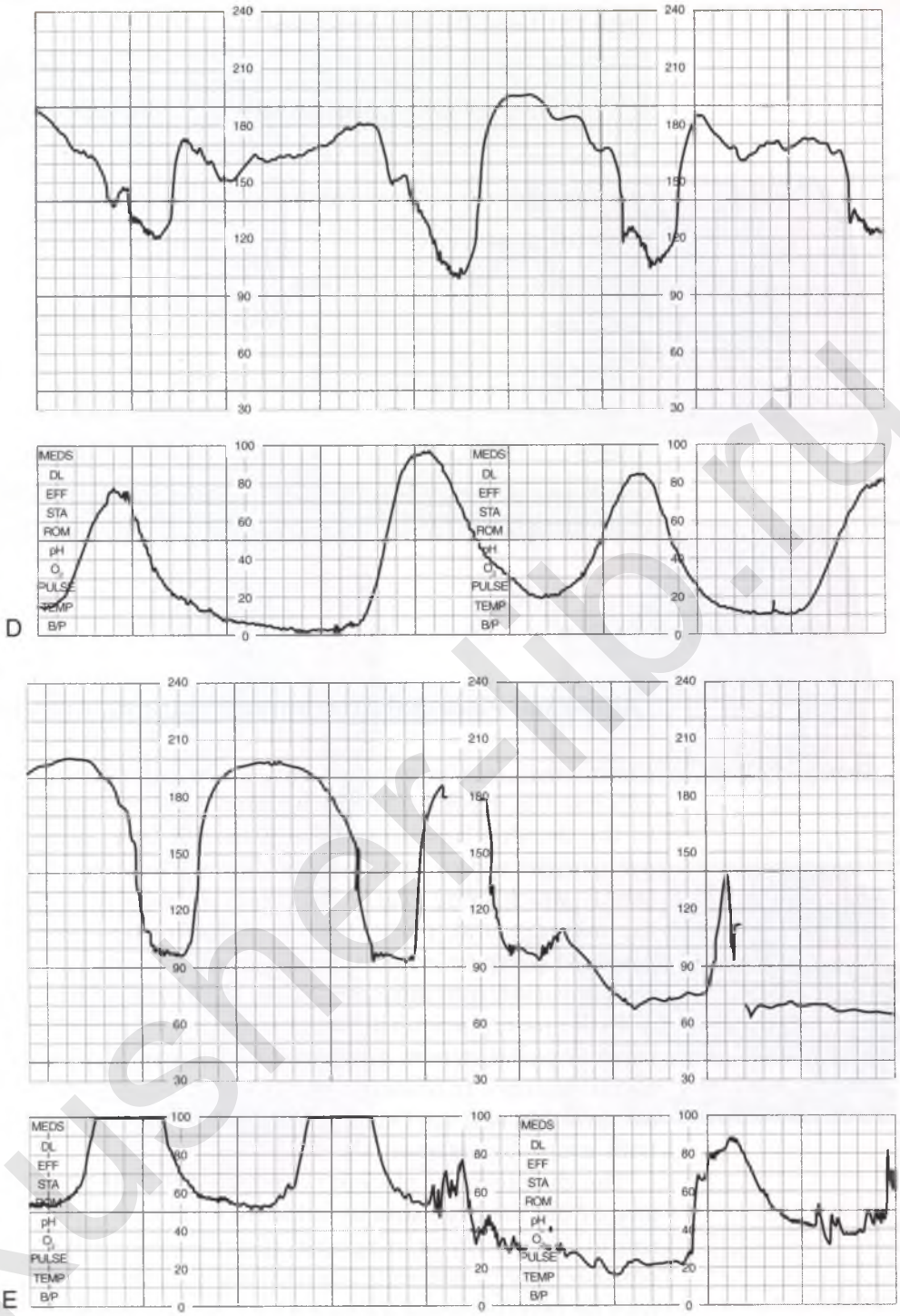

Figure 3-9, cont'd
D, Repetitive severe variable decelerations with a late component variability, and fetal tachycardia. **E,** FHR revealing bradycardia indicating uterine rupture.

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Table 3-11 Perinatal Morbidity and Mortality*

	Fetal Extrusion		
	Total (n = 28)	**Partial (n = 13)**	**No Extrusion (n = 58)**
Perinatal death	4 (14)	0	2 (2)
5-minute Apgar <7	20 (71)	6 (46)	13 (22)
Umbilical artery pH			
Total <7.00	19 (68)	8 (62)	15 (25)
6.90–6.99	4	2	3
6.80–6.89	3	3	2
6.70–6.79	8	3	8
<6.70	4	0	2

*Modified from Leung AS, Leun EK, Paul RH: Uterine rupture after previous cesarean delivery: maternal and fetal consequences. Am J Obstet Gynecol 1993;169:945–950.

Abnormal Placental Attachment (Placenta Accreta)

Placenta accreta/percreta is defined as abnormal penetration of the chorionic villi into the myometrium or beyond. The pathophysiology is related to a defect in the decidua basalis and the Nitabuch layer allowing for the villi to invade parts or the entire myometrium layer. There are three types of abnormal placental attachment: placenta accreta defined as superficial invasion of villi to the myometrium, placenta increta (invasion is deep in the myometrium), and placenta percreta when there is complete invasion through the myometrium with invasion into adjacent pelvic structures (Fig. 3-10).

In the past, placenta accreta used to be a rare obstetric complication; however, recent studies report a substantial increase in incidence probably related to recent changes in obstetric practice and demographics. Risk factors for placenta accreta/percreta are listed in Table 3-12. The risk is markedly increased in those with multiple cesarean sections.

The diagnosis of placenta accreta should be considered in the presence of any of the factors listed in Table 3-12. The diagnosis is usually made by ultrasound (Fig. 3-11) or magnetic resonance imaging (MRI). Radiologic findings consistent with placenta accreta/percreta are summarized in Table 3-13. MRI may be helpful in case of posterior placental location or for assessing the degree of placental invasion into surrounding tissues (Fig. 3-12).

Most patients with placenta accreta are asymptomatic unless they have associated previa. In this case, they usually present with vaginal bleeding with or without labor. In contrast, in patients with placenta percreta, the presenting symptom may be hematuria or intraabdominal bleeding.

Maternal and perinatal morbidities are increased in patients with placenta accreta/percreta. Fetal morbidities are usually related to preterm delivery (Table 3-14). On the other hand, both maternal mortality and morbidity are markedly increased in patients with accreta/percreta (Table 3-15). Maternal complications depend on degree of placental invasion, whether delivery was planned or was an emergency, and the management used.

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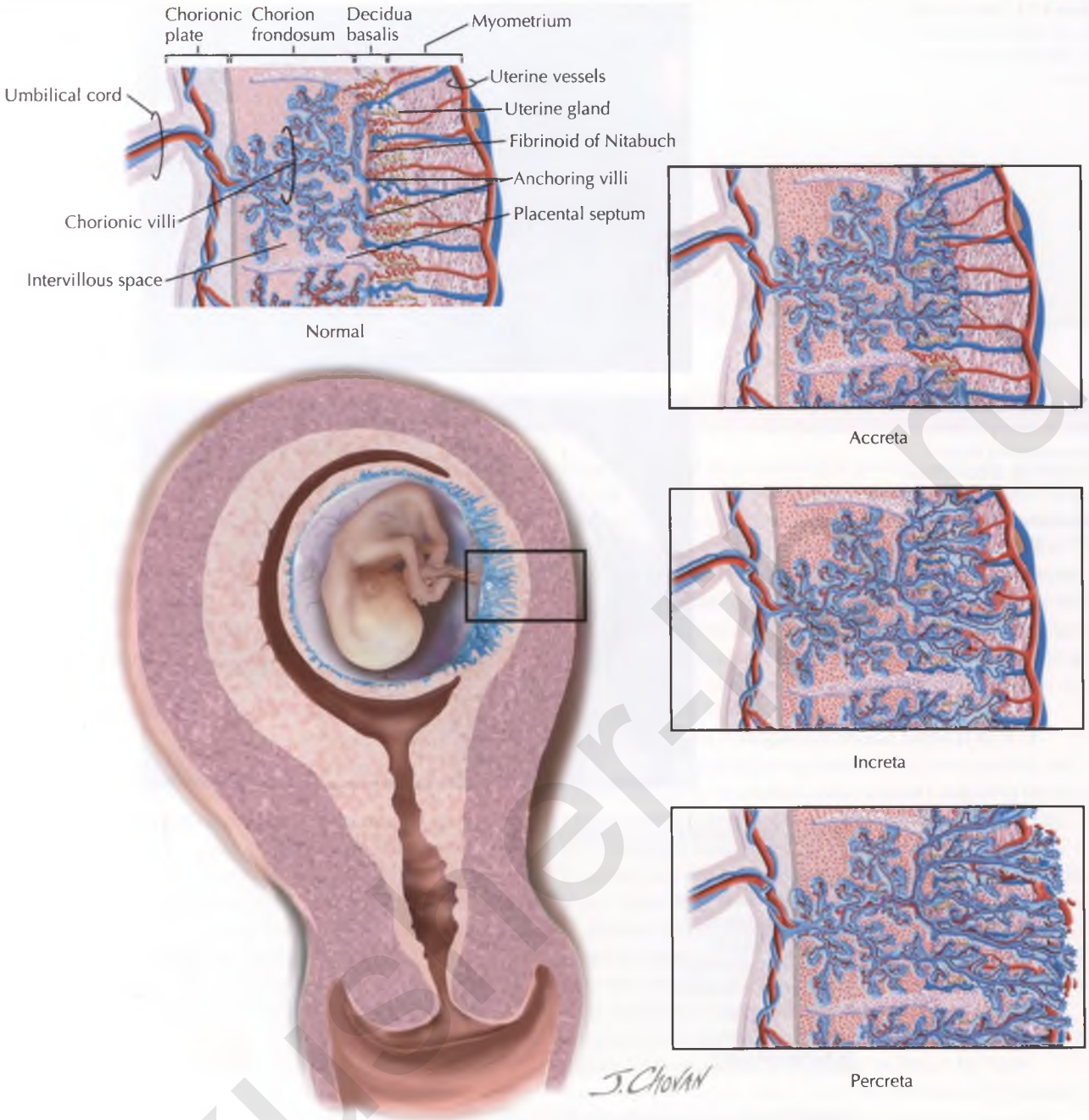

Figure 3-10 Various types of adherent placenta. In placenta increta there is invasion up to the uterine serosa, whereas in perceta there is invasion beyond the serosa into adjacent structures. (A Netter illustration from www.netterimages.com. © Elsevier Inc. All rights reserved.)

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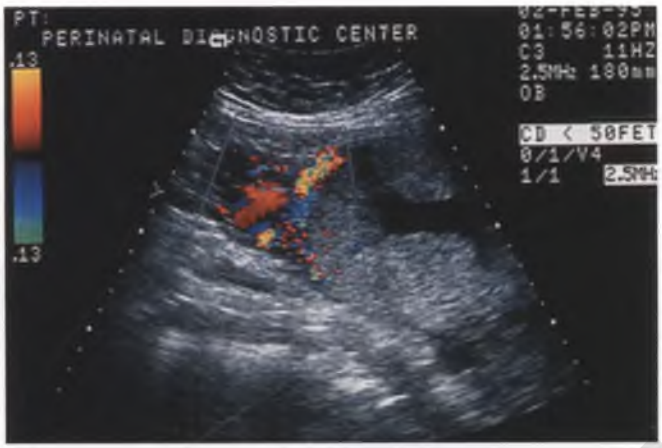

Figure 3-11 Note invasion into the myometrium by placenta tissue in placenta increta.

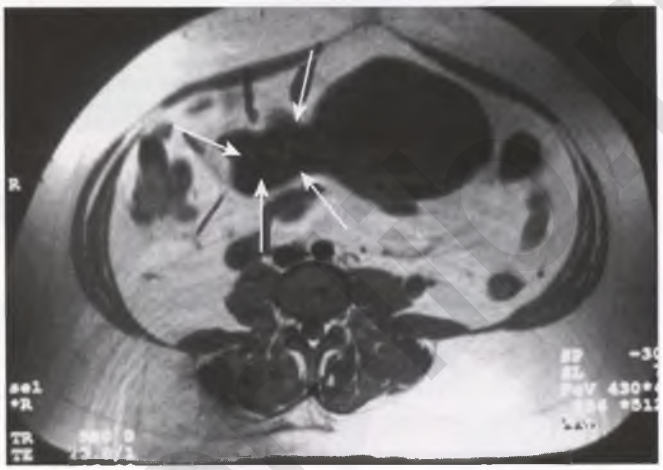

Figure 3-12 MRI of the placenta. *Arrows,* Placenta previa extending beyond uterine serosa. (From O'Brien JM, et al: The management of placenta percreta. *Am J Obstet Gynecol* 1996;175:1635.)

Table 3-12 Risks Factors for Adherent Placenta

- Placenta previa
- Prior uterine surgery
  - Cesarean section
  - Myomectomy
  - Suction curettage
  - Cornual resection
  - Hysteroscopy
- Advanced age
- Increased parity

Table 3-13 Ultrasound Findings Suggestive of Accreta

- Placental lacunae
- Loss of retroplacental hypoechoic zone
- Patients with previous cesarean and previa
  - Low-lying gestational sac
  - Intraplacental lakes with turbulent flow
  - Absent decidual basalis (myometrial thickness <1 mm)
- Loss of smooth interface with bladder
- Focal nodular projections into the bladder (percreta)

Table 3-14 Gestational Age at Delivery in Known Placenta Accreta (n = 62)*

- Planned at 34 to 35 weeks (n = 53)
- Mean ± SD at delivery 33.9 ± 1•1 week
- Planned at ≥36 weeks (n = 9)
- 22 (35%) required emergency C/S
- 4 of 9 planned at ≥36 weeks had emergency hemorrhage

*Data from Warshak CR, Ramos GA, Eskander R, et al: Effect of predelivery diagnosis in 99 consecutive cases of placenta accreta. Obstet Gynecol 2010;115:65–69.

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Table 3-15 Maternal Complications in Accreta and Percreta

- Severe intrapartum/postpartum bleeding
  - Massive transfusion of blood/products
- Hysterectomy
- Bladder/ureteral injury
- Bowel injury
- Pulmonary edema/acute respiratory distress syndrome (ARDS)
- Pelvic infection/abscess
- Admission to intensive care unit
- Hypoxic ischemic encephalopathy
- Acute tubular necrosis
- Repeat surgical intervention
- Death

Case 4: Placenta Accreta

A 20-year-old G2P1, with a history of prior cesarean section, had marked elevations in maternal serum alpha fetoprotein level 14.6 multiples of the median (MOM) (normal <2). Subsequent ultrasound examination revealed placenta previa with an area of abnormal blood flow within the myometrium suggestive of placenta percreta (see Fig. 3-11). Because of this finding, MRI was performed confirming that the placenta extended beyond the uterine serosa (see Fig. 3-12). The patient was then hospitalized at 33 weeks' gestation for close monitoring and received corticosteroids for fetal lung maturity. At 34 weeks' gestation, there was evidence of oligohydramnios and a decision was made for planned delivery. The patient desired further pregnancies, and requested avoiding hysterectomy if possible at time of laparotomy. As a result, she had planned repeat cesarean section. At time of laparotomy, there was evidence of placenta percreta (Fig. 3-13A); the uterine fundus was identified and placental edge was located (see Fig. 3-13B). Following that, the uterine incision was made in the fundus away from the site of percreta (see Fig. 3-13C). The infant was delivered by breech extraction from vertex presentation. The newborn weighed 2240 g and had Apgar scores of 8 and 9.

The umbilical cord was ligated close to its placental insertion, and uterine incision was closed (see Fig. 3-13D and E). The estimated blood loss was 600 mL, and no blood transfusion was needed. Patient received prophylactic antibiotics and intramuscular methotrexate was given the next day and repeated weekly at a dose of 1 mg/kg ideal body weight. She had follow-up with ultrasound, β-human chorionic gonadotropin (β-hCG), CBC, and liver enzymes. The therapy was continued for 6 weeks until two consecutive undetectable β-hCG values were reported. At 10 weeks after surgery, there was rupture of membranes with prolapse of the umbilical cord stump into the vagina. This was later excised and patient received IV antibiotics because of fever, and then discharged home. Eight months after delivery, patient was doing well.

Discussion

Early diagnosis and planned delivery are the keys to improved pregnancy outcome in patients with adherent placenta. The diagnosis should be considered in patients with one or more of the risk factors listed in Table 3-12. Once a diagnosis of placenta accreta is made (usually by ultrasound), the next step is to evaluate the degree of penetration toward the uterine serosa or beyond into adjacent structures such as the bladder, bowel, or parametrial tissues. This should be confirmed by MRI as happened in this case. The degree of invasion and extension beyond the uterine wall is important for maternal counseling and for planning of surgery as it relates to timing, location, and resources (Table 3-16).

Preparation for management should begin at time of diagnosis. A multidisciplinary team should be organized, and patient should be counseled about the risks including those of preterm delivery and organ injury, blood transfusions, early and prolonged hospitalization, and potential admissions to intensive care unit. Counseling should also include discussion about desire to preserve fertility (keep the uterus).

Because planned delivery is important, it is critical to schedule delivery prior to onset of labor or bleeding. It is also important to administer corticosteroids for fetal lung maturity at 33 to 34 weeks' gestation depending on whether there is accreta or percreta with extension. Those with placenta previa/increta are hospitalized at 34 weeks' gestation for close monitoring and preparation, and then delivered at 35 weeks. If the patient has placenta percreta with extension to pelvic structures, she is hospitalized at 33 weeks with planned delivery at 34 weeks' gestation.

If the patient presents with labor and bleeding or if placenta increta/percreta is first diagnosed at time of laparotomy, then management should be as described in Table 3-17. At time of laparotomy, it is important to evaluate for active bleeding, assess extent and location of placental invasion, and consider the available help and resources. If possible, avoid making a uterine incision until resources and help are available. During this period, apply moist laparotomy pads over the uterus or apply packing over bleeding vessels while waiting for help, blood, and blood products. If the patient is stable and resources are not available at your facility, close the abdominal incision and consider transfer of the patient to a tertiary facility.

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Case 4: Placenta Accreta—cont'd

At time of delivery, surgical options will depend on patient desire for future child birth, degree and site of bleeding, and extent of extension and invasion of placental tissue. In some instances it is appropriate to plan cesarean hysterectomy immediately after delivery of the infant, whereas in other situations, it is advisable to leave the placenta in situ. A management plan for placenta accreta/percreta is summarized in Figure 3-14.

After laparotomy in patients with placenta percreta, the uterine incision should be made away from the placental margin. In some of these cases, intraoperative ultrasound can be used to delineate the placental location. The uterine incision is made in the fundus in case of placenta previa (see Fig. 3-13C), whereas it should be made in the lower uterine segment in case of fundal percreta.

If the decision is made to leave the placenta in situ, the umbilical cord is ligated in proximity to its insertion and the uterine incision is closed. The patient is then observed in the hospital for 7 to 10 days for possible bleeding or infection. During this period, broad-spectrum antibiotics are given, and the patient is started on intramuscular methotrexate of 50 mg/m^2 every week, and serial ultrasound examination including 3-D angiography. Other testing includes weekly β-hCG titer, CBC, coagulation studies, and serum evaluation of liver enzymes and creatinine to monitor for methotrexate effects and side effects. During this management, profuse hemorrhage requiring transfusions and hysterectomy may occur from hours to 6 weeks postsurgery. In addition, some patients can develop delayed onset of DIC (usually 3 to 6 weeks after surgery), and late-onset infection.

If a decision is made for cesarean hysterectomy, profuse bleeding can be a problem in case the placenta begins to separate or because of uterine bleeding at the site of placenta previa. In this situation, a tourniquet maneuver technique can be temporarily used to reduce or prevent bleeding, allow time for blood transfusions, and allow for an experienced surgeon to become available. The tourniquet can be a 7 to 9 French sterile Rulibex tube, a heavy Penrose drain, or a piece of plastic IV tubing. The tourniquet is first applied to encircle both uterine vessels and ovarian arteries (Fig. 3-15).

When the surgeon is ready to begin the cesarean hysterectomy, two openings are created through the avascular space in both broad ligaments just below the uterine incision. The previous tourniquet is then removed, and a new one is passed through the created holes and then tied securely in place. This tourniquet will now shut off blood flow from the uterine arteries (Fig. 3-16). Cesarean hysterectomy is then begun until it is time to clamp the uterine vessels. At this stage, the second tourniquet is cut, and uterine vessels are clamped and sutured. The tourniquet technique can be very valuable in patients who present with bleeding from accreta with previa where blood and blood products are not readily available and/or when proper staff and surgeons are also not readily available.

In summary, all obstetric units should be prepared to deal with antepartum and intrapartum hemorrhage. This requires anticipation and development of policies and protocols to identify those who are at high risk and to make available all of the resources needed to manage such complications. This requires a multidisciplinary team that includes the obstetrician, nursing and blood bank staff, anesthesia staff, and others as needed.

Table 3-16 Planned Management of Accreta and Percreta

- Reduces the risks of adverse outcome
- Proper counseling of patient
  - Potential loss of fertility
  - Need for more frequent monitoring
  - Need for early and prolonged hospitalization
  - Risks of preterm delivery and organ injury
    - Need for transfusion of blood and blood products
    - Admission to intensive care unit
- Delivery in well-equipped and staffed OR
  - Avoids chaos
  - Availability of blood and blood products
  - Availability of multidisciplinary team
    - Skilled surgeon
    - Anesthesia
    - Gynecologic oncologist
    - Urologist
    - Nursing and OR personnel
    - Intervention radiology
    - Blood bank staff

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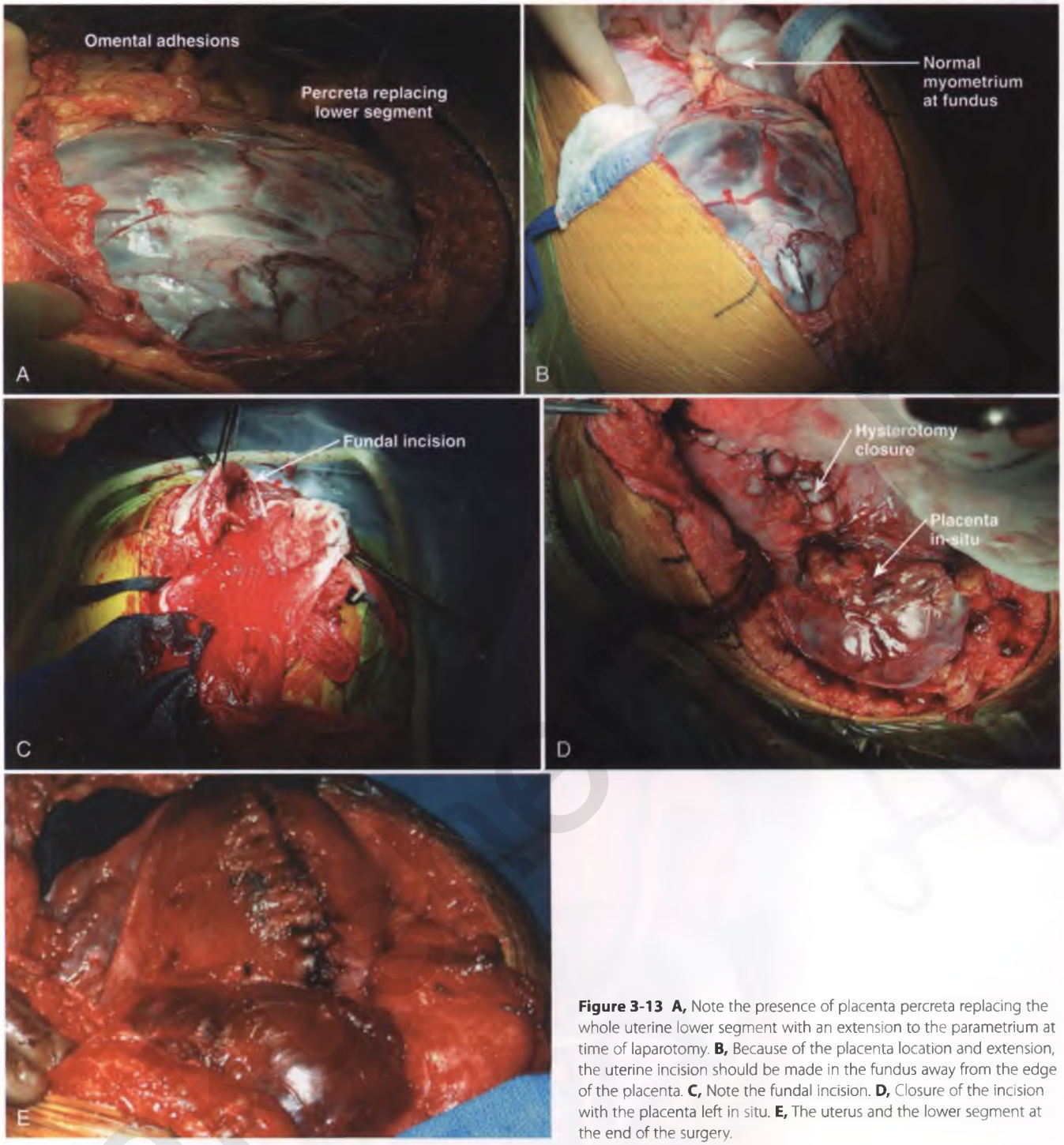

Figure 3-13 **A,** Note the presence of placenta percreta replacing the whole uterine lower segment with an extension to the parametrium at time of laparotomy. **B,** Because of the placenta location and extension, the uterine incision should be made in the fundus away from the edge of the placenta. **C,** Note the fundal incision. **D,** Closure of the incision with the placenta left in situ. **E,** The uterus and the lower segment at the end of the surgery.

Table 3-17 Management of Unsuspected Placenta Increta/Percreta

- Assess location and extent of placental invasion
- Evaluate for presence of active bleeding
- Inquire about availability of assistance/resources
- Delay uterine incision if things look abnormal
  - Distorted lower segment
  - Blood vessels on uterine serosa
  - Invasion into the bladder or surrounding tissue
- If patient is stable and facility is not prepared:
  - Cover the uterus with warm towels and call for help
  - Close the abdominal incision and consider transfer

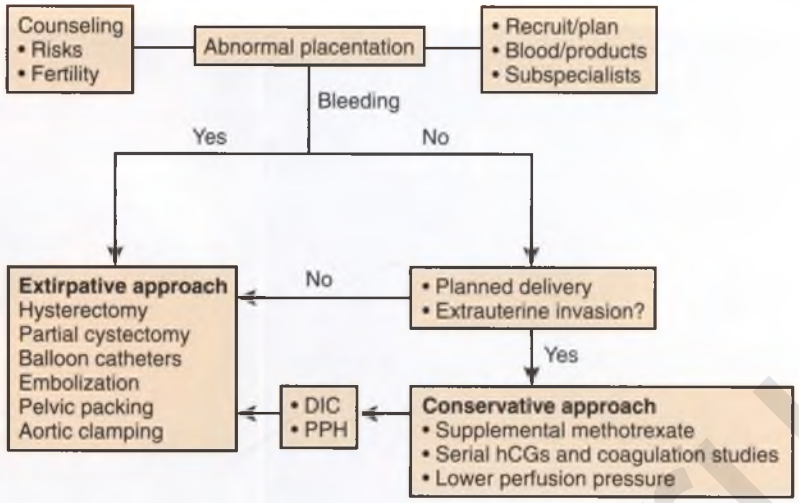

Figure 3-14 Algorithm for the management of placenta accreta. *DIC*, Disseminated intravascular coagulopathy; hCGs, human chorionic gonadotropins; *PPH*, postpartum hemorrhage.

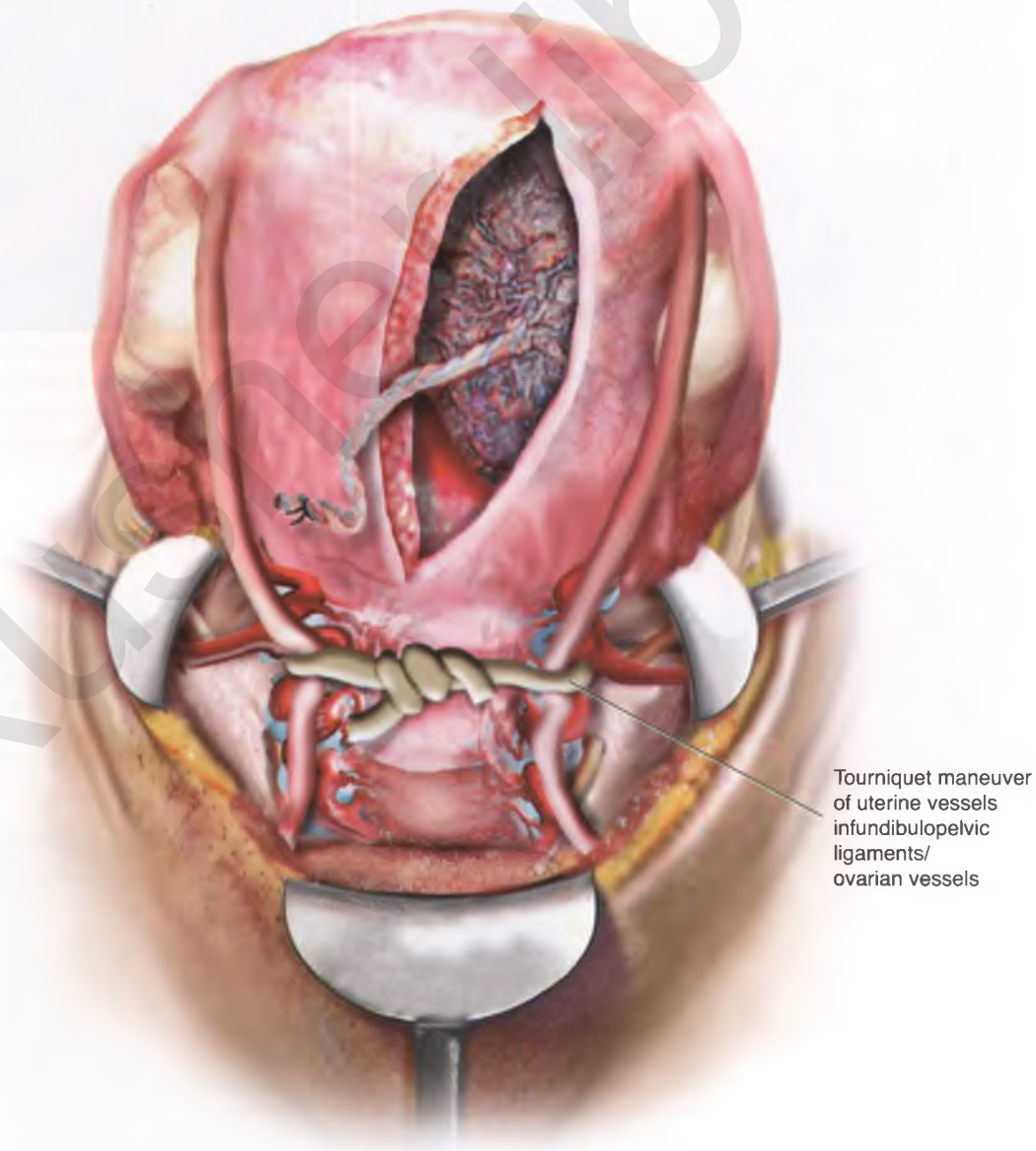

Figure 3-15 Tourniquet technique as a temporary measure to reduce bleeding prior to hysterectomy in patient with placenta accreta.

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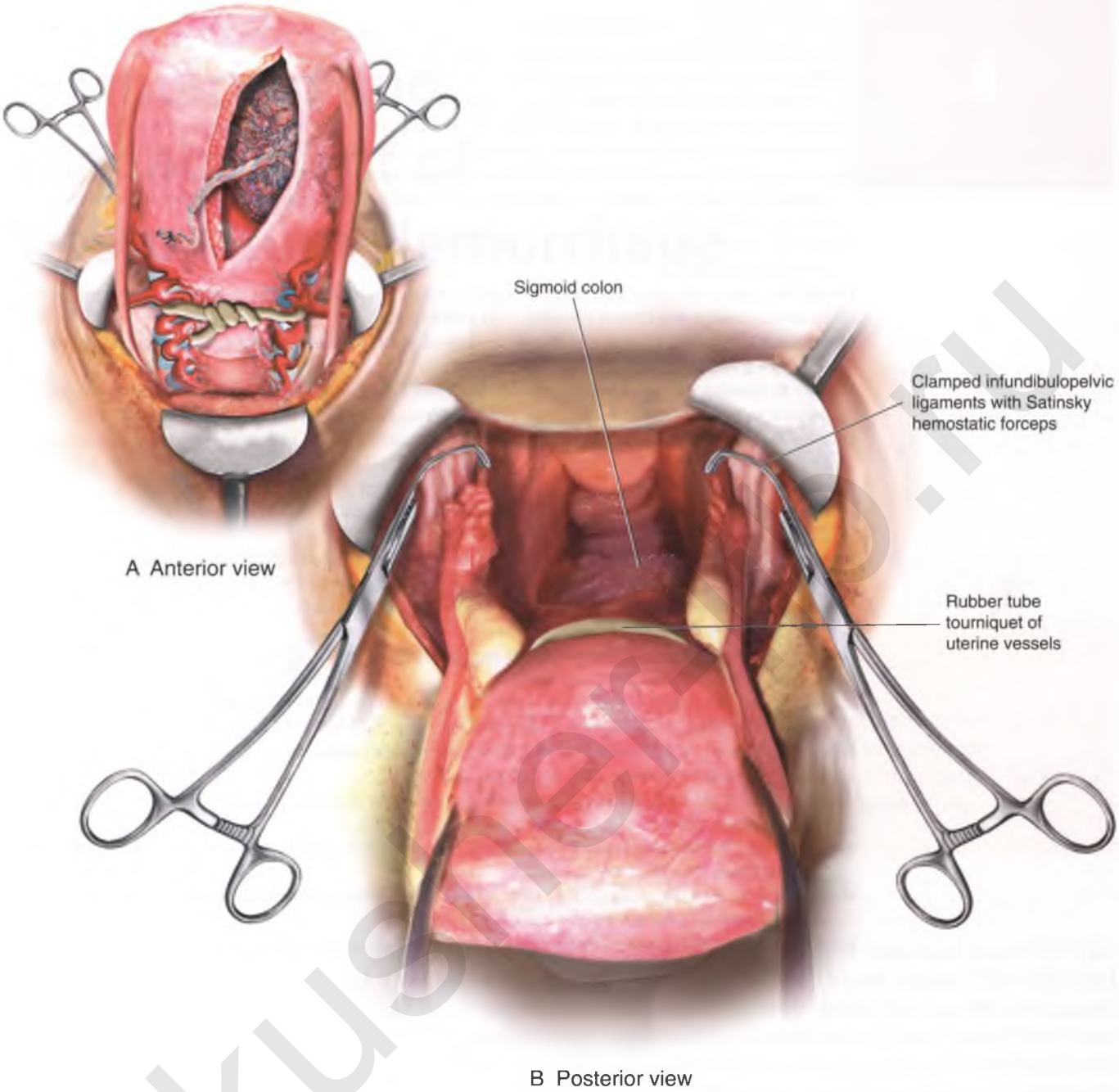

Figure 3-16 Anterior and posterior views of the location of the tourniquet around uterine arteries.

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4

Evaluation and Management of Postpartum Hemorrhage

Baha M. Sibai M.D.

Video Clips on DVD

4-1 PowerPoint Discussion of Protocols and Review of Management of Postpartum Hemorrhage
4-2 Techniques and Equipment Required to Manage Uterine Inversion
4-3 Technique of Balloon Tamponade for Postpartum Hemorrhage
4-4 Technique for How Best to Perform a Cesarean Hysterectomy

Postpartum hemorrhage (PPH) is the leading cause of maternal deaths worldwide and a major cause of maternal death in the United States. The reported incidence ranges from 5% to 10% of all deliveries depending on the definition used. Postpartum hemorrhage is defined as an estimated blood loss of ≥500 mL after vaginal delivery or ≥1000 mL following cesarean section. It is also classified as primary (onset within 24 hours after delivery of the baby) or secondary (onset >24 hours postpartum). Hemorrhage is also classified as mild if the blood loss is ≤1500 mL, severe if >1500 mL, and massive if >2500 mL. The incidence of severe postpartum hemorrhage (0.5% to 1%) is increasing because of recent changes in obstetric demographics (see Chapter 1). Severe postpartum hemorrhage is a life-threatening emergency that develops in association with one or more of the factors listed in Table 4-1.

In general, uterine atony is the most frequent cause of PPH. Risk factors for uterine atony are summarized in Table 4-2. Persistent vaginal bleeding after delivery of the fetus and the placenta is the first sign of PPH. The bleeding can be in the form of slow and continuous oozing or it can be profuse vaginal bleeding. The bleeding is also associated with maternal tachycardia with narrow pulse pressure and reduced urine output. In severe or unrecognized cases, PPH leads to hypovolemic shock (Table 4-3).

*Acknowledgement. To Dr. Nader Susseinzadeh for his contribution of Video 4-4.

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Table 4-1 Causes of Postpartum Hemorrhage

- Uterine atony
- Severe cervicovaginal-perineal lacerations
  - Operative vaginal delivery
  - Precipitate labor
- Uterine inversion
- Retained products of conception
- Vascular lacerations during cesarean section
- Adherent placenta
- DIC/other coagulopathies

Table 4-2 Risk Factors for Uterine Atony

- Previous history associated with postpartum hemorrhage
- Uterine overdistention
  - Multifetal gestation
  - Macrosomia
  - Polyhydramnios
  - Marked hydrocephaly/fetal tumors
- Uterine muscle fatigue
  - Grand multipara
  - Prolonged labor/cesarean section in labor
  - Prolonged use of uterotonic agents
  - Advanced maternal age (≥40 yr)
- Uterus-relaxing medications
  - Magnesium sulfate
  - Nifedipine
  - β-mimetic drugs
  - General anesthetic drugs
- Morbid obesity
- Chorioamnionitis

Table 4-3 Signs and Symptoms of Hypovolemic Shock

Signs	Symptoms
Systolic pressure ≤90 mm Hg	Anxiety
Diastolic pressure ≤50 mm Hg	Confusion
Heart rate ≥110 bpm	Lethargy
Narrow pulse pressure	Air hunger
Cold and clammy	Tachypnea
Pale looking	Dizziness
Oliguria/anuria	

Case 1: Uterine Atony

A 20-year-old G1 was admitted at 38 weeks' gestation for induction of labor because of severe preeclampsia. Cervical examination revealed vertex presentation at −2 station, and the cervix was long and closed. IV magnesium sulfate was started as a 6-g loading dose over 20 minutes followed by 2 g/hr as maintenance. Because of severe hypertension, the patient received two doses of 5 mg IV hydralazine. Laboratory testing revealed a hematocrit of 39% and hemoglobin (Hgb) of 13 g/dL, the platelet count was 140,000/mm^3, with normal serum creatinine (0.8 mg/dL), normal liver enzymes, and normal fibrinogen, prothrombin, and partial thromboplastin time. Cervical ripening was started at 6 PM with misoprostol at 25-mg vaginal suppositories every 4 hours. At 3 PM the next day, cervical exam revealed 2 cm dilation, 70% effacement with presenting part at −1 station. Oxytocin induction was started and 3 hours later membranes were ruptured when she was

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Case 1: Uterine Atony—cont'd

4 cm dilated. The oxytocin dose was progressively increased and she reached full dilation at midnight. At that time she started pushing and after 3 hours of pushing, the vertex was at 3+ station with subsequent delivery by vacuum of a 3800-g infant with good Apgar scores.

Following delivery of the placenta, the patient was noted to have moderate amount of vaginal bleeding. Oxytocin 40 IU was added to the IV and ran wide open. Pelvic examination revealed no retained products of conception, but the uterus was noted to be soft but full with clots of blood. Uterine massage was initiated for about 10 minutes, but she continued to bleed. Inspection of the vagina revealed no cervical or vaginal laceration. Because of continued vaginal bleeding, 800 µg of misoprostol was inserted per rectum. Vital signs obtained at that time revealed a blood pressure of 110/70 mm Hg, pulse of 110 beats per minute (bpm) and a respiratory rate of 22/min. The patient was also noted to be pale. Blood was obtained and sent for complete blood count (CBC) and crossmatch of 2 units of packed red blood cells (PRBCs), and patient received a bolus of 500 mL of lactated Ringer's.

Uterine massage was initiated again because of continued heavy vaginal bleeding. Approximately 15 minutes later, the blood pressure was 90/50 mm Hg, the pulse was 130 bpm, and respiratory rate was 26/min. The rate of IV infusion was increased to 200 mL/hr. Approximately 15 minutes later, the patient was complaining of dizziness and was noted to be lethargic. Repeat vital signs revealed blood pressure of 80/40 mm Hg, the pulse was 140 bpm, and respiratory rate was 28/min. The patient received 0.25 mg of 15-methyl prostaglandin $F_2\alpha$ IM, and anesthesia was called to help in the management. Another IV access was established, PRBCs were started, and large boluses of fluid were administered. Blood was sent for coagulation studies and for 2 more units of PRBCs. Because of continued blood loss, another dose of 0.25 mg of 15-methyl prostaglandin $F_2\alpha$ was given, and the decision was made for surgery. The results of initial CBC revealed a hematocrit of 26%, an Hgb of 7 g/dL, and a platelet count of 100,000/mm^3.

Exploratory laparotomy performed approximately 80 minutes postpartum revealed an intact, boggy uterus that was full of clots. A diagnosis of uterine atony was confirmed. The patient had bilateral uterine artery ligation followed by bilateral uterotubal artery ligation. Despite this, the uterus remained atonic. A third dose of 0.25 mg of 15-methyl prostaglandin $F_2\alpha$ was administered in the uterine fundus. Results of coagulation studies arrived while patient was in the OR and revealed presence of disseminated intravascular coagulopathy (DIC) with a hematocrit of 21% and Hgb of 5.4 g/dL. Two additional units of PRBCs were given, and anesthesia called for 4 more units of PRBCs, 4 units of fresh frozen plasma (FFP), and 10 units of platelets. Because of continued uterine atony, the decision was made for B-Lynch sutures (uterine compression sutures), which was performed.

During surgery, blood pressures were <90 mm Hg systolic and ≤50 mm Hg diastolic. Following the B-Lynch sutures, uterine blood loss was reduced, and the abdomen was closed. The patient remained intubated and was transferred to the intensive care unit (ICU) for postoperative management. Her postoperative course was complicated by DIC (requiring 12 more units of PRBCs, 8 units of FFP, and 10 units of platelets), acute renal failure (requiring four sessions of dialysis), pulmonary edema, and acute respiratory distress syndrome (ARDS). Because of neurologic symptoms (visual changes and upper extremity weakness) the patient had magnetic resonance imaging (MRI), which revealed findings consistent with hypoxic ischemic changes. She remained in the ICU for 10 days and then discharged home 7 days later. At time of discharge, she had moderate renal dysfunction with memory loss and continued motor weakness in upper extremities.

Discussion

This patient had prolonged induction of labor requiring large doses of uterotonic agents. In addition, she was in labor for a total of 30 hours including prolonged second stage. Moreover, she received large doses of magnesium sulfate for more than 24 hours. Thus she was at high risk for uterine atony (see Table 4-2). Consequently, the managing providers should have anticipated this complication and been prepared for prompt diagnosis, availability of blood products, adequate staff for close monitoring, and calling for additional help very early in the process. As a result, she developed unrecognized massive blood loss for which she received "too little" replacement of fluids and blood and "too late" replacement of blood products and surgical intervention.

Early recognition and prompt treatment are important to reduce adverse maternal outcome in patients with PPH. The first step in management should include planning for its development based on the presence of risk factors, and to call for help when PPH is recognized. Subsequent management should include finding the cause of bleeding with simultaneous assessment of blood loss and maternal vital signs (Table 4-4). It is important to keep in mind that blood loss is usually underestimated or it can be difficult to assess because large amounts of blood can accumulate in the uterus (uterine atony), in the vagina (obese women with cervical or vaginal laceration), or in the peritoneal cavity (extension of vertical cervical laceration). In addition, considering that blood flow to the uterus at term is approximately 600 mL/min, the patient can lose about 50% of her blood volume within 5 to 10 minutes in case of severe uterine atony. Therefore, two short large-bore IV catheters should be secured immediately to help in administration of large amounts of crystalloids, blood, and blood products as needed. This also requires the availability of rapid infusion systems and forced-air warming devices. An arterial line is also very helpful for continuous monitoring of vital signs,

Case 1: Uterine Atony—cont'd

arterial gases and CBC, platelet count, and coagulation studies. This requires help from anesthesia staff or staff with expertise in massive volume replacement therapy. Subsequent management can be medical (Table 4-5) or surgical (Table 4-6) depending on etiology and response to initial therapy.

Finally, the decision to proceed with prompt hysterectomy should depend on one or more of the following factors: maternal hemodynamic status, age and parity, number of cesarean sections (≥3), and presence of preexisting conditions such as accreta/percreta.

Uterine Atony

Management of uterine atony should include uterine massage (very difficult in obese women), use of uterotonic agents such as continuous infusion of oxytocin (40 IU/L), 0.2 mg of IM methylergonovine (avoid using in women with hypertension or preeclampsia), IM 0.25 mg prostaglandin $F_2\lambda$ (Hemabate), or oral or rectal misoprostol (600 mg). If these measures fail, then uterine packing or tamponade with a balloon or a Foley catheter should be performed. Proper positioning of the

Table 4-4 Evaluation and Management of Postpartum Hemorrhage

- Anticipate based on risk factors
- Call for help
- Assess blood loss and hemodynamic status
- Establish etiology (four T's)
  - Tone (atony)
  - Tissue (retained products, uterine inversion)
  - Trauma (uterine rupture, lacerations)
  - Thrombin (coagulation)
- Prompt IV access and therapy
  - Fluids, blood, blood products
- Medical therapy
- Surgical therapy

Table 4-5 Medical Management of Postpartum Hemorrhage

- Continuous monitoring
  - Vital signs, urine output, mental status
  - Blood loss
- Resuscitation
  - Fluids, blood, blood products
- Uterine massage
- Uterotonic agents
  - Oxytocin, ergotamine, prostaglandins
- Tamponade (balloon) or uterine packing (see Figs. 4-4, 4-5, and 4-6)
- Replace uterus if uterine inversion

Table 4-6 Surgical Management of Postpartum Hemorrhage

- Dilation and curettage of retained products
- Repair of lacerations
- Uterine compression sutures
  - B-Lynch brace (see Fig. 4-4)
  - Hayman (see Figs. 4-5 and 4-6)
  - Quahba (see Fig. 4-7)
- Arterial ligation
  - Uterine, utero-ovarian, hypogastric (see Fig. 4-9)
- Hysterectomy
- Pelvic embolization

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uterine balloon is crucial to stop bleeding. It is important to emphasize that if uterine atony continues despite the use of massage and uterotonic agents, the patient should be moved to the operating room for proper evaluation (rule out the presence of retained tissues, cervical or vaginal lacerations), and for insertion of the balloon for tamponade. If bleeding continues despite uterine packing or tamponade, the patient should receive immediate laparotomy and then considering the use of one of the uterine compression sutures described later.

Balloon Tamponade of Uterine Cavity

This procedure consists of insertion of a silicone or rubber-shaped balloon in the uterine cavity with subsequent filling of the balloon with a certain volume of normal saline. Balloon tamponade can be used as a temporary measure to slow down the bleeding in preparation for surgery and/or to allow for resuscitation and availability of blood and blood products. It may also be useful during maternal transfer of the patient from a level-1 hospital to a tertiary care facility. In vaginal delivery, the balloon is inserted after complete examination of the uterine cavity, cervix, and vagina to exclude the presence of retained products of conception or lacerations. The balloon should be inserted above the cervical internal os, and is then filled with 300 to 400 mL of warm saline. The balloon is held in place with vaginal packing in case of complete cervical dilation to prevent it from prolapsing into the vagina. It is important to observe for cessation of bleeding after insertion (the tamponade test). If bleeding continues, uterine tamponade will not work. Balloon tamponade can also be used to treat uterine atony following cesarean section. Following balloon tamponade, the patient should be closely monitored with serial measurements of vaginal bleeding, vital signs, and fluid intake and output. Broad-spectrum antibiotics and continuous IV oxytocin should be administered as long as the balloon is in place. The balloon is then gradually deflated over a 24-hour period. Balloon tamponade is successful in approximately 80% of cases.

Balloon tamponade can be achieved with a Foley catheter balloon that usually holds a maximum of 80 mL, which is readily available in any labor and delivery unit. In this case, at least four or five Foley balloons may be needed to be effective in the case of a term uterus. There are also several uterine balloons that are available for uterine tamponade in cases of uterine atony. The SOS Bakri catheter balloon was the first to be introduced for this purpose. It can hold up to 500 mL of fluid. Proper positioning of the balloon for this purpose is described in Figure 4-1A and improper positioning in Figure 4-1B. The BT-Cath catheter is also effective for this purpose (Fig. 4-2). The U.S. Food and Drug Administration (FDA) has approved the Epps balloon for uterine tamponade in cases of postpartum hemorrhage (Fig. 4-3). Note that this balloon has a large uterine bag and a smaller cervical bag that can be used to tamponade bleeding from the cervix. It can also be used for pelvic packing in case of diffuse oozing in the pelvis. For proper inflation and insertion of these balloons, please refer to the DVD.

Uterine Compression Sutures

The B-Lynch suture requires the patient be in Lloyd-Davis (frog-legged) position. At the time of surgery, bimanual compression by the surgeon should be performed to test for potential benefit. During compression, a nurse should

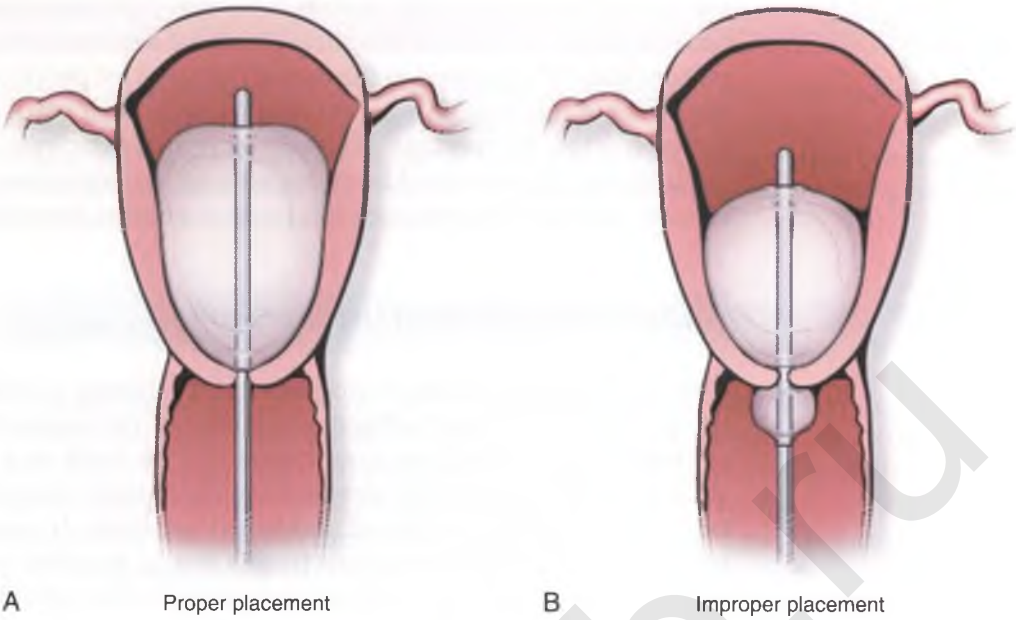

Figure 4-1 SOS Bakri balloon catheter. **A,** Demonstrates proper placement. **B,** Demonstrates improper placement.

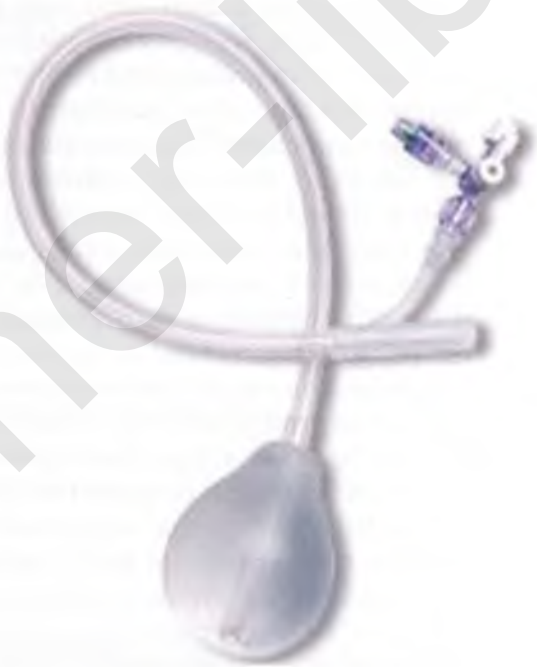

Figure 4-2 BT-Cath balloon catheter for uterine tamponade.

observe for decrease or cessation of vaginal bleeding. If successful, incision is made in the lower uterine segment (if patient had a vaginal delivery), the uterine cavity is emptied from blood and clots, and a No. 1 chromic or Vicryl long suture is placed as described in Figure 4-4A. The two ends of the suture are then pulled tight while the uterus is compressed by an assistant. At this time, the vagina is checked again for bleeding; if none is observed the suture is tied while the uterus is compressed (see Fig. 4-4B). The benefits of the B-Lynch suture involve checking for absence of clots in the uterine cavity and leaving the endometrial cavity patent, which allows for subsequent drainage of blood and lochia. At the end of the procedure, the vagina is checked again for adequate hemostasis before the abdomen is closed.

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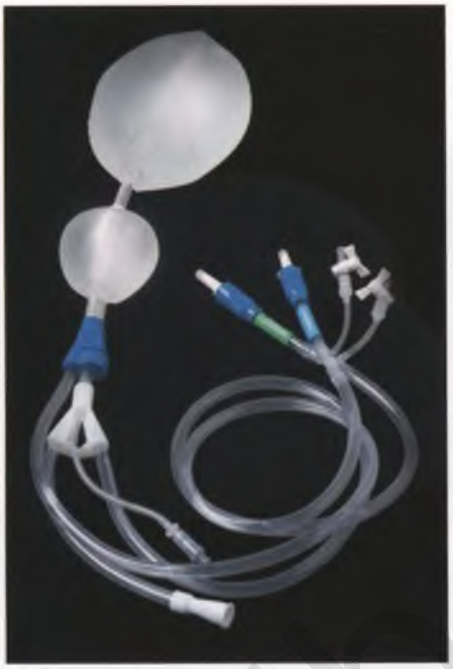

Figure 4-3 Epps balloon catheter for uterine tamponade.

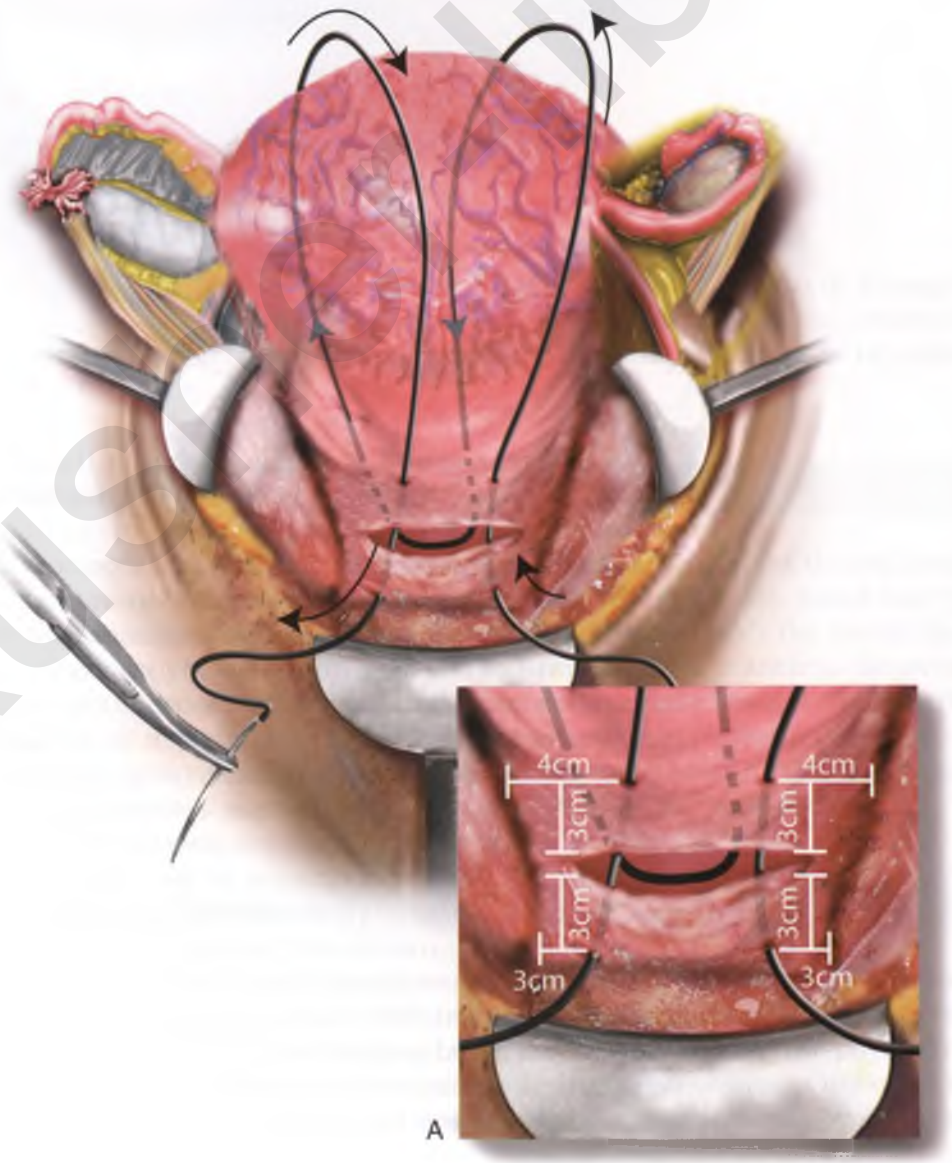

Figure 4-4 A, B-Lynch uterine compression suture.

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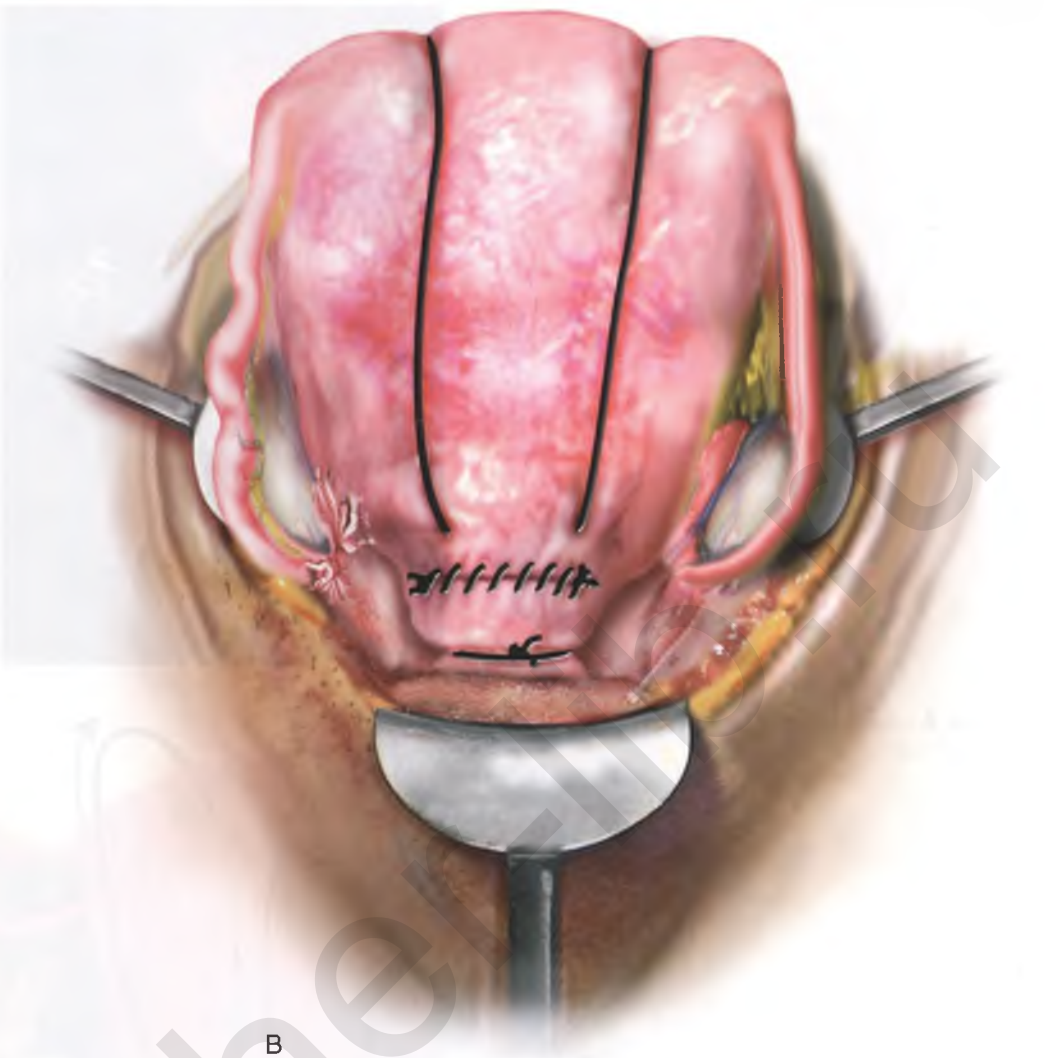

Figure 4-4, cont'd B, B-Lynch suture demonstrating uterine compression.

The Hayman suture is similar to that of the B-Lynch except that it is performed without inspecting or opening the uterine cavity. Two vertical sutures are usually used to compress the uterine cavity from anterior to posterior wall. The needle is inserted starting from the front to the back of the uterus above the bladder reflection. The compression sutures are tied at the top of the fundus. If bleeding continues, two additional vertical sutures are added (Fig. 4-5). In case of bleeding from placenta previa extending down to the cervical internal os, two isthmic cervical compression sutures can be used to control bleeding from this area. In this case, it is advisable to pass a closed artery forceps in the cervical lumen before tying the suture to ensure patency of the cervical os (Fig. 4-6). The Hayman compression suture is quicker to perform; however, it may result in complete obliteration of the uterine cavity and it does not allow for direct visualization of the cavity. In addition, there are limited data about future pregnancies after this procedure.

The Ouahba sutures involve two lateral sutures placed near the uterine horns and two transverse sutures placed above and below the lower segment uterine incision (Fig. 4-7A). The two top lateral sutures control bleeding in the fundus and obstruct blood supply from utero-ovarian blood vessels. The lower transverse suture is effective in controlling bleeding from the descending branch of uterine arteries (see Fig. 4-7B).

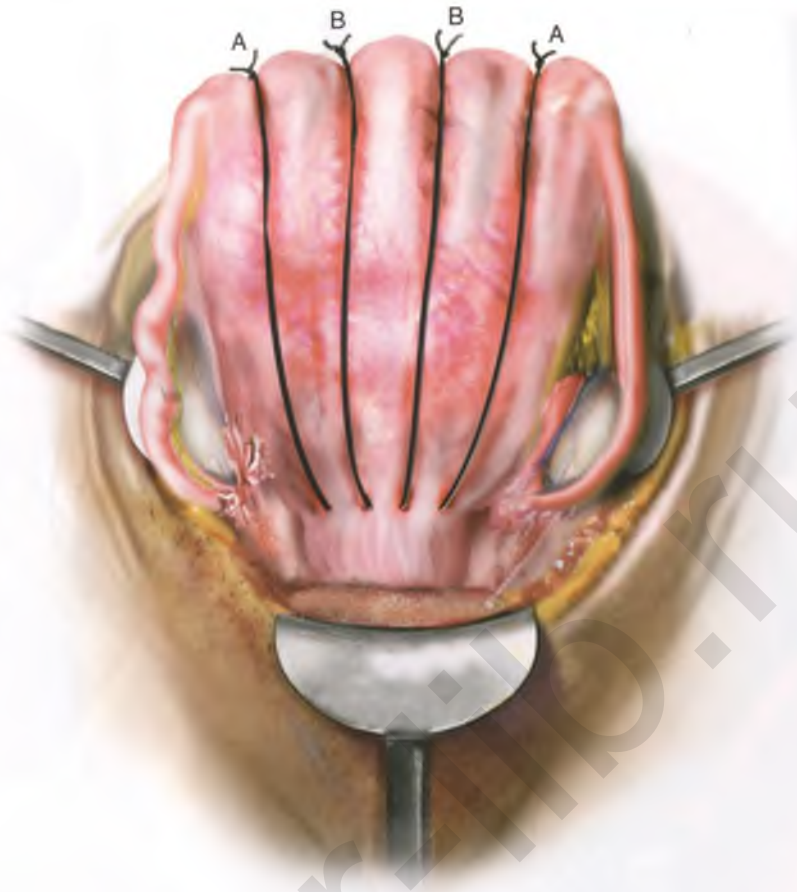

Figure 4-5 Hayman uterine compression suture.

If the uterine compression sutures are not successful, subsequent management can include either stepwise uterine devascularization sutures, cesarean hysterectomy, or selective pelvic arterial embolization (if patient is in stable condition).

Ligation of Pelvic Vessels

Vascular supply to the uterus and pelvic organs is very complex during pregnancy because of the extensive collateral circulations (Fig. 4-8). Blood supply to the uterus is mostly (90%) from the uterine arteries, with the remainder being supplied by the ovarian arteries and other pelvic branches. Stepwise pelvic arterial ligation (uterine arteries, utero-ovarian arteries and hypogastric arteries) should only be used when tamponade and compression sutures fail to stop bleeding in a young patient who desires future childbirth. The technique of uterine artery and utero-ovarian ligation is shown in Figure 4-9.

Embolization of Uterine or Pelvic Arteries

Uterine artery embolization should be considered only in patients who desire future childbirth and who are hemodynamically stable. This procedure requires transfer of the patient to a radiology suite and the availability of physicians with the expertise to perform these procedures. The procedure is effective in almost 90% of cases with uterine atony; however, it is less effective in the

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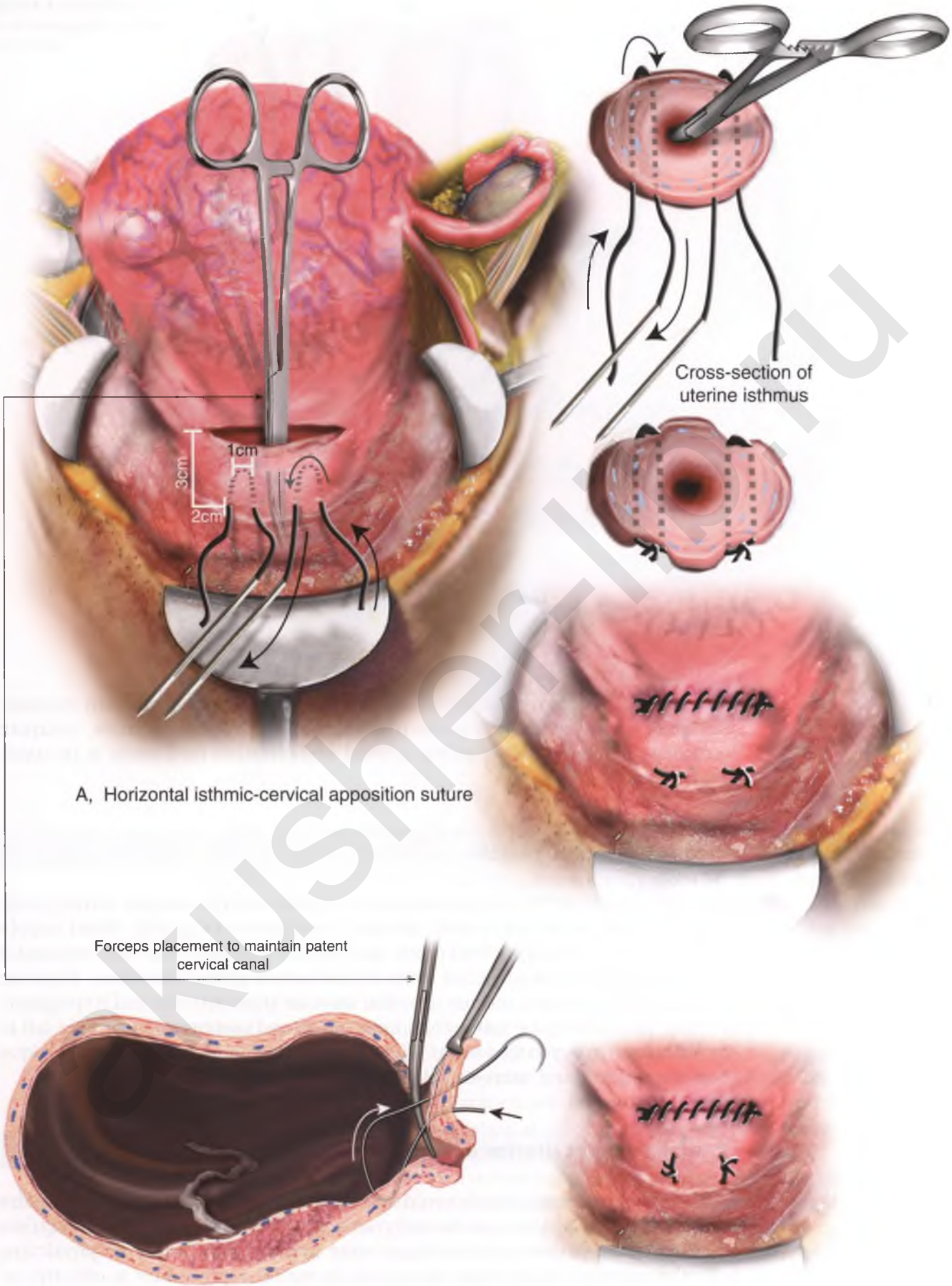

Figure 4-6 **A** and **B,** Hayman cervical compression suture.

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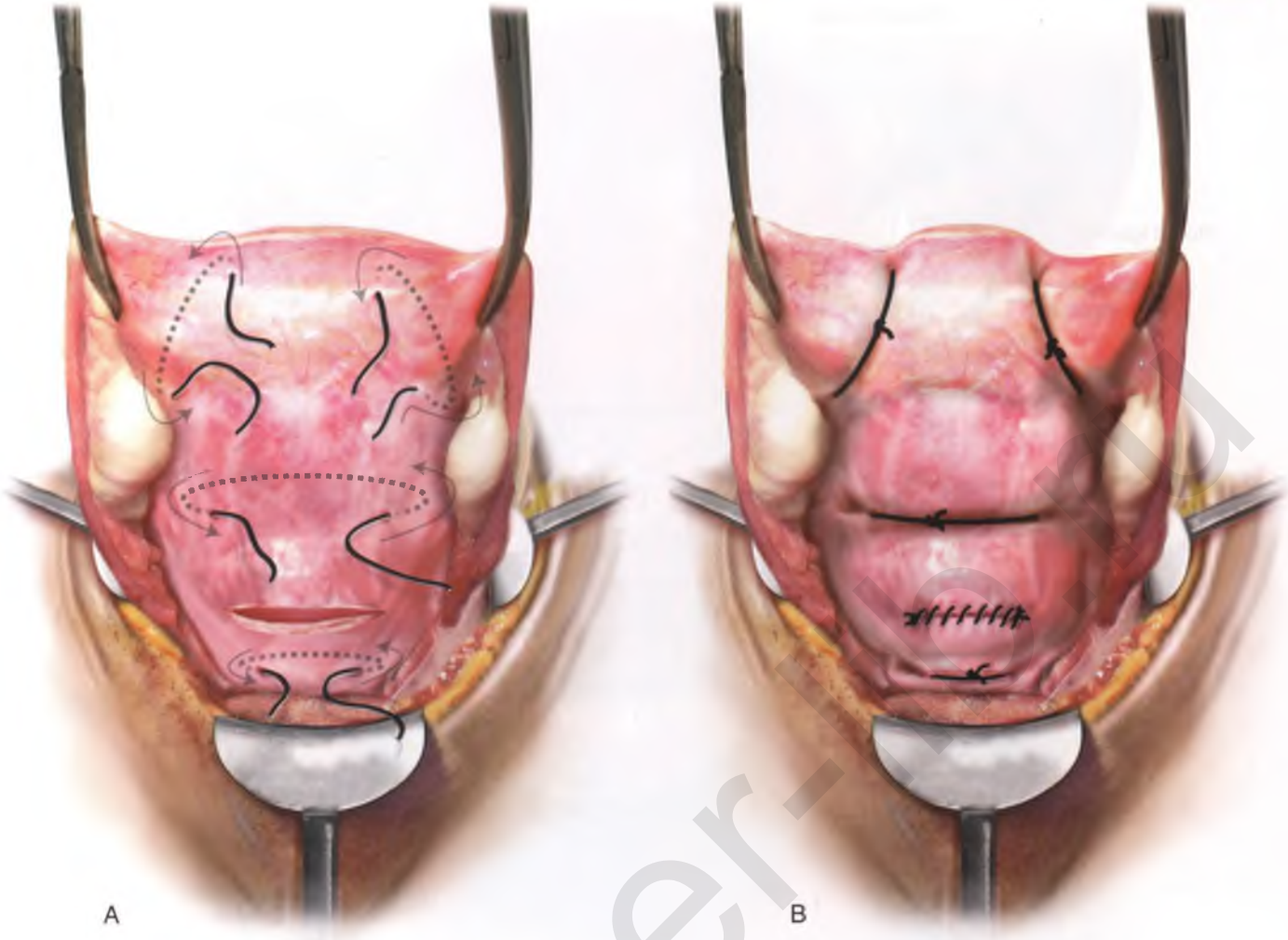

Figure 4-7 **A** and **B,** Quahba uterine compression sutures.

presence of placenta accreta/percreta. Pelvic arterial embolization is also useful when the bleeding is secondary to vessels other than the uterine arteries. In this case, the bleeding vessel is visualized and selectively embolized to control the source of the bleeding.

Cesarean Hysterectomy

This procedure can be lifesaving when the bleeding is either from the uterus or cervix, and it does not respond to other measures discussed earlier. The decision for hysterectomy should be made as soon as possible, particularly prior to the development of DIC. It should be considered the treatment of choice in older patients, those with multiple cesarean section, and those with complete previa or accreta. For a detailed description and method of cesarean hysterectomy, refer to the DVD.

The performance of cesarean hysterectomy requires planning if possible (placenta accreta, multiple cesarean sections, or previous cesarean plus central

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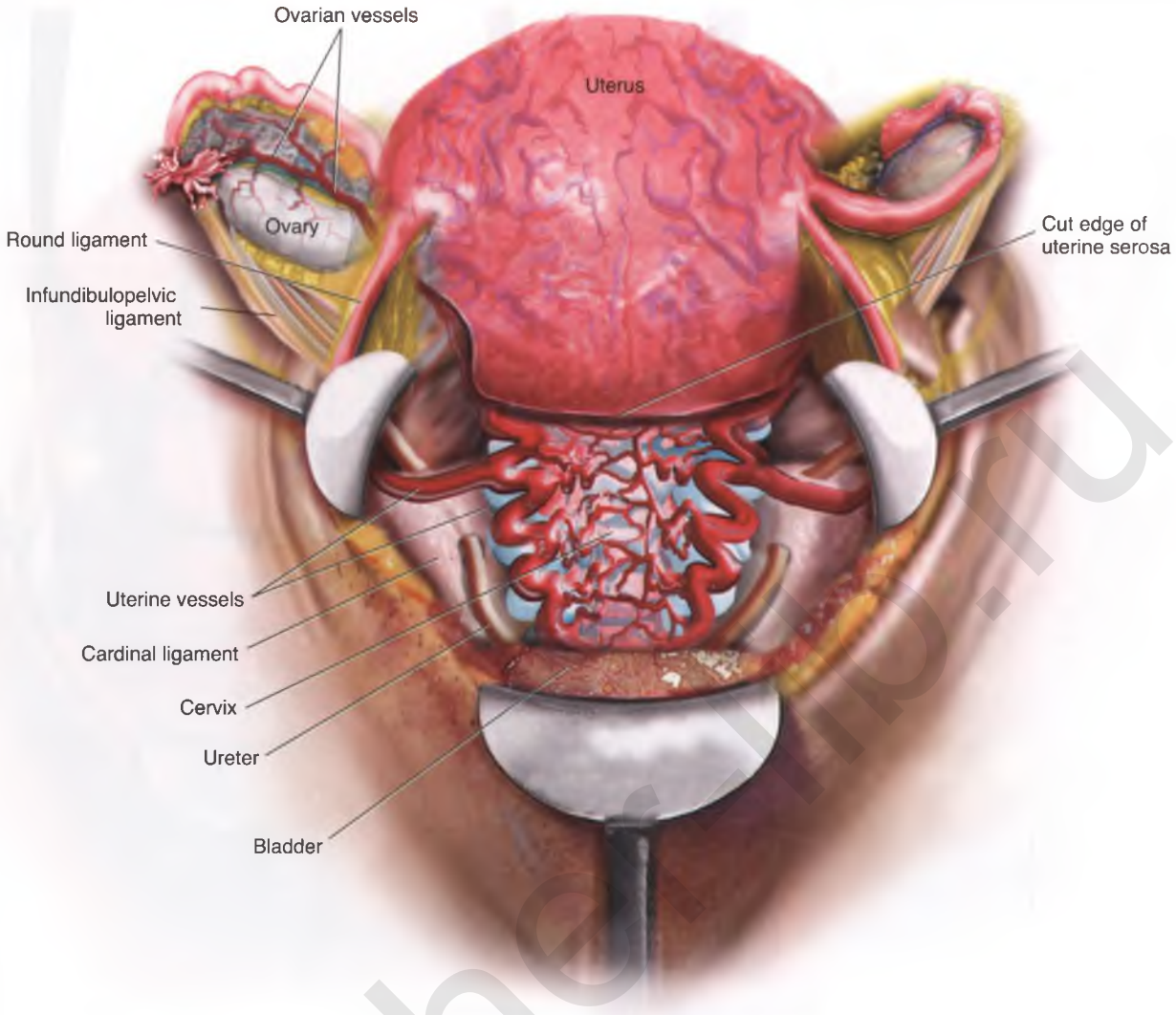

Figure 4-8 Vascular supply to the uterus showing collateral circulation.

placenta previa), and the availability of surgeons with the required skills to perform such procedure. Cesarean hysterectomy is usually more difficult to perform as compared to nonpregnant hysterectomy because during pregnancy the pelvic vessels are much larger and tortuous, which requires careful dissection, and the pelvic tissues are usually edematous and friable, particularly in patients with prolonged labor or prolonged second stage or chorioamnionitis. In such cases, it is difficult to identify the external os of the cervix, and the bladder wall can be very edematous. In addition, pelvic visualization of anatomic structures can be difficult because of bleeding. The decision between total and supracervical hysterectomy should be based on maternal condition, presence of DIC, and the source of bleeding (atony versus accreta, cervical previa, cervical lacerations).

In order to have adequate visualization of the various pelvic structures, it is advisable to perform a vertical abdominal incision and vertical uterine incision if applicable (total placenta previa or accreta with previa), and to place a Bookwalter or Balfour abdominal retractor.

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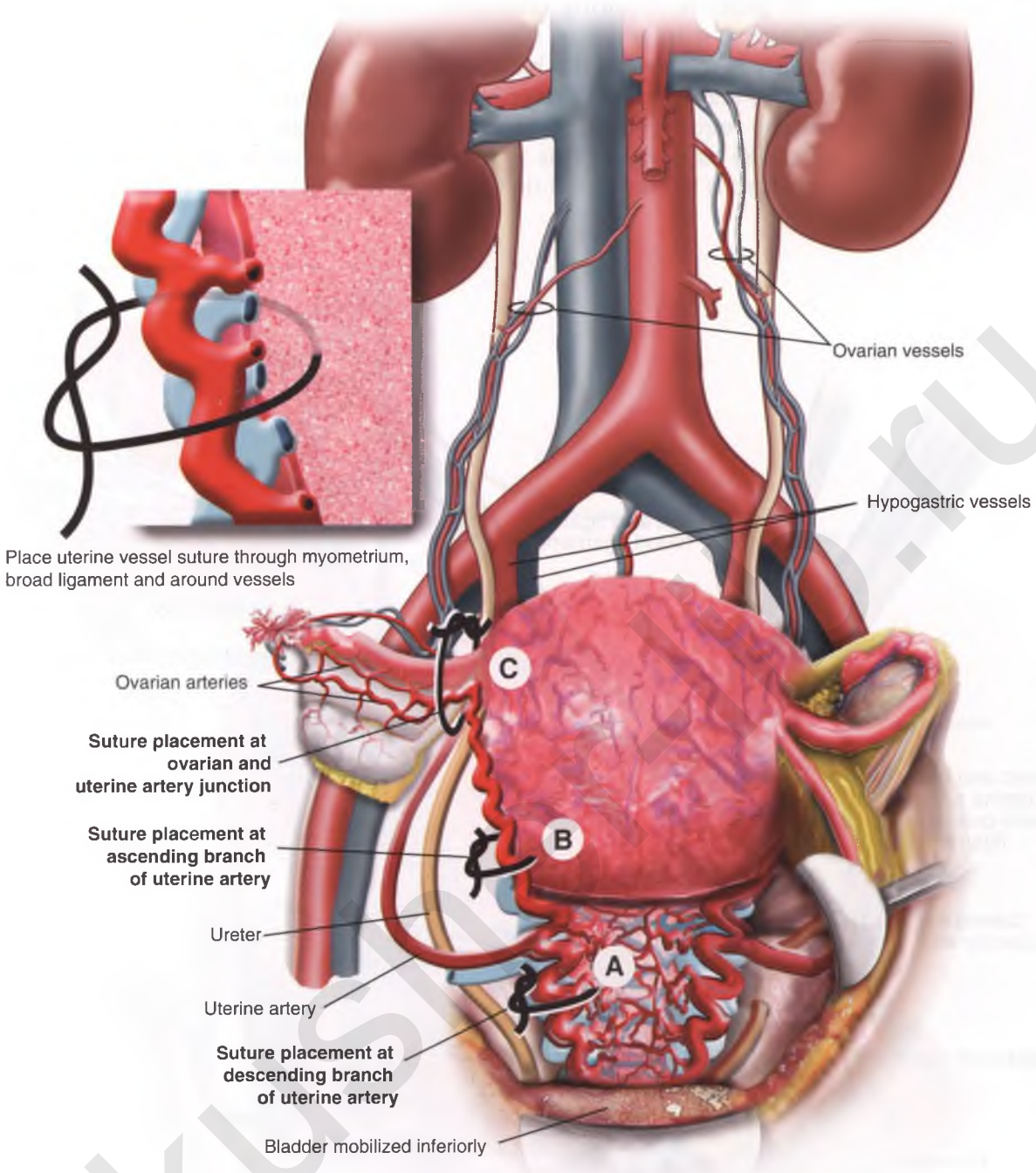

Surgical ligation locations of uterine blood supply

Figure 4-9 Uterine artery and utero-ovarian artery ligation.

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In patients requiring emergency hysterectomy following vaginal delivery because of uterine atony, rupture, or major lacerations, the first step after laparotomy is to develop the bladder flap that can be a opened with Metzenbaum scissors and extended from one edge of the round ligament to the other. If the patient requires hysterectomy during cesarean section, the bladder flap is extended as needed. This is followed by closure of the uterine incision. The next step is to clamp and tie the round ligament on each side (Fig. 4-10A). The

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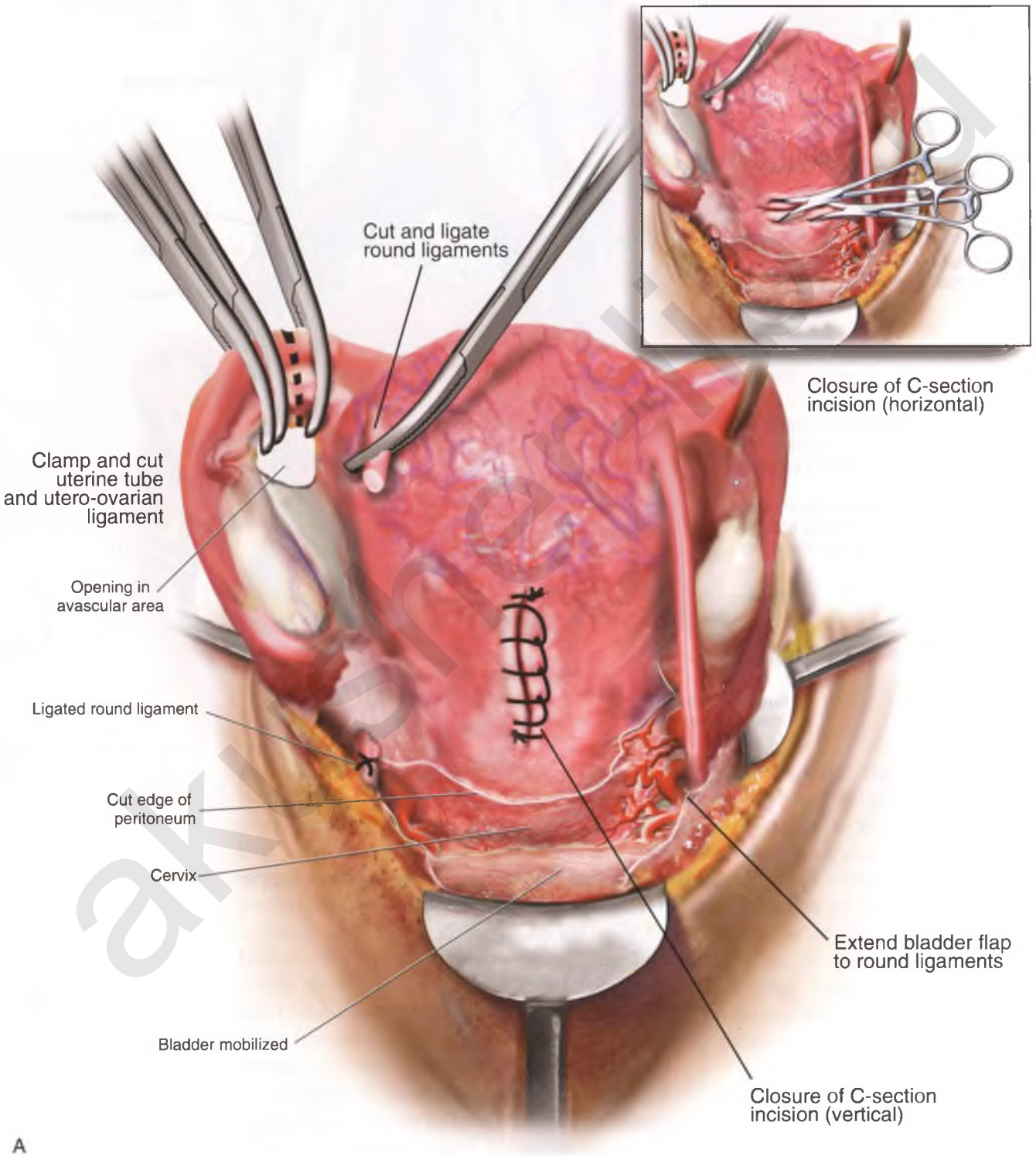
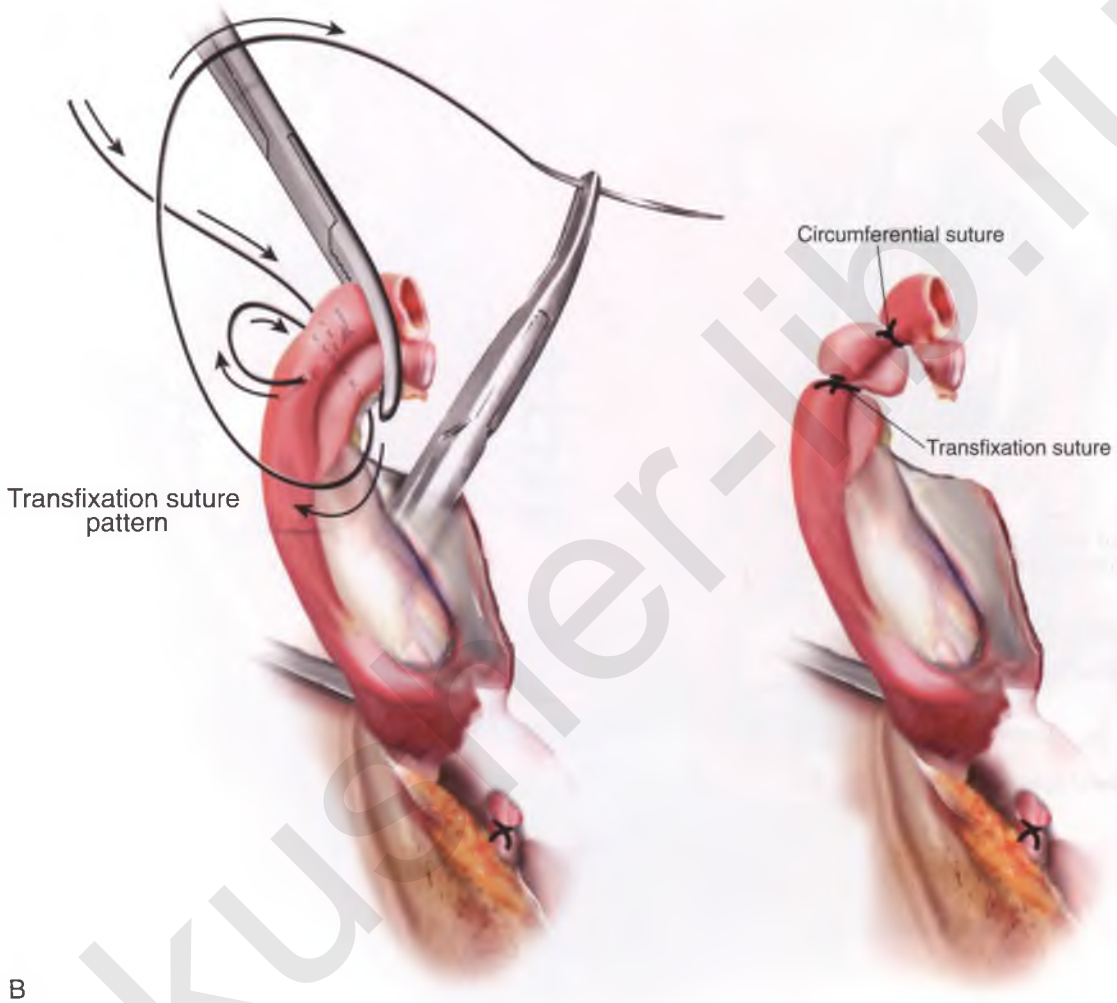


Figure 4-10 A, Technique of cesarean hysterectomy showing development of bladder flap, ligation of round ligament.



B

Figure 4-10, cont'd B, Technique of clamping and ligation of uterotubal and ovarian vessel.

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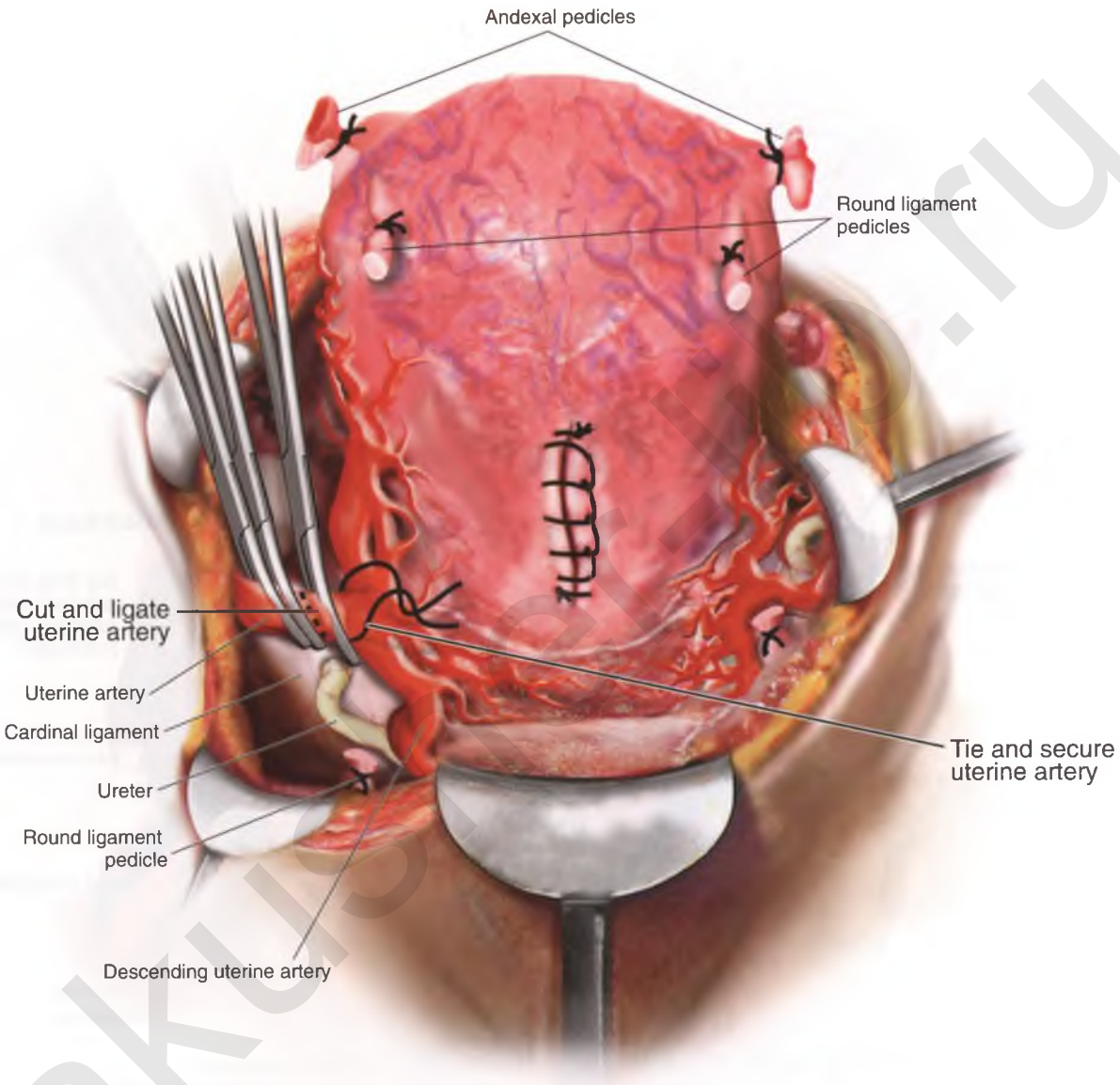

C

Subtotal (supracervical) cesarean hysterectomy

Figure 4-10, cont'd C, Clamping and ligation of uterine artery.

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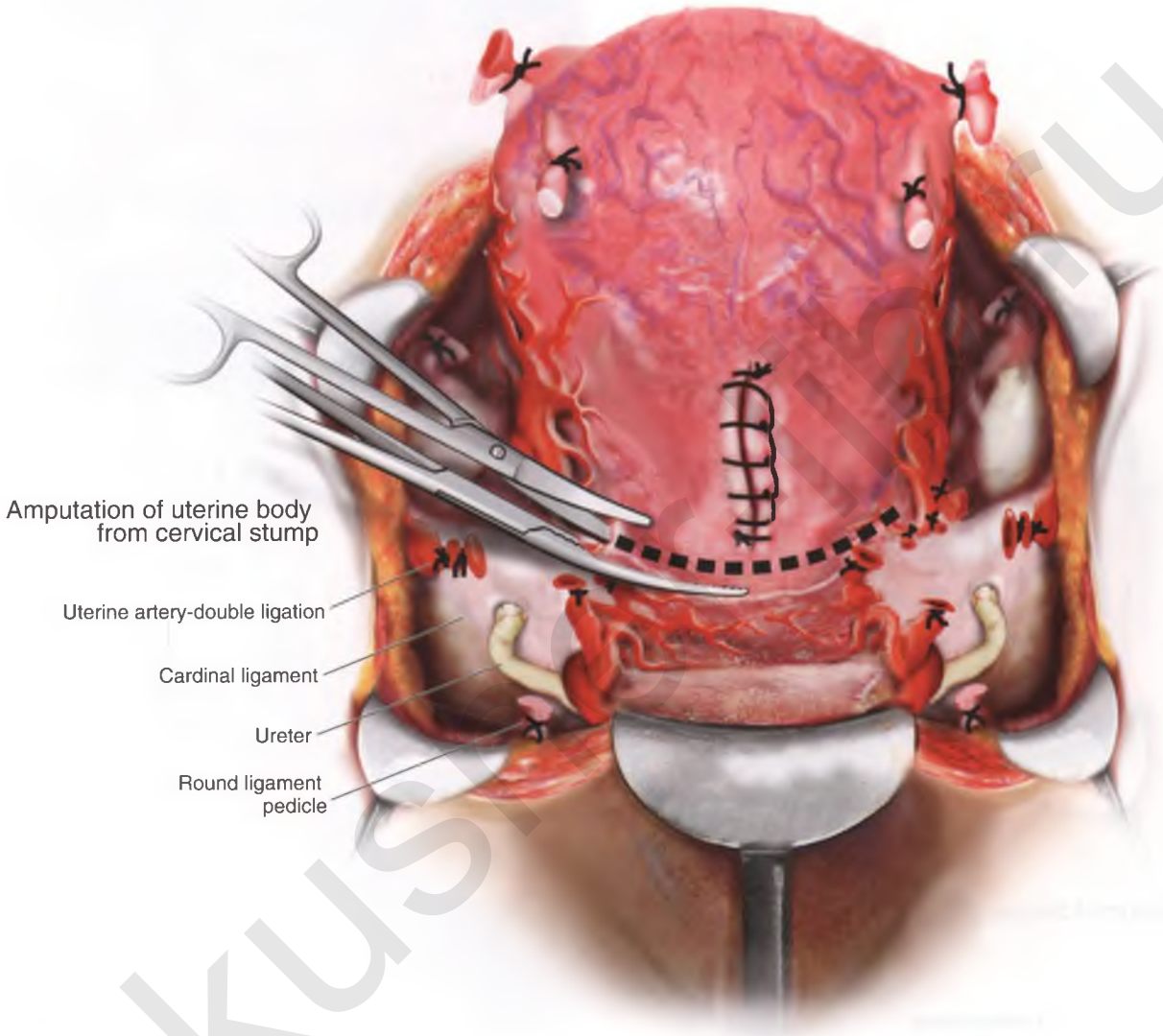

D

Subtotal (supracervical) cesarean hysterectomy

Figure 4-10, cont'd D, Technique for supracervical hysterectomy showing amputation of uterine fundus.

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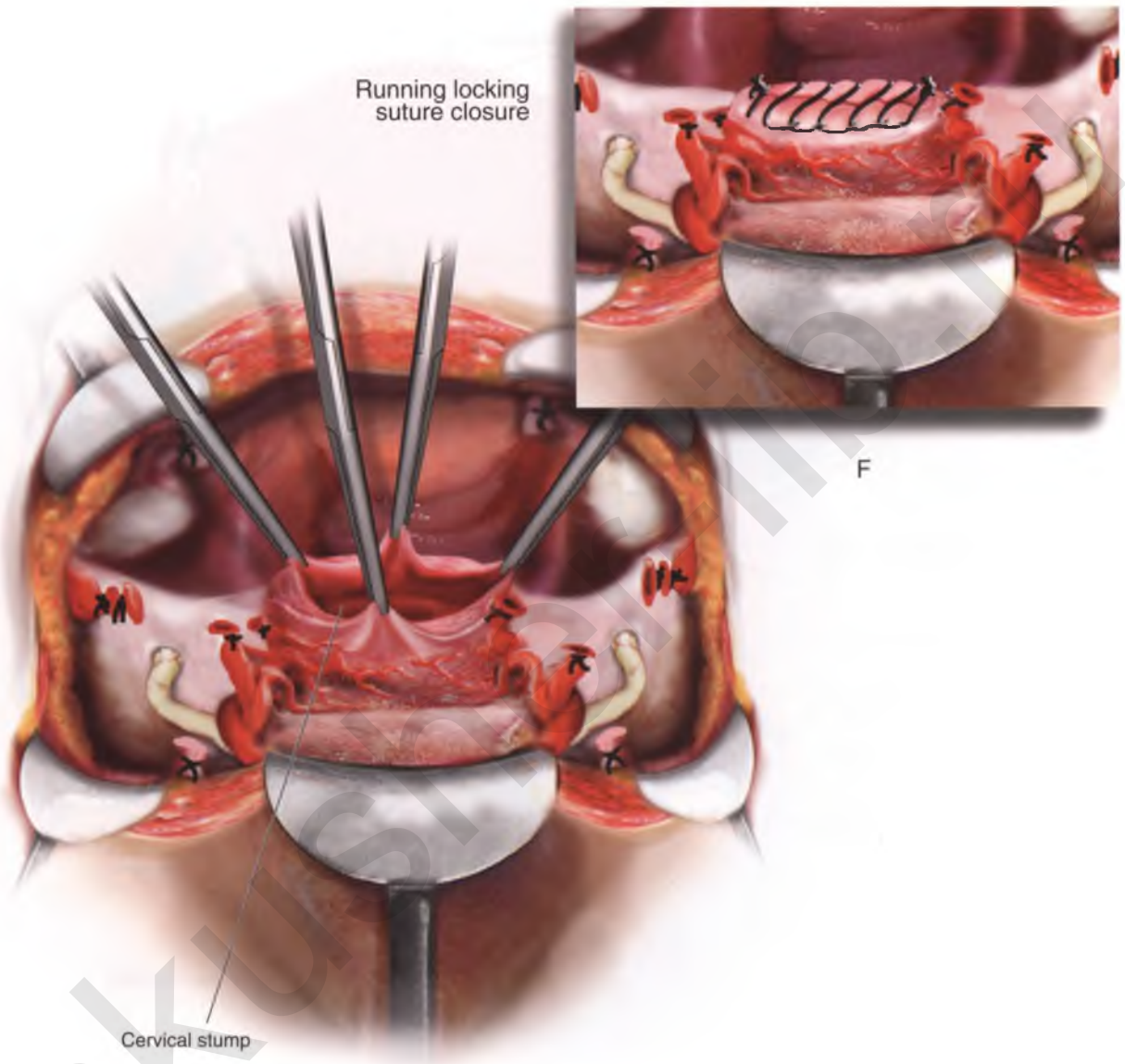
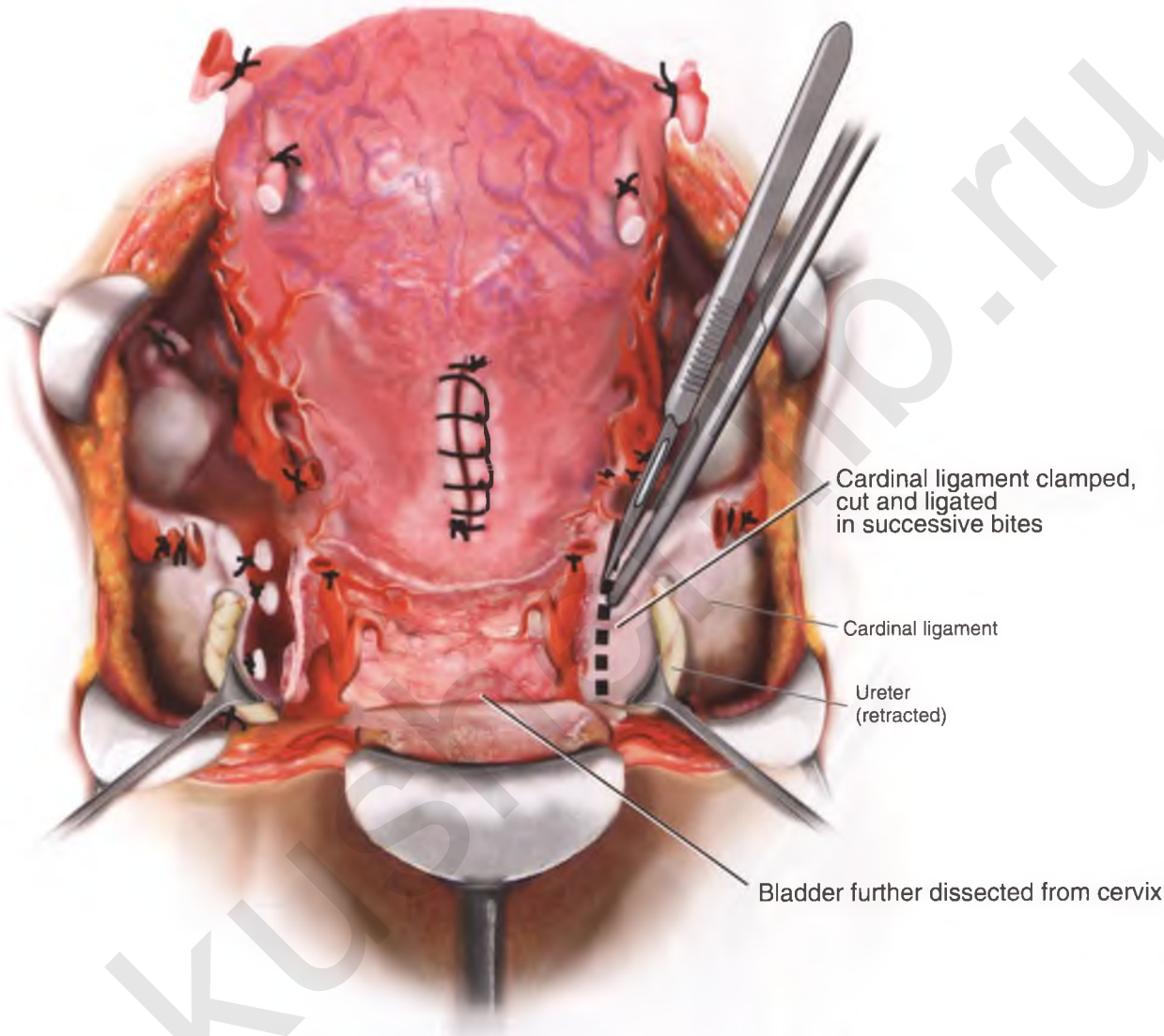


Subtotal (supracervical) cesarean hysterectomy

Figure 4-10, cont'd E and **F,** Demonstration of cervical stump and closure after removal of uterus.



G **Total cesarean hysterectomy**

Figure 4-10, cont'd G, Cardinal ligament being cut and ligated in steps. Note the location of the ureter retracted away from the clamp.

Continued

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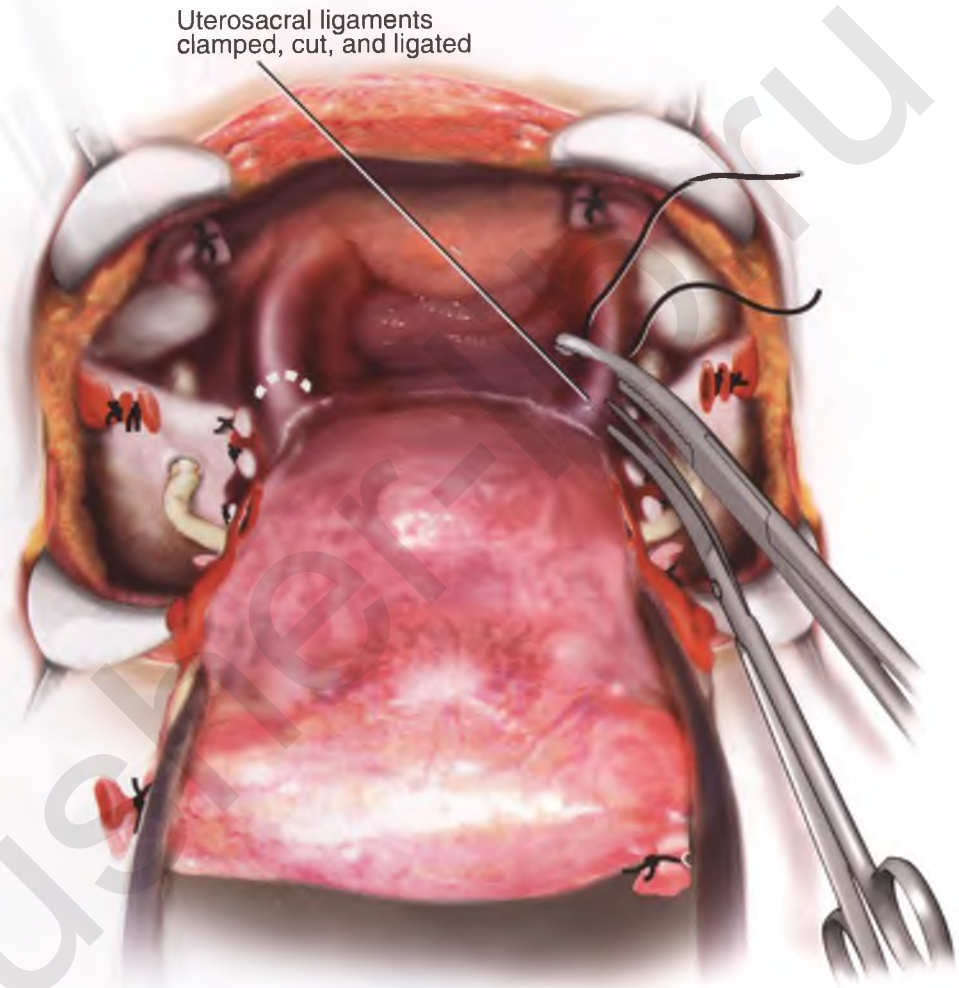

H **Total cesarean hysterectomy**

Figure 4-10, cont'd H, Clamping and ligation of uterosacral ligament.

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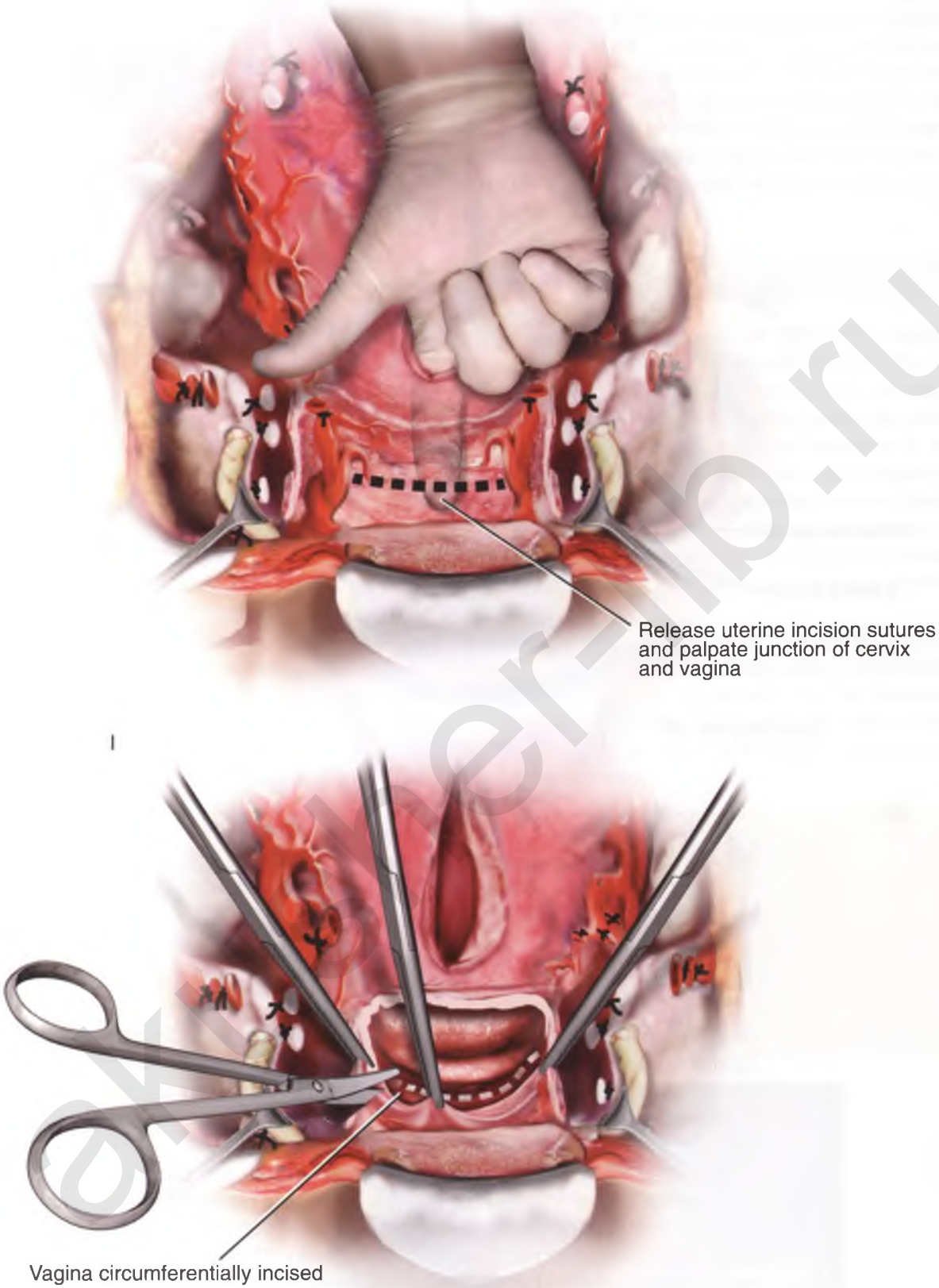

Total cesarean hysterectomy

Figure 4-10, cont'd I, Amputation of the uterus from the cervical stump with uterine and cardinal ligaments cut and ligated.

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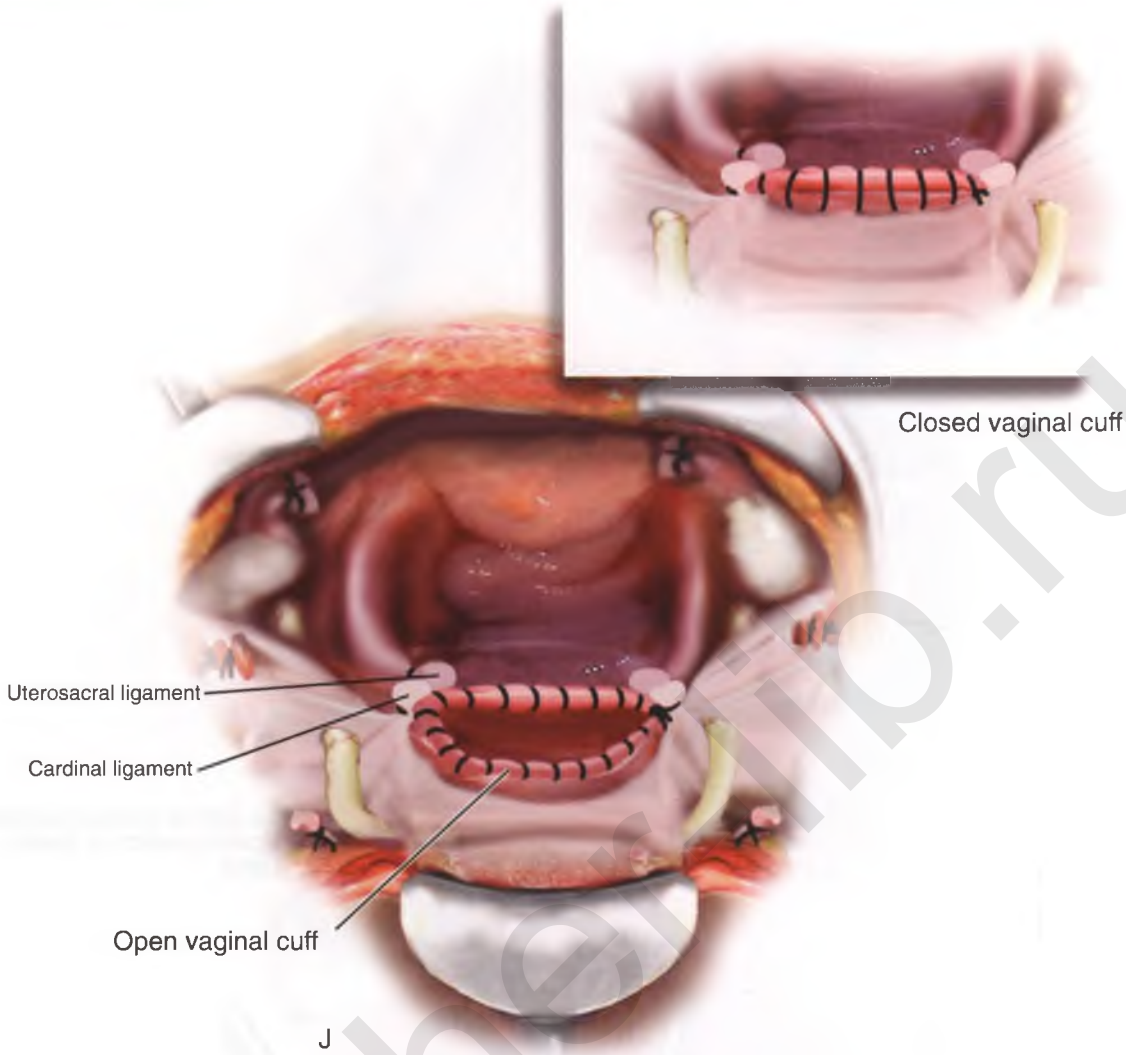

Total cesarean hysterectomy

Figure 4-10, cont'd J, An open and closed vaginal cuff.

next step is to identify and develop a pedicle that includes the tube, uterotubal ligament, and utero-ovarian vessels. The pedicle on each side is then clamped adjacent to the uterus using Heaney clamps. This may include either a three-clamp or a two-clamp technique. The tissue between the clamps is then cut and the pedicles are suture ligated (see Fig. 4-10B).

The ascending branches of the uterine artery are then identified near the junction of the cervix and uterus. The artery is clamped with a Heaney clamp with the tip resting along the lateral edge of the uterus and perpendicular to the vessel (two or three clamps can be used). The uterine vessels are then cut and suture ligated. The suture should be placed just below the tip of the clamp and next to the uterine wall (see Fig. 4-10C). After completion of this step, the surgeon can proceed with a supracervical hysterectomy by removing the fundus from the lower segment (cervix) using scissors (see Fig. 4-10D). The top edges of the remaining cervical tissue are then closed with a running locking suture (see Fig. 4-10E and F).

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For total hysterectomy the anterior wall of the cervix should be dissected free from the bladder wall, and then the cardinal ligaments are clamped and cut and ligated using successive bites with a straight Heaney or Ballantine clamp. It usually takes an average of three bites of tissue of 1.5 cm in size (see Fig. 4-10G). The next step is to clamp, cut, and ligate the uterosacral vessels (see Fig. 4-10H). The junction of cervix and vagina is then identified and the vagina is circumferentially incised with complete removal of the cervix (see Fig. 4-10I). Finally, the vaginal cuff edges are sutured with the cuff kept open or completely closed (see Fig. 4-10J).

Uterine Inversion

Uterine inversion is a rare but life-threatening cause of PPH. The exact incidence is unknown, but ranges from 1:1000 to 1:4000 deliveries. The exact etiology is unknown, but it usually develops in association with certain risk factors (Table 4-7). Uterine inversion can be complete in which case the inverted fundus is seen at or beyond the introitus (Fig. 4-11), or incomplete in which the inverted uterus is palpated inside the vagina. The signs and symptoms of uterine inversion include failure to palpate the uterine fundus, severe hypotension, shock, and profuse bleeding. The hypotension may be associated with reflex bradycardia because of vagal response secondary to traction on uterine ligaments. In certain cases, the degree of shock can be out of proportion to the amount of bleeding.

Puerperal uterine inversion can result in substantial maternal morbidity and mortality, particularly when there is delay in diagnosis and prompt replacement and correction of maternal shock. It is important not to remove the placenta in case of uterine inversion since this will result in severe hemorrhaging. In addition, keeping the placenta in place will make it easier for the replacement of the inverted uterus.

Table 4-7 Risk Factors for Uterine Inversion

- Uterine atony in association with:
  - Relaxed lower segment
  - Relaxed cervix
- Premature/strong traction on the cord
  - Excessive fundal pressure
  - Fundal placenta
  - Short cord
- Adherent placenta
- Chronic uterine overdistention

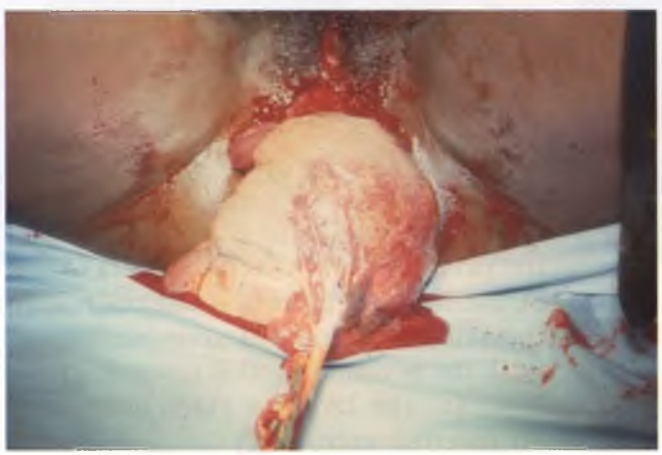

Figure 4-11 Complete uterine inversion.

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Case 2: Uterine Inversion

A 34-year-old G2P0 with gestational diabetes mellitus, fetal macrosomia, and polyhydramnios presents at 33 5/7 weeks' gestation with preterm uterine contractions and cervical effacement. A diagnosis of preterm labor was confirmed for which she was treated with corticosteroids for fetal lung maturity, IV insulin for control of blood sugars, and magnesium sulfate for preterm labor. IV magnesium sulfate was started at a 6-g loading dose and a continuous infusion of 2 g/hr and again to 4 g/hr because of contractions and cervical dilations. IV magnesium sulfate was continued for 4 hours and then discontinued because of advanced cervical dilation and spontaneous rupture of membranes with immediate delivery of a live infant weighing 3480 g. Following delivery of the placenta, there was profuse vaginal bleeding and the patient became hypotensive with a blood pressure of 70/50 mm Hg, and a pulse of 58/min. Pelvic examination revealed the presence of complete uterine inversion. The obstetrician called for help, and maternal resuscitation was started with the plan for manual correction of the inversion. An anesthesiologist administered 100 mg of IV glyceryl trinitrate for uterine relaxation, and the obstetrician attempted to push the uterine fundus through the cervical ring as described in Figure 4-12. This maneuver was attempted twice, but failed. Following this, the O'Sullivan hydrostatic reduction technique was used with successful uterine replacement (Fig. 4-13). Immediately after replacement, continuous infusion of oxytocin was started, and 0.2 mg of methylergonovine was given to restore uterine tone. The estimated blood loss was 1500 mL, and the patient received 2 L of crystalloids and 4 units of packed red blood cells to correct hypovolemia and hypotension. Her blood pressure and pulse subsequently stabilized, and she was discharged home 2 days later with a hematocrit of 27%.

Discussion

This patient had chronic uterine muscular stretch because of macrosomia and polyhydramnios, and prolonged uterine relaxation from the use of large doses of IV magnesium sulfate. As a result, both the uterus and the cervix failed to contract after delivery of the placenta, increasing her chance for uterine inversion. The diagnosis was promptly made, and the managing physician appropriately called for help for management of maternal shock, and for rapid correction of uterine inversion by manual uterine replacement.

Management of the patient with uterine inversion includes early diagnosis, maternal resuscitation for correction of hypovolemia and shock, and uterine replacement. A stepwise approach for management of puerperal uterine inversion, including medical and surgical procedures, is described in Figure 4-14.

Manual Replacement (See Fig. 4-12)

This procedure requires the administration of uterus-relaxing agents such as IV magnesium sulfate, terbutaline, or nitroglycerin. It takes approximately 10 minutes for magnesium sulfate to be effective, whereas the effects of IV terbutaline (0.25 mg) are seen within 2 minutes. In contrast, IV nitroglycerin has immediate onset of action with very short half-life, which makes it the ideal drug of choice. If the patient is hemodynamically unstable, it is best to use halothane with general anesthesia. This procedure is usually successful in 30% to 40% of cases.

The O'Sullivan Replacement Technique (See Fig. 4-13)

This procedure involves infusing 2 to 3 L of warm saline into the vagina under pressure. The fluid is attached to a pole 3 to 4 feet high and the nozzle inserted in the vagina. The introitus is obliterated by approximating the labia together with the fingers of the operator, or by attaching the opening of the infusing tube to a silicone vacuum suction cup held in the vagina at the introitus. This will generate enough intravaginal hydrostatic pressure to push the inverted fundus upward. This procedure has a success rate of almost 80%. See the DVD for video demonstration of technique for replacement of an inverted uterus.

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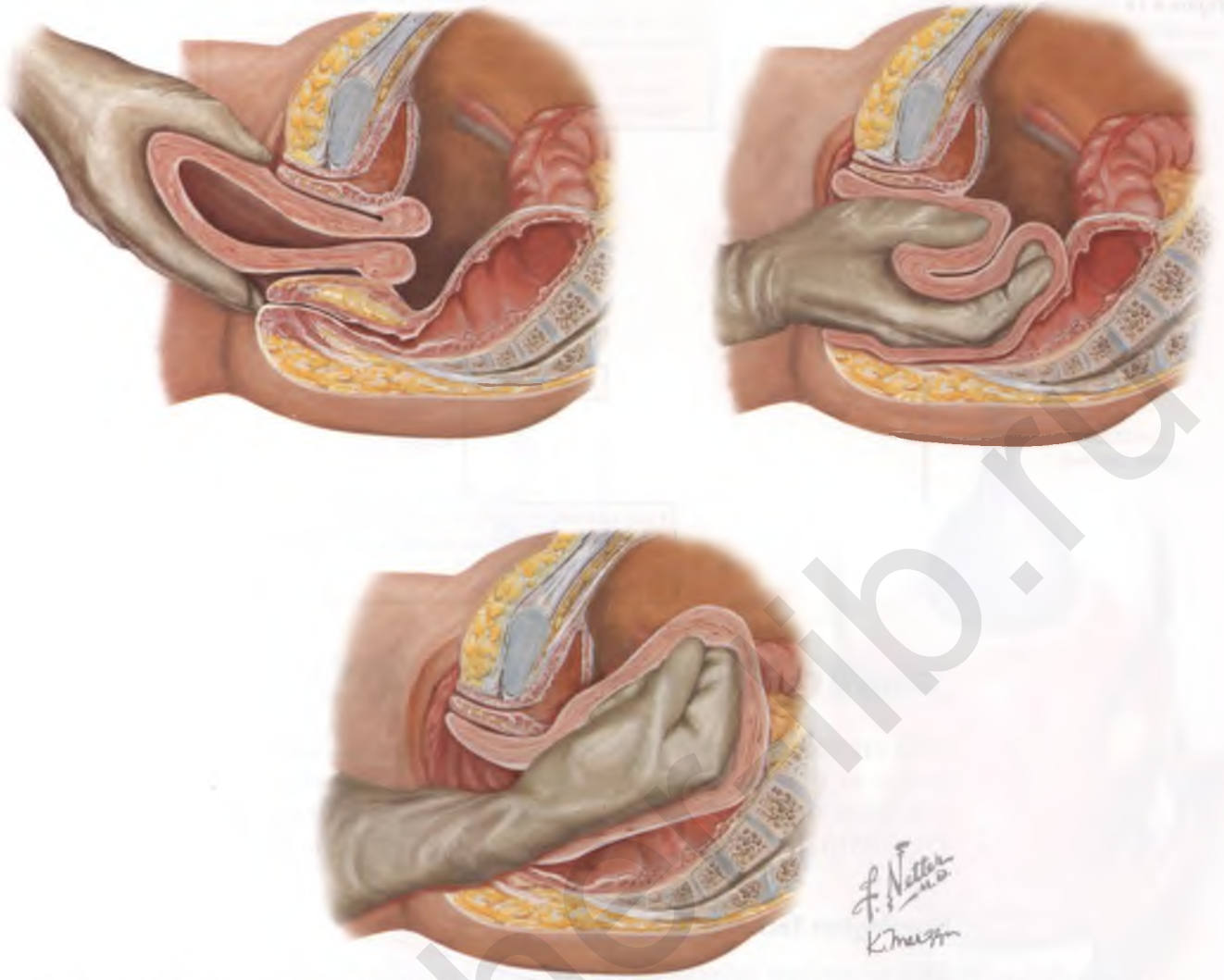

Figure 4-12 Technique for manual uterine replacement. (A Netter illustration from netterimages.com. © Elsevier Inc. All rights reserved.)

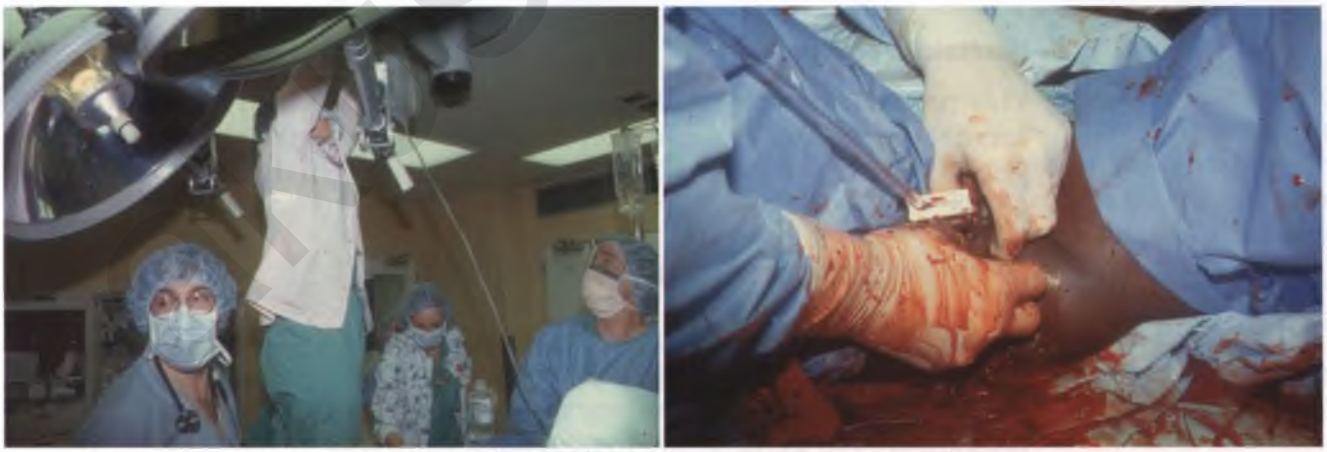

Figure 4-13 O'Sullivan hydrostatic technique for reduction for uterine inversion.

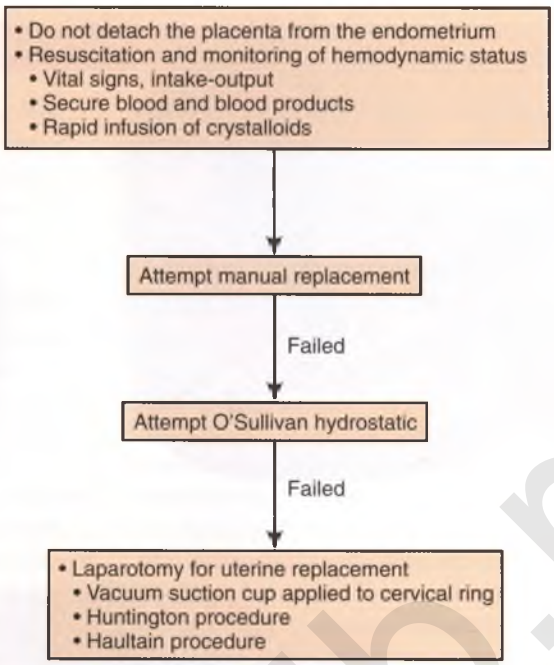

Figure 4-14 Algorithm for management of uterine inversion.

Surgical Correction of Uterine Inversion

This procedure requires laparotomy. A silicone vacuum suction cup is applied firmly to the cup of the uterus created by the inversion (Fig. 4-15A). Using gentle traction, the cup is pulled upward to turn the inside of the uterus out (see Fig. 4-15B). If this fails, the next step is to attempt the Huntington technique.

Huntington Technique

This procedure involves the application of Allis clamps at the site of inversion below the cervical ring with subsequent gentle upward traction. The clamping and traction are repeated several times until the uterine fundus is replaced in the uterine cavity. If this fails, the next step is to attempt the Haultain procedure.

Haultain Procedure

This procedure involves making an incision in the posterior portion of the cervix at the site of the inversion ring. This increases the size of the cervical ring to allow replacement of the fundus.

Preparing for Obstetric Hemorrhage

Because intrapartum-postpartum hemorrhage complicates at least 5% of all deliveries, it is essential that all labor and delivery units be prepared to anticipate and manage PPH. This requires the availability of the proper staff, equipment, and knowledge of blood bank capability at the hospital. In addition, in tertiary care hospitals, it is important to have a massive transfusion protocol in place, which requires a coordinated effort between the massive transfusion protocol team and the blood bank staff. This should also include development of training and performance of clinical drills to test competence and compliance with the protocols. These protocols should take into consid-

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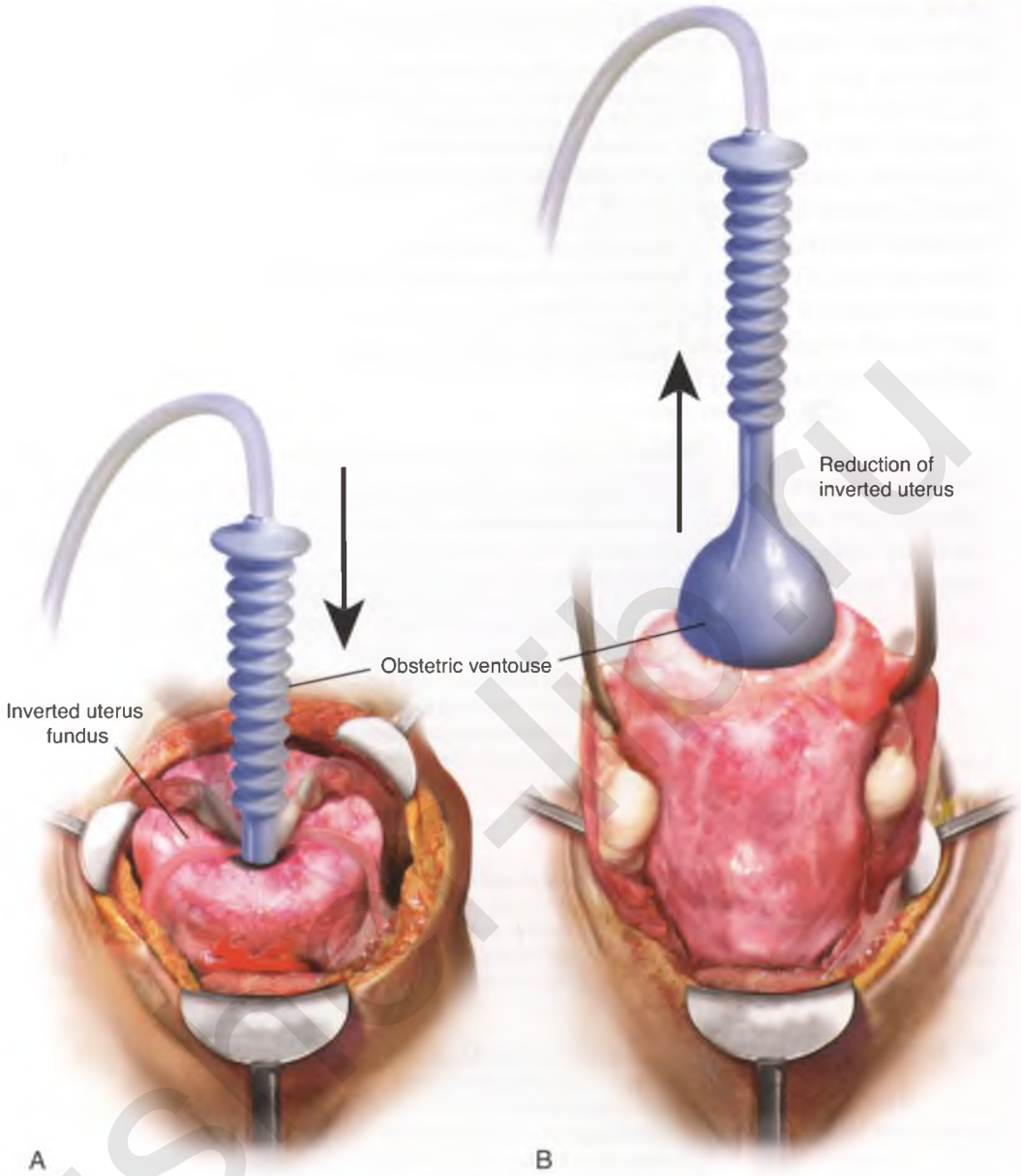

Figure 4-15 **A** and **B,** Use of silicone vacuum suction cup for correction of uterine inversion at time of laparotomy.

eration the available resources at a certain hospital (Table 4-8). Because time is critical in replacing blood and blood products, it is essential that all personnel be aware and educated about the time it takes at their hospitals to order and obtain various blood types, blood products, and coagulation factors, and to know the amount needed, the equipment used for rapid infusion and warning, as well as potential complications associated with massive transfusions (Table 4-9).

Massive obstetric hemorrhage defined as blood loss of more than 2000 mL complicates approximately 0.5% of all pregnancies. It is usually seen in cases of delayed management of uterine atony, abruptio placentae with DIC, placenta accreta/increta, or the need for cesarean hysterectomy. It is associated with hypovolemic shock, a hemoglobin of ≤6 g/dL, DIC, acidosis, and hypothermia. Delay in treatment results in ischemic renal injury, myocardial and cerebral ischemia, ARDS, and death. Therefore, it requires immediate detection and management as described in Table 4-10. A suggested protocol for massive transfusion is described in Table 4-11.

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Table 4-8 Policy and Procedures for Postpartum Hemorrhage

- Uniform policy for early detection and monitoring for PPH
- Consider available resources (individualize)
  - Rural hospital (basic protocol)
    - Criteria for immediate referral
    - Temporary measures for stabilization
  - Secondary hospitals
  - Tertiary hospitals
- Identify a multidisciplinary team
  - Obstetrician, anesthesiologist, other specialists
  - Nurse-midwives, nurses
  - Blood bank, hematologists
  - Regular training and education (drills)

Table 4-9 Complications from Massive Transfusion of Packed Red Blood Cells (More Than 6 Units)

- Dilutional DIC (requires FFP, cryoprecipitate)
- Hypothermia (blood is stored at 1 to 6° C)
  - Monitor core temperature
  - Adequate warming of patient/infused fluids
- Hypocalcemia (citrate in blood binds to calcium)
  - Give calcium gluconate
- Acidosis (pH of stored blood is 6.9-7.0)
  - Monitor blood gases and correct as needed
- Hyperkalemia (K+ leaves red blood cells in stored blood)
  - Monitor K+ levels, electrocardiogram (ECG) and correct if needed
- ARDS (antibody-antigen mediated)
  - Monitor in ICU

Table 4-10 Management of Massive Obstetric Hemorrhage (>2000 mL)

- Activate massive transfusion protocol
- Continuous monitoring of maternal status
  - Vital signs; intake-output
  - Foley catheter
  - ECG
  - Hematocrit and coagulation tests
  - Arterial gases and electrolytes
- Correct hypothermia
  - Bair Hugger
  - Warm all IV fluids
  - Warm OR suite to 80° F (27° C)
- Correct acidosis, electrolytes
- Identify and correct the source of bleeding

Table 4-11 Massive Transfusion Protocol for Severe Hemorrhage

Cycle	1	2	3
Red blood cells	6 units	6 units	6 units
Plasma	4 units FFP	4 units FFP	4 units FFP
Platelets	5 units pooled	5 units pooled	5 units pooled
Cryoprecipitate	—	10 units pooled	10 units pooled
Recombinant FVIIa	—	Consider	Administer 60-90 µg/kg
Fibrinogen concentrate*	—	2 g	4 g

*Can be used instead of cryoprecipitate.
FFP = Fresh frozen plasma

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A massive transfusion protocol should be available and activated when there is severe hemorrhage with maternal shock. It includes immediate and serial evaluation of CBC, coagulation studies, arterial blood gas, lactic acid, and electrolytes (every 2 hours). The goals of transfusion are to keep Hgb levels at ≥7 g/dL, fibrinogen levels at ≥100 mg/dL, PT and PTT values <1.5 × mean control, platelet count >50,000/mml^3, and to stop the bleeding and improve tissue perfusion. The transfusions should be given in cycles and repeated every 2 hours as needed according to results of CBC, coagulation, and electrolyte studies. Fibrinogen concentrate can be used instead of cryoprecipitate (it can be available quickly). It is ideal in case of low fibrinogen values related to either abruptio placentae or amniotic fluid embolism. In addition, recombinant factor VIIa (FVIIa) should be used during the third cycle. (See video presentation regarding indications and complications of massive transfusion protocol.)

SUMMARY: Early recognition and prompt management of PPH are key to reducing adverse maternal mortality and morbidities. Management of PPH should be approached in a stepwise fashion that begins by identifying those at high risk, followed by aggressive resuscitation and search for the cause. Once the cause is identified, this should be followed by either medical therapy, surgical therapy, or both. Such management will always require a multidisciplinary team.

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Amniotic Fluid Embolism

5

Baha M. Sibai M.D.

Video Clips on DVD

5-1 PowerPoint Discussion of Amniotic Fluid Embolism

Amniotic fluid embolism (AFE) is a rare but unpredictable and rapidly developing obstetric emergency. The exact incidence of AFE is unknown; however, the reported incidence ranges from 1 per 10,000 to 1 per 20,000 singleton births. Although there are no known risk factors for AFE, the reported incidence increases in the presence of one or more associated factors listed in Table 5-1. Despite its rarity, AFE is a major cause of maternal mortality in the Western countries accounting for 8% to 10% of all maternal deaths in the United States, Canada, and England. The majority of cases of AFE develop during the active phase of labor, mostly during delivery. However, AFE can also develop at the time of cesarean section and even in the immediate postpartum period.

Case 1: Amniotic Fluid Embolism

A 34-year-old G4P3 who was admitted at 41 weeks' gestation for induction of labor. Ultrasound evaluation revealed an estimated fetal weight of 3942 g with an amniotic fluid index of 7 cm. Fetal heart rate revealed reassuring tracing with good variability and presence of accelerations. Cervical examination revealed vertex presentation with a Bishop score of 5. Physical examination revealed normal findings with blood pressure of 100/62 mm Hg. Laboratory findings revealed normal hematocrit and platelet count.

Oxytocin induction was started and 4 hours later she had artificial rupture of membranes with evidence of meconium staining. Cervical examination revealed cervix 4 cm dilation and 90% effaced. Approximately 1 hour later, the fetal heart rate tracing was reassuring, but uterine contractions were increased (Fig. 5-1A). Approximately 10 minutes later, cervical examination revealed anterior lip, increased uterine activity, and reassuring fetal heart rate (see Fig. 5-1B). After the examination, there were variable decelerations, and 10 minutes later the patient complained of sudden onset of shortness of breath, became cyanotic, and lost consciousness. At that time, fetal monitoring revealed bradycardia and marked increase in uterine activity and tone (see Fig. 5-1C). She had immediate resuscitation with oxygen and bagging followed by intubation, and the fetal heart rate revealed bradycardia and absent variability (see Fig. 5-1D). Six minutes after cardiac arrest she had cesarean delivery in the room with delivery of a live infant weight of 4020 g with Apgar scores of 1, 4, and 7 at 1, 5, and 10 minutes, respectively. Maternal resuscitation efforts were discontinued after 30 minutes. Autopsy findings found massive amniotic fluid embolism. The infant had metabolic acidosis, but no seizures and was discharged home 7 days later.

Discussion

The etiology and pathogenesis of AFE remains poorly understood. It usually results from amniotic fluid entering the maternal circulation producing acute cardiovascular, hemodynamic, and hematologic abnormalities similar to those seen with anaphylaxis. The portal of entry of the amniotic fluid after membranes rupture could be at the site of placental implantation or at site of tears in the cervix or lower uterine segment. The fetal cells and biochemical mediators in the amniotic fluid are responsible for the majority of signs and symptoms seen in this syndrome (Fig. 5-2).

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Case 1: Amniotic Fluid Embolism—cont'd

AFE is characterized by sudden onset of acute hypoxia, hypotension, and cardiovascular arrest during labor or at time of cesarean section or within 60 minutes after delivery. In most patients there is sudden change in maternal and/or fetal condition. The presenting signs and symptoms are listed in Table 5-2. Laboratory findings in AFE are listed in Table 5-3.

Table 5-1 Factors Associated with Increased Incidence of AFE

- Advanced maternal age
- Multiparity
- Abnormal placentation
- Medical induction of labor
- Uterine overdistention
  - Multifetal gestation
  - Polyhydramnios
- Cervical or uterine lacerations
- Fetal demise

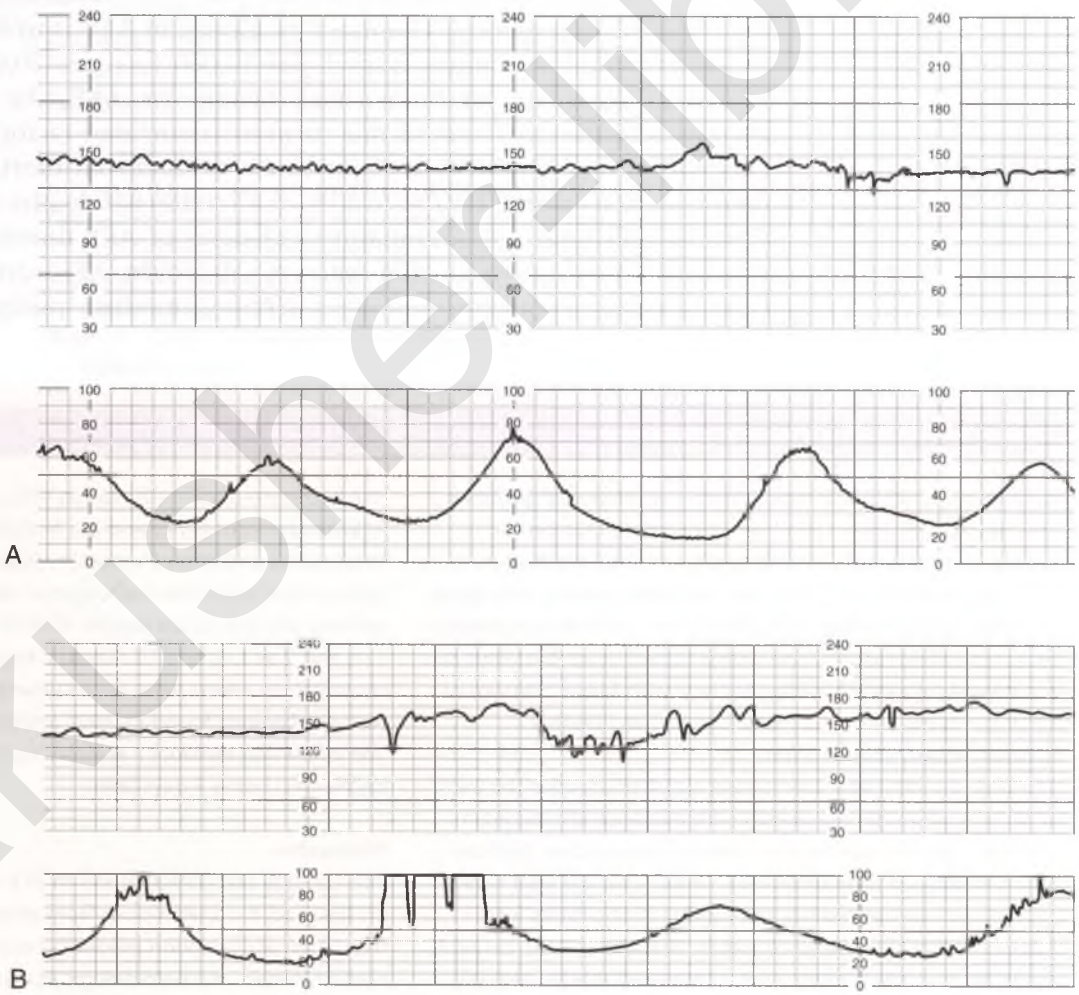

Figure 5-1 **A,** Uterine activity tracing revealing tachysystole with reassuring fetal heart rate tracing. **B,** Fetal heart rate tracing reveals the appearance of variable decelerations.

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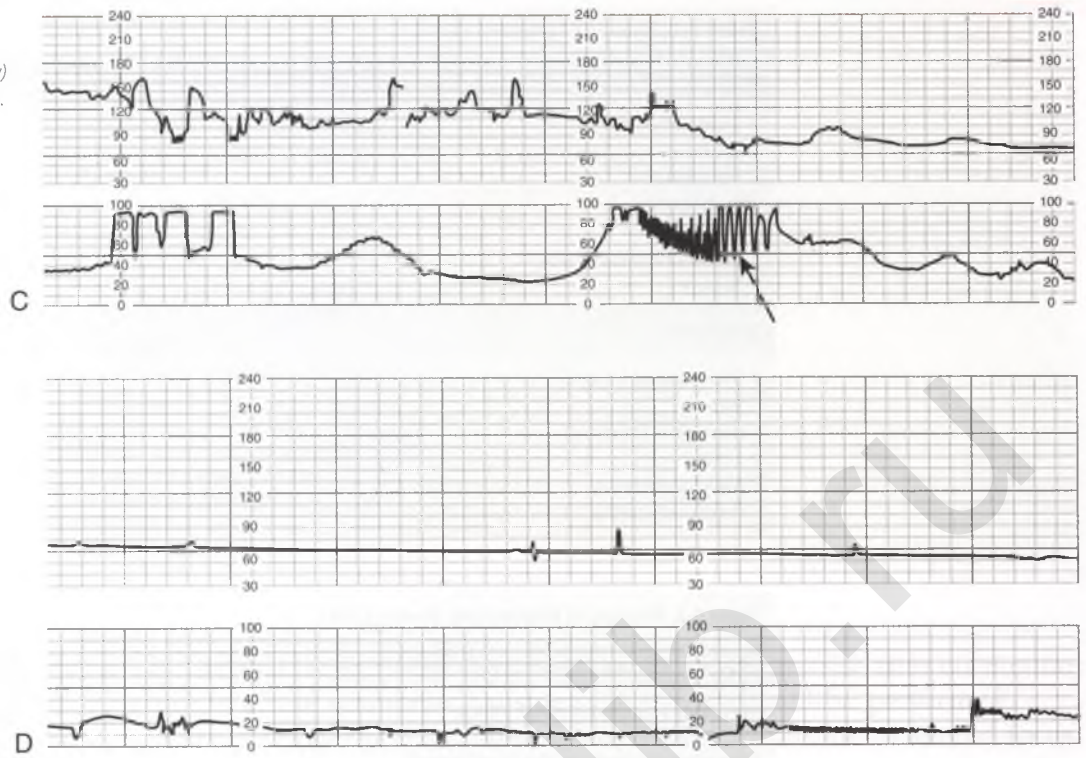

Figure 5-1, cont'd C, Note tetanic uterine contractions during maternal seizure *(arrow)* with onset of fetal bradycardia. **D,** Fetal bradycardia with absent variability.

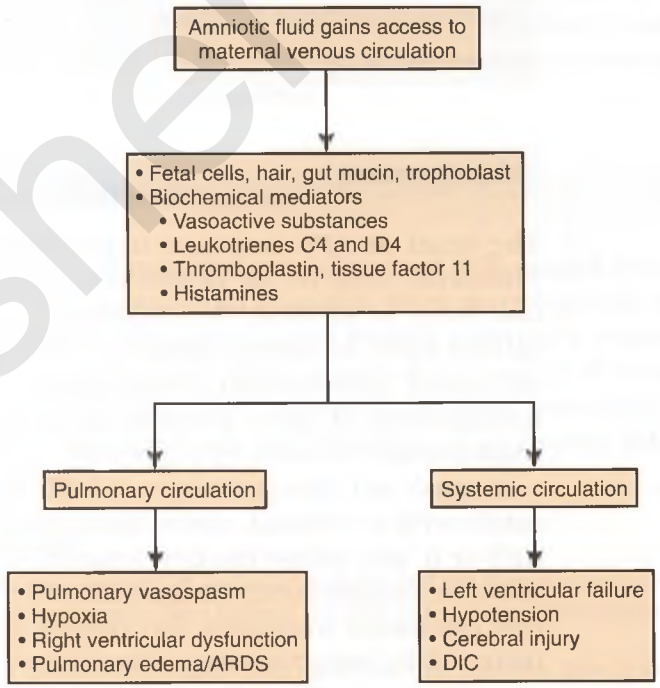

Figure 5-2 Pathophysiology of amniotic fluid embolism.

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Table 5-2 Signs and Symptoms of AFE

Signs
• Breathlessness • Cyanosis • Hypotension • Tachycardia • Arrhythmias • Cardiovascular collapse • Seizures
Symptoms
• Shortness of breath • Anxiety • Change in behavior • Chills • Nausea/vomiting • Fetal bradycardia

Table 5-3 Findings of Investigative Studies in AFE

	%
Hypoxemia (pulse oximetry <90%)	100
Disseminated intravascular coagulation	60-83
Pulmonary edema/ARDS	70-90
Electrocardiogram (abnormal rhythm)	80
Echocardiogram (ventricular dysfunction)	100
Left ventricular failure	

Management

The onset of AFE is always unpredictable, sudden, and rapidly progressive. Therefore, most of the deaths occur within a few hours of onset as was the case here. In addition, the majority of survivors have life-threatening complications such as severe hypoxia, cardiogenic shock, pulmonary edema, disseminated intravascular coagulopathy (DIC), and hemorrhage. Therefore, management of these patients should first focus on rapid cardiorespiratory resuscitation followed by supportive care of vital organs (Table 5-4).

A rapid and abrupt change in fetal heart rate pattern during labor (bradycardia with or without severe decelerations) may be the first manifestation of AFE or it may follow the development of maternal cardiovascular collapse. In addition, uterine contraction monitoring reveals the presence of hyperstimulation (increased frequency and resting tone). These changes are secondary to maternal hypoxia resulting in both uterine and fetal hypoxia (see Fig. 5-2). If asystole or pulseless electrical activity continues beyond 5 minutes despite resuscitation, delivery should be performed as part of cardiopulmonary resuscitation (CPR) to improve chances of resuscitation as well as to improve fetal outcome. Keep in mind that the outcome for the neonate (both acute and long term) depends on timing of delivery in relation to the arrest. Neonatal survival and intact neurologic outcome start to decrease if delivery is accomplished beyond 6 minutes after the arrest. Therefore, if there is no maternal response beyond this time, prompt delivery should be performed in the labor room. In

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Table 5-4 Management of Various Clinical Presentations of AFE

- Asystole/pulseless electrical activity
  - Advanced life support protocols
  - Delivery if no response after 5 minutes
- Respiratory failure/severe hypoxia
  - Intubation
  - Oxygen at high concentration
- Cardiogenic shock
  - Crystalloids/blood
  - Inotropic and vasoactive agents
    - No epinephrine, dopamine
- Pulmonary edema/ARDS
  - Invasive hemodynamic monitoring
  - Dobutamine, furosemide
  - Limit fluid replacement
- Disseminated intravascular coagulation
  - Blood and blood products
  - Recombinant factor Vila
- Hysterectomy (atony, lacerations)

patients without asystole, if the fetal heart rate changes persist after correction of maternal hypoxia, emergency cesarean delivery should be performed.

After restoring maternal cardiorespiratory functions, attention should focus on treatment of cardiogenic shock, pulmonary edema or acute respiratory distress syndrome (ARDS), and DIC. Thus these women should have close monitoring in an intensive care facility with aggressive fluids and blood product replacement with central hemodynamic monitoring guidance. If the measures listed in Table 5-4 fail, patients may require pulmonary vasodilators (IV prostacyclin, nitric oxide), extracorporeal membrane oxygenation (ECMO), or cardiac bypass as life-saving measures.

Maternal and Perinatal Outcomes

In the past, the reported maternal mortality from AFE ranged from 60% to 80%, and among women who survived only 40% were neurologically intact. In contrast, recent data from population-based studies reported a maternal mortality of 13% to 37%. In addition, one study reported that only 2 of 31 survivors (7%) had neurologic impairment. The reported perinatal mortality was approximately 20%; however, there are limited data on long-term infant outcomes among the survivors.

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Pulmonary Embolism in Pregnancy

6

Robert Egerman M.D.

Accounting for 20% to 30% of maternal deaths, pulmonary embolism ranks as the most frequent cause of maternal death in developed countries. Its incidence during pregnancy is 5 to 10 times that of the general population. Originating mostly from the venous system of the lower extremities, undiagnosed or untreated thrombosis can embolize through the heart into the pulmonary vasculature, leading to right-sided heart failure, hypoxemia, and vascular collapse. The early presentation of this disease may be obscured by common symptoms and signs attributable to pregnancy or to the early puerperium, necessitating diagnostic consideration of this entity throughout gestation.

Case 1: Thromboembolism

A 23-year-old nulliparous patient at 31 weeks' gestation presented with contractions and pelvic pressure for 2 days. Her past medical history included asthma that was well controlled on a low-dose corticosteroid inhaler and obesity (body mass index 40 kg/m^2). On physical examination her weight was 278 lb, temperature 98° F, pulse 87 beats per minute (bpm), respiratory rate 12 breaths/min, and blood pressure 130/78 mm Hg. Generally she appeared anxious and in moderate distress during uterine contractions. Cardiovascular and pulmonary findings were unremarkable. Her abdomen was obese, nontender and the cervical examination revealed 3 cm dilation and 1.5 cm in length, and the presenting part was at –3 station. The fetal heart rate was reactive and uterine contractions occurred at 3-minute intervals.

The patient was admitted to labor and delivery, a urinary catheter was placed, IV fluids were administered along with IM betamethasone and IV magnesium sulfate. Rapid testing for group B β–hemolytic streptococci was sent and later reported as negative. Over the next 24 hours the contractions abated despite cervical dilation progressing to 4 cm. She was transferred to the antepartum unit 36 hours after betamethasone therapy and remained on bed rest with allowance to use the bathroom. On hospital day 4, uterine contractions returned and the patient was transferred to labor and delivery, where labor ensued. A vigorous female infant was delivered spontaneously and the patient was transferred to the postpartum unit 6 hours after delivery.

On day 5 of the hospitalization, she reported having chest tightness and difficulty breathing over the past 2 hours. The patient felt this was likely to be an "asthma attack." Examination revealed an alert, anxious patient in moderate respiratory distress. Vital signs indicated temperature 98.8° F, pulse 117 bpm, respiratory rate 20 breaths/min, and blood pressure 140/82 mm Hg. Bilateral breath sounds were heard without wheezes, and tachycardia was noted on cardiac auscultation with no murmurs. Extremities revealed 2+ pedal edema bilaterally and the abdominal examination was benign. Reassurance was provided and a short-acting β–agonist inhaler was prescribed. A helical computed axial tomographic scan was ordered to "cover all the bases."

Fours hours after receiving the inhalation treatment, the physician was called because of increased respiratory effort. Vital signs revealed temperature 98.7° F, pulse 132 bpm, respiratory rate 26 breaths/min, and blood pressure 80/30 mm Hg. One liter of normal saline was administered and repeat blood pressure was 92/48 mm Hg. A computed tomographic (CT) scan had not been performed at this time. IV heparin was administered and dosed on a weight-based nomogram. The patient was transferred to the intensive care unit (ICU), where on arrival she became obtunded and progressively hypotensive. She went into a pulseless tachycardic rhythm and respiratory arrest. Advanced cardiac life support was initiated including chest compressions, endotracheal intubation, and the administration of epinephrine and fluid boluses. A femoral

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Case 1: Thromboembolism—cont'd

venous catheter was inserted during the resuscitative efforts and multiple doses of epinephrine were given as well as vasopressin. The rhythm deteriorated into asystole and external pacing was attempted. After a total of 41 minutes of circulatory collapse and unsuccessful resuscitative efforts, the patient was pronounced deceased.

Discussion

Notably, the patient had numerous risk factors for having a thromboembolic event: obesity, pregnancy, hospitalization with limited ambulatory activity, pregnancy and delivery. Consideration should have been given to thromboembolic prophylaxis using a sequential compression device and thromboembolic deterrent stockings throughout her admission. For patients at high risk for thromboembolism, with multiple risk factors (including two of the following: age >35 years, obesity, medical illness, cesarean section, infection, immobility, or gross varicosities) heparin prophylaxis should be considered as well. In patients who have had a prior thromboembolism or thrombophilia, prophylactic heparin should be considered. For the patient described, clinical indicators suggesting a pulmonary embolism occurred early in her course. Anticoagulation should not have been withheld pending the results of thoracic imaging. Had a pulmonary embolism not been confirmed, further anticoagulation could have been discontinued.

Table 6-1 Risk Factors for Pulmonary Embolism in Pregnancy

- Obesity
- Immobilization
- Advanced age
- Surgery
- Spinal cord injury
- Trauma
- Acute medical illness
- Nephrotic syndrome
- Malignancy
- Central venous catheterization
- Heparin-induced thrombocytopenia
- Thrombophilia
  - Factor V Leiden
  - Protein C or S or antithrombin deficiency
  - Prothrombin gene mutation
  - Antiphospholipid antibodies:
    - Lupus anticoagulant, anticardiolipin antibodies
  - Possible factors:
    - Elevated homocysteine, factors VIII, IX or XI, fibrinogen
    - Thrombin-activated fibrinolysis inhibitor

Pathophysiology and Risk Factors

The pathophysiology of a pulmonary embolism begins with the hypercoagulable state induced in the gravida: increased clotting factors, decreased fibrinolysis, platelet activation, and venous stasis. A reduction in venous flow in the lower extremities is not uncommon beginning in the late second trimester and lasting until several weeks postdelivery. Risk factors for thromboembolic disease are widely known and are listed in Table 6-1. A preponderance of lower extremity deep venous thromboses occurs on the left side due to compression of the left iliac vein by the right iliac artery as shown in the schematic in Figure 6-1.

Fifteen percent of untreated deep venous thrombi embolize to the lungs causing death in 15% of patients. Once an embolism occurs, vasoactive substances are released into the pulmonary vasculature causing bronchoconstriction and vasoconstriction (Fig. 6-2). Further, the mechanical obstructive effects of the clot produce right ventricular failure. Right ventricular dysfunction has

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Figure 6-1 Compression of the left iliac vein by the right iliac artery.

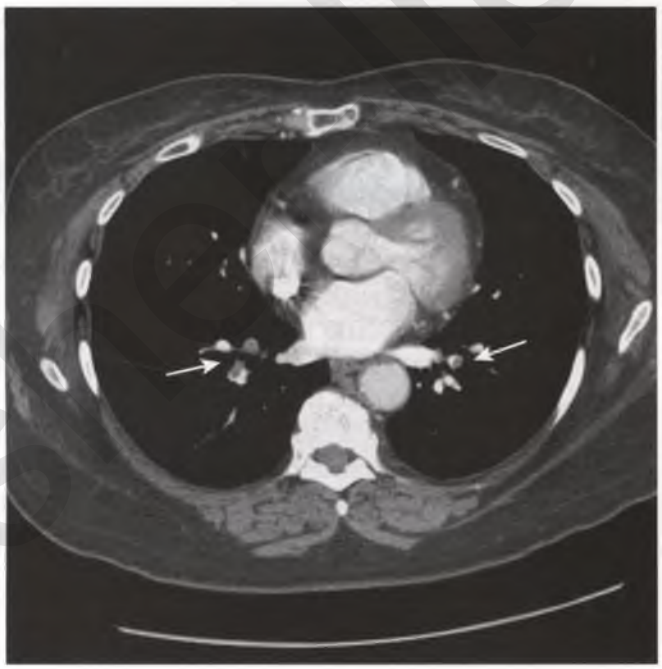

Figure 6-2 Helical CT revealing intraluminal filling defects *(arrows)* in the pulmonary vascular tree.

been described in this setting using echocardiography with evidence of ventricular dilatation, free-wall hypokinesia or paradoxical movement, a 30 mm Hg pressure gradient between the right ventricular and right atrial chambers, or a decreased pulmonary flow acceleration time. Sometimes a clot can be visualized within the right side of the heart (atrium, ventricle, or pulmonary artery), as well as visualized sonographically protruding through a patent foramen ovale. In severe cases, cardiac output is not able to be sustained and cardiopulmonary arrest occurs.

Patients can be stratified into various risk categories depending on their risk of death. High-risk patients have hypotension and right ventricular dysfunction (massive pulmonary embolism), whereas intermediate-risk patients remain

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normotensive with right ventricular dysfunction (submassive pulmonary embolism). The lowest risk patient is one who has neither hypotension nor right ventricular dysfunction.

Presentation and Diagnosis

Frequently the patient presents with nonspecific complaints; however, given the potential severity of a pulmonary embolism and the underlying risk factor of pregnancy, the provider should be familiar with this diagnosis. Signs and symptoms of a pulmonary embolism can be variable and may be subtle. These are presented in Table 6-2.

Options for the diagnostic workup are listed in Table 6-3; however, if the diagnosis is suspected (unless contraindicated), anticoagulation should be initiated and helical CT (specifically looking for a pulmonary embolism) should be obtained. Other imaging modalities are both less specific and sensitive. Venous Doppler may only detect 20% of proximal deep vein thrombosis in cases of pulmonary embolism. D-dimer testing can be omitted in the evaluation of the pregnant woman. Moreover, D-dimer testing should not be used in patients with a high probability for a pulmonary embolus because of its low negative predictive value. Further, basing diagnostic testing on clinical probability scores is not appropriate until these have been validated for pregnancy.

Interestingly, the fetal radiation exposure is less with CT than with ventilation perfusion scanning. A diagnostic algorithm is presented in Figure 6-3.

Management

Hemodynamic stability and adequate oxygenation are the first steps in managing a patient with a pulmonary embolism. If hypotension is present, IV lactated Ringer's or saline should be administered. (A massive pulmonary embolism is defined as systolic blood pressure <90 mm Hg or a drop from baseline of >40 mm Hg. In this instance, IV fluids, vasopressors [dopamine] should be considered because anticoagulation alone is not adequate.) Oxygenation should be provided to maintain a saturation >95%, and the patient placed on telemetry and bed rest initially. The patient should be transferred to an ICU and the ICU team should be consulted (Table 6-4). A multidisciplinary approach should be sought.

Table 6-2 Signs and Symptoms of Pulmonary Embolism

Signs	Symptoms
Tachycardia	Chest or pleuritic pain (sudden or gradual)
Tachypnea	Cough or wheezing or hemoptysis
Hypotension	Dyspnea (may be sudden or over weeks)
Right ventricular lift	Fever
Accentuated second heart sound (loud P_2)	Anxiety
Jugular venous distention	Leg swelling
Hypoxemia (not always seen initially)	Lightheadedness or syncope

Table 6-3 Laboratory Studies and Diagnostic Imaging for Pulmonary Embolism

Test	Comment
Arterial blood gas	Normal values in 30% of affected patients; may show hyperventilation, hypoxemia may be present
Pulse oximetry	Poor sensitivity, hypoxemia may be present
A-a gradient	Normal values in 30% of affected patients, widened A-a gradient may be present
12-lead ECG	Sinus tachycardia most likely, may show right ventricular strain, S wave in lead I and Q wave and inverted T wave in lead III
Chest x-ray	Can be normal. May see effusion, parenchymal abnormalities: atelectasis, Hampton's hump (infarction near pleura), regional oligemia (Westermark sign)
D-dimer	Not useful as a stand-alone test in pregnancy given the increased baseline risk for embolism
Troponin or brain natriuretic peptide	May be increased
V/Q scan (ventilation/perfusion scan)	An option if CT scan cannot be performed, limited sensitivity
Computed axial tomography (helical CT with pulmonary embolism protocol) (shield fetus)	Preferred method for ruling out a pulmonary embolism given there are no contraindications to the procedure. Minimal fetal radiation exposure. Shield abdomen and breasts. If negative CT and suspicion high for embolic event, perform venous Doppler of lower extremities.
Magnetic resonance imaging	Limited data presently
Echocardiography	May indicate a hemodynamically significant embolus
Pulmonary angiography	Definitive study; however, carries the greatest maternal risk for morbidity/mortality

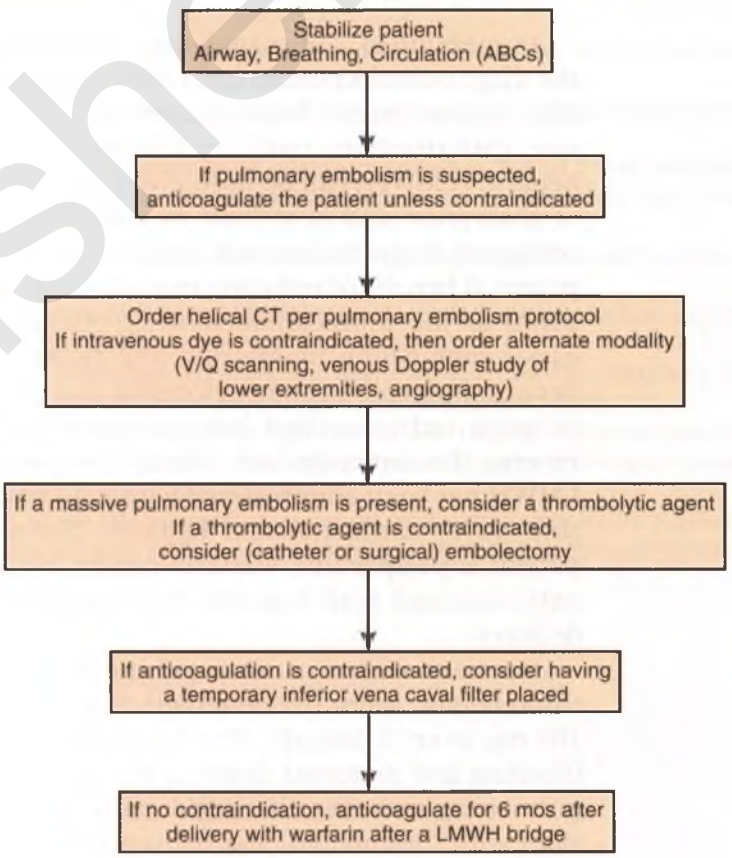

Figure 6-3 Management of pulmonary embolism.

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Table 6-4 Monitoring of a Patient with a Pulmonary Embolism

- Admit to obstetric or medical ICU
- Notification of ICU team
- Large IV access
- Blood tests for CBC, PT/PTT/INR, CMP
- Assessment for hypotension, tachypnea
- Continuous pulse oximetry
- ± Arterial blood gas
- Oxygen supplementation by face mask
- Continuous ECG monitoring
- Central hemodynamic monitoring
- Continuous fetal heart rate monitoring (≥24 wk)

Table 6-5 Treatment of Pulmonary Embolism

1. Anticoagulation
   LMWH (use with intermediate or low risk)
      Enoxaparin 1 mg/kg every 12 hr
      Confirm Xa level is therapeutic (0.6-1.2 units/mL 4 hr after dosing)
      Screen for heparin-induced thrombocytopenia
   Unfractionated heparin (use for massive embolism)
      See various published nomograms
      80 units/kg bolus then 18 units/kg/hr
      Frequent PTT monitoring
      Screen for heparin-induced thrombocytopenia
2. IV fluids
   Normal saline 500-1000 mL bolus if hypotension is present
   Maintenance IV fluids
3. Bed rest
4. See text for thrombolytic discussion

As mentioned, anticoagulation is typically initiated at the consideration of the diagnosis of a pulmonary embolism. In cases of massive pulmonary embolism, unfractionated heparin is the anticoagulant of choice. In cases of submassive (intermediate risk) or low-risk embolic events, low-molecular-weight heparin (LMWH) can be used given its advantage of a more predictable pattern of absorption and is at least as effective at treating thromboembolism when compared to unfractionated heparin. In nonpregnant subjects, LMWH has the potential benefit of reducing mortality and recurrent clot formation when compared to the unfractionated form. Additional advantages to LMWH include easier therapeutic monitoring and reduced risk of adverse effects including heparin-induced thrombocytopenia and osteopenia. Conversely, an advantage to using unfractionated heparin could be its shorter half-life and ability to reverse the anticoagulant effects. (Regional anesthesia is contraindicated if LMWH has been administered within 24 hours.) Standard nomograms are available for IV unfractionated heparin. Dosing is reviewed in Table 6-5. If the patient is postpartum, warfarin can be initiated once she has been adequately anticoagulated with heparin. Anticoagulation is continued for 6 months after delivery.

A patient with a massive pulmonary embolism is likely to benefit from a thrombolytic agent (recombinant tissue plasminogen activator, r-tPA [alteplase 100 mg over 2 hours]). The hazards of this treatment include intracranial bleeding and maternal death (1%); however, the risk of death from a massive pulmonary embolism is considerably greater (20%). Contraindications to consider with thrombolytic use include bleeding, recent surgery, or intracranial or

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spinal disease. A catheter embolectomy or thrombolytic may be an alternative to systemic thrombolytic administration. Additionally, if a clot is visualized freely floating within the right heart or in the left atrium (potential paradoxical embolism), catheter embolectomy is indicated. Surgical embolectomy can be used if other modalities fail; however, it is associated with relatively high perioperative mortality. The benefit of thrombolytic therapy in patients with a submassive pulmonary embolism (right ventricular dysfunction) is under study. Certainly consideration can be given in this setting if maternal cardiac function deteriorates.

Fetal and Neonatal Outcomes

Maternal death would likely be catastrophic to the fetus. Heparins, either LMWH or unfractionated, do not cross the placenta. Coumarin derivatives are teratogenic between 6 and 12 weeks' gestation and can contribute to intraventricular hemorrhage in the third trimester. In this setting these agents are avoided until after delivery. Coumarin derivatives can be used by lactating patients. Alteplase is a polypeptide that does not cross the placenta. Surgical embolectomy including cardiopulmonary bypass may have an increased fetal risk; however, these procedures are used in extremis.

Suggested Readings

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7

Peripartum Cardiomyopathy and Pulmonary Edema

Ryan Loftin M.D.
James Van Hook M.D.

Video Clips on DVD

7-1 PowerPoint Discussion of the Diagnosis and Management of Pulmonary Edema and Peripartum Cardiomyopathy

Pulmonary edema is a secondary disease process characterized by pulmonary interstitial and alveolar fluid collection resulting in hypoxemia and increased work of breathing. Conceptually, pulmonary edema may be classified into the four categories of hydrostatic, increased capillary permeability, lymphatic insufficiency, and unclear etiology (Fig. 7-1). As Figure 7-1 implies, pregnancy-related causes of pulmonary edema typically encompass three of the four categories. The most frequent causes of pulmonary edema in pregnancy postpartum are listed in Table 7-1. Both tocolytic-associated pulmonary edema (Table 7-2) and pulmonary edema from preeclampsia (Table 7-3) are complex entities about which some controversy still exists regarding their etiology; however, changes in vascular permeability and gradient between pulmonary wedge pressure and colloid oncotic pressure play an important role in its etiology (Fig. 7-2). Nonetheless, successful management of pulmonary edema is logically addressed in a standardized fashion.

Peripartum cardiomyopathy in which maternal cardiac left ventricular systolic dysfunction and symptoms of heart failure develop between the last month of pregnancy and the fifth month postpartum. The disorder is of unknown cause and is a diagnosis of exclusion in pregnant or puerperal patients who present in congestive heart failure late in pregnancy or early in the postpartum period. Despite being traditionally classified as a late pregnancy or postpartum entity, a similar clinical presentation may be seen earlier in pregnancy and has been reported early in the second trimester of pregnancy. In the United States, the incidence of peripartum cardiomyopathy is estimated to be 1 in 3000 to 4000 live births. The worldwide incidence varies; certain populations have a reported risk of 1 in 100. Characteristics and associations seen with the diagnosis of peripartum cardiomyopathy are listed in Table 7-4. Pulmonary edema in the patient with peripartum cardiomyopathy typically is hydrostatic in nature, with mechanistic specifics from left ventricular systolic dysfunction.

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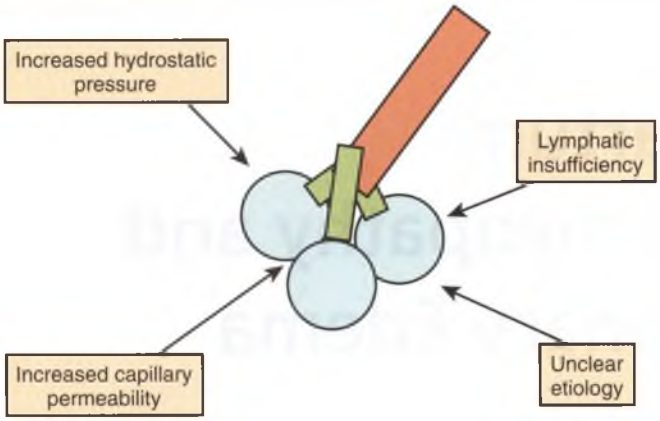

Figure 7-1 Potential etiologies of pulmonary edema.

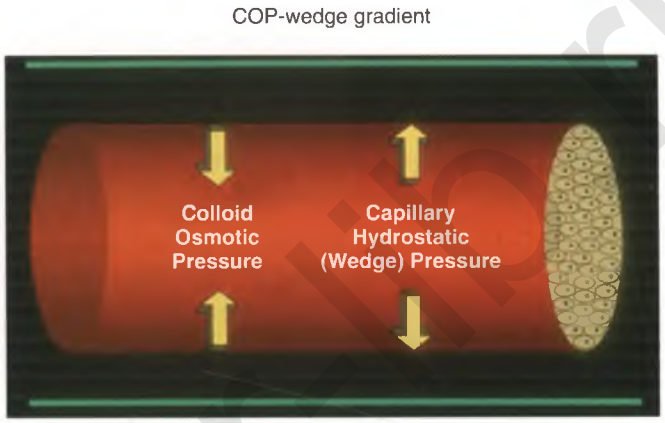

Figure 7-2 Mechanisms governing development of pulmonary edema in patients with preeclampsia and those receiving tocolytics. This describes forces inside the capillaries and those in the interstitium.

Table 7-1 Causes of Pulmonary Edema in Pregnancy Postpartum

- Preeclampsia-eclampsia
- Tocolytics
- Cardiac disease
  - Left ventricular dysfunction
  - Mitral stenosis
  - Cardiomyopathy
- Infections
  - Pyelonephritis/sepsis
  - Varicella pneumonia
- Thyroid storm

Table 7-2 Factors Associated with Pulmonary Edema in Patients with Preterm Labor

- Subclinical infection
- Multifetal gestation
- Prolonged tocolytic therapy
- Need for multiple tocolytics
- Excessive fluid therapy (positive balance)
- Undiagnosed cardiac disease

Table 7-3 Factors Associated with Pulmonary Edema in Severe Preeclampsia

- Fluid overload
  - Prehydration for epidural
  - Prolonged magnesium sulfate and oxytocin
  - Postpartum mobilization
- Renal dysfunction/acute tubular necrosis
- Untreated severe hypertension
- Low serum colloid oncotic pressure
- Sepsis

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Table 7-4 Characteristics Associated with Peripartum Cardiomyopathy

- Age at extremes of childbearing years
- Use of tocolytic therapy
- African descent
- Parity
- Gestational hypertension or preeclampsia
- Multifetal gestation

Case 1: Peripartum Cardiomyopathy

A 34-year-old G3P2 presents to labor and delivery 6 weeks postpartum complaining of profound lower extremity edema and shortness of breath. Review of her pregnancy history indicates that she delivered a 36 twin gestation via vaginal delivery after spontaneously presenting in labor. Her pregnancy was complicated by gestational hypertension, although she never manifested the criteria to make the diagnosis of mild preeclampsia. Since discharge she has felt tired, with a progressive increase in fatigue that she initially attributed to around-the-clock care of newborn twins. Approximately 2 days prior to her presentation to the emergency department, she developed a cough, and increasing dyspnea on exertion. In the evening prior to presentation, she awoke unable to remain supine in her bed.

On admission her blood pressure was 140/65 mm Hg and her pulse was 130 beats per minute (bpm). Respirations were labored, with a respiratory rate of 35 per minute. Digital pulse oximetry saturation (room air) was 86%. Temperature was normal. Physical exam of the patient's cardiovascular system demonstrated diffuse rales and rhonchi during lung auscultation, an S_3 gallop, and jugular venous distention. Pitting lower extremity edema was also observed. Initial arterial blood gas (100% Fio_2) revealed a pH of 7.46 Pao_2 of 140 mm Hg, $Paco_2$ of 32 mm Hg, and HCO_3^- of 24 mEq/L.

The patient was immediately managed with oxygen administration, bilevel positive airway pressure (BIPAP), judicious administration of 2 mg morphine sulfate IV and furosemide 40 mg IV. Portable chest x-ray showed bilateral air-space disease, perihilar infiltrates, and bilateral small pleural effusions. Cardiomegaly was also suspected. Complete blood count (CBC) and metabolic panel were drawn; results were unremarkable. Electrocardiogram (ECG) was consistent with left atrial enlargement, sinus tachycardia, diffuse T-wave inversion, increased QRS voltage in horizontal plane leads (V_1-V_6) and low QRS amplitude in limb leads. D-dimer and cardiac enzyme panel results were normal. B-type natriuretic peptide (BNP) was elevated at 900 pg/mL.

As initial therapy and diagnostic workup were begun, the patient received a bedside echocardiograph. Diffuse hypokinesis of the dilated left ventricle was immediately evident. There was also mitral valve regurgitation, left atrial enlargement, and a calculated left ventricular ejection fraction (LVEF) of 23%. A diagnosis of dilated cardiomyopathy, congestive heart failure, and pulmonary edema were made. The patient's management rapidly became multidisciplinary in nature, with cardiovascular and ventilatory management directed by appropriately credentialed acute care physicians. The patient received her care in an acute care unit that provides continuous monitoring of ECG, blood pressure, pulse oximetry, and clinical assessment. The ability to manage acute cardiovascular and/or respiratory instability was present.

After load reduction with the angiotensin-converting enzyme inhibitor (ACE) captopril was begun. Judicious additional diuresis with furosemide (20 to 40 mg IV) was accomplished. Venous thromboembolic prophylaxis with 40 mg enoxaparin subcutaneously daily was ordered. Dobutamine and IV nitroglycerin were initiated. However, the patient became hypotensive (<90/60 mm Hg). Noninvasive assessment of cardiac output demonstrated a reduced cardiac output. At the recommendation of the patient's consulting cardiologist, norepinephrine was started IV at 2 mcg/min, with titration to blood pressure as obtained by continuous arterial blood pressure monitoring. A central venous catheter was placed. Hypoxemia worsened and the patient was intubated, sedated, and maintained on mechanical ventilation. The use of a pulmonary artery catheter was considered, however, the patient's condition stabilized and noninvasive assessment of the patient was sufficient for management.

On hospital day 3, the dose of norepinephrine was gradually reduced and was eventually discontinued. The patient's systolic blood pressure remained above 90 mm Hg. Nesiritide, a recombinant human BNP used for acute decompensated heart failure, was begun by the cardiologist as a 2-mcg/kg bolus followed by continuous infusion at 0.01 mcg/kg/min. Hypoxemia resolved and work of breathing lessened as the hydrostatic pulmonary edema caused by congestive heart failure improved. The patient was successfully extubated and progressively weaned from oxygen. Nesiritide was discontinued and the patient was maintained on a daily regimen of furosemide, captopril, and eplerenone, an aldosterone agonist. The patient was eventually discharged and had an improving clinical status and echocardiographic picture. Metoprolol was begun early in the outpatient period. A search for other specific causes of the patient's dilated cardiomyopathy was not suggestive of any other likely etiology. A presumptive diagnosis of peripartum cardiomyopathy was made. The patient was discouraged from becoming pregnant immediately and had a careful 6-month follow-up of her status by both her OB-GYN provider and her cardiologist arranged. Review of status at that time formed

Case 1: Peripartum Cardiomyopathy—cont'd

the basis of long-term recommendations for both general and reproductive follow up.

Discussion

This case entails both the immediate management of hydrostatic pulmonary edema and the underlying initiating pathophysiologic process of peripartum cardiomyopathy. This case is typical in that the presentation is suggestive of a cardiorespiratory process. The case example as presented is straightforward in that sufficient time has elapsed from delivery to lessen confusion from most other primary pregnancy-related diagnoses (Table 7-5). Presentation of peripartum cardiomyopathy in the antenatal period or within the first 2 weeks postpartum is often confounded by confusion on the part of physicians caring for the patient in eliciting the primary diagnosis. Preeclampsia and other pregnancy-related diagnoses may be initially confused with the primary cardiac etiology of peripartum cardiomyopathy.

This patient presented with congestive heart failure and pulmonary edema. It is important to approach a patient with cardiorespiratory insufficiency in a systematic fashion to determine the etiology and target management accordingly. As a secondary disease process, pulmonary edema is initially approached by delineating the subtype of edema. However, unlike typically seen pregnancy complications, immediacy in primary stabilization is important. Shortness of breath is often seen as a principal symptom in the previously otherwise healthy gravida or postpartum patient. Chest pain and other manifestations of myocardial ischemia are often relatively late findings. Most (but not all) pregnant and postpartum patients are young enough as to be able to tolerate tachycardia and moderate hypoxia without the predominant symptom—chest pain.

Cough and dyspnea are frequently more common symptoms. Often younger patients may have a surprising degree of tachycardia and hypoxia. As with postpartum hemorrhage, severity of symptoms may not be a good guide as to severity of disease process. The patient presented here has a significant risk of cardiorespiratory arrest. These patients, although often initially ambulatory, are sicker than they may casually seem to be. Oxygen saturation via digital pulse oximetry is an easy way to assess a patient for hypoxemia. Please note that once receiving oxygen therapy, this patient would have had a normal pulse oximetry saturation reading in the face of significant illness and risk of death. Therefore, arterial blood gas analysis and recognition of hemodynamic compromise are also important.

As outlined in subsequent sections determination of cardiac or noncardiac etiology, along with timely exclusion of other causes is crucial. Pulmonary edema and congestive heart failure are operationally processes that deal with derangement of breathing and circulation. Therefore, evaluation in the context of airway, breathing, and cardiovascular systems should be the primary tenet of initial assessment. These patients generally need a multidisciplinary approach to care. The role of the obstetrician should be that of ombudsmen—the care provider that is first and foremost interested in overall care and management.

Peripartum cardiomyopathy may present during pregnancy. In one of the larger series of peripartum cardiomyopathy, an identical subset of patients with presentation during pregnancy was seen in approximately 20% of cases, highlighting the need for vigilance for this condition even during pregnancy. Evaluation of the fetus, including assessment of gestational age, consideration for delivery planning, corticosteroids for fetal lung maturation if delivery is medically indicated, and gestational age between 24 and 34 weeks are all important components of management of the fetus. Pulmonary edema may of course present during or after pregnancy, depending on the etiology.

The following are steps suggested for workup of the complicated patient with pulmonary edema, with emphasis on the exclusionary diagnosis of peripartum cardiomyopathy.

Remember the ABCs

Providing an adequate airway ("A"), along with support of oxygenation and ventilation ("B") are always initial steps in the evaluation of any sick patient. Respiratory rate minimally changes during normal pregnancy (although minute ventilation does increase by approximately 20%). Therefore, an elevated respiratory rate (>20 breaths/min) is possibly indicative of increased work of breathing. Pulse oximetry is indicative of hemoglobin oxygen saturation. At sea level, pregnant and nonpregnant patients without underlying chronic pulmonary disease should manifest an oxygen saturation of at least 94% if readings are measured while the patient is inspiring room air. Conversely, apparently normal oxygen saturation in a patient with significant underlying baseline hypoxemia may be seen in the patient breathing mask oxygen of higher oxygen content than room air. Nonetheless, pulse oximetry is a quick way of initial rapid assessment of oxygenation. Ventilation must also assess gas exchange in addition to oxygenation. Therefore, arterial blood gas measurement is a necessary secondary evaluation.

Circulation ("C") refers to evaluation of both blood pressure and pulse. A markedly elevated pulse (>110 bpm) may be suggestive of hypoxia or a compensatory attempt to maintain cardiac output if contractility is diminished. Blood pressure may be elevated, lowered or unchanged initially.

Assess the Fetus (if Undelivered)

If the patient is pregnant or very recently postpartum, the fetal status and gestational age may give clues as to the possible etiology of maternal cardiorespiratory insufficiency. Preeclampsia may present with respiratory insufficiency from pulmonary edema. Tocolytics and/or intrauterine infection as may be seen in preterm labor or prolonged preterm rupture of amniotic membranes may be an additional etiology. If the fetus is of sufficient gestational age (≥24 weeks' gestation), fetal

Case 1: Peripartum Cardiomyopathy—cont'd

heart rate monitoring may be needed to assess the moment to moment status of the fetus as well as serve as at least some guide to overall maternal perfusion. Immediate delivery may not always be indicated, although best management depends on the etiology. Evaluation of fetal weight, gestational age, growth, and presence of anomalies via ultrasound once the patient is immediately stabilized is recommended.

Perform General Assessment for Immediately Addressable Causes

Several diagnostic tests are recommended as a part of primary assessment. As mentioned, arterial blood gas analysis determines if metabolic acidosis is present, quantifies the degree of hypoxemia, and assesses ventilatory status. Additionally, once stable, a chest radiograph quickly assesses the presence or absence of pleural effusion, pneumothorax, pulmonary consolidation, and the general presence of pulmonary edema. Although the chest radiography may be performed quickly via portable exam, if pulmonary embolism is suspected, equivalent or better information can be obtained via a helical spiral computed tomography (CT) radiograph. ECG will provide clues regarding acute coronary syndrome, dysrhythmia, cardiomyopathy, and cardiac axis abnormalities. Serum electrolyte analysis, complete blood count, and cardiac enzyme testing (troponin T) complete an initial laboratory analysis, unless presenting clues are more suggestive of particular etiology (obtain urinalysis, blood or sputum cultures, coagulation profile, or liver function testing as indicated clinically).

Determine if Etiology is Pulmonary, Cardiac, or Some Other Cause

Although conceptually thought of in more complex fashion as outlined in the opening paragraphs, initial diagnostic assessment should address if hypoxemia is primarily a respiratory or a nonrespiratory cause. Although interactions may be far more complex, the original event, cause, or pathophysiology undoubtedly gives clues as to principal assessment. Therefore, if one first thinks of pulmonary- and nonpulmonary-inciting processes, some degree of simplification may be accomplished.

Primary pulmonary processes are generally addressed via treatment of the underlying condition and reduction in lung water (see following section). Primary cardiac processes may also result in increased lung water. However, increased edema and hypoxemia are the result of hydrostatic edema due to cardiac pump failure. Transmigration of pulmonary alveolar fluid is dependent on several interrelated factors.

If hypoxia is thought of mechanistically, treatment may be directed against the underlying etiology and the related and unrelated influences affecting passage of fluid. Capillary permeability depends on the inherent properties of the membrane and intrinsic and/or pathophysiologic processes affecting permeability. Membrane capillary incompetence is best addressed by treatment of the underlying condition. Oncotic and hydrostatic components of fluid leakage are directly addressed with therapy. Data are mixed regarding alterations of oncotic forces via the use of plasma expanders and colloid. Hydrostatic derangements are either from an imbalance (excess) of intravascular volume or an apparent excess from cardiac failure (pump failure). Irrespective of the etiology, the most directly addressable determinants are intravascular volume and cardiac pump manipulation. Therefore, treatment of a primarily oncotic problem may be improved by reduction in lung water, with support until improvement of the patient occurs.

Balance Perfusion with Lung Water

As stated, the most addressable options for management of pulmonary edema are hemodynamic and intravascular manipulation. However, often conflicting effects and results occur with inappropriate manipulation of intravascular volume. Marked improvement in oxygenation may occur if massive diuresis, with resulting reduction in pulmonary hydrostatic pressure. However, a concomitant reduction in myocardial preload may cause reduction in cardiac output and tissue perfusion. Hypotension may result, with resulting adverse consequences. Therefore, an assessment of cardiac function and intravascular volume status is the next step in the assessment of a patient such as presented here.

Central venous catheter monitoring provides some direct assessment of right-sided cardiac filling. However, prediction of left-sided filling is often not directly possible with central venous catheter monitoring alone. Evaluation of left-sided cardiac performance and vascular filling has traditionally been addressed with pulmonary artery catheter monitoring. However, lack of demonstrated efficacy when the technology has been studied in specific disease states makes pulmonary artery catheter monitoring a methodology that is used far less now than in the past. With the generalized availability of bedside echocardiography, clinicians have a noninvasive means to ascertain both cardiac function (diminished function may be suggestive of a cardiac etiology) and intravascular volume status. If echocardiography is used in conjunction with clinical assessment and other available monitoring modalities (central venous catheter monitoring, pulse oximetry monitoring, and clinical clues), a reasonable idea can be had of both the underlying pathophysiology of hypoxia and hemodynamic derangement.

Therefore, primary management depends on addressing the modifiable factors producing hypoxia and (if present) cardiac failure. In Figure 7-3, after ABCs are addressed, and determination of cardiac filling is made. In hypervolemic high-filling states, diuresis with loop diuretics (such as furosemide 40 mg IV in the nonpregnant patient or 20 mg IV in the pregnant patient with monitored fetus) is good initial therapy. Left ventricular filling should be optimized to a left ventricular preload of approximately 14 to 18 mm Hg (or as

Case 1: Peripartum Cardiomyopathy—cont'd

estimated). With ventricular filling optimized, the next step is that of perfusion. Vasopressors, inotropic agents, and afterload reduction all may be necessary. Blood pressure and perfusion need to be balanced.

Use Consultants Wisely

The exampled patient as presented had markedly diminished cardiac contractility. Her pulmonary edema resulted from increased pulmonary hydrostatic pressure producing increased lung edema. Her disorder therefore primarily was that of a cardiac etiology. She required subspecialty care from a cardiologist in addition to primary care from an obstetrician. Specific consultation and management of congestive heart failure by the cardiologist afforded best care and follow-up. However, the obstetrician, as the physician with the best general knowledge of pregnant, peripartum, and postpartum women, is also vital to this sick patient's care. Consultants may be confounded by the possibility that the differential diagnosis may contain diseases and disorders unique to pregnancy that they may not be familiar with. Conversely, the obstetrician has an everyday understanding of typically seen pregnancy-related diseases such as preeclampsia and may help guide and assist consultants who do not normally see sick pregnant women. The obstetrician should perform as a good primary care physician and operationally oversee the overall care of the sick patient in his or her care.

Have Correct Location of Care

Correct location of care is critical to the success of dealing with the sick patient with pulmonary edema and congestive heart failure. Each hospital is different in the suite of resources available for care and the repertoire of services that its acute care units provide. Stable, pregnant, or immediately postpartum patients, without the need or anticipated need for mechanical ventilation or invasive monitoring, may be better served through primary care in a labor and delivery environment. The patient requiring multiple inotropic agents and vasopressor therapy or the patient undergoing mechanical ventilation may be more appropriately cared for in a cardiac care unit or general intensive care unit setting. Careful consideration of resources available with agreed-upon location suitable to all physician and nonphysician stakeholders should be an ongoing part of daily management decisions.

Table 7-5 Differential Diagnosis of Pulmonary Edema

Pulmonary	Cardiac
• Pneumonia	• Congestive heart failure
• Pulmonary embolus	• Peripartum cardiomyopathy
• Intrinsic lung disease	• Valvular heart disease (especially mitral stenosis)
• Asthma	• Acute coronary syndrome (ACS), infarction
• Tocolytic therapy	• Other cardiomyopathy (viral or drug induced)
• Preeclampsia	
• Amniotic fluid embolism	
• Acute respiratory distress syndrome (ARDS)	
• Sepsis	

Postpartum and Recovery Management

When compared with most other causes of cardiomyopathy, peripartum cardiomyopathy has a higher rate of eventual spontaneous recovery of ventricular function. Approximately 15% of those diagnosed with peripartum cardiomyopathy die as a result of the diagnosis during the index admission of diagnosis. Approximately 25% recover left ventricular function after 6 months of treatment and approximately one third overall recover after 5 years. Management of failure postdischarge involves periodic assessment of cardiac function via echocardiography until recovery or final stabilization occurs. Treatment by a cardiologist with specific expertise in congestive heart failure is often advised because there is a fair to good chance of recovery of function in some of these patients. Secondary workup of other unusual causes (viral disorders) also should accompany the recovery phase of treatment.

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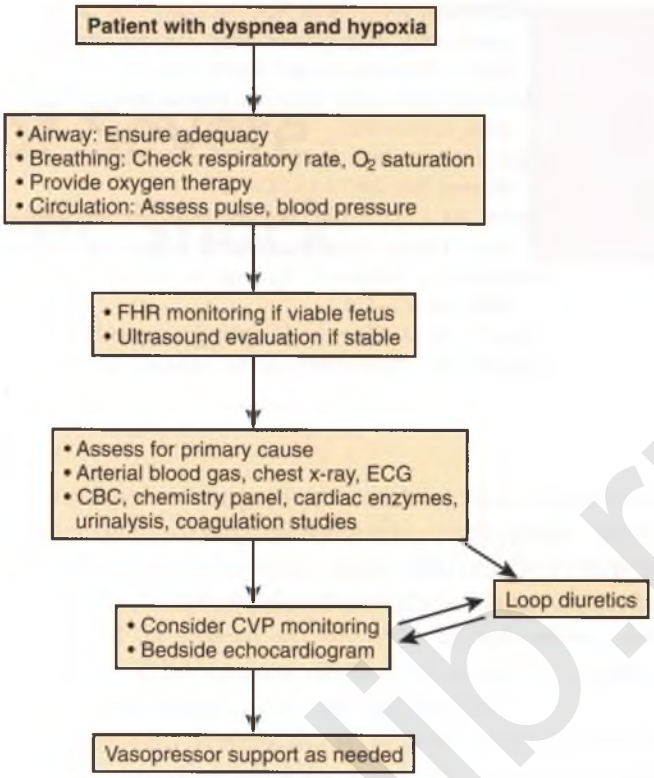

Figure 7-3 Flow diagram/algorithm for treatment of patient with hypoxia. *CBC,* Complete blood count; *CVP,* central venous pressure; *ECG,* electrocardiogram; *FHR,* fetal heart rate.

Studies of subsequent pregnancy outcomes in those patients who have recovered from an initial episode of peripartum cardiomyopathy have an increased risk of heart failure in a subsequent pregnancy. Approximately 20% of those with apparently normal left ventricular function prior to a subsequent pregnancy develop congestive heart failure during that pregnancy. Approximately 45% of those with impaired cardiac function who later attempt pregnancy exhibit congestive heart failure during their next pregnancy. In one study, 7% of those in the impaired function group died during a next pregnancy. A risk may also be present that prepregnancy normal function may deteriorate and remain diminished after the next pregnancy. Therefore, pregnancy should be discouraged in those who previously were diagnosed with peripartum cardiomyopathy who have abnormal residual function. Even those patients who have regained normal function need to understand the potential risks involved in becoming pregnant again. Prepregnancy counseling and contraceptive counseling need to be addressed.

Suggested Readings

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Management of Severe Sepsis and Septic Shock

8

John R. Barton M.D.
Baha M. Sibai M.D.

Sepsis is defined as suspected or proven infection plus a systemic inflammatory response syndrome (e.g., fever, tachycardia, tachypnea, and leukocytosis). Severe sepsis is defined as sepsis with organ dysfunction (hypotension, hypoxemia, oliguria, metabolic acidosis, thrombocytopenia or obtundation), whereas septic shock is defined as severe sepsis with hypotension, despite adequate fluid resuscitation. Potential causes of severe sepsis or septic shock during pregnancy and the postpartum are listed in Table 8-1.

The presenting signs and symptoms in severe sepsis during pregnancy can be variable depending on the etiology as well as the duration of infection (Table 8-2). The most common presenting symptom is fever (>39.0° C [>102° F]) with or without chills; however, in cases of advanced sepsis the patient can develop hypothermia (temperature <36.0° C [<96° F]) with tachycardia (heart rate [HR] >110 beats per minute [bpm]) and tachypnea (respiratory rate [RR] >24/min). In most cases, the location of pain or tenderness assists in determining the etiology of the underlying infection. For example, patients with pyelonephritis present with flank or back pain and the tenderness localized at the costovertebral angle, whereas those presenting with either cholecystitis or pancreatitis have right upper quadrant abdominal pain and tenderness or generalized abdominal pain. On the other hand, patients with endomyometritis, chorioamnionitis, and septic abortion have lower abdominal and pelvic pain in association with uterine and cervical motion tenderness.

The laboratory findings in patients with severe sepsis or septic shock depend on the etiology, the duration of infection, presence of preexisting medical or obstetric disorders, as well as the quality of management used (Table 8-3). It is important to note that traditional laboratory values to define sepsis in nonpregnant women may not apply to pregnant women. The most common laboratory abnormality in patients with septic shock during pregnancy is leukocytosis (usually a white blood count >15,000/mm^3); however, in cases of advanced sepsis the patient can develop leukopenia and neutropenia. In addition, patients with viral sepsis usually have leukopenia. Moreover, most patients have abnormal serum creatinine levels (>1 mg/dL).

Pregnancies complicated by severe sepsis or septic shock are associated with increased rates of preterm labor, fetal infection, and preterm delivery resulting in increased rates of perinatal morbidity and mortality. These complications are related to prematurity or maternal hypoxemia and acidosis. Although septic shock is rare during pregnancy, its development may result in substantial morbidities and even maternal death (Table 8-4). Indeed, the reported maternal mortality from septic shock ranges from 20% to 50% (with higher rates seen in patients with multiple organ failure).

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Table 8-1 Causes of Severe Sepsis/Septic Shock in Pregnancy/Postpartum

- Acute pyelonephritis
- Pneumonia
- Neglected chorioamnionitis
- Endomyometritis
- Wound infections including episiotomy
  - Unrecognized
  - Inadequately treated
- Septic abortion with retained products of conception
- Ruptured appendix
- Infarcted bowel
- Necrotizing pancreatitis
- Acute cholecystitis

Table 8-2 Signs and Symptoms of Severe Sepsis and Septic Shock

- Fever
- Temperature instability (>38° or <36° C)
- Tachycardia (HR >110 bpm), tachypnea (RR >28/min)
- Diaphoresis
- Clammy/mottled skin
- Nausea/vomiting
- Hypotension/shock
- Oliguria
- Pain (location based on site of infection)
- Altered mental state (confusion, decreased alertness)

Table 8-3 Laboratory Findings in Severe Sepsis and Septic Shock

- Leukocytosis or leukopenia
- Hypoxemia
- Thrombocytopenia
- Metabolic acidosis
  - Increased serum lactate
  - Low pH
  - Increased base deficit
- Elevated liver enzymes
- Disseminated intravascular coagulopathy (DIC)
- Elevated serum creatinine

Table 8-4 Maternal Complications of Severe Sepsis and Septic Shock

- Pulmonary edema
- Acute respiratory distress syndrome (ARDS)
- Acute renal failure
- Shock liver (ischemic hepatitis)
- Septic emboli to other organs
- Myocardial ischemia
- Cerebral ischemia
- Disseminated intravascular coagulopathy (DIC)
- Death

Case 1: Severe Sepsis

A 21-year-old G1P0 at 31 weeks of gestation presents during a routine prenatal office visit. She complains of burning on urination, right flank pain, and nausea and vomiting. A urinalysis with a urine culture is obtained. She refuses antibiotics stating, "It might hurt the baby," and the physician does not insist on treatment.

The patient presented 48 hours later to the labor and delivery unit with worsening pain and complaints of fever and chills. Clinical evaluation revealed the presence of right costovertebral angle tenderness in association with the following:

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Case 1: Severe Sepsis—cont'd

- Temperature (100.4° F oral)
- RR (22/min)
- HR (110/min)
- Systolic blood pressure (SBP) 90 mm Hg
- O_2 saturation on room air 94%

Laboratory evaluation revealed:

- White blood cell count (20,400/mm^3 with 15% bands)
- Platelet count (160,000/mm^3)
- Serum creatinine (1.3 mg/dL)
- Serum lactate (3.1 mmol/L)

The organism on urine culture from her previous office visit returned *Escherichia coli;* however, antibiotic sensitivities were not yet available. The patient had no known drug allergies and was started on IV ampicillin 2 g every 6 hours. IV fluid of D_5 ½ normal saline (NS) was started at a rate of 125 mL/hr with no initial bolus of fluid. She was also started on 2 L of oxygen/minute by nasal cannula for tachypnea. Fetal heart rate monitoring revealed fetal tachycardia with moderate variability and absent periodic decelerations. Uterine monitoring revealed the presence of contractions (1 every 10 minutes). There was no cervical change or evidence of rupture of membranes on pelvic examination.

Eight hours following admission to the hospital her clinical evaluation changed to the following:

- Temperature (102.4° F oral)
- RR (60/min)
- HR (140/min)
- SBP (70 mm Hg)
- O_2 saturation on 2 L oxygen by nasal cannula 88%
- Urine output for 8 hours (120 mL)

Repeat laboratory evaluation showed the following:

- White blood cell count (31,200/mm^3 with 20% bands)
- Platelet count (90,000/mm^3)
- Serum creatinine (2.1 mg/dL)
- Serum lactate (5.8 mmol/L)
- Serum AST (100 IU/L)

Fetal heart rate monitoring continued to reveal fetal tachycardia, but now also with minimal variability, absent accelerations, and no decelerations. Uterine contraction monitoring revealed a contraction frequency of 1 every 6 minutes. Repeat cervical examination was unchanged. Because of her severe hypoxia and tachypnea, an anesthesiologist was paged to intubate the patient, which was accomplished without difficulty. Following intubation, the patient was transferred to the intensive care unit. An immediate chest x-ray noted appropriate placement of the endotracheal tube but lung findings were consistent with acute respiratory distress syndrome (ARDS). A diagnosis of severe sepsis was made and a severe sepsis protocol was initiated that included infectious disease, pulmonary medicine, and pharmacy consultations, as well as the initiation of broad-spectrum antibiotics and central hemodynamic monitoring.

Subsequent management included a change in antibiotic therapy to the third-generation cephalosporin, ceftazidime 1 g IV every 8 hours. Aminoglycosides such as gentamicin could also have been chosen for antibiotic coverage for *E. coli* but must be used in caution with impaired renal function due to their potential for nephrotoxicity. Management should also include placement of invasive hemodynamic monitoring for fluid management and evaluation of cardiac function as well as acetaminophen therapy to reduce maternal fever to potentially improve fetal tachycardia. The urine culture sensitivity results revealed that the *E. coli* was resistant to ampicillin but sensitive to ceftazidime. With aggressive therapy the patient had gradual improvement including extubation 48 hours later and becoming afebrile 72 hours after the change in antibiotic therapy. Fetal heart rate monitoring revealed a normal baseline with moderate variability and presence of accelerations. Uterine monitoring revealed no contractions. The patient was discharged home on suppression antibiotics and subsequently had a spontaneous vaginal delivery at term.

Discussion

Early detection of disease process and intervention can improve the outcome and survivability in severe sepsis and septic shock. Early provision of time-sensitive therapies and standardized therapies of best practice have been shown to decrease mortality, hospital cost, and hospital length of stay in randomized studies in complicated patients. This finding underscores the importance of developing and implementing a severe sepsis protocol that includes goal-directed therapy. This requires the involvement of a multidisciplinary approach that includes physicians, nursing, pharmacy staff, and hospital administration.

In this case, the initial antibiotic choice of ampicillin alone was inappropriate because many *E. coli* isolates in pregnancy are resistant to this agent. The patient should have received a combination of agents such as ampicillin and gentamicin or initial treatment with an alternate agent such as a cephalosporin. In addition, fluid resuscitation should have been more aggressive and used normal saline or lactated Ringer's solution. In patients presenting with acute pyelonephritis in pregnancy with hypotension, initial fluid resuscitation should be performed initially as rapid infusions of warmed fluids (1 to 2 L over 60 minutes). Subsequent IV infusions are guided by maternal vital signs, pulse oximetry, and urine output to avoid the development of pulmonary edema. Colloids do not appear to be superior to crystalloids but attempts should be made to avoid excess free water (i.e., with use of 0.9% NS).

During the observation period, the patient was having a gradual deterioration in her cardiorespiratory status that culminated with the development of septic shock. Appropriate management should have included the initiation of the severe sepsis protocol. This would have consisted of admission to an

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Case 1: Severe Sepsis—cont'd

intensive care unit or a special obstetric care unit for close and continuous monitoring of vital signs, oxygenation, and fluid intake/output. To achieve adequate organ perfusion and avoid the development of pulmonary edema, fluid resuscitation should be directed by central hemodynamic monitoring with physiologic perfusion endpoints including mean arterial pressure ≥65 mm Hg, central venous pressure 8 to 12 mm Hg or urine output >25 L/hr. This management could have avoided the need for mechanical ventilation and progression to septic shock.

Patients with acute pyelonephritis during pregnancy can develop uterine contractions (with or without cervical change) as a result of release of endotoxins. In general, most patients respond to hydration and the contractions resolve after treatment. As a result, true preterm labor is not common. However, on occasion, a patient develops true preterm labor with cervical dilation, suggesting the need for tocolytic therapy. A major concern with tocolytic therapy in this clinical setting is increasing the risk of pulmonary edema particularly if β-agonists are used. Therefore, in certain instances such as gestational age less than 32 weeks, tocolytic therapy with magnesium sulfate to allow time for corticosteroid administration for fetal benefit may be considered.

Management of Septic Shock in Pregnancy and Postpartum

The outcome and survivability in severe sepsis and septic shock in pregnancy are improved with early detection, prompt recognition of the source of infection, and targeted therapy. This can be achieved by formulating a stepwise approach that consists of early provision of time-sensitive interventions such as aggressive hydration, initiation of appropriate antibiotics, central hemodynamic monitoring, and the involvement of pharmacy, infectious disease specialists, and critical care specialists. When there is evidence of infected tissue requiring operative intervention for source control, the appropriate gynecologic or surgical specialists should be involved as the patient is stabilized. Even with appropriate antibiotic therapy, however, the patient will continue to deteriorate unless septic foci (i.e., abscess, necrotic tissue) are surgically excised. The initial step in management in goal-directed therapy is to identify sepsis and the site of infection. These patients will require prompt surgical intervention (Table 8-5). Another important question relates to the need for delivery in pregnant women with severe sepsis/septic shock. Indications for delivery in such women are listed in Table 8-6. If septic shock is diagnosed, the physician should then activate septic shock standard orders (Table 8-7). Therapy goals in the management of severe sepsis/septic shock are outlined in Table 8-8. Appropriate cultures should be obtained and antibiotic therapy initiated within 1 hour. Those at high risk for septic shock should be identified by SBP <90 mm Hg despite fluids (20 to 40 mL/kg) and serum lactate level >4 mmol/L. In addition, there are indicators that suggest a poor outcome in patients with established septic shock (Table 8-9).

Table 8-5 Indications for Surgical Intervention in Severe Sepsis/Septic Shock

- Cholecystitis with bile duct obstruction
- Necrotizing pancreatitis
- Perinephric abscess
- Acute appendicitis
- Obstructing renal stone
- Retained product of conception
- Uterine microabscess/gas gangrene
- Bowel infarction
- Pelvic abscess
- Necrotizing fasciitis
- Infected episiotomy site

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Table 8-6 Indications for Delivery

- Maternal
  - Intrauterine infection
  - Development of DIC
  - Compromised cardiopulmonary function by uterine size and/or peritoneal fluid
    - Compartment syndrome
    - Hydramnios
    - Multifetal gestation
  - Severe ARDS/barotrauma
  - Cardiopulmonary arrest
- Fetal
  - Fetal demise
  - Gestational age associated with low neonatal morbidity/mortality

Table 8-7 Septic Shock Standard Orders

Hemodynamic Management

- Central line and arterial line placement
  - Vasoactive agents if mean arterial pressure (MAP) <65 mm Hg
  - Inotropes if $Scvo_2$ remains <70%
- Fluid resuscitation (6 to 10 L can be needed)
  - Use warm normal saline or lactated Ringer's
  - Rapid infusion (3 L over 20 minutes)
  - Delay in restoring circulating volume increases morbidity/mortality
  - Physiologic perfusion endpoints
    - CVP 8 to 12 mm Hg
    - Mean arterial pressure >65 mm Hg
    - Urine output >25 mL/hr
- Oxygen and mechanical ventilation
  - Sedation, analgesia, neuromuscular blocker

Antimicrobial Therapy

- Prompt cultures
  - Don't delay therapy awaiting cultures
  - Survival differences seen in delay of antibiotic therapy of only 1 hr
- Prompt empiric antibiotic therapy
  - Gentamicin 1.5 mg/kg IV, then 1 mg/kg IV every 8 hr
  - Clindamycin 900 mg IV every 8 hr
  - Penicillin 3 million units IV every 4 hr

  or
  - Vancomycin 15 mg/kg IV, then dosing by pharmacy
  - Piperacillin/tazobactam (Zosyn) 4.5 g IV stat, then every 6 hr

  or
  - Per standard hospital antibiotic protocol for severe sepsis/septic shock

Search and Eliminate Source of Sepsis

- Retained products or necrotic uterus
- Appendix or gallbladder
- Pancreatitis
- Debridement of infected tissue (incision, episiotomy, fascia)
- Abscess

Table 8-8 Therapy Goals in the Management of Severe Sepsis and Septic Shock

Initial Resuscitation Phase (First 6 Hours)

- Blood cultures obtained (goal within 1 hr)
- Antibiotics initiated (goal within 1 hr)
- Central line placed (goal within 4 hr)
- CVP >8 mm Hg (goal within 6 hr)
- Lactic acid level drawn
- Norepinephrine drip started if indicated (MAP <65 mm Hg after volume resuscitation)
- Transfusion of packed red blood cells if indicated

Maintenance Phase (6 to 24 Hours)

- Drotrecogin (Xigris) administration considered
- Insulin protocol initiated if indicated

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Table 8-9 Prognostic Indicators of Poor Outcome in Septic Shock

- Delay in initial diagnosis
- Delay in appropriate antibiotic therapy
- Preexisting debilitating disease process
- Poor response to massive IV fluid resuscitation
- Depressed cardiac output
- Reduced oxygen extraction
- Presence of ARDS or renal failure
- High serum lactate (>5 mmol/L)

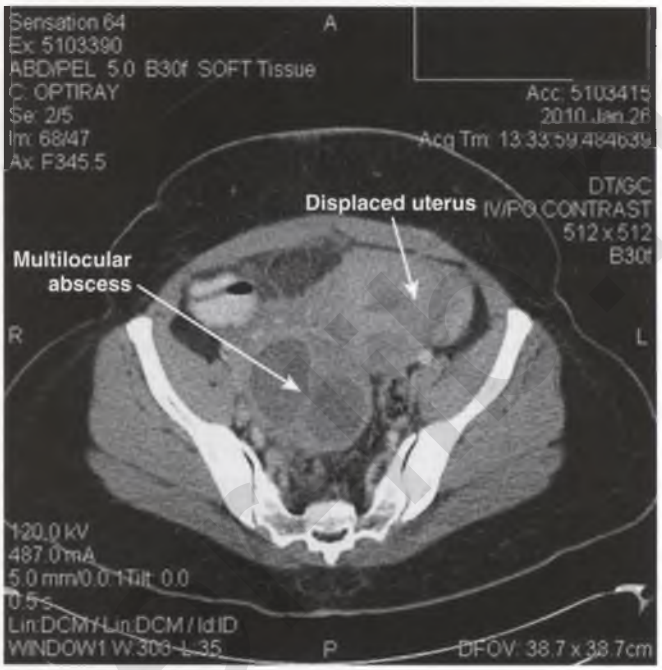

Figure 8-1 Postdelivery abdominal CT scan revealing multiloculated abscess.

Once the hemodynamic status has been addressed and antibiotic therapy initiated, the next step is to search for and eliminate a potential surgical source of infection. For patients who have endomyometritis following uterine surgery or a septic abortion and who fail to respond despite aggressive antibiotic therapy, it is important to rule out the presence of pelvic or abdominal abscess or microabscesses of the uterus. Imaging of the abdomen or pelvis should be performed to search for abscess (Fig. 8-1) or intramyometrial gas formation (Fig. 8-2) with prompt surgery for abnormal findings including hysterectomy and bilateral salpingo-oophorectomy. Patients with an abdominal incision or episiotomy, particularly obese women with medical disorders such as diabetes mellitus, nephritic syndrome, and autoimmune disorders requiring immunosuppressive therapy should be considered at very high risk for wound complications including abscess formation and necrotizing fasciitis. If these patients have severe sepsis that does not respond to aggressive therapy, they warrant prompt imaging and a detailed physical examination.

In cases of necrotizing fasciitis, physical examination will reveal skin that is tender, erythematous, and warm. The skin often will be without sensation secondary to necrosis of the superficial nerves. X-ray or computed tomography (CT) imaging usually shows subcutaneous gas suggestive of clostridial infection. The diagnosis of necrotizing fasciitis is a surgical emergency. Even with appropriate antibiotic therapy, the patient's condition will continue to deteriorate unless the septic foci are eliminated. Management of this condition includes

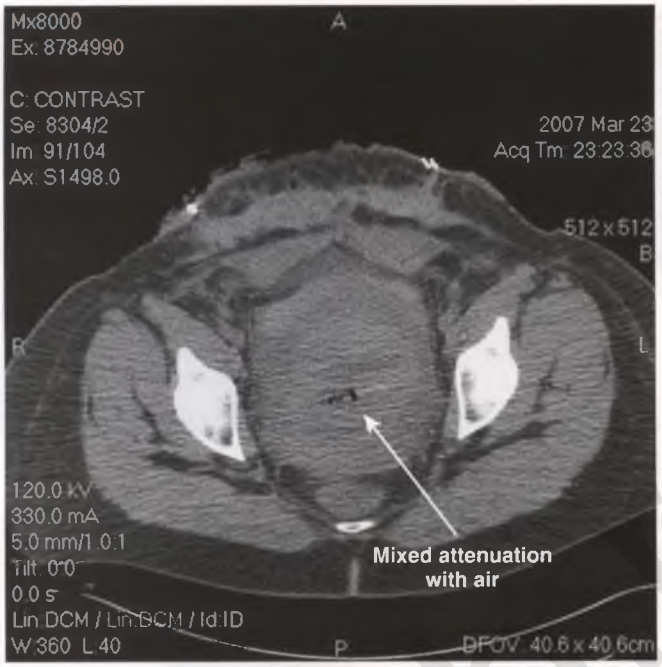

Figure 8-2 Postdelivery abdominal CT scan revealing mixed attenuation within the endometrial cavity containing air.

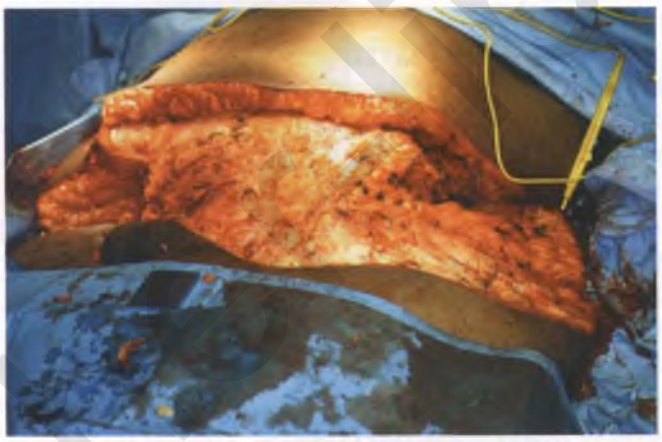

Figure 8-3 Extensive abdominal wall and fascia dissection necessitated by postcesarean delivery necrotizing fasciitis.

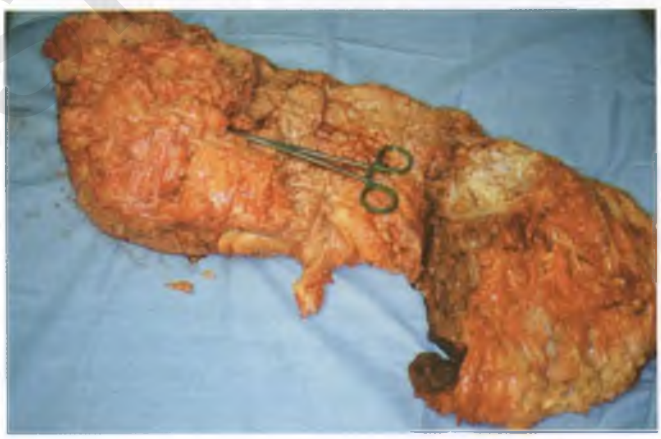

Figure 8-4 Surgical debridement specimen for patient in Figure 8-3 with a diagnosis of necrotizing fasciitis.

resection of the involved tissue. The incision should be made through the involved tissue down to the fascia with extensive dissection (Fig. 8-3) and removal of the affected tissue (Fig. 8-4) until well vascularized healthy tissue is reached at the margins. Further, tissue should be submitted for pathology evaluation (Fig. 8-5) as well as culture and bacterial sensitivities. The incision is packed open and then debrided on a daily basis as necessary. Once recovered,

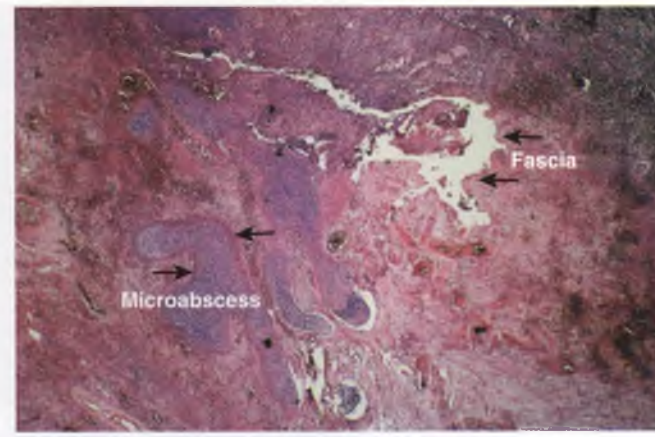

Figure 8-5 Pathology sections revealing microabscess in the debridement specimen for patient in Figure 8-3.

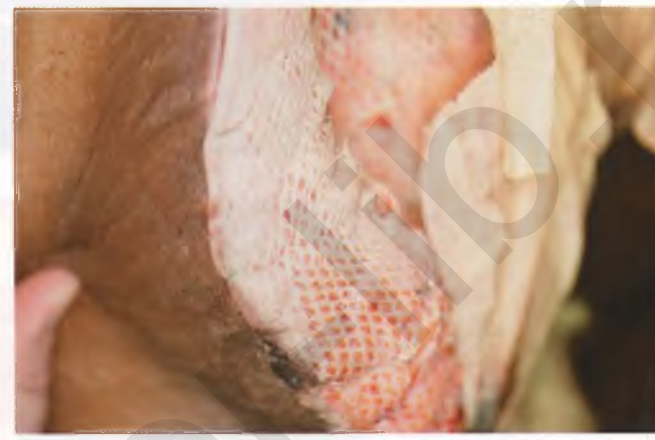

Figure 8-6 Skin grafts placed to improve wound healing after debridement of incision in a patient with necrotizing fasciitis.

allograft or xenograft skin can be used to cover the open incision (Fig. 8-6). Finally, it is always advisable to seek surgical consultation to rule out nongynecologic sources of tissue necrosis such as appendiceal or pancreatic abscesses and infarcted bowel.

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9

Management of Hypertensive Crisis Including Stroke

John R. Barton M.D.
Baha M. Sibai M.D.

Cerebrovascular accidents (hypertensive encephalopathy and hemorrhage) are rare complications during pregnancy and the postpartum period. The exact incidence of these complications during pregnancy and the first 6 weeks postpartum is unknown. Several population-based studies, however, have consistently shown that the risk of these complications is increased compared with the nonpregnant state. The issue of cerebral vascular accidents during pregnancy and the postpartum period should be of increasing relevance to obstetricians, anesthesiologists, emergency department physicians, neurologists, and critical care physicians. The incidence of stroke increases with age, and women are becoming pregnant at older ages than ever before; obstetric patients ages 45 to 50 years have become increasingly common. Obesity is also a risk factor for stroke, and pregnant women mirror American society at large, in which obesity has become epidemic. Long-standing hypertension and diabetes mellitus, both associated with obesity, further increase the risk of stroke. The risk of stroke in the postpartum period is almost certainly higher still. Postpartum strokes generally occur from 3 days to 6 weeks after delivery—a vulnerable time when significant physiologic, hematologic, and hemodynamic changes occur.

Stroke can happen at any time during pregnancy and in the postpartum period. The clinical presentation is similar to that seen in nonpregnant patients; however, the presenting signs and symptoms can mimic those seen in preeclampsia/eclampsia, and the possibility of cerebrovascular accidents may be overlooked. Table 9-1 lists the potential etiologies of cerebrovascular accidents during pregnancy and postpartum.

Hypertension is one of the most common medical disorders affecting pregnancy. It complicates 12% of pregnancies and is responsible for 18% of maternal deaths in the United States. Cerebrovascular accidents have been recognized as a complication of hypertensive disorders of pregnancies for many years. Several studies have reported that hypertensive disorders of pregnancy are associated with 11% to 47% of cases of hypertensive encephalopathy and with 8% to 50% of cerebral hemorrhage during pregnancy and within the first 6 weeks postpartum.

The presenting signs and symptoms of cerebrovascular accidents in pregnancy and postpartum are summarized in Table 9-2. In general, there is potential for the delay in making the correct diagnosis and treatment of these patients

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Table 9-1 Potential Etiologies of Cerebrovascular Accidents During Pregnancy and Postpartum

Vascular Causes
• Migraine • Aneurysms • AV malformation or angiomas • Arterial or venous thrombosis • Cerebral vasospasm syndromes • Arterial dissection • Collagen vascular disease • Cerebral angiitis • Hypotensive hypoxic ischemic encephalopathy
Hypertensive Causes
• Severe acute hypertension • Untreated long-standing hypertension • Preeclampsia-eclampsia
Disorders of Coagulation
• Thrombophilias (congenital and acquired) • Hemoglobinopathies • Thrombotic thrombocytopenic purpura • Disseminated intravascular coagulopathy (DIC) • Leukemias
Medications/Drugs
• Cocaine • Ergot derivatives • Sympathomimetics or serotonergics • Catecholamines (pheochromocytoma)
Other
• Metastatic choriocarcinoma • Postdural puncture syndrome

Table 9-2 Signs and Symptoms of Hypertensive Encephalopathy or Cerebral Hemorrhage

- Persistent headaches
- Visual changes
  - Blurred vision
  - Photophobia
  - Scotomata
  - Retinal detachment
  - Blindness
- Epigastric pain
- Nausea and vomiting
- Shortness of breath/chest tightness
- Neurologic deficits
  - Seizures
  - Altered mental status
  - Motor deficits
  - Coma
- Severe hypertension
- Low to normal pulse

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because these signs and symptoms can mimic those of preeclampsia-eclampsia. In addition, patients may hesitate to seek immediate medical care when experiencing symptoms of headache, nausea, vomiting, and epigastric pain because they are common in pregnancy and postpartum, and some women might not consider them serious enough for evaluation unless they become severe. Moreover, the presence of these symptoms may not be aggressively evaluated by obstetricians and emergency department physicians, particularly in women who are normotensive and in the late postpartum period. Therefore, it is prudent to counsel and educate patients, especially those with relevant risk factors (diagnosed gestational hypertension, preeclampsia, chronic hypertension) to seek care for these symptoms.

The laboratory findings (Table 9-3) and the neuroimaging findings (Table 9-4) in patients with cerebrovascular accidents overlap with those in severe preeclampsia-eclampsia. In making the diagnosis of cerebrovascular accident, history and physical examination are critically important, although neuroimaging and other procedures such as lumbar puncture may be needed to further clarify the diagnosis and establish the etiology. A potential delay in making the correct diagnosis and initiating proper therapy may arise because of failure to perform neuroimaging and other relevant procedures secondary to the concern of theoretic risks to the pregnancy and fetus.

Maternal and perinatal complications associated with cerebrovascular accidents depend on etiology, timing of the diagnosis, maternal and fetal conditions, and gestational age at time of diagnosis, as well as the quality of management used. In general, maternal complications include death and residual neurologic deficit, whereas perinatal complications may include fetal/neonatal death, fetal hypoxia, and prematurity.

Table 9-3 Hypertensive Encephalopathy or Cerebral Hemorrhage

Laboratory Findings
• Thrombocytopenia • Elevated liver enzymes • Hemolysis • Renal function changes • Proteinuria • Electrocardiogram changes

Table 9-4 Cerebrovascular Accidents During Pregnancy and Postpartum

Neuroimaging Findings

CT/MRI
• Focal or diffuse cerebral edema
• Posterior reversible encephalopathy syndrome (PRES)
• Infarction
• Hemorrhage
  • Intraparenchymal
  • Intraventricular
  • Subarachnoid
  • Subdural
Magnetic resonance angiography (MRA) or four-vessel traditional angiography
• Thrombosis
• Vasoconstriction/vasospasm, angiopathy
Magnetic resonance venography (MRV)
• Sinus venous thrombosis
• Cortical vein thrombosis

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Case 1: Hypertensive Encephalopathy

A 40-year-old G1P0 presents in the first trimester with blood pressure 170/120 mm Hg. The patient gives a history of chronic hypertension for the past 5 years that was treated with angiotensin-converting enzyme (ACE) inhibitor and diuretics. With the diagnosis of pregnancy, her primary care physician discontinued the ACE inhibitor due to concern for fetal effects and switched her to oral methyldopa at a dose of 250 mg twice daily. Due to continued hypertension, the methyldopa dose was increased to 500 mg twice daily. During the subsequent prenatal visits, her blood pressure continued to worsen and the dose of methyldopa was increased to 1000 mg three times daily.

At 35 weeks' gestation she was seen in the office with a blood pressure 170/105 mm Hg. The patient complained of a severe headache but denied shortness of breath, vision changes, and epigastric pain. Urine dipstick for protein was trace. Because of concern for preeclampsia, the patient was sent to the obstetric triage for nonstress testing and repeat blood pressure measurements. She was observed for 1 hour during which acetaminophen was given and her headache improved. A nonstress test was performed, which was reactive. Repeat blood pressure measurements during the observation period ranged between 160 to 180 and 100 to 115 mm Hg. The physician ordered oral labetalol with a first dose of 200 mg to be given prior to discharge with a prescription for 200 mg twice a day. In addition, he instructed the patient to take acetaminophen as needed if her headache returned. The triage nurse questioned the physician as to the appropriateness of these recommendations because of her concerns of the degree of hypertension and suggested admission for further monitoring and evaluation. Unfortunately, the physician did not order further evaluation in part because the urine dipstick was trace for protein, and preeclampsia laboratory tests had recently been preformed and were normal.

Previous laboratory studies as an outpatient:

AST	30 units/L (normal <40 units/L)
ALT	35 units/L (normal <60 units/L)
Uric acid	4.5 mg/dL (normal <6 mg/dL)
Creatinine	0.7 mg/dL (normal <1 mg/dL)
Platelet count	200,000/mm^3 (normal >100,000/mm^3)
Hemoglobin	12.3 g/dL (normal >10 g/dL)

The patient was instructed to return to the office in 1 week. Because of her concern, the nurse instructed the patient to notify her physician's office if she developed visual changes or epigastric pain.

The following evening, the patient's headache worsened despite acetaminophen. She was unable to sleep due to the headache and shortness of breath. At 6 AM, her husband noticed her to be confused with inability to see. He attempted to call her physician's office and was instructed to bring his wife to the hospital. Within 5 minutes the patient started seizing and fell to the floor unconscious. He called 911. On arrival the paramedics noted she was unresponsive with a blood pressure of 240/130 mm Hg with a pulse of 50 beats per minute (bpm). The patient was intubated, IV access was obtained, and she was transferred immediately to the hospital.

On arrival to the hospital, the fetal heart rate tracing was Category III for which the patient was rushed for emergency cesarean section. She delivered a live infant with a birth weight of 2500 g with Apgar scores of 0 at 1 minute, 2 at 5 minutes, and 4 at 10 minutes. Prior to cesarean section the patient was noted to have bilateral rales. The pulse oximetry was 90%. During the postoperative period she was rushed for a cranial computed tomography (CT) scan that revealed diffuse cerebral edema with uncal herniation (Fig. 9-1). She was later declared brain dead with ultimate demise 3 days postdelivery.

Discussion

This patient had several risk factors for hypertensive encephalopathy including advanced age, long-standing chronic hypertension requiring two medications for control when not pregnant, and severe hypertension in the first trimester not adequately controlled with methyldopa. At 35 weeks' gestation when she presented with an acute exacerbation of severe hypertension with probable superimposed preeclampsia, the patient should have been admitted to the hospital for further evaluation and treatment. This should include laboratory evaluation with a complete blood count, platelet count, and liver function tests as well as assessment for pulmonary edema with pulse oximetry or chest x-ray. Early detection of superimposed preeclampsia is critical to reduce the risk of maternal and fetal complications.

Sudden increases in blood pressure to severe ranges (diastolic >120 mm Hg) are associated with increased maternal mortality and morbidity (acute and long term). Such severe episodes of hypertension can result in the development of intracerebral hemorrhage, hypertensive encephalopathy, acute renal failure, congestive heart failure, malignant ventricular arrhythmias, or abruptio placentae with resultant disseminated intravascular coagulopathy. As a hypertensive emergency, attempts should be made to reduce the blood pressure to a safe range within 1 hour of diagnosis.

Continuous fetal monitoring should be used during antihypertensive medication titration to assess fetal well-being in all women with a gestational age beyond 24 weeks. Further, therapy goals are not for the return of normal blood pressure, but rather reduction of blood pressure to a level associated with a decreased risk of cerebrovascular accidents or loss of cerebral autoregulation. Targets for blood pressure control vary considering whether the patient is antepartum or postpartum as well as the presence of chronic hypertension or thrombocytopenia, defined in this setting as <100,000 mm^3. Generally, sustained blood pressure values of at least 160 mm Hg systolic or at least 110 mm Hg diastolic are indications to initiate antihypertensive therapy antepartum

Case 1: Hypertensive Encephalopathy—cont'd

and intrapartum. For women with thrombocytopenia and those in the postpartum period, sustained blood pressures of at least 150 mm Hg systolic or 100 mm Hg diastolic are indications to initiate antihypertensive therapy. Pregnant patients with essential hypertension without evidence of preeclampsia or end-organ damage can be managed as per JNC-7 guidelines. It is important that the obstetric service or obstetrician be involved as early as possible in women with a gestational age beyond 24 weeks to assist in antihypertensive management decisions that may affect the fetal status.

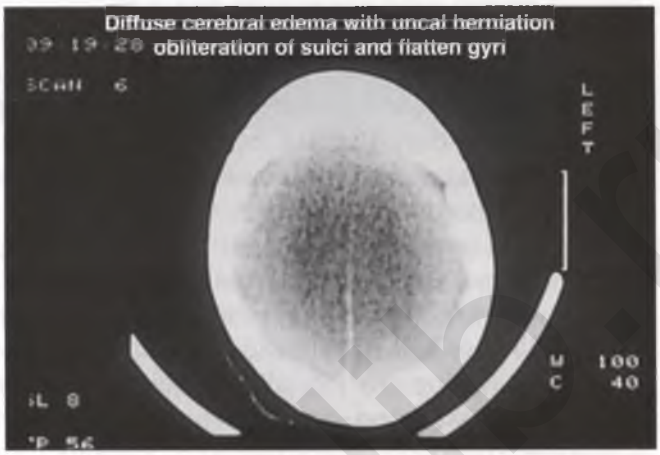

Figure 9-1 Cranial CT scan revealing diffuse cerebral edema with uncal herniation.

Hypertensive Encephalopathy

Untreated essential hypertension progresses to a hypertensive crisis in up to 1% to 2% of cases for unknown reasons. Hypertensive encephalopathy is usually seen in patients with a systolic blood pressure (BP) above 250 mm Hg or a diastolic BP above 150 mm Hg. Patients with acute onset of hypertension may develop encephalopathy at pressure levels that are generally tolerated by those with chronic hypertension. Normally, cerebral blood flow is approximately 50 mL/100 g tissue per minute. When the BP falls, cerebral arterioles normally dilate, whereas when BP increases they constrict to maintain constant cerebral blood flow. This mechanism usually remains operative between 60 and 120 mm Hg diastolic BP. Hypertensive encephalopathy is considered to be a derangement of the autoregulation of cerebral arterioles, which occurs when the upper limit of autoregulation is exceeded (Fig. 9-2). With severe

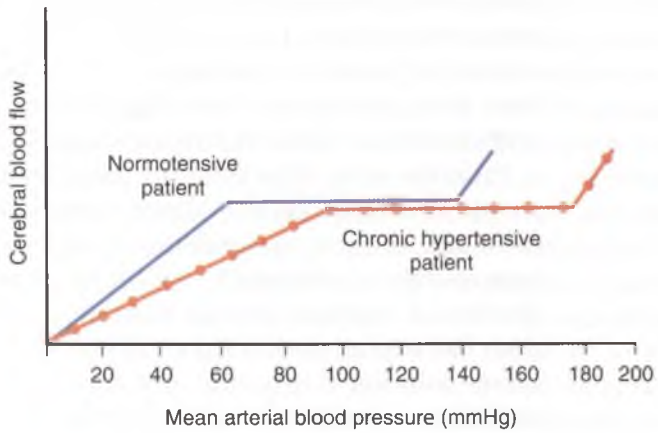

Figure 9-2 Relationship between mean arterial blood pressure and cerebral blood flow in normotensive and chronic hypertensive patients. (Adapted from Standgaard S: Autoregulation of cerebral circulation in hypertension. Acta Neurol Scan 1978;57[suppl 66]:1.)

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hypertension (mean arterial pressure of 130 to 150 mm Hg), cerebral blood vessels constrict as much as possible and then reflex cerebral vasodilatation occurs. This results in overperfusion, damage to small blood vessels, cerebral edema, and increased intracranial pressure (breakthrough theory). Others believe that hypertensive encephalopathy results from an exaggerated vasoconstrictive response of the arterioles resulting in cerebral ischemia (overregulation theory). Patients who have impaired autoregulation involving the cerebral arterioles may experience necrotizing arteriolitis, microinfarcts, petechial hemorrhage, multiple small thrombi, or cerebral edema. Typically, hypertensive encephalopathy has a subacute onset over 24 to 72 hours.

During a hypertensive crisis, other evidence for end-organ damage may be present: cardiac, renal, or retinal dysfunction secondary to impaired organ perfusion and loss of autoregulation of blood flow. Ischemia of the retina (with flame-shaped retinal hemorrhages, retinal infarcts, or papilledema) may occur causing decreased visual acuity. Impaired regulation of coronary blood flow and marked increase in ventricular wall stress may result in angina, myocardial infarction, congestive heart failure, malignant ventricular arrhythmia, pulmonary edema, or dissecting aortic aneurysm. Necrosis of the afferent arterioles of the glomerulus results in hemorrhages of the cortex and medulla, fibrinoid necrosis, and proliferative endarteritis resulting in elevated serum creatinine (>3 mg/dL), proteinuria, oliguria, hematuria, hyaline or red blood cell casts, and progressive azotemia. Severe hypertension may be associated with abruptio placentae with resultant disseminated intravascular coagulopathy (DIC). In addition, high levels of angiotensin II, norepinephrine, and vasopressin accompany ongoing vascular damage. These circulating hormones increase relative efferent arteriolar tone resulting in sodium diuresis and hypovolemia. Because levels of renin and angiotensin II are increased, the aldosterone level is also elevated. The effect of these endocrine changes may be important in maintaining the hypertensive crisis.

Treatment of Hypertensive Encephalopathy

The ultimate goal of therapy is to prevent the occurrence of hypertensive organ damage. Patients at risk for a hypertensive crisis should receive intensive management during labor and for a minimum of 48 hours after delivery. Although pregnancy may complicate the diagnosis, once the life-threatening conditions are recognized, pregnancy should not in any way slow or alter the mode of therapy. The only reliable clinical criterion to confirm the diagnosis of hypertensive encephalopathy is prompt response of the patient to antihypertensive therapy. The headache and sensorium often clear dramatically—sometimes within 1 to 2 hours after treatment. The overall recovery may be somewhat slower in patients with uremia and those in whom the symptoms have been present for a prolonged period before the therapy is given. Sustained cerebrovascular deficits should suggest other diagnoses.

Patients with hypertensive encephalopathy or other hypertensive crisis should be hospitalized. IV lines should be inserted for fluids and medications. Although there is a tendency to restrict sodium intake in patients with a hypertensive emergency, volume contraction from sodium diuresis may be present. A marked drop in diastolic BP with a rise in heart rate on standing from the supine position is evidence of volume contraction. Infusion of normal saline solution during the first 24 to 48 hours to achieve volume expansion should be considered. Saline infusion may help decrease the activity of the

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renin-angiotensin-aldosterone axis and result in better BP control. Simultaneous repletion of potassium losses and continuous monitoring of BP, volume status, urinary output, electrocardiographic readings, and mental status are mandatory. An intraarterial line provides the most accurate BP information. Laboratory studies include a complete blood count with differential, reticulocyte count, platelets, and blood chemistries. A urinalysis should be obtained for protein, glucose, blood, white cells, casts, and bacteria. Assessment for end-organ damage in the central nervous system, retina, kidneys, and cardiovascular system should be done periodically. Antepartum patients should have continuous fetal monitoring if gestational age is beyond 24 weeks.

Lowering Blood Pressure

There are risks associated with too rapid or excessive reduction of elevated BP. The aim of therapy is to lower mean BP by no more than 15% to 25%. Small reductions in BP in the first 60 minutes, working toward a diastolic level of 100 to 110 mm Hg are recommended. Although cerebral blood flow is maintained constantly over a wide range of BPs, there is a lower as well as an upper limit to autoregulation (see Fig. 9-2). In chronic hypertensive women who have a rightward shift of the cerebral autoregulation curve secondary to medial hypertrophy of the cerebral vasculature, lowering BP too rapidly may produce cerebral ischemia, stroke, or coma. Coronary blood flow, renal perfusion, and uteroplacental blood flow also may deteriorate, resulting in acute renal failure, myocardial infarction, fetal distress, or death. Hypertension that proves increasingly difficult to control is an indication to terminate the pregnancy.

Desirable antihypertensive agent properties for use in hypertensive emergencies in pregnancy include a rapid onset of action after administration and short duration of action in the event of overtitration. Previously the most commonly used agent for the acute treatment of severe hypertension in pregnancy was IV hydralazine, a direct arteriolar vasodilator, given as bolus injections of 5 to 10 mg every 15 to 20 minutes for a maximum dose of 30 mg. Several drugs were compared with hydralazine in small, randomized trials. The results of these trials were the subject of a systematic review that suggested that IV labetalol or oral nifedipine is as effective as hydralazine. The final recommendation was that agents chosen should be based on the clinician's experience and familiarity with a particular drug, and on what is known about adverse effects. The recommended dose of labetalol is 20 to 80 mg IV every 10 to 15 minutes for a maximum of 220 mg, and the dose of nifedipine is 10 to 20 mg orally every 30 minutes for a maximum dose of 50 mg in 1 hour.

Sodium nitroprusside is an extremely effective agent for the emergent treatment of patients in hypertensive crises. Due to its immediate onset of action and very short duration of action (1 to 10 minutes), it must be given as a continuous IV infusion. Pharmacologically it relaxes arteriolar and venous smooth muscle equally by interfering with both influx and the intracellular activation of calcium. It is hepatically metabolized and renally excreted. Because preeclamptic patients have a propensity for depleted intravascular volume, they are especially sensitive to its effects. The initial infusion dose should therefore be 0.2 μg/kg/min rather than 0.5 μg/kg/min as is customary in nonpregnant patients. Cyanide and thiocyanate are both metabolic products of sodium nitroprusside metabolism. Thiocyanate toxicity, manifested as tinnitus delirium or blurred vision, should be monitored. Further, although cyanide intoxication is rare, it can be of particular concern in the neonate for patients treated intrapartum. In addition,

preeclamptic patients have a propensity for depleted intravascular volume; these patients are especially sensitive to nitroprusside and may have overshoot hypotension when nitroprusside is used.

Nitroglycerin is an arterial but mostly venous dilator with an immediate onset of action. It is given as an IV infusion of 5 μg/min that is gradually increased every 3 to 5 minutes to titrate for blood pressure control with a maximum dose of 300 μg/min. It is the drug of choice in preeclampsia associated with pulmonary edema and for control of hypertension associated with tracheal manipulation. Side effects such as headache, tachycardia, and methemoglobinemia may develop. It is contraindicated in hypertensive encephalopathy because it increases cerebral blood flow and intracranial pressure.

The use of ACE inhibitors in pregnancy has been associated with an increased risk of intrauterine fetal demise, renal dysgenesis, oligohydramnios, and neonatal renal dysfunction. As such, ACE inhibitors are contraindicated during pregnancy but may be considered for hypertensive control in postpartum patients. Esmolol should also be avoided in pregnancy given the risk of fetal bradycardia as well as neonatal hyotonia and bradycardia. The initial dose, maximum dose, peak effects, and potential side effects of various antihypertensive drugs used in pregnancy or postoperatively are summarized in Table 9-5.

Table 9-5 Antihypertensive Drugs for Use in Hypertensive Emergencies in Pregnancy

Drug	Route	Initial Dose	Onset of Action	Peak Effect	Repeat Dose	Comments
Hydralazine	IV	5-10 mg	5-20 min	10-80 min	10 mg	Avoid with severe headache and tachycardia
Labetalol	IV	20 mg	2-5 min	5-15 min	20-80 mg	Avoid in bradycardia and severe asthma
Nifedipine	PO	10 mg	20 min	0.5-2 hr	10-20 mg	Avoid with headache and tachycardia
Nitroprusside	IV infusion	0.2 μg/kg/min	30-60 sec	1-2 min	Max dose 10 μg/kg/min	Caution for cyanate and thiocyanate accumulation
Nitroglycerin	IV infusion	5 μg/min	Immediate	Immediate	Max dose 300 μg/min	Ideal in setting of pulmonary edema
Enalapril	IV	1.25 mg	0.5-4 hr	Up to 4 hr for 1st dose	2.5 mg	Use only postpartum given potential for adverse fetal effects

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Case 2: Postpartum Cerebral Vasoconstriction or Angiopathy

The patient is a 33-year-old G3P2 with normal blood pressure and absent proteinuria at each of nine prenatal visits starting in the first trimester. During pregnancy she was noted to have a weight gain of 55 pounds and complained of increased swelling, but denied headaches, blurred vision, or epigastric pain. At 39 weeks' gestation her blood pressure was 112/70 mm Hg with negative proteinuria on dipstick. Because of her weight gain and swelling, she was admitted to a hospital for induction of labor.

During induction she received oxytocin and a successful epidural analgesia with subsequent delivery of a live term infant with appropriate weight and normal Apgar scores. Her blood pressures through labor and delivery were all normal. She was then discharged home the next day with a blood pressure of 110/68 mm Hg, and no symptoms.

Two days following discharge (postpartum day 3), the patient presented to the emergency department because of new onset severe headaches that were unrelated to position change. Blood pressure values measured in the emergency department ranged between 139/82 and 152/90 mm Hg, and there was no proteinuria. A CT scan of the brain was normal. The patient was given IV analgesics, 2 L of IV fluids, and was discharged home on oral narcotics because of the headaches and instructed to call her physician the next day.

The patient presented to the emergency department again on postpartum day 5 because of persistent headaches. She was found to have persistent elevations in blood pressure with three of the systolic values above 160 mm Hg. The patient was given IV labetalol, two doses, one dose of IV furosemide and was started on oral labetalol for blood pressure control. During the next several hours she had a massive diuresis (approximately 4500 mL of urine) and her blood pressures ranged between normal and mild hypertension values. Her headaches persisted, however, for which she was evaluated by both an anesthesiologist and a neurologist. A diagnosis of muscle spasm was made and the patient was discharged home on oral labetalol for the elevated blood pressures, analgesics for headaches, and a muscle relaxant. Instructions were given to call back if she developed severe headache or visual changes.

Approximately 36 hours after discharge (postpartum day 7), she woke up agitated with blurred vision and became confused. A family member called 911, and she was taken to a local hospital. On arrival to the admission area, the patient developed a grand mal seizure and lost consciousness. Magnetic resonance imaging (MRI) of the brain demonstrated the presence of parietal hemorrhage (Fig. 9-3). The patient was treated with antihypertensive medications and magnesium sulfate for presumed diagnosis of eclampsia. However, all tests including 24-hour urine protein values were normal. The next day she regained consciousness but was noted to have muscle weakness with paralysis, but her blood pressure was normal.

On postpartum day 9, the patient became agitated and then developed a grand mal seizure for which she was treated with phenytoin. Neurologic consultation was obtained with recommendation for four-vessel arteriogram. The results of the arteriogram were consistent with postpartum cerebral vasculopathy (Fig. 9-4).

Discussion

This patient presented in the late postpartum period with three episodes of persistent headaches and increased blood pressure. Despite the initial normal findings on the cranial CT scan on postpartum day 3, the patient should have received cranial MRI evaluation during her subsequent admission on postpartum day 5. The diagnosis of late postpartum eclampsia was incorrect based on available data.

As stated, the diagnosis of cerebral vasospasm syndrome is confirmed by the presence of abnormal contrast cerebral angiography. The classic findings reveal multifocal areas of segmental narrowing and dilation, particularly in large and medium-sized arteries and their branches. The intracranial portions of the arteries of the circle of Willis are usually selectively involved. Similar cerebrovascular abnormalities may be seen using magnetic resonance angiography (MRA). CT, MRI scan, and diffusion-weighted (DW) MRI show multifocal areas of infarction mostly in the posterior parietal and temporal occipital regions of the brain. The findings are usually related to hypoperfusion secondary to severe vasoconstriction.

Management of this syndrome depends on etiology as well as on clinical findings. In general, treatment is supportive care. Vasodilators with calcium channel blockers may be used to treat vasospasm. Primary cerebral vasculitis is treated with steroids and other immunosuppressive agents. In the majority of cases, there is spontaneous resolution of the vasospasm without any treatment. In most patients the cerebral abnormalities resolve within weeks but may last for months. Both transcranial Doppler flow velocity studies and MRA can be used in follow-up to establish reversal of the vasoconstriction. The prognosis and long-term outcome are usually favorable in the absence of major cerebral hemorrhage or primary vasculitis. There is usually full recovery in patients with late postpartum eclampsia as well as in those with idiopathic postpartum angiopathy; however, in some patients the effects can result in residual neurologic deficit as in this case. Alternatively, if the vasoconstriction is recurrent or continuous (not responsive to therapy), it may result in severe intracerebral hemorrhage and death (Fig. 9-5).

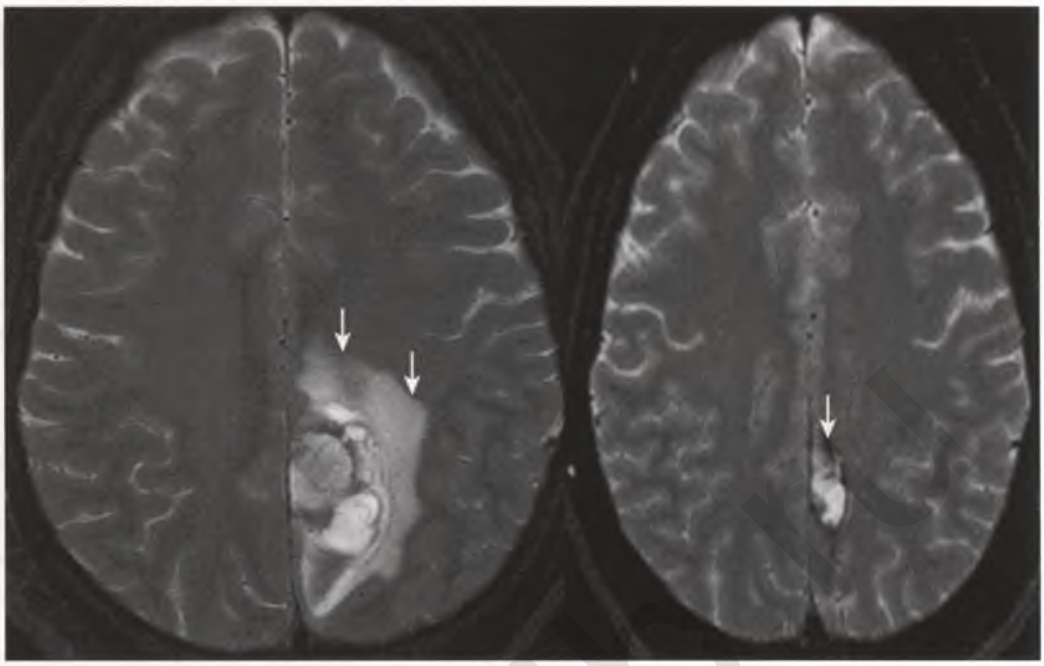

Figure 9-3 Cranial MRI revealing parietal hemorrhage *(arrows)*.

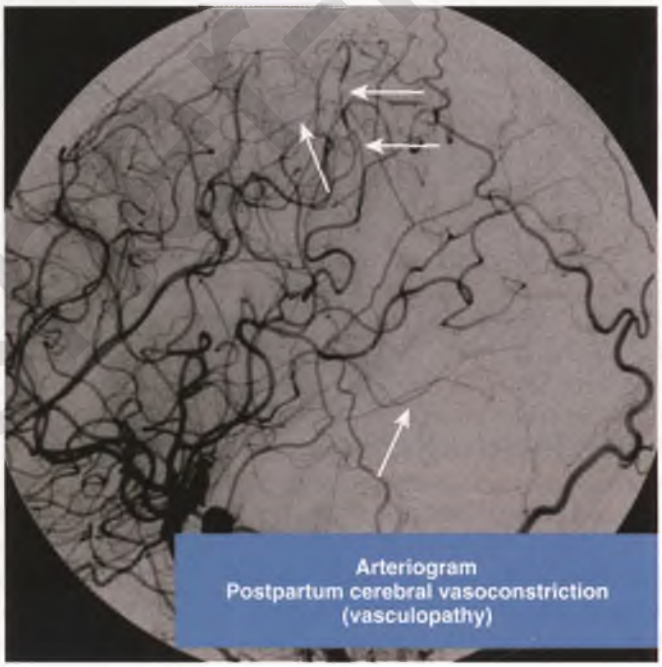

Figure 9-4 Cerebral arteriogram demonstrating vasospasm *(arrows)*.

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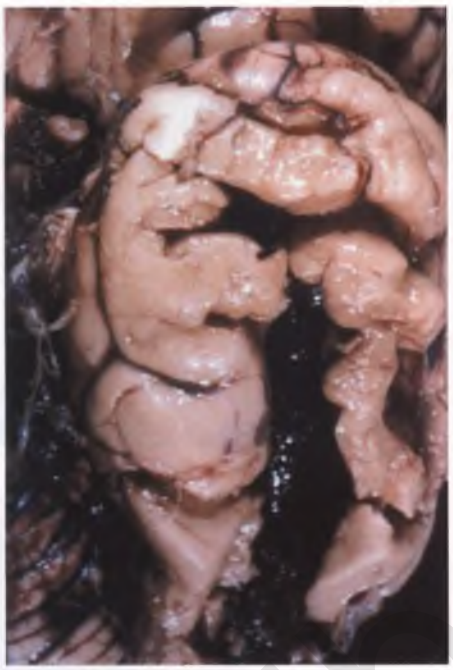

Figure 9-5 Postmortem occipital and parietal hemorrhages.

Case 3: Ruptured Cerebral Aneurysm

A 25-year-old white G2P1 presents at 36 weeks' gestation with a benign prenatal course thus far. She describes a symptom of a sudden onset of a severe headache that she states is "the worst in my life." She also complains of nausea and vision change but denies vomiting as well as numbness or weakness in either extremity. Her initial blood pressure was 150/90 mm Hg. Urine protein was negative. The patient was admitted to the labor and delivery unit for evaluation of possible gestational hypertension. Twenty-four urine studies were ordered and admission laboratory evaluation including:

Platelet count	205,000/mm^3 (normal >100,000/mm^3)
AST	12 units/L (normal <60 units/L)
ALT	14 units/L (normal <40 units/L)
Uric acid	4.8 mg/dL (normal <6 mg/dL)
Hemoglobin	12.1 g/dL (normal >10 g/dL)

During evaluation the patient was mildly confused which progressed to unconsciousness. She developed a sudden cardiopulmonary arrest. Cardiopulmonary resuscitation was begun immediately but attempts at resuscitation failed. Because this was a witnessed cardiopulmonary arrest, perimortem cesarean section was initiated 4 minutes after the onset of arrest with delivery of a viable infant. Despite continued aggressive efforts, the resuscitation failed and the patient expired. The presumed diagnosis at that time was eclampsia with possible intracranial hemorrhage.

An autopsy was performed that initially revealed the presence of a subarachnoid hemorrhage with obliterated sulci and flattened gyri (Fig. 9-6). After removal of the brain and further examination of the base of the brain, there was evidence of a ruptured cerebral aneurysm (Fig. 9-7). The final autopsy diagnosis was, therefore, intracranial hemorrhage secondary to a rupture of a berry aneurysm in the circle of Willis.

Discussion

As stated, the differential diagnosis of preeclampsia/eclampsia should include cerebral aneurysm rupture and arteriovenous malformation (AVM). Patients with intracranial hemorrhage can present with hypertension, proteinuria, and cerebral manifestations that are consistent with the findings of preeclampsia. The classic presentation for a ruptured cerebral aneurysm includes acute onset of a severe headache often described as "the worst headache in my life," suggestive of a subarachnoid hemorrhage. In such situations, it is important to evaluate the cerebral vessels.

Patients who receive a diagnosis of AVM or aneurysm before hemorrhage should be referred for surgical embolization or clipping as it is believed that patients with AVM may be at increased risk of bleeding during pregnancy. Patients with AVMs are also prone to bleed during delivery. Once an intracerebral hemorrhage has occurred, the extent of the bleeding will determine the course of treatment. If the brainstem is compromised, surgical decompression is necessary. Surgery may also be necessary if the bleed is subarachnoid in origin; however, surgery itself may damage overlying normal brain tissue, and surgical morbidity is high. If the bleeding and the patient are stable, surgery can be avoided. Blood pressure should be well controlled and seizures prevented. Steroids have not proven beneficial.

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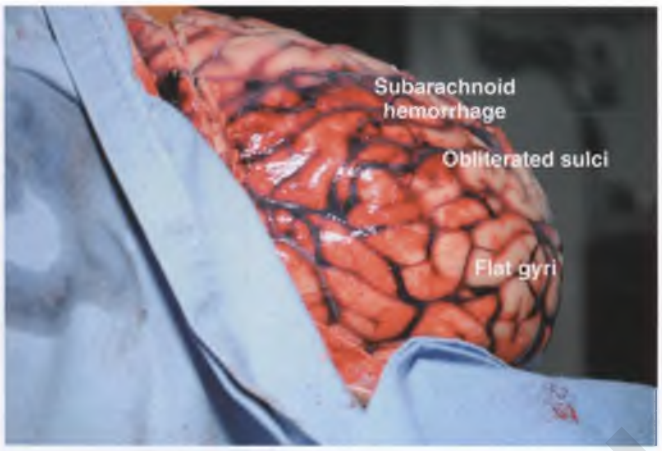

Figure 9-6 Postmortem abnormal intracranial findings showing cerebral edema and subarachnoid hemorrhage.

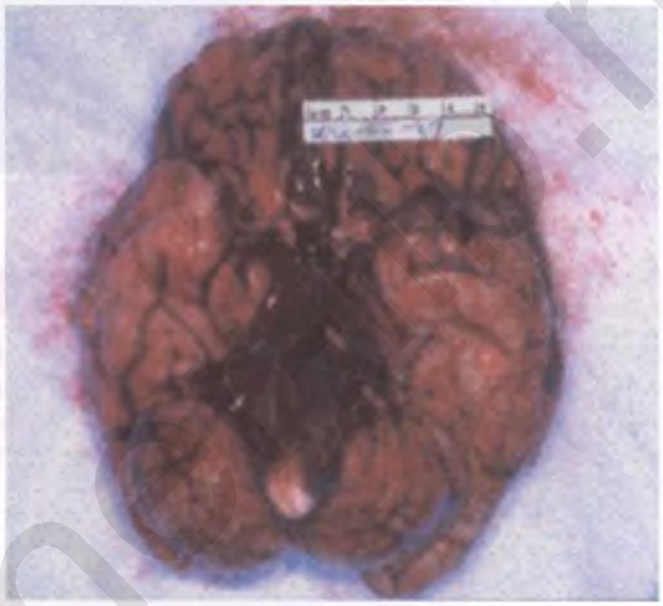

Figure 9-7 Gross picture of the base of the brain showing the site of ruptured aneurysm.

SUMMARY: Stroke can happen at any time. The clinical presentation is similar to that seen in non-pregnant patients; however, the symptoms can mimic those seen in preeclampsia and eclampsia. Obstetricians are therefore encouraged to become well versed in the symptoms of stroke and to have a low threshold for clinical suspicion of symptoms that may mimic common complaints of pregnancy. A rapid diagnosis and close consultation with an interdisciplinary team of colleagues may maximize outcomes in patients suffering one of the most feared and serious complications of pregnancy.

Suggested Readings

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Management of Eclampsia

10

Baha M. Sibai M.D.

Video Clips on DVD

10-1 PowerPoint Discussion of Evaluation and Management of a Patient with Eclampsia

10-2 Discussion with Dr. Sibai on Emergent Management of a Woman Presenting with an Eclamptic Seizure

Eclampsia is defined as the onset of seizures and/or unexplained coma during pregnancy (usually not before 20 weeks of gestation) or postpartum in patients with signs and symptoms of preeclampsia. In the Western world, the incidence ranges from 1 in 2000 to 1 in 3448 pregnancies. Incidence is higher in tertiary referral centers, in the developing countries, and in patients with no prenatal care. Although most cases (90%) present in the third trimester or 48 hours following delivery, there have been rare cases (1.5%) at or before 20 weeks and as late as 23 days postpartum. Risk factors for eclampsia are described in Table 10-1.

Case 1: Eclampsia with Eclamptic Seizure

An ambulance rushes Jessica, a 19-year-old G1P0 with an intrauterine pregnancy at 36 weeks' gestation, to labor and delivery. She's had no prenatal care. Her family says she has been complaining of headache and visual disturbance for the last few days. They've witnessed a 3-minute seizure during which she drooled saliva but had no urinary or fecal incontinence. Her medical/surgical and social history are completely negative.

On admission, her blood pressure is 170 mm Hg systolic over 110 mm Hg diastolic. She has 2+ protein on her urine dip. Bedside ultrasound confirms a singleton intrauterine pregnancy consistent with 36 weeks of gestation. Fetal heart rate tracing is reassuring, and cervical exam reveals a Bishop score of 7.

The 19-year-old patient underwent immediate suctioning of secretions and was placed in a lateral semirecumbent position and given oxygen by face mask. IV access was secured and laboratory tests were obtained for complete blood count (CBC), including platelet count, liver enzymes, and a metabolic profile. A loading dose of 6 g of magnesium sulfate was given over 20 minutes and that was followed by a maintenance dose of 2 g/hr. Because of her severe hypertension, a 10-mg bolus of IV hydralazine was administered. Blood pressures were monitored every 5 to 10 minutes, and after 30 minutes, the systolic was 150 mm Hg and diastolic was 105 mm Hg. Maternal urine output and reflexes were monitored every hour.

The results of the blood tests revealed a platelet count of 120,000/mm^3, a hematocrit of 37%, and normal liver enzymes. Once maternal and fetal conditions were considered stable, IV oxytocin was started to initiate labor.

The patient subsequently spontaneously delivered vaginally an infant weighing 2700 g with Apgar scores of 7 and 9 at 1 and 5 minutes, respectively. Postpartum magnesium sulfate was continued for 24 hours. In addition, maternal vital signs, intake and urine output and patellar reflexes, were monitored every hour. She was started on oral nifedipine, 10 mg every 6 hours, because of elevated blood pressures and asked to return within 1 week for postpartum follow-up.

Discussion

This is a typical presentation of a patient with eclampsia in the absence of prenatal care. It's terrifying to witness a life-threatening eclamptic convulsion develop. First the patient's face becomes distorted and her eyes bulge; then

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Case 1: Eclampsia with Eclamptic Seizure—cont'd

she becomes red faced. Lasting usually 60 to 75 seconds, convulsions occur in two phase. The first—which lasts for 15 to 20 seconds—begins with facial twitching; then the body becomes rigid with generalized muscular contractions. During the 60-second second phase, the muscles of the body alternately contract and relax in rapid succession starting in the jaw muscles, then moving to the eyelids and the other facial muscles before spreading throughout the whole body. Apnea develops not just during but also immediately after the seizure. Coma sometimes follows seizures and the woman usually remembers nothing of the recent events. The woman often foams at the mouth and usually bites her tongue, unless it's protected. A period of hyperventilation occurs after the tonic-clonic seizure to compensate for the respiratory acidosis that developed during the apneic period.

The majority of patients with eclampsia have hypertension (systolic blood pressure ≥140 and/or diastolic pressure ≥90 mm Hg), plus proteinuria (≥1+ on dipstick), and edema. In addition, as was the case in this patient, more than 50% will give a history of headaches, visual changes, and/or epigastric pain prior to seizures. Although hypertension is considered the hallmark for the diagnosis of eclampsia, it can be absent in 15% to 20% prior to seizures. In these cases, the patient will have proteinuria and associated central nervous system or epigastric pain with nausea and vomiting.

There is no single laboratory test that is diagnostic of eclampsia. The patient usually has hemoconcentration and elevated serum creatinine and uric acid, but normal platelet count and liver enzymes. Thrombocytopenia (platelet count <100 K/mm^3) is seen in approximately 20% of cases, and the full HELLP (*h*emolysis, *e*levated *l*iver enzymes, and *l*ow *p*latelet count) syndrome in 10% to 15% of cases. Most patients demonstrate reduced oxygen saturation during and immediately after the seizure. If these persist, consider aspiration or pulmonary edema.

Although eclampsia is the most common cause of convulsions in association with hypertension in pregnancy and/or proteinuria, don't overlook the other causes that—although rare—can produce convulsions in pregnancy or postpartum and can mimic eclampsia (Table 10-2).

The basic goals of therapy are to support maternal condition, prevent maternal injury and aspiration, prevent recurrent convulsions, and control blood pressure and other associated maternal complications. Once maternal and fetal conditions are optimized, the next step is to initiate process of delivery (Fig. 10-1). Specifics of detailed steps are described following.

Table 10-1 Risk Factors for Eclampsia

- First pregnancy
- Previous eclampsia
- Multifetal gestation
- Chronic hypertension/renal disease
- Collagen vascular disease
- Molar pregnancy/partial mole
- Gestational hypertension-preeclampsia *plus*
  - Severe headache
  - Persistent visual changes
  - Severe epigastric/right upper quadrant pain
  - Altered mental status

Table 10-2 Differential Diagnosis of Eclampsia

- Cerebrovascular accidents
  - Hemorrhage
  - Ruptured aneurysm or malformation
  - Arterial embolism or thrombosis
  - Cerebral venous thrombosis
  - Hypoxic ischemic encephalopathy
  - Angiomas
- Hypertensive encephalopathy
- Seizure disorder
- Amniotic fluid embolism
- Metabolic diseases
  - Hypoglycemia, hyponatremia, thyroid storm
- Reversible posterior leukoencephalopathy syndrome
- Thrombophilia
- Thrombotic thrombocytopenic purpura
- Postdural puncture syndrome
- Cerebral vasculitis

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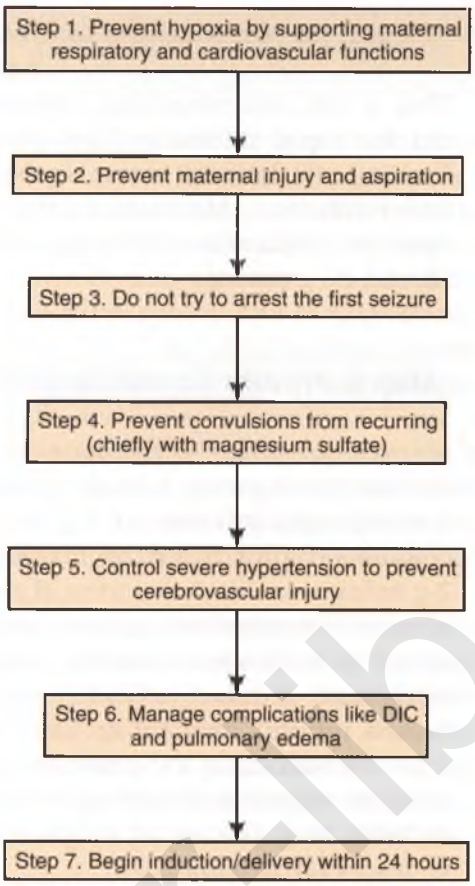

Figure 10-1 Steps in managing an eclamptic seizure.

Step 1. Support Maternal Respiratory and Cardiovascular Functions to Prevent Hypoxia

Establish airway patency and maternal oxygenation during or immediately after the acute episode. Even if the initial seizure is short, it's important to maintain oxygen levels by administering oxygen via face mask with or without reservoir at 8 to 10 L/min. The goal is to prevent the hypoxia and respiratory acidosis that often occur during the convulsions. I advise pulse oximetry to monitor oxygenation in these patients. Arterial blood gas analysis is required if the pulse oximetry is abnormal (oxygen saturation <92%), or aspiration or pulmonary edema is suspected. Sodium bicarbonate is not given unless the pH is less than 7.

Step 2. Prevent Maternal Injury and Aspiration

Secure the bed's side rails by elevating them, and make sure they're padded. Also place a padded tongue blade between the patient's teeth to prevent her from biting her tongue. Avoid forcing the tongue blade too deeply into the back of the throat. Position the woman in a lateral decubitus position to minimize aspiration of oral secretions and vomitus, should she vomit. Be aware that aspiration can be caused by forcing the padded tongue blade to the back of the throat, which stimulates the gag reflex with resultant vomiting.

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Step 3. Do Not Attempt to Arrest the First Seizure

This is not recommended, especially when no IV access or skilled personnel for rapid intubation are available. In addition, most seizures are self-limiting. If there is status epilepticus, the patient needs muscle paralysis and intubation. Medications that abolish the laryngeal reflex such as diazepam or midazolam (Versed) also increase the risk of aspiration and thus should be avoided.

Step 4. Prevent Convulsions from Recurring

Magnesium sulfate is the drug of choice to treat and prevent further seizures. I recommend giving a loading dose of 6 g over 15 to 20 minutes followed by a continuous infusion of 2 g/hr. Up to 10% of patients can have a second convulsion after receiving magnesium sulfate. If that happens, give another 2-g bolus over 3 to 5 minutes. If the patient is still having seizure activity after adequate magnesium sulfate dosing, give 250 mg of sodium amobarbital IV over 3 to 5 minutes. Consider intubation in those women who do not respond to therapy. Monitor patient for signs of magnesium toxicity by serial evaluation of reflexes, respiratory rate, and urine output. Serum magnesium levels are only needed in the presence of renal dysfunction. These signs and symptoms of magnesium toxicity in relation to the magnesium level are described in Table 10-3. In case of magnesium toxicity, management for these patients is described in Table 10-4.

Table 10-3 Magnesium Toxicity

Manifestations	Level (mg/dl)
Loss of patellar reflex	8-12
Double vision	8-12
Feeling of warmth, flushing	9-12
Somnolence	10-12
Slurred speech	10-12
Muscular paralysis	15-17
Respiratory arrest	15-17
Cardiac arrest	30-35

Table 10-4 Management of Magnesium Toxicity

- D/C magnesium sulfate
- Obtain Mg level
- Restart or reduce dose according to Mg level

Repiratory compromise

- Give calcium gluconate 1 gram IV
- Intubate
- Assisted ventilation

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Step 5. Prevent Cerebrovascular Injury by Controlling Severe Hypertension

Cerebrovascular injury, which can be a major cause of maternal morbidity and even death, can develop in the form of hypertensive encephalopathy with massive increases in intracranial pressure with resultant cerebral edema or intracerebral hemorrhage. Rarely, it's associated with massive increases in intracranial pressure. For details regarding cerebral injury, see Chapter 9. Imaging is not necessary for diagnosis and management unless there are other associated findings (Table 10-5). Imaging findings (computed tomography [CT] scan or magnetic resonance imaging [MRI]) in eclampsia are similar to those in patients with hypertensive encephalopathy. CT and MRI results reveal the presence of edema and infarction within the subcortical white matter and adjacent gray matter, mostly in the parieto-occipital lobes. These findings are labeled posterior reversible encephalopathy syndrome (Fig. 10-2). In more severe cases, the patient might have massive cerebral edema with the potential for herniation (Fig. 10-3).

To avoid such complications it's important to reduce and maintain blood pressure in a safe range without compromising cerebral perfusion and uteroplacental flow (which is already reduced in these patients). In addition, it prevents congestive heart failure by reducing the afterload. The goal is to keep systolic blood pressure between 140 and 160 mm Hg and diastolic blood pressure between 90 and 110 mm Hg. This can be achieved with IV bolus doses of 5 to 10 mg of hydralazine or 20 to 40 mg of labetalol every 15 minutes, as

Table 10-5 Indications for Cerebral Imaging in Eclampsia

- Presence of focal neurologic deficits
- Prolonged coma
- Repeat seizures despite adequate magnesium levels
- Presence of blindness
- Onset <20 weeks' gestation
- Onset >48 hours postpartum

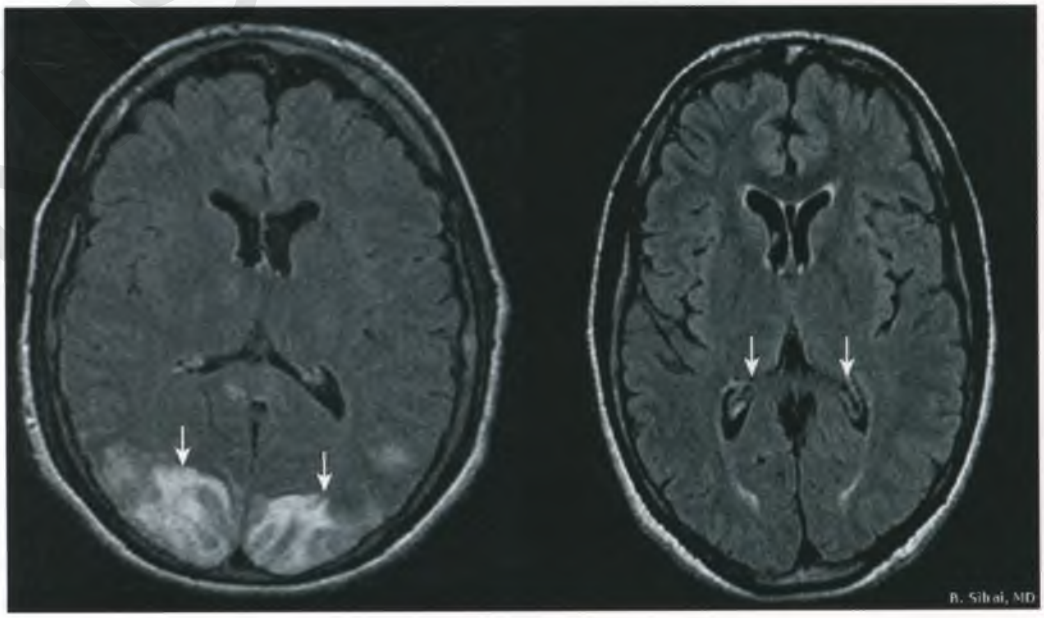

Figure 10-2 MRI of the brain revealing posterior reversible encephalopathy syndrome *(left)*, and repeat MRI 10 days later revealing resolution of vasogenic edema *(right)*. *Arrows*, Vasogenic anemia.

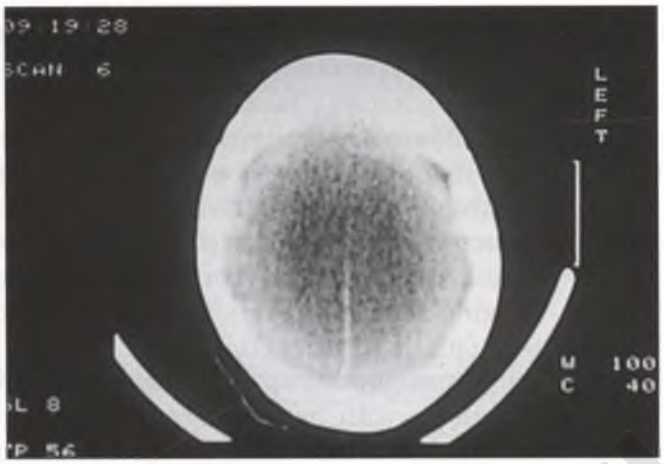

Figure 10-3 CT scan of the brain demonstrating massive cerebral edema with herniation.

Table 10-6 Treating Patients with Increased Intracranial Pressure

- Continuously monitor intracranial pressure
- Improve cerebral venous drainage
  - Elevate head of bed 30 to 45 degrees
- Intubation and ventilation
  - Arterial Pco_2 ≤25 mm Hg
  - O_2 saturation ≥95%
- Osmotic diuresis
  - Mannitol, furosemide
- Withdrawal of cerebrospinal fluid (CSF)

needed, or 10 to 20 mg of nifedipine orally every 30 minutes for a maximum dose of 50 mg in 1 hour. Sodium nitroprusside or nitroglycerin is rarely needed in patients with eclampsia. Diuretics are not used unless a woman has pulmonary edema. Rarely, patients can become confused, lethargic, or have prolonged coma due to an increase in intracerebral pressure. In this event, we recommend intracerebral pressure monitoring with treatment described in Table 10-6.

Step 6. Manage Complications Such As Disseminated Intravascular Coagulopathy (DIC) and Pulmonary Edema

DIC (7% to 11% Incidence)

Although DIC usually occurs in the setting of placental abruption and/or fetal demise, excessive blood loss can also cause the condition to develop. However, do not delay treatment if you strongly suspect DIC. Maintain blood volume and blood pressure with aggressive replacement with crystalloids and/or blood products as indicated (packed red blood cells [PRBCs], platelets, fresh frozen plasma, cryoprecipitate). Treatment of DIC is mainly supportive in nature, provided delivery takes place.

Pulmonary Edema (3% to 5% Incidence)

This diagnosis is usually made clinically, beginning with symptoms or dyspnea and chest discomfort. Tachypnea, tachycardia, and basal rales are detected on exam. In addition, pulse oximetry reveals values less than 94%. Two things confirm the diagnosis: arterial blood gas analysis and chest x-ray findings. The

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latter will reveal interstitial edema and Kerley B lines or frank alveolar edema. Initial management includes administering oxygen and stimulating greater urine output by giving 20 to 40 mg of IV furosemide over 1 to 2 minutes. If no adequate diuresis begins within 1 hour, slowly administer an 80-mg dose. For more details regarding diagnosis and management of pulmonary edema, see Chapter 7.

Step 7. Begin Induction/Delivery Within 24 Hours

Intrapartum Management: Fetal Heart Rate (FHR) Monitoring and Tocometry

During the convulsion, there's usually a prolonged deceleration or bradycardia, or both. After the convulsion, on the other hand—due to maternal hypoxia and hypercarbia—FHR monitoring can show compensatory tachycardia, decreased beat-to-beat variability, and transient late decelerations. In addition, uterine contraction monitoring shows increases in both uterine tone and frequency (Fig. 10-4). These changes in FHR and uterine activity usually last from 3 to 15 minutes.

Because the FHR pattern usually returns to normal after maternal resuscitation (Figs. 10-5 and 10-6) consider other conditions if an abnormal pattern persists. It may take longer for the heart rate pattern to return to baseline in an eclamptic woman whose fetus is preterm with growth restriction. Placental abruption can occur after the convulsion and should be considered if uterine hyperactivity remains or if there are repetitive late decelerations or if fetal bradycardia persists (Fig. 10-7).

Anesthesia Options

Either systemic opioids or epidural anesthesia can relieve maternal pain during labor and delivery. Epidural, spinal, or combined anesthesia is not contraindicated unless coagulopathy or severe thrombocytopenia (platelet count less than 90,000/mm^3) is present. General anesthesia in women with eclampsia increases the risk of aspiration and failed intubation due to airway edema. In addition, general anesthesia appears to significantly increase systemic and intracerebral pressures during intubation and extubation. Giving labetalol injections prior to intubation can ameliorate these changes.

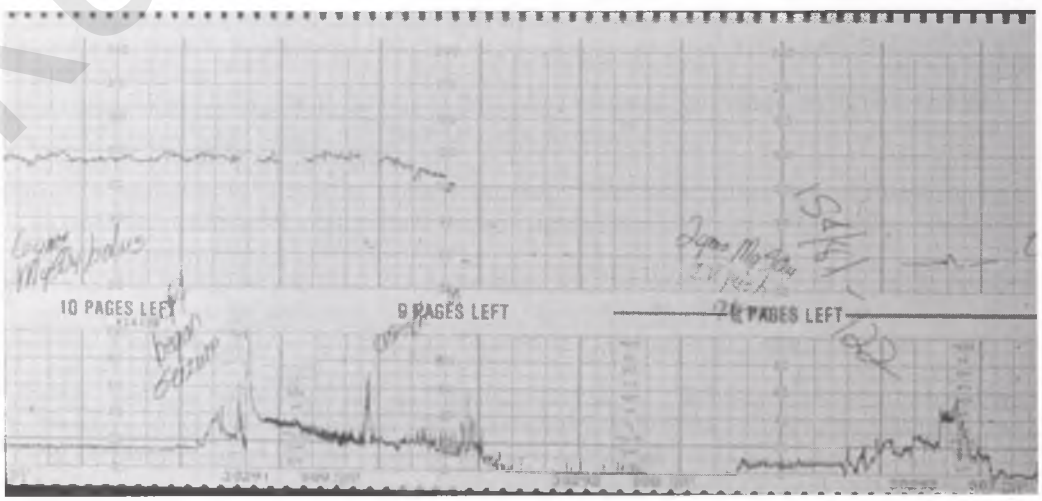

Figure 10-4 FHR tracing revealing increased uterine activity and fetal bradycardia.

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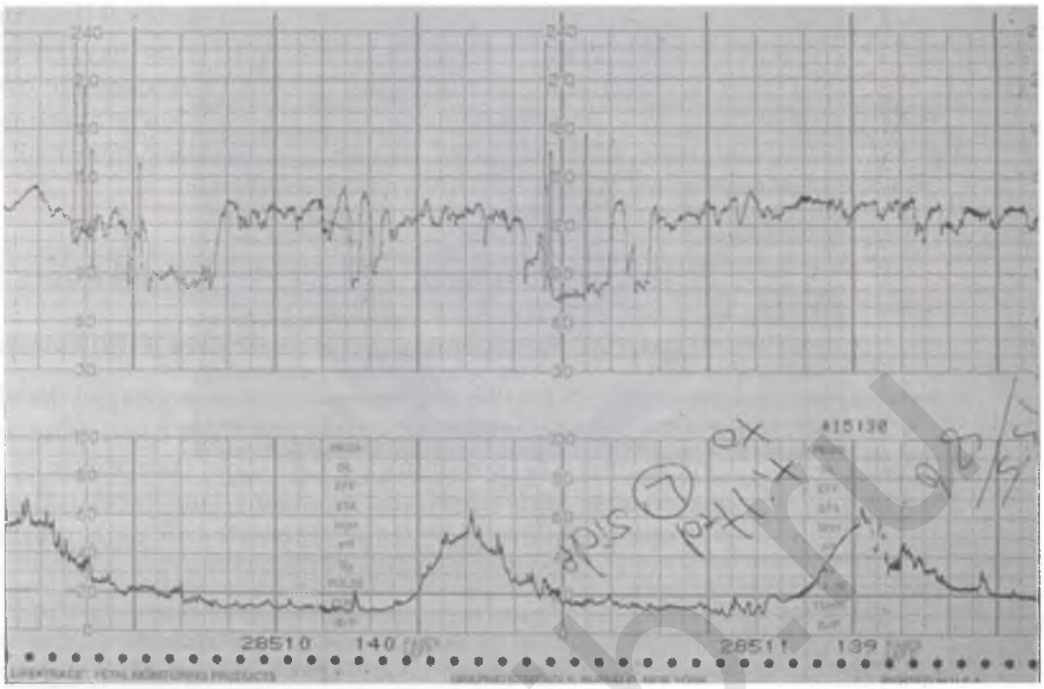

Figure 10-5 After maternal resuscitation, FHR tracing reveals variable decelerations with moderate variability.

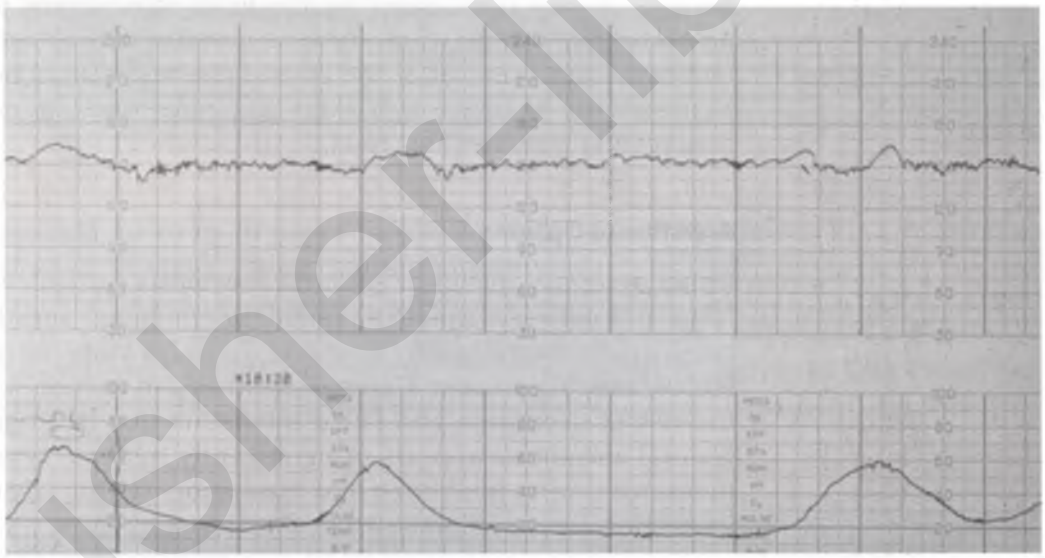

Figure 10-6 FHR now reassuring with good variability and accelerations.

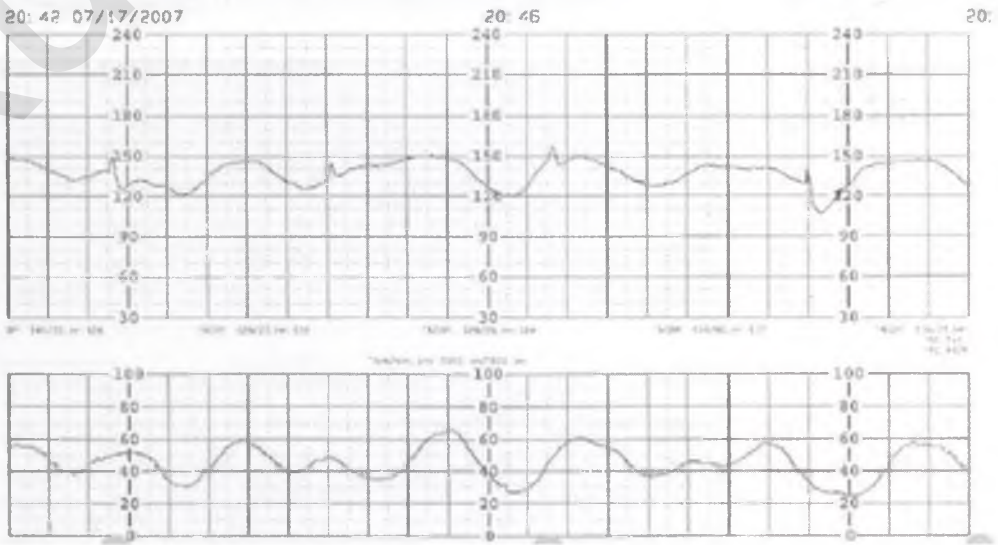

Figure 10-7 Fetal monitoring revealing uterine tachysystole with repetitive late decelerations suggesting abruptio placentae.

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Eclampsia itself is not an indication for cesarean section. Do not rush a patient to the operating room, especially if her condition is unstable. It is to the fetus's advantage to first be resuscitated in utero. However, if bradycardia or persistent late decelerations occur despite resuscitative measures, consider a diagnosis of abruptio placentae or nonreassuring fetal status. Base your decision on whether to proceed with cesarean section after maternal stabilization on the gestational age of the fetus, fetal condition, the presence of labor, and the cervical Bishop score. I recommend cesarean section for those with eclampsia before 30 weeks who are not in labor and whose cervical Bishop score is below 5. You can induce labor with either oxytocin or prostaglandins in all patients after 30 weeks of gestation regardless of Bishop score. A similar approach is used in patients with gestations before 30 weeks if the Bishop score is at least 5.

Postpartum Management

Due to the large amount of IV fluid these patients receive, I recommend strict evaluation of input and output. Pulse oximetry and frequent pulmonary auscultation are important, especially in patients with preexisting chronic hypertension, abnormal renal function, and those with abruptio placentae.

Continue magnesium sulfate for at least 24 hours after delivery or after the last convulsion, or both. If renal insufficiency occurs, reduce both magnesium sulfate and the rate of fluid administration. After delivery, oral antihypertensive medication can be used to maintain systolic blood pressure below 155 mm Hg and diastolic blood pressure below 105 mm Hg. You may use 200 mg of labetalol every 8 hours (maximum dose 2400 mg/day) or 10 mg of nifedipine every 6 hours. Oral nifedipine is a beneficial diuretic postpartum, and there are no risks in giving the drug concomitant with magnesium sulfate. An oral diuretic may also be used if needed to promote diuresis.

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Subcapsular Liver Hematoma

11

John R. Barton M.D.
Baha M. Sibai M.D.

Video Clips on DVD

11-1 PowerPoint Discussion of the Diagnoses and Management of Subcapsular Liver Hematoma

It is estimated that 10% to 20% of patients with severe preeclampsia/eclampsia will have hepatic involvement. This liver pathology has been described in association with a constellation of signs, symptoms (nausea, vomiting, upper gastric pain and mucosal bleeding), and laboratory abnormalities called HELLP (*h*emolysis, *e*levated *l*iver enzymes and *l*ow *p*latelet count) syndrome. The laboratory criteria for HELLP syndrome are summarized in Table 11-1. Because of low platelets and abnormal platelet function, patients could have petechiae or ecchymosis (Fig. 11-1) or they can have bleeding from the gums (Fig. 11-2). In addition, because of hemolysis, the urine appears tea colored (Fig. 11-3).

Subcapsular liver hematoma is a rare but life-threatening complication of HELLP syndrome. In most instances rupture involves the right lobe of the liver and is preceded by the development of a parenchymal hematoma. Most patients with a subcapsular hematoma of the liver are seen in the late second or third trimester of pregnancy, although the diagnosis might not be made until the immediate postpartum period. Frequent signs and symptoms of hepatic rupture and hepatic hemorrhage include epigastric pain, hypotension, shock, nausea and vomiting, shoulder pain, evidence of massive ascites, respiratory difficulty, or pleural effusions and often with a dead fetus (Table 11-2). The degree of hypertension and proteinuria may not mirror the degree of liver involvement. Further, there is a wide variation in presentation and severity of the symptoms of hepatic rupture particularly in their time course in relation to hepatic distention and hepatic rupture. The diagnosis of liver hematoma can be confirmed by one or more of radiologic modalities described in Table 11-3. An ultrasound or computed axial tomography of the liver should be performed to rule out the presence of subcapsular hematoma of the liver and assess for the presence of intraperitoneal bleeding (Fig. 11-4). Chest x-ray findings will reveal the presence of plural effusions and elevation of the diaphragm on the right side (Fig. 11-5). The diagnosis can also be made either by computed tomography (CT) scan (Fig. 11-6) or magnetic resonance imaging (MRI) (Fig. 11-7). Clearly, once rupture of the Glisson's capsule occurs, hypotension and hypovolemic shock are the rule. Rupture of an intact hematoma can occur spontaneously or may

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Table 11-1 Laboratory Findings in HELLP Syndrome

- Hemolysis (at least two of these)
  - Peripheral smear (schistocytes, burr cells)
  - Serum bilirubin (≥1.2 mg/dL)
  - Low serum haptoglobin
  - Severe anemia, unrelated to blood loss
- Elevated liver enzymes
  - AST or ALT ≥ twice upper level or normal
  - LDH ≥ twice upper level or normal*
- Low platelets (<100,000/mm^3)

*Also elevated in severe hemolysis.

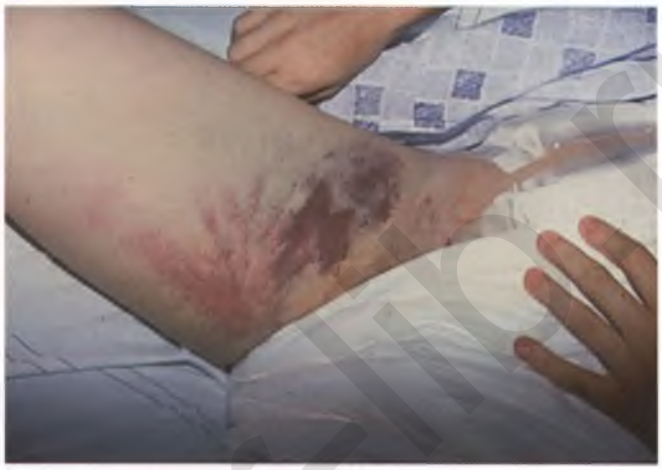

Figure 11-1 Severe ecchymoses in a patient with HELLP syndrome.

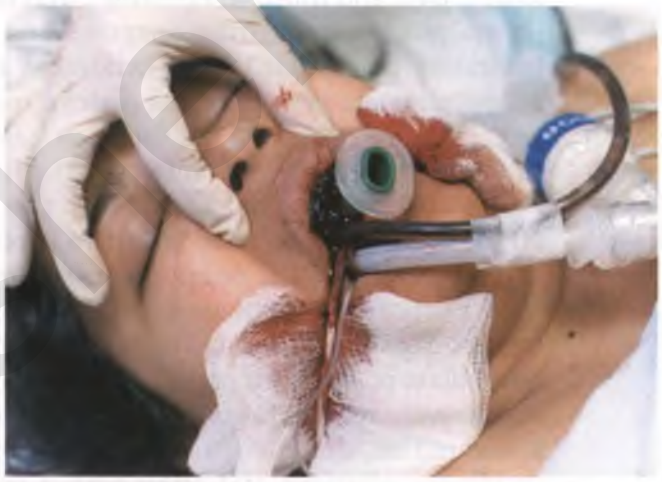

Figure 11-2 Bleeding gums.

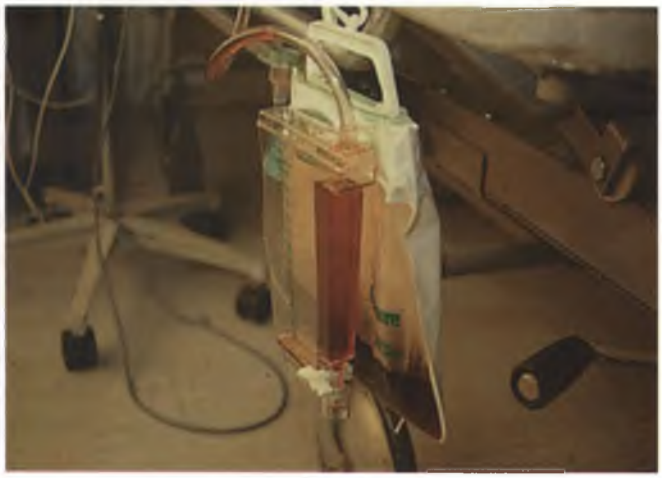

Figure 11-3 Tea-colored urine revealing hemolysis.

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Table 11-2 Sign and Symptoms of Subcapsular Liver Hematoma

- Severe epigastric pain
- Severe nausea or vomiting
- Shoulder/neck pain
- Respiratory difficulty or pain on inspiration
- Acute onset of hypotension and tachycardia
- Relapsing hypotension
- Disseminated intravascular coagulopathy (DIC)
- Abrupt elevations in liver enzymes
- Massive ascites or pleural effusions
- Change in the fetal heart rate tracing
  - Tachycardia with decelerations
- Fetal death

Table 11-3 Radiologic Imaging in Subcapsular Liver Hematoma

- Chest x-ray
  - Pleural effusion
  - Elevated right diaphragm
- Ultrasound
  - Hematoma
  - Intraperitoneal fluid
- CT scan
- MRI

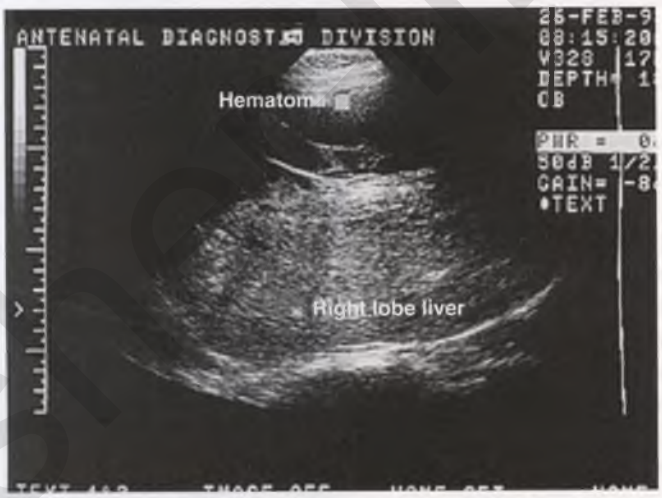

Figure 11-4 Ultrasound of the liver revealing hematoma.

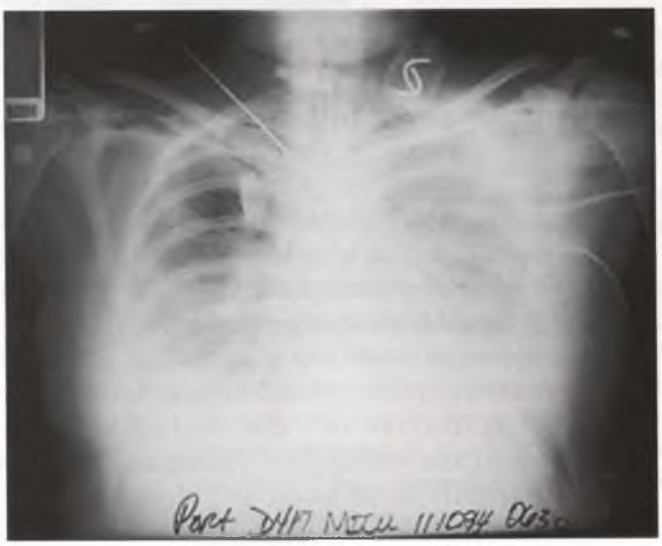

Figure 11-5 Chest x-ray revealing bilateral pleural effusions.

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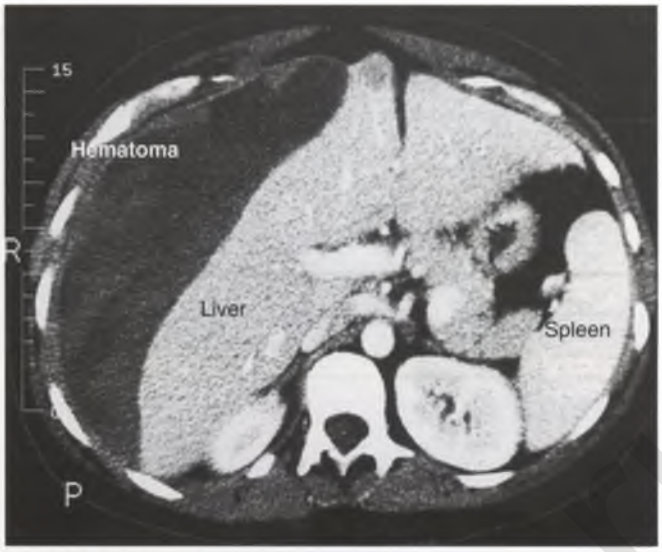

Figure 11-6 CT scan revealing intact liver hematoma in the postpartum period.

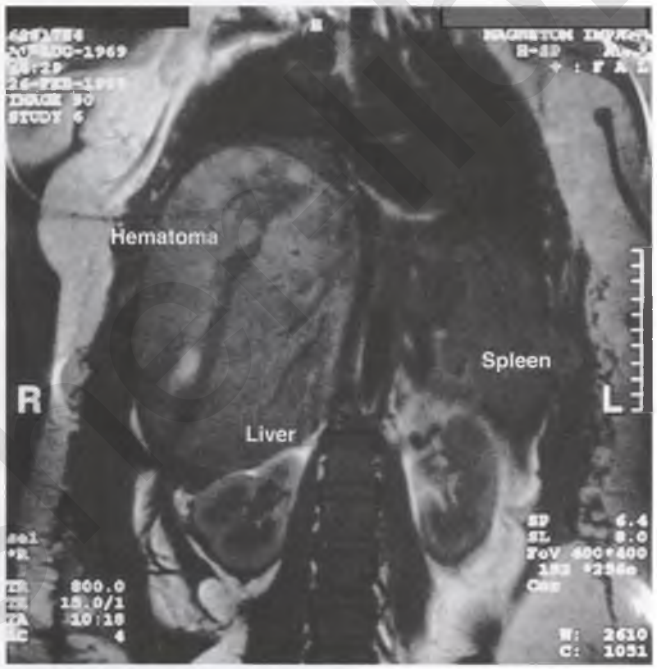

Figure 11-7 MRI revealing liver hematoma.

be associated with exogenous forces of trauma including abdominal palpation, convulsions, or vomiting.

The differential diagnosis for patients with physical examination findings consistent with peritoneal irritation, hepatomegaly, hemolysis and low platelet count should also include acute fatty liver of pregnancy, abruptio placentae with disseminated intravascular coagulopathy (DIC), ruptured uterus, thrombotic thrombocytopenic purpura, and a ruptured splenic artery aneurysm.

Pregnancies complicated by subcapsular hematoma are associated with extremely high rates of maternal and perinatal mortality and morbidity (Table 11-4). The rate of these morbidities depends on whether the hematoma is ruptured, the availability of blood and blood products, the administration of massive transfusions of blood and blood products, and the surgical management used. The risks for the fetus are usually related to prematurity and hypoxia.

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Table 11-4 Maternal and Perinatal Complications in Hepatic Hematomas

Maternal
• Need for large volumes of blood transfusions • Prolonged hospitalization in intensive care unit • Pulmonary edema • ARDS • Renal failure • Hepatic failure   • Potential for liver transplant • Sepsis • DIC • Intracerebral hemorrhage • Death
Perinatal
• Preterm delivery • Perinatal asphyxia • Cerebral palsy • Mental retardation • Fetal/neonatal death

Case 1: Ruptured Liver Hematoma

A 32-year-old G3P2 presents to her obstetrician at 32 weeks' gestation with complaints of intermittent headaches and visual disturbances. Her current pregnancy represents new paternity. Her blood pressure in the office was 140/88 mm Hg with a baseline blood pressure at 8 weeks' gestation of 124/70 mm Hg. Urine protein was 2+ on dipstick. The decision was therefore made to admit the patient for evaluation of preeclampsia.

Laboratory studies on admission:

AST	30 units/L (normal <40 units/L)
ALT	35 units/L (normal <60 units/L)
LDH	180 units/L (normal <200 units/L)
Platelet count	200,000/mm^3 (normal >100,000/mm^3)
Hemoglobin	12.3 g/dL (normal >10 g/dL)

Fetal ultrasound noted a breech presentation, normal fetal growth, and normal amniotic fluid volume as well as a reassuring biophysical profile. The 24-hour urine protein measurement was 2010 mg/day. Given preeclampsia remote from term, continued hospitalization was followed and the patient received steroids for fetal lung maturity enhancement. Forty-eight hours after admission the patient complained of sudden onset of severe epigastric pain with vomiting and had right upper quadrant tenderness.

Repeat laboratory studies:

AST	123 units/L
ALT	100 units/L
LDH	250 units/L
Platelet count	70,000/mm^3
Hemoglobin	9.3 g/dL

A diagnosis of HELLP syndrome was made and a cesarean section was performed for a breech presentation with delivery of a viable infant with normal Apgar scores. Delivery was performed through a Pfannenstiel incision. Because there was no oozing and no hemoperitoneum, drains were not placed.

Twenty-four hours postoperatively she complained of sudden onset of right upper quadrant abdominal pain that radiated to her neck and shoulder as well as retrosternal pain sensation. Ultrasound of the upper abdomen was concerning for intraabdominal hemorrhage. Laboratory tests revealed a hemoglobin of 6 g/dL, massive elevations in liver function values, and the presence of coagulopathy (DIC). A ruptured subcapsular liver hematoma was suspected. The patient received immediate resuscitation with large amounts of IV fluids and blood and blood products (packed red blood cells, fresh frozen plasma, cryoprecipitate, and platelets). Once the patient was stabilized, a computed tomography (CT) scan of the upper abdomen confirmed a rupture of a subcapsular hematoma of the liver (Fig. 11-8). A surgical team was immediately called and the decision was made to make a separate midline upper abdominal incision to evaluate the upper abdomen and liver. Rupture of the Glisson's capsule over the right lobe of the liver was noted (Fig. 11-9). Blood and clots were evacuated. There was no evidence of liver laceration. The bleeding sites on the surface of the liver were coagulated with an argon laser (Fig. 11-10). Laparotomy sponges were packed against the liver surface (Fig. 11-11) and closed drains were placed in each paracolic gutter. During surgery, the patient required transfusion of 20 units of packed red blood cells, 10 units of fresh frozen plasma, 20 units of cryoprecipitate, and 6 units of platelets. The patient was then transferred to the

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Case 1: Ruptured Liver Hematoma—cont'd

intensive care unit (ICU), where transfusions were continued as needed based on laboratory evaluations and maternal hemodynamic findings. The next day, a repeat laparotomy was performed in which laparotomy sponges were removed and the surface of the liver was inspected and not found to be bleeding. Her postoperative course was complicated by acute renal failure requiring dialysis and pulmonary edema treated with diuretic therapy and ventilatory support (Fig. 11-12). Coagulopathy was corrected by transfusion of blood and blood products. Her laboratory values returned to normal over the next 10 days and she was discharged to home 19 days postdelivery.

Discussion

HELLP syndrome may be complicated by hepatic rupture with the development of a hematoma beneath Glisson's capsule. The hematoma may remain contained, or rupture, with resulting hemorrhage into the peritoneal cavity. Women who develop a hepatic hematoma typically have abdominal pain and many have severe thrombocytopenia, shoulder or neck pain, nausea, and vomiting. If hepatic rupture occurs, swelling of the abdomen from hemoperitoneum and shock rapidly ensue. The aminotransferases are usually modestly elevated, but values of 4000 to 5000 IU/L can occasionally be seen.

The presence of ruptured subcapsular liver hematoma resulting in shock is a surgical emergency requiring acute multidisciplinary treatment. Resuscitation should consist of massive transfusions of blood, correction of coagulopathy with fresh frozen plasma and platelets, and immediate laparotomy. A team experienced in liver trauma surgery should be consulted. If hepatic rupture is suspected, an incision in the upper abdomen is necessary for adequate surgical exposure. A lower abdominal midline incision can be extended superiorly. If a Pfannenstiel incision was used for operative delivery, a separate upper abdominal incision should be made to maximize visualization of the upper abdomen and liver. Options at laparotomy include packing and drainage, use of an argon beam coagulator, surgical ligation of the hemorrhaging hepatic segments, embolization of the hepatic artery to the involved liver segment, and loosely suturing omentum or surgical mesh to the liver to improve integrity. Intraabdominal closed drains should be placed at laparotomy to monitor postoperative intraabdominal bleeding (Fig. 11-13). Even with appropriate treatment, maternal and fetal mortality is more than 50%. Mortality is most commonly associated with exsanguination and coagulopathy. Those who survive are also at increased risk for developing serious morbidities such as acute respiratory distress syndrome (ARDS), pulmonary edema, and acute renal and hepatic failure in the postoperative period.

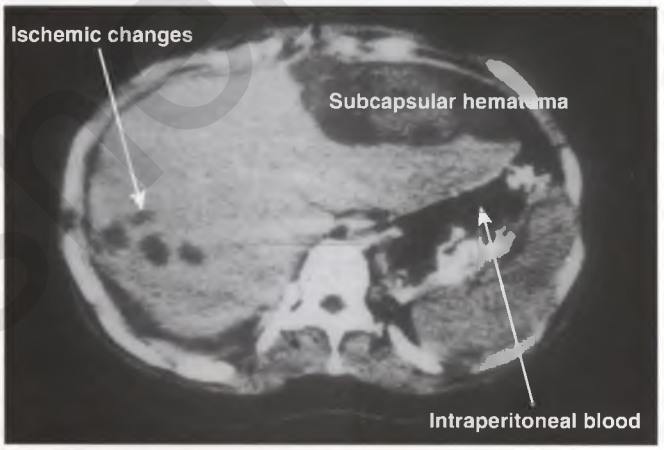

Figure 11-8 CT scan revealing ruptured subcapsular hematoma.

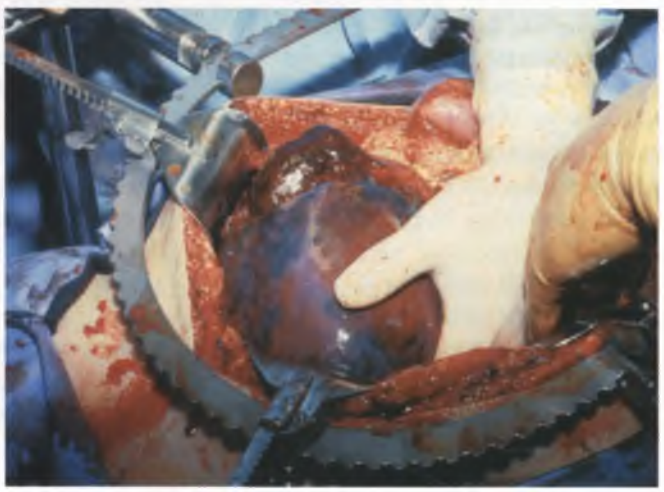

Figure 11-9 Ruptured liver hematoma at time of laparotomy.

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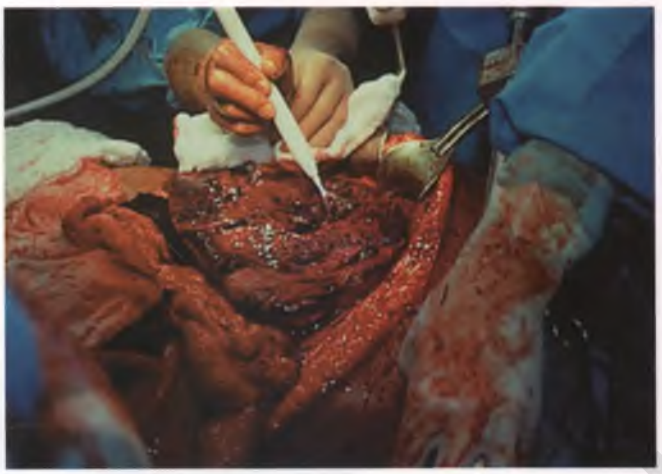

Figure 11-10 Use of argon beam to control bleeding from liver surface.

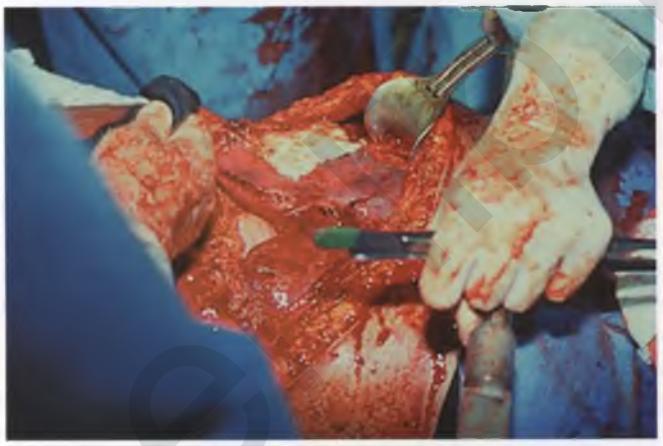

Figure 11-11 Liver packing.

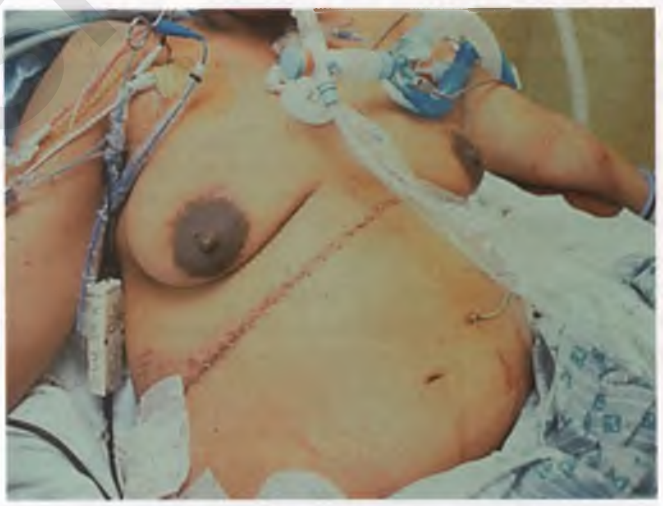

Figure 11-12 Patient in intensive care unit after surgery with all catheters, intubated.

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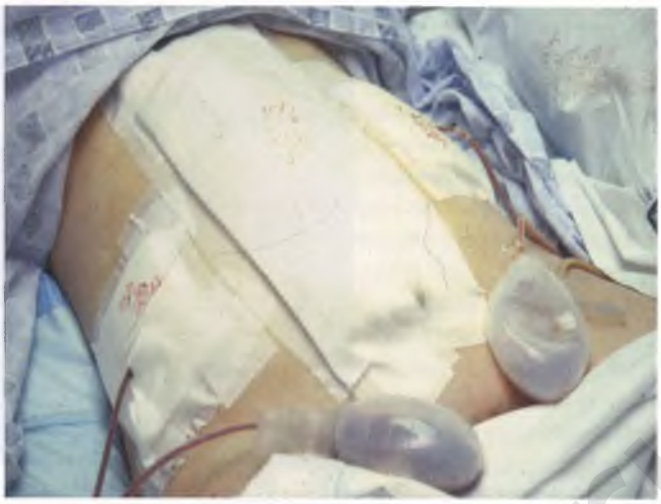

Figure 11-13 Jackson-Pratt drains in patient with liver hematoma.

Case 2: Unruptured Liver Hematoma

A 22-year-old G3P2 with uncomplicated prenatal course, presented in active labor at 39 weeks' gestation. During labor she had mild hypertension, urine protein was not tested, and no blood tests were ordered because she progressed rapidly with spontaneous vaginal delivery of a live infant with good Apgar scores. Her hematocrit and platelet count immediately postpartum were normal. A few hours postpartum she had bilateral tubal ligation. Approximately 8 hours after delivery she was having severe epigastric pain with vomiting that was attributed to surgery. This was followed up by complaints of pain on inspiration, tachypnea, and tachycardia with acute severe hypotension. Diagnosis of intraabdominal bleeding was considered to probably be related from the site of tubal ligation.

Blood was sent for complete blood count (CBC), coagulation studies, crossmatch for packed red blood cells, fresh frozen plasma, and platelets. Transfusions were initiated and the patient underwent laparotomy for evaluation of hemorrhage. There was about 2 to 5 L of blood in the abdomen, but the sutures around the tubes were intact. Surgical consult was obtained, the incision was enlarged, and the patient was found to have liver laceration with intact subcapsular hematoma (Fig. 11-14). The liver laceration was sutured, and the abdomen was packed and the patient referred to a tertiary care center.

Evaluation of hematoma and hemodynamic status of the patient at a tertiary center revealed that both were stable. As a result, the decision was made to also close the abdomen after insertion of two drains and to transfuse the patient with close monitoring (see Fig. 11-13). The hematoma was serially followed with CT scan of the liver (Figs. 11-15 through 11-17). The patient was discharged home 11 days after surgery with a follow-up CT scan obtained at 4 months after surgery showing partial resorption of the hematoma (see Fig. 11-17).

Discussion

Although surgical repair has been recommended by some authors for hepatic hemorrhage without liver rupture, this complication can be managed conservatively in patients who remain hemodynamically stable. Management should include close monitoring of hemodynamics and coagulation status with volume replacement and blood transfusion, as needed. It is important with conservative management to avoid exogenous sources of trauma to the liver such as abdominal palpation, convulsions, or emesis, and to use care in transportation of the patient. Indeed, any sudden increase in intraabdominal pressure could lead to rupture of the subcapsular hematoma. Serial assessment of the subcapsular hematoma with ultrasound or CT is necessary. If the size of the hematoma remains stable and the laboratory abnormalities are resolving, the patient may be discharged home with outpatient follow-up. It may take months to years for the hematoma to resolve completely.

Management of Subcapsular Hematoma

A stepwise management plan of hepatic hematoma in women with HELLP syndrome is described in Figure 11-18. The first step in management in these patients should be to admit to an intensive care unit facility with close monitoring of hemodynamic parameters and fluid status to avoid the potential for

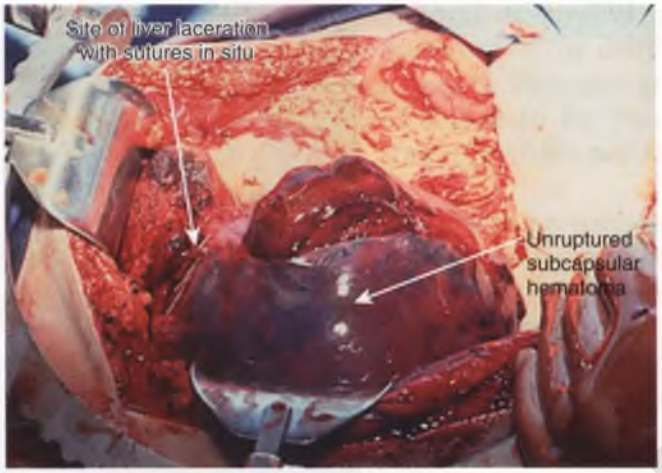

Figure 11-14 Intact liver hematoma with evidence of liver laceration.

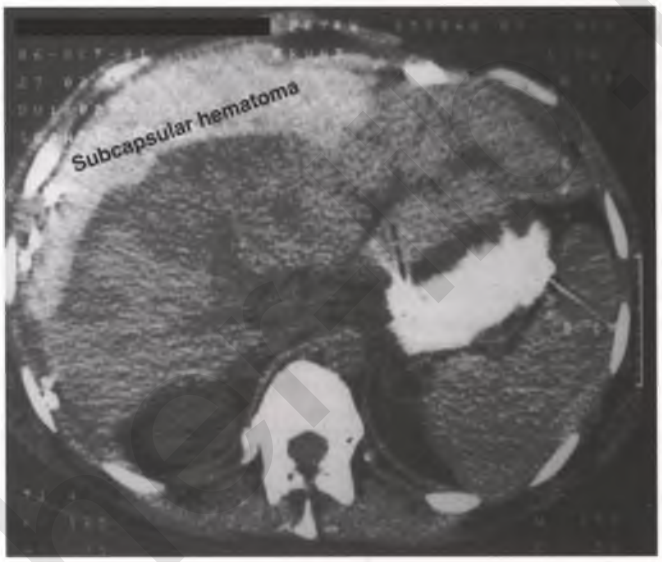

Figure 11-15 Unruptured hematoma, first day postop.

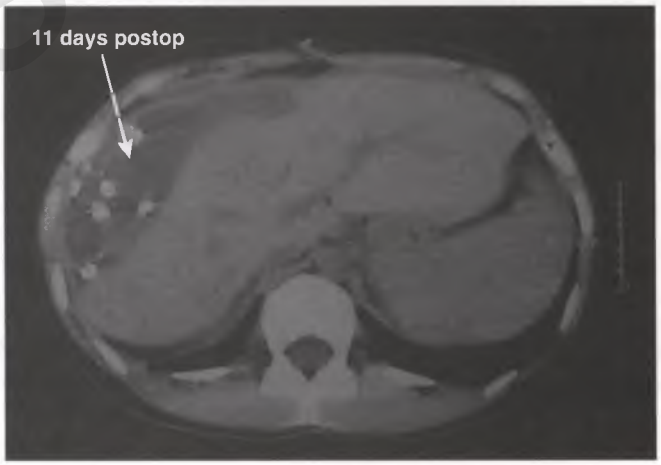

Figure 11-16 Stable hematoma 11 days postop.

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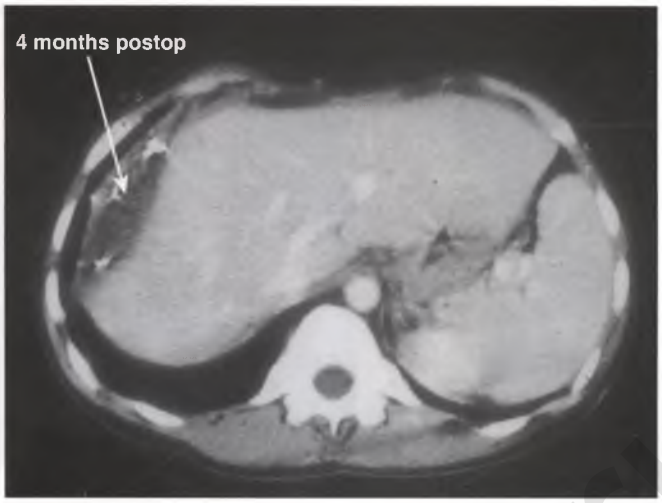

Figure 11-17 Partial resolution of hematoma 4 months postop.

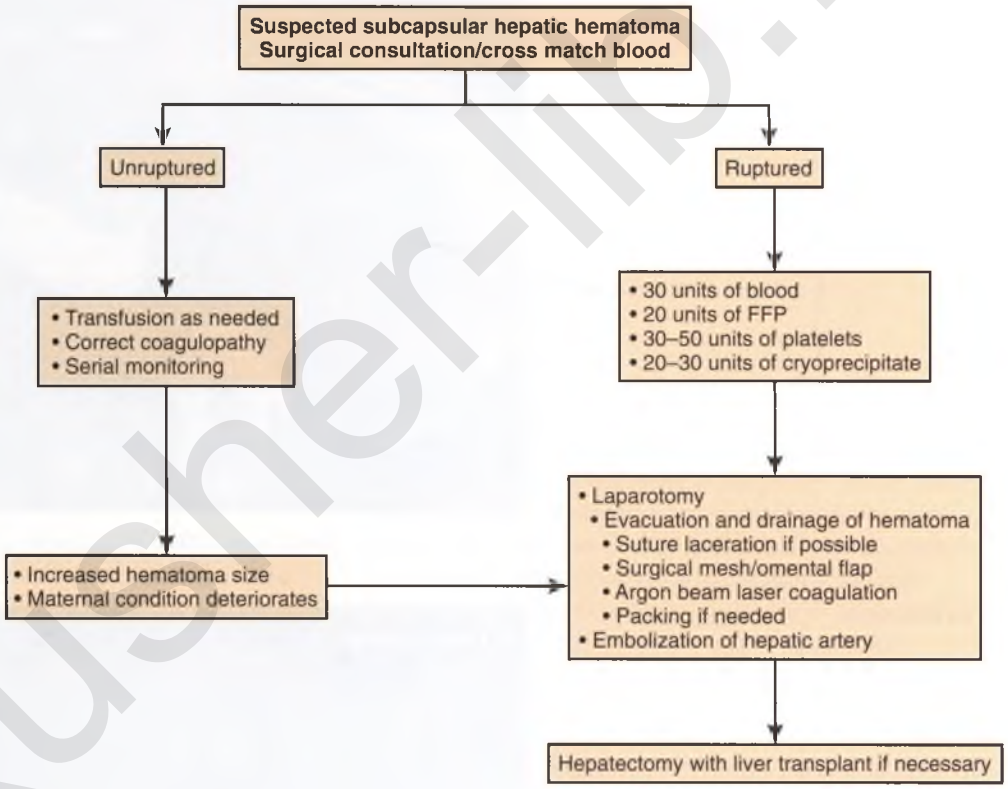

Figure 11-18 Algorithm for the management of a subcapsular hematoma of the liver.

pulmonary edema or respiratory compromise. The next step is to anticipate the potential need for transfusion of large amounts of blood and blood products and the need for aggressive intervention if rupture of the hematoma is suspected. We recommend 30 units of packed red blood cells, 20 units of fresh frozen plasma, 30 units of platelets, and 20 units of cryoprecipitate be available if rupture of a subcapsular hematoma is suspected. Constant monitoring must continue during this management, however, because patients can rapidly become unstable after rupture of the hematoma. Coagulopathy must be aggressively managed because failure to do so is associated with an increased rate of organ failure. Because of organ failure, the patient requires prolonged

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hospitalization for close monitoring in a maternal or surgical intensive care unit. Some patients may require ventilator support and/or dialysis. A patient with an unruptured subcapsular hematoma who is hemodynamically stable can be conservatively managed. Postpartum follow-up for patients with an intact subcapsular hematoma of the liver should include serial CT, MRI, or ultrasonography until the defect resolves.

Survival for patients with a ruptured subcapsular hematoma of the liver clearly is associated with rapid diagnosis and immediate medical or surgical stabilization. For those receiving numerous transfusions, hepatitis and human immunodeficiency virus (HIV) status as well as isoantibody development should be assessed. Although the data on subsequent pregnancy outcome after a subcapsular hematoma of the liver in pregnancy are limited, we and others have managed seven such patients who had successful normal maternal and fetal outcomes in their subsequent pregnancies.

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Diabetic Ketoacidosis

12

Baha M. Sibai M.D.

Video Clips on DVD

12-1 PowerPoint Discussion of Appropriate Management of Diabetic Ketoacidosis

12-2 Discussion with Dr. Sibai on Acute Management of a Patient with Diabetic Ketoacidosis

Diabetic ketoacidosis (DKA) is an infrequent complication of diabetes in pregnancy, but in the absence of prompt diagnosis and treatment it can be life threatening to mother and fetus. The reported incidence in pregnancies complicated by diabetes mellitus ranges from 1% to 5%. The incidence depends on the presence or absence of one or more of the risk factors listed in Table 12-1.

Case 1: Diabetic Ketoacidosis

A 37-year-old G5P3 presented at approximately 31 weeks of gestation complaining of nausea, vomiting, diarrhea for 2 days, and decreased fetal movement followed by no fetal movement for 24 hours. She gives a history of only one prenatal visit, and no previous prenatal blood work.

She was admitted with diagnosis of gastroenteritis with dehydration. On admission, the patient was dizzy and tachypneic with a respiratory rate of 26 beats per minute (bpm). Her temperature was 98.6° F, pulse was 127 bpm, and blood pressure of 124/80 mm Hg.

Laboratory blood tests revealed a glucose level of 983 mg/dL, K+ of 5.3 mEq/L, anion gap of 34, and creatinine of 1.8 mg/dL. A complete blood count and platelet count were normal. Arterial blood gas revealed a pH of 7.26, a bicarbonate of 13 mEq/L, and base excess of −11.2. Serum and urine ketones were positive. Electrocardiogram (ECG) revealed sinus tachycardia. Fetal heart rate monitoring revealed absent accelerations, absent variability and presence of spontaneous decelerations (Fig. 12-1A). Ultrasound examination revealed normal fluid and a biophysical profile (BPP) of 2/10.

A diagnosis of DKA was made, and the patient received 15 µ of regular insulin as a loading dose followed by continuous IV infusion at a rate of 10 µ/hr. She also received large doses of fluids (4 L of normal saline during first 5 hours) and potassium replacement. After control of plasma glucose levels and correction of maternal acidosis and electrolytes, fetal heart rate tracing continued to be nonreassuring (see Fig 12-1B) and repeat BPP was still 2/10. Despite that, delivery was not performed. Over the next several hours the fetal tracing continued to be nonreassuring and was followed by reduced base line and repetitive decelerations (see Fig. 12-1C). Umbilical artery Doppler revealed absent diastolic flow, BPP was still 2/10. A decision for cesarean section was made, but the fetus died 20 minutes after the last testing and the patient underwent induction of labor with subsequent vaginal delivery of a stillborn fetus weighing 2000 g.

Discussion

Pregnancy is characterized by increased insulin resistance (higher insulin requirement), a relative state of accelerated starvation (increased free fatty acids), and a lowered buffering capacity (low serum bicarbonate levels). These changes are increased with advanced gestation as a result of increasing anti-insulin hormone production such as human placental lactogen, prolactin, cortisol, and progesterone. As a result, DKA

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Case 1: Diabetic Ketoacidosis—cont'd

usually develops after 20 weeks' gestation, and predisposes diabetic pregnant women to develop DKA very rapidly (over hours) and at lower blood glucose levels (<300 mg/dL) than is seen in nonpregnant patients. In patients with pregestational or gestational diabetes, DKA results from inadequate circulating insulin and reduced glucose utilization in peripheral tissues (Fig. 12-2).

This patient presented with classical signs and symptoms of hyperglycemia and ketoacidosis in a pregnant woman with poorly controlled diabetes. The clinical findings in patients with DKA are related to hyperglycemia, osmotic diuresis, severe volume depletion, acidosis, and electrolyte imbalance. The presenting signs and symptoms are listed in Table 12-2.

The laboratory findings in DKA are related to the magnitude of insulin deficiency and the amount of ketoacids and ketones production (3-β-hydroxybutyrate, acetoacetate, and acetone). She also had all the laboratory findings expected in DKA (Table 12-3).

DKA in pregnancy requires prompt management with intensive maternal and fetal monitoring (Table 12-4). The principles of management consist of aggressive volume replacement, insulin therapy, correction of acidosis and electrolytes, correction of potential risk factors, and monitoring response to treatment in the mother and fetus. Table 12-5 describes fluid replacement, insulin therapy, and correction of acidosis and electrolytes during the initial 24 hours of management.

In patients with DKA, the fluid deficit is approximately 100 mL/kg body weight. It is important to replace 75% of fluid deficit during the first 24 hours and to be completed within 48 hours. Use regular (short-acting) insulin as an IV bolus and then as continuous infusion until serum bicarbonate and anion gap become normal and serum ketones become negative. It is important to remember that 3-β-hydroxybutyrate is the predominant ketone body (five times as high as acetoacetate) in patients with DKA. The nitroprusside reaction tests (Acetest, Ketostix) measure only acetoacetate. Therefore, blood ketones should be monitored with the Optium meter, which measures capillary β-hydroxybutyrate.

In DKA, potassium deficit is approximately 5 to 10 mEq/L of body weight. The deficit results from renal loss (osmotic diuresis) and potassium shift from extracellular to intracellular space with the use of insulin and correction of acidosis during the first 4 hours of treatment. The onset of significant hypokalemia could lead to serious cardiac arrhythmias. Therefore, it is important to keep serum potassium above 4.5 meq/L by IV potassium replacement with either potassium chloride or potassium phosphate (if serum phosphate is low).

Maternal DKA results in acute changes in uterine blood flow, reduced tissue perfusion, and increased hemoglobin affinity for oxygen (reduced fetal oxygen delivery). Maternal hyperglycemia also results in fetal hyperglycemia and hyperinsulinemia, which increase fetal oxygen requirements. These changes result in fetal hypoxemia and acidosis which can influence all modes of fetal testing (Table 12-6). However, these changes are not an indication for immediate delivery because most resolve after correction of the metabolic condition. In addition, performing emergency cesarean delivery in the presence of untreated DKA could lead to increased maternal morbidities and even mortality. In this case, priority was to correct the maternal DKA first. This took about 6 hours during which maternal condition was improving. Despite improvement in maternal condition, the fetal heart rate tracing remained nonreactive and there were episodes of deceleration, and the BPP remained at 2/10. As a result, the patient should have been delivered because of nonreassuring fetal testing. Delayed delivery ultimately resulted in fetal death secondary to presumed fetal hypoxia and acidosis. However, if fetal compromise continues (nonreassuring fetal heart rate or BPP) after maternal stabilization, then prompt delivery is warranted to prevent fetal demise and/or injury.

Table 12-1 Risk Factors for DKA in Pregnancy

- Noncompliance with insulin therapy
- Undiagnosed diabetes or gestational diabetes
- Acute infections, particularly pyelonephritis or pneumonia
- Protracted vomiting
- Use of β-sympathomimetic drugs for preterm labor
- Corticosteroids for fetal lung maturity

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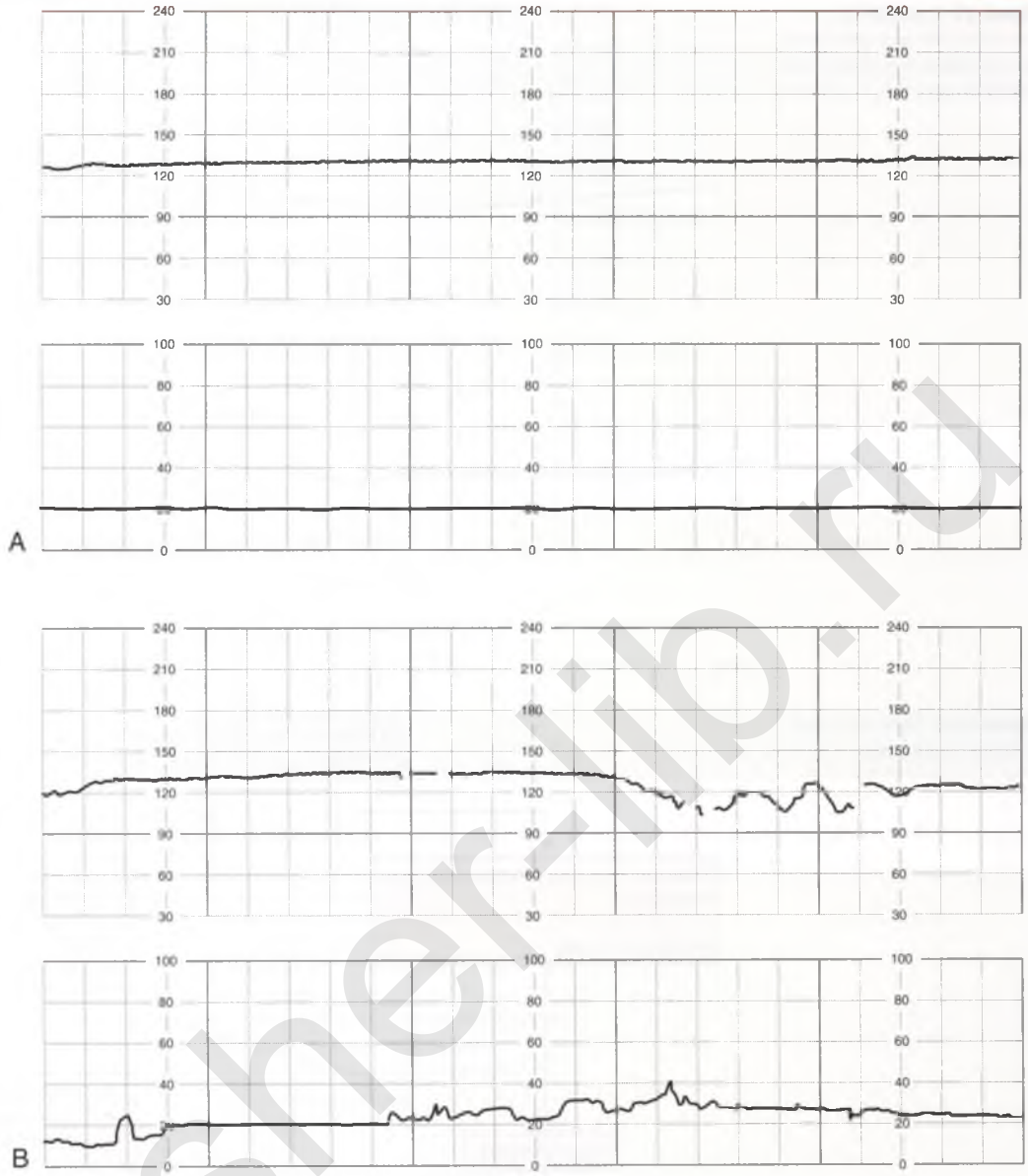

Figure 12-1 A, Fetal heart rate (FHR) tracing demonstrating absent accelerations, absent variability, and FHR decelerations during DKA. **B,** Nonreassuring FHR tracing after correction of maternal acidosis.

Continued

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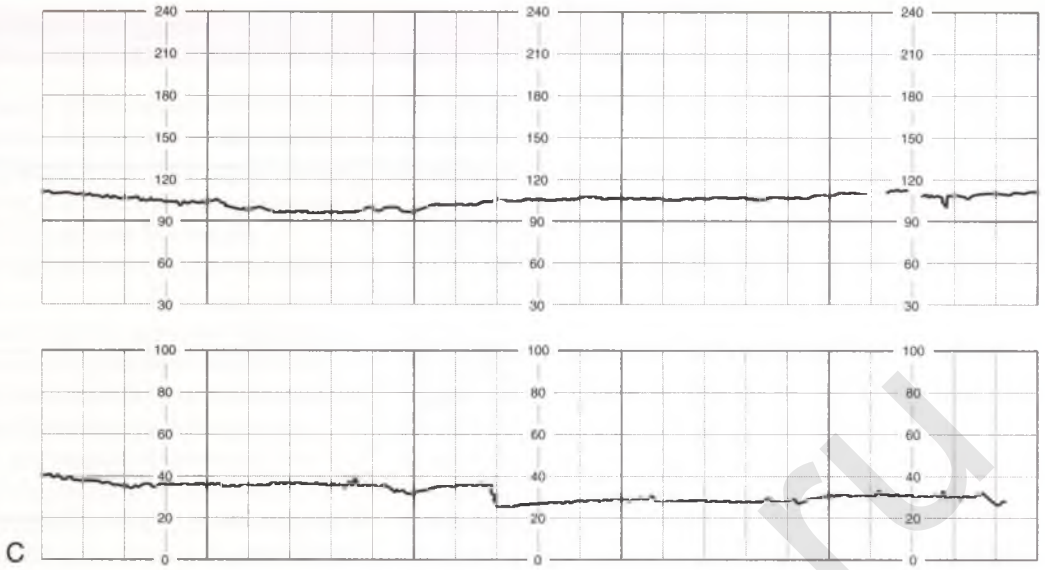

Figure 12-1, cont'd C, Ominous FHR tracing recorded approximately 30 minutes prior to fetal demise.

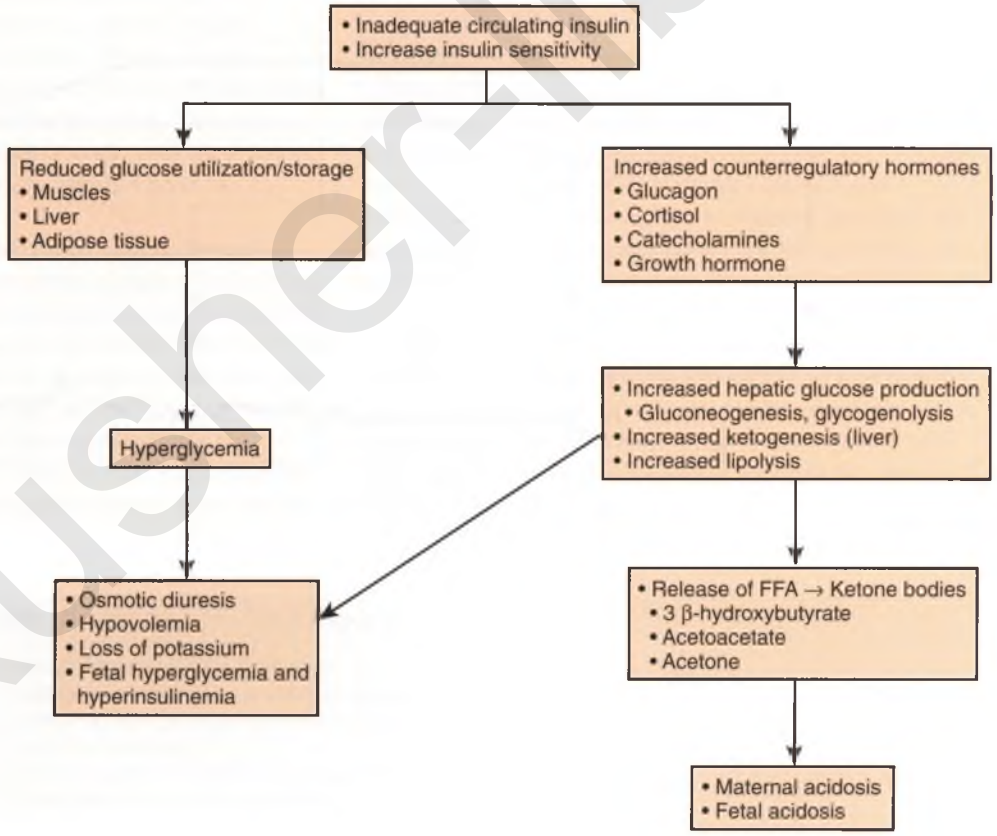

Figure 12-2 Pathophysiology of DKA in pregnancy.

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Table 12-2 Signs and Symptoms of DKA

Signs	Symptoms
Hyperventilation-tachypnea	Polyuria
Sinus tachycardia	Polydipsia
Hypotension	Nausea/vomiting
Dehydration (dry mucous membranes)	Abdominal pain
Change in sensorium/disorientation	Blurred vision
Fruity breath (increased acetone)	Muscle weakness
Coma	

Table 12-3 Laboratory Findings in DKA

- Serum glucose >300 mg/dL (>200 mg/dL)*
- Arterial pH <7.30 (<7.25)*
- Serum bicarbonate <15 mEq/L*
- Anion gap >12 mEq/L
- Serum ketones (+)†

*These values are variable.
†Acetest or Ketostix only detect acetoacetate.

Table 12-4 Monitoring of DKA in Pregnancy

- Medical or obstetric intensive care unit
- Vital signs every 15 minutes
- Large bore IV catheter or central line
- Obtain arterial gases, serum glucose, electrolytes, ketones
  - Serum glucose and ketones every 1 to 2 hours
  - Serial arterial gases and anion gap, and electrolytes
- Obtain urine for analysis, culture, ketones
- Oxygen by face mask at 6 L/min
- Continuous pulse oximetry
- Continuous fetal heart rate monitoring (≥24 weeks)
- Evaluate for infection
- Bedside flow-sheet
  - Intake/output
  - Results of serial blood tests
  - Medications

Table 12-5 Treatment of DKA

- Insulin replacement (bolus 10-15 units, maintenance 0.1 units/kg/hr)
- Fluid replacement (deficit ≈ 100 mL/kg body weight)
  - 1 L (0.9% NS) first hr
  - 1 L (0.9% NS) second hr
  - 0.5 L/hr third hr
  - 0.25 L/hr 4-24 hr
- Add 5% dextrose to IV if glucose <250 mg/dL
- Monitor serum glucose and ketones every hour
- Potassium replacement if serum K+ <5 mEq/L
  - Potassium chloride (20-40 mEq/hr)
  - Potassium phosphate
- Continuous fetal heart rate monitoring

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Table 12-6 Transient Changes in Fetal Testing in DKA

- Fetal heart rate
  - Tachycardia
  - Absent accelerations
  - Poor variability
  - Late decelerations
- Abnormal BPP
- Doppler findings (redistribution of blood flow)
  - Increased umbilical artery pulsatility index
  - Reduced middle cerebral artery pulsatility index

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Thyroid Storm in Pregnancy

13

Baha M. Sibai M.D.
Annette Bombrys D.O.

Video Clips on DVD

13-1 PowerPoint Discussion of Management of Thyroid Storm

13-2 Discussion with Dr. Sibai on Acute Management of a Patient with Thyroid Storm

Thyroid storm is a rare but potentially lethal complication of uncontrolled hyperthyroidism during pregnancy. The estimated incidence is 1% to 2% of pregnant women with hyperthyroidism. It is a life-threatening condition that usually develops in patients with poorly controlled disease in association with one or more of the factors listed in Table 13-1.

Case 1: Thyroid Storm

A 32-year-old in the 24th week of her fourth pregnancy arrives at the emergency department complaining of cough and congestion; shortness of breath; and swelling in her face, hands, and feet; diarrhea; and racing heart. The swelling and shortness of breath have become worse over the past 2 weeks, and she had several episodes of bloody vomiting the day before her visit. In addition, she had a 15-pound weight loss during the past 2 weeks. The patient says she has not experienced any leakage of fluid, vaginal bleeding, or contractions. She reports good fetal movement.

Physical findings revealed exophthalmia and an enlarged thyroid with a nodule on the right side, as well as bilateral rales, tachycardia, tremor, and increased deep tendon reflexes. The blood pressure was 170/111, pulse of 125 beats per minute (bpm), and temperature of 99° F. Laboratory tests revealed thyroid-stimulating hormone (TSH) of <0.01 IU/L (normal, 0.35 to 5.50), free T_4 (FT_4) of 5 ng/dL (normal, 0.8 to 11.8), free T_3 (FT_3) of 842 pg/dL (normal, 230 to 420), thyroid-stimulating immunoglobulin (TSI) 234% (normal <125), and thyroid peroxidase antibody (TPo) of 984 IU/mL (normal, 0 to 35). Complete blood count, platelet count, liver enzymes, and metabolic profile were all normal. Pulse oximetry was 90%, chest x-ray revealed bilateral pulmonary edema and pleural effusions, and electrocardiogram (ECG) revealed sinus tachycardia. A computed tomography (CT) scan of the chest showed bilateral pleural effusions indicative of high-output cardiac failure. Thyroid ultrasonography (US) revealed a diffusely enlarged thyroid gland with a right-sided mass.

Fetal heart rate (FHR) was in the 170s, with normal variability and occasional variable deceleration (Fig. 13-1). Fetal US was consistent with the estimated gestational age and showed adequate amniotic fluid and no gross fetal anomalies and no evidence of fetal cardiac failure.

A diagnosis of thyroid storm was made, and the patient received propylthiouracil (PTU) 800 mg orally as a loading dose followed by 200 mg every 6 hours. One hour later she received Lugol's solution. In addition, she received 2 mg IV propranolol followed by 2 mg as needed, and dexamethasone 2 mg IV every 6 hours. Because of congestive heart failure, she received IV doses of furosemide, and for severe hypertension she received IV labetalol. As her symptoms diminished, fetal tachycardia resolved (Fig. 13-2). The patient's (FT_4) level began to decline, consistent with appropriate treatment, and she was discharged home and instructed to continue PTU and labetalol and to follow up at the endocrinology and high-risk obstetric clinics as soon as possible.

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Case 1: Thyroid Storm—cont'd

The patient did not follow this advice. Consequently, she presented at 33 5/7 weeks in a hypertensive crisis (blood pressure 182/125 mm Hg), with symptoms similar to those she first exhibited plus acute pulmonary edema. Laboratory testing again revealed hyperthyroidism, with hemoconcentration (hematocrit 43%), elevated liver enzymes (AST, ALT, and bilirubin), and proteinuria of 2+ on dipstick. Chest x-ray revealed pulmonary edema. She was treated accordingly. Because of active labor and diagnosis of severe preeclampsia, she was given IV magnesium sulfate and IV labetalol to control severe hypertension. During labor, FHR revealed repetitive late decelerations (Fig. 13-3) and emergency cesarean section was performed. A male infant was delivered, weight 2390 g, Apgar scores 9 at 1 minute and 9 at 5 minutes. Cord gases revealed pH of 7.21, Pco_2 of 63 mm Hg, HCO_3 of 26 mmol/L, and base deficit of –3.2 mmol/L.

Treatment of her hyperthyroidism and pulmonary edema was continued in the postpartum period and she was discharged after stabilization on various medications.

Discussion

The pathophysiologic abnormalities in thyroid storm are related to a state of excessive synthesis and release of T_4 in association with peripheral conversion of T_4 to T_3. The excess free T_4 and free T_3 levels in the maternal circulation results in a state of hypermetabolism with cardiovascular decompensation and central nervous system abnormalities. These abnormalities are exaggerated in the presence of anemia, labor, infection, molar pregnancy, and stress. Therefore US evaluation is important to rule out the presence of molar pregnancy when patients present with these signs and symptoms in the first half of pregnancy. If molar pregnancy is detected, then management should include evacuation of the molar pregnancy. The clinical signs and symptoms usually reflect derangement in multiorgan systems that are affected by the actions of thyroid hormones. The presenting signs and symptoms in thyroid storm are listed in Table 13-2, and the laboratory findings are summarized in Table 13-3.

This patient had the classical signs and symptoms of hyperthyroidism. They were present before pregnancy and early in pregnancy. Unfortunately they were not appreciated by her physician. If diagnosed early and if treated appropriately, this could have prevented the development of thyroid storm.

Pregnancy complicated by a thyroid storm is associated with increased maternal and perinatal morbidities and mortality. Consequently these patients require admission with close maternal and fetal monitoring in an intensive care facility (Table 13-4). The goals of therapy include to reduce the synthesis and release of thyroid hormone, to block peripheral conversion of T_4 to T_3, and to block the actions of thyroid hormones on target tissue and should include treatment of potential risk factors and associated maternal complications such as dehydration, electrolyte imbalance, congestive heart failure, arrhythmias, or seizures (Table 13-5). Treatment should be started based on clinical findings and should not await the results of thyroid function tests. A stepwise approach for management of thyroid storm including medications and doses is detailed in Figure 13-4.

In this case the patient initially responded to treatment with improvement in her clinical laboratory findings as well as in FHR tracing. It is important to emphasize that the patient not be rushed for emergency cesarean delivery because of nonreassuring fetal testing. This could prove detrimental to maternal and perinatal outcome. In general, stabilization of maternal condition will result in improved FHR tracing.

This patient did not follow the medical advice following hospital discharge. She was noncompliant with taking her antithyroid medications. As a result, she presented again with an acute emergency that was worse than the previous presentation. Despite aggressive medical therapy, the FHR tracing did not improve (see Fig. 13-3B) suggesting cesarean delivery was required. At delivery she was found to have 25% abruptio placentae, which is one of the complications of untreated hyperthyroidism.

Table 13-1 Risk Factors for Thyroid Storm in Pregnancy

- Undiagnosed hyperthyroidism
- Molar pregnancy
- Noncompliance with antithyroid medications
- Inadequate control of hyperthyroidism
- Infection
- Preeclampsia
- Labor and delivery

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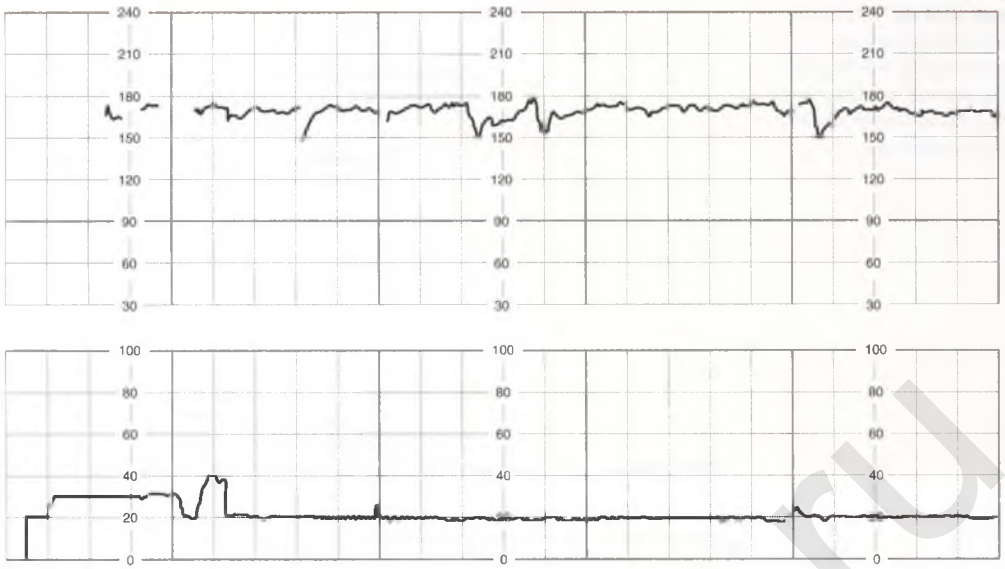

Figure 13-1 FHR tracing demonstrating fetal tachycardia with occasional deceleration.

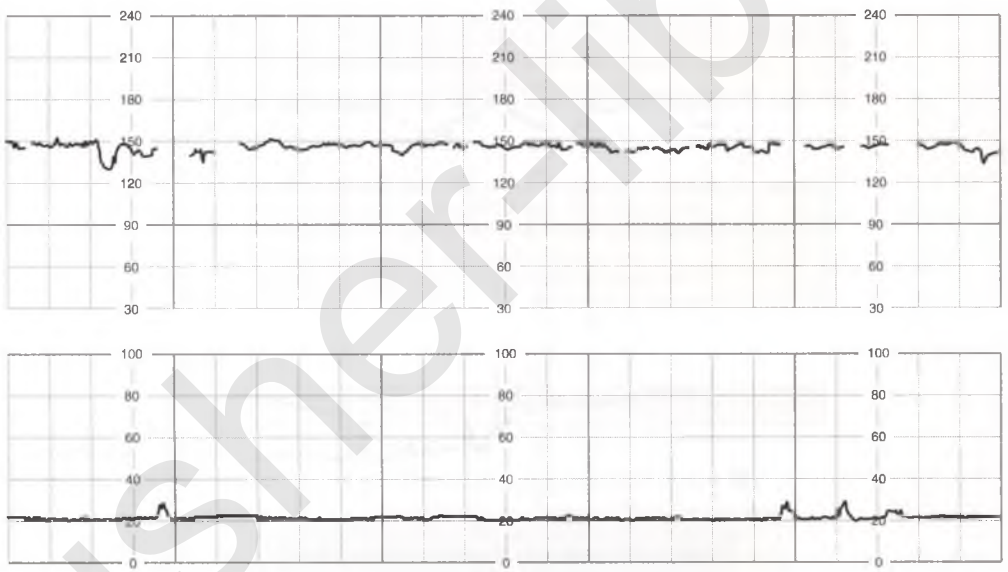

Figure 13-2 FHR tracing demonstrating normal baseline and variability after control of hyperthyroidism.

Table 13-2 Signs and Symptoms of Thyroid Storm

Signs	Symptoms
Fever, flushed skin	Nausea/vomiting
Tachycardia	Diarrhea
Hypertension	Weight loss, sweating
Wide pulse pressure	Palpitations, tremors
Cardiac arrhythmias	Nervousness/restlessness
Congestive heart failure	Swelling
Altered mental state	Shortness of breath
Seizures/coma	

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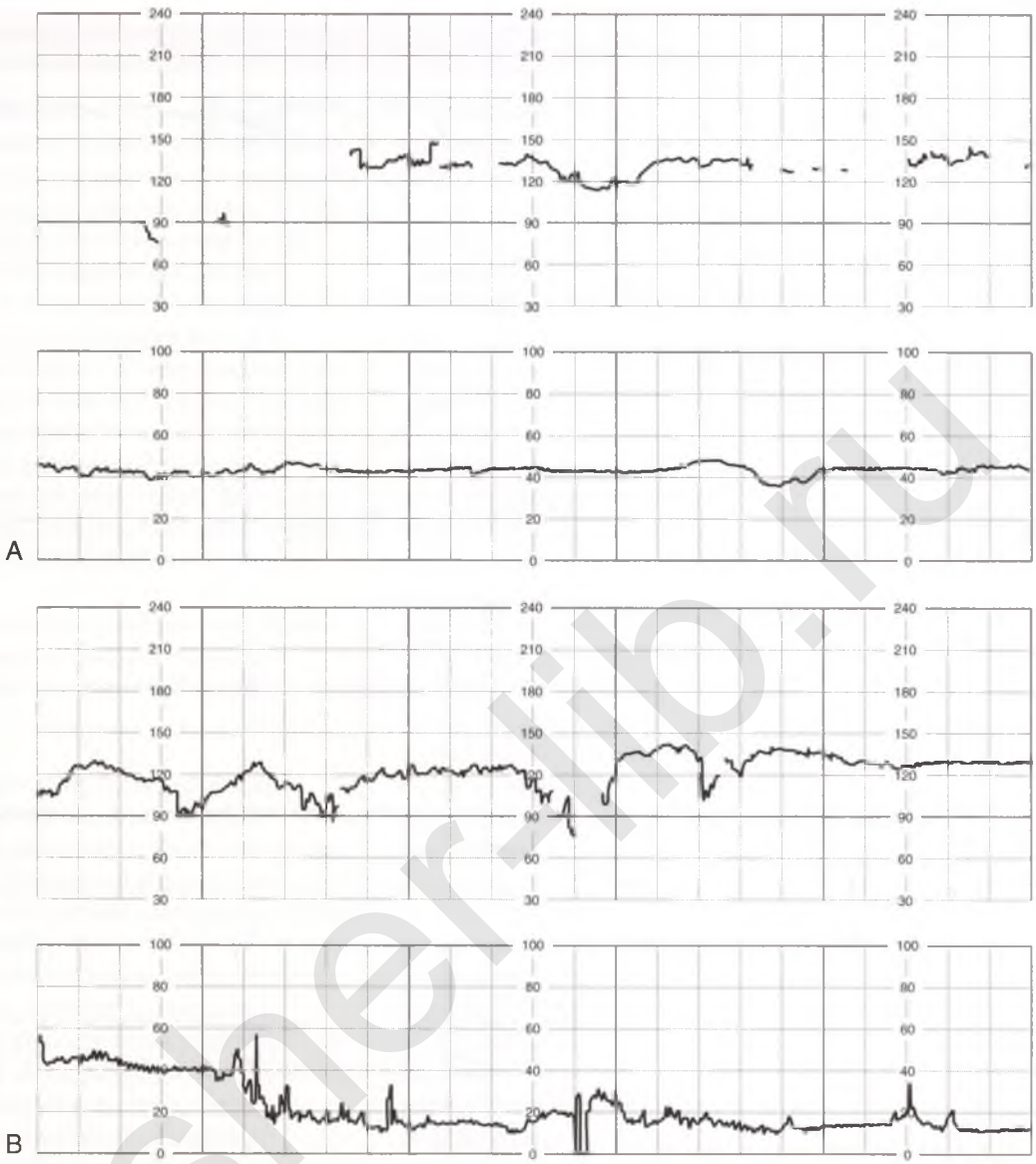

Figure 13-3 **A,** FHR tracing demonstrating variable deceleration. **B,** FHR tracing demonstrating repetitive late decelerations. Patient also had severe hypertension.

Table 13-3 Laboratory Findings in Thyroid Storm

- Low to undetectable TSH
- Increased FT_4
- Increased FT_3
- Leukocytosis
- Elevated liver enzymes
- Chest x-ray (congestive heart failure)

Table 13-4 Monitoring of Thyroid Storm in Pregnancy

- Admit to obstetric or medical ICU
- Large IV access
- Blood tests for TSH, FT_4, FT_3, CBC, electrolytes
- Arterial blood gases
- Insert nasogastric tube if needed
- Oxygen supplementation by face mask
- Continuous pulse oximetry
- Continuous ECG monitoring for arrhythmias
- Central hemodynamic monitoring
- Continuous FHR monitoring (≥24 wk)
- Evaluate for infection, preeclampsia

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Table 13-5 Treatment of Thyroid Storm

- Reduce synthesis of thyroid hormone (thioamide)
- Prevent release of stored hormones
  - Iodides
  - Glucocorticoids
- Block peripheral conversion of T_4 to T_3
  - Thioamides
  - Glucocorticoids
- Block peripheral actions of hormones
  - Phenobarbital
  - Antiemetics
- Reduction of fever
  - Rectal acetaminophen
  - Cooling blanket/sponge bath
- Fluid and electrolyte replacement
- Control of arrhythmias, heart failure, seizures

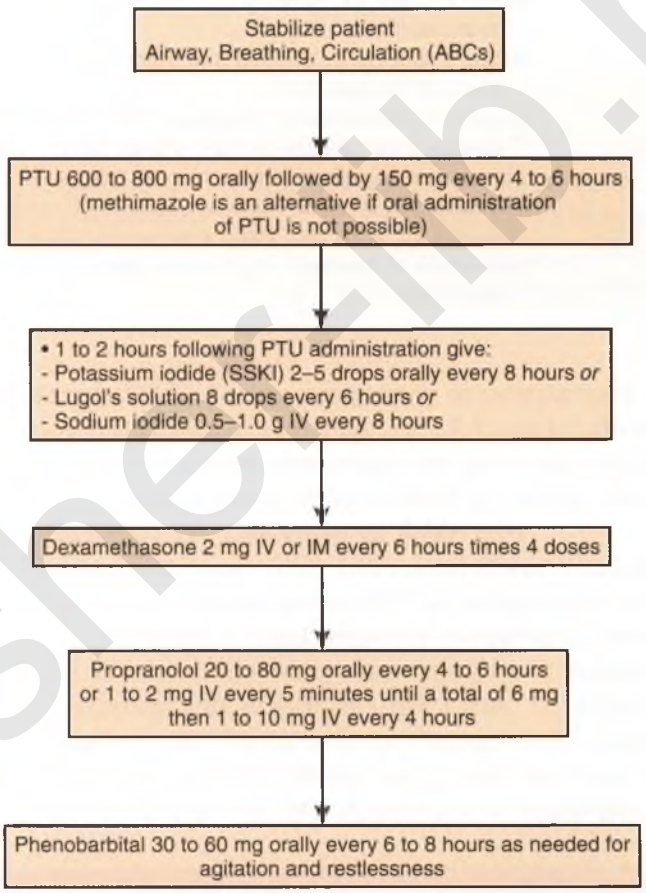

Figure 13-4 Management of thyroid storm.

Fetal and Neonatal Effects

Hyperthyroidism in the fetus or newborn can be caused by placental transfer of maternal TSI. It is usually encountered in women with Graves' disease. The clinical findings of hyperthyroidism in the fetus include tachycardia, fetal growth restriction, oligohydramnios, and congestive heart failure. During a thyroid storm, FHR tracing can reveal tachycardia and decreased variability (see Fig. 13-1). Ultrasound may demonstrate reduced fluid and signs of fetal congestive heart failure. It is important to note that treatment of the thyroid storm in the mother will reverse the findings in the fetus. If fetal condition

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does not improve after treatment of the maternal condition, then delivery is indicated if there is evidence of nonreassuring fetal testing. Following delivery, the pediatrician should be informed about the presence of hyperthyroidism in order to observe the infant for signs of hyperthyroidism, which are usually transient and related to transplacental passage of thyroid-stimulating antibodies.

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Cardiorespiratory Arrest and Cardiorespiratory Resuscitation in Pregnancy

14

Candice Snyder M.D.
Katherine Wolfe D.O.
Ryan Loftin M.D.
Baha M. Sibai M.D.

Video Clips on DVD

14-1 PowerPoint Discussion of Cardiorespiratory Arrest and Resuscitation in Pregnancy

14-2 A Step-by-Step Video Demonstration of How to Manage a Pregnant Patient Who Develops Cardiorespiratory Arrest

Cardiorespiratory arrest (CRA) during pregnancy or postpartum is a rare complication with unknown incidence. The onset of CRA can be abrupt and unpredictable (as in the case of a massive pulmonary or amniotic fluid embolism) or gradual and expected (as seen with hypovolemic or septic shock). Potential causes of CRA in pregnancy are described in Table 14-1.

Maternal and perinatal outcomes after CRA depends on the cause as well as the speed of cardiopulmonary resuscitation (CPR), including timing of delivery. Similar to nonpregnant patients, a rapid response is essential; however, in cases of pregnancy, timing is extremely important because it relates to adequate resuscitation of the mother and effects on the fetus. In addition, there are factors unique to pregnancy as it relates to physiologic and anatomic changes (Table 14-2), the technique of CPR (Table 14-3), and the need to empty the uterus within 5 minutes of onset of CPR if there is no response.

Case 1: Cardiorespiratory Arrest

A 40-year-old patient is pregnant for the first time with a twin gestation as a result of in-vitro fertilization. She has a 5-year history of chronic hypertension that required two antihypertensive medications for blood pressure management during this pregnancy. At 34 weeks' gestation, she presented to an emergency department with shortness of breath, cough, and inability to sleep flat. Her blood pressure was 180 to 200 (systolic), 105 to 115 mm Hg (diastolic), and her heart rate was 115 beats per minute (bpm) with a respiratory rate of 24 breaths/min. Pulse oximetry was between 91% and 93%. Blood tests and urine were sent to rule out superimposed preeclampsia. Heart and lung examinations were noted to be normal. The patient received 10 mg of IV labetalol with a plan to observe response to treatment. During the observation period, the blood pressures remained between 174 to 186 and 100 to 110 mm Hg, the pulse was 120 bpm, and the respiratory rate was 28 breaths/min. The pulse oximeter was reading between 90% and 91%. After 30 minutes the patient was noted to be cyanotic, agitated, and sitting up on edge of the bed; this was followed by acute CRA. The physician was called and CPR was initiated in the emergency department using standard nonpregnancy techniques. This was continued for at least 5

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Case 1: Cardiorespiratory Arrest—cont'd

minutes with no response. At that time, the obstetrician in the room called for emergency cesarean delivery for which the patient was transferred to the operating room. A low-transverse uterine incision was performed 12 minutes after the arrest with delivery of two live infants that were severely depressed. Both infants had evidence of severe metabolic acidosis and developed neonatal encephalopathy. After delivery of the infants, CPR was continued, but the patient expired 30 minutes later.

Discussion

Early detection and prompt management of potential causes of CRA during pregnancy are extremely critical because they may allow for interventions that will prevent adverse maternal and fetal outcomes as was evident in this case. This patient had several risk factors for pulmonary edema, and on presentation to the emergency department, her clinical findings were suggestive of early onset heart failure. No chest x-ray was ordered, and blood pressure was not adequately controlled. She then developed unobserved progressive hypoxia leading to respiratory and then cardiac arrest. The code team was called and CPR was started promptly, but the resuscitation team was not familiar with the appropriate techniques relevant to pregnancy. As a result, CPR was performed with the patient in supine position and the uterus was not displaced from compressing the inferior vena cava and aorta, particularly in the presence of twins. In addition, after CPR for 4 minutes, no attempts were made to deliver the infants both for maternal and fetal benefits. Finally, when the decision for delivery was made, the patient was moved to the operating room resulting in additional delay in timing of delivery.

This case demonstrates the lack of knowledge of CPR in pregnancy by the various providers responding to the code, particularly as to the importance of left uterine displacement to relieve aortocaval compression, and of prompt delivery within 5 minutes into CPR. In addition, it highlights the need for development of protocols for CPR in pregnancy including mechanisms for education, training, and methods to maintain skills in CPR for all physicians and staff providing obstetric services.

Several studies have reported that most physicians that may respond to perform CPR in pregnancy lack the necessary knowledge and skills to manage maternal resuscitation. These studies have identified limited knowledge of the importance of anatomic and physiologic changes in pregnancy, the technique to be used, and the potential life-saving benefit of cesarean delivery within 5 minutes. In addition, there was a lack of knowledge among providers regarding the safety and doses of medication to be administered during CPR.

Table 14-1 Potential Causes of Cardiorespiratory Arrest in Pregnancy

- Pulmonary embolism
- Untreated pulmonary edema
- Trauma/hypovolemic shock
- Coronary artery disease/dysrhythmia
- Cardiotoxic medications
- Amniotic fluid/air embolism
- Failed intubation
- Magnesium overdose
- Cerebrovascular accident
- Septic shock

Table 14-2 Factors Unique to CPR in Pregnancy

- Physiologic changes
  - Airway/laryngeal edema
  - Increased O_2 consumption
  - Increased risk of aspiration
  - Diminished chest compliance and functional residual capacity
- Anatomic changes
  - Gravid uterus makes rise and fall of chest difficult to evaluate
  - Chest compressions are technically challenging
    - Large breasts in obese women
    - Flared ribs
    - Elevated diaphragm
  - Compression of inferior vena cava impairs venous return
  - Compression of aorta by uterus—exaggerated by obesity

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Table 14-3 Techniques of CPR Unique to Pregnancy

- Patient position (relieve compression off IVC/aorta)
  - Left side tilt with back at 30 degrees
  - Place wedge under right side
  - One rescuer to patient's left side
    - Pull uterus laterally and toward head
- Chest compressions
  - Place on hard board to be effective
  - Pendulous breasts held back by assistant
  - Hand positioned up on sternum (2 cm above tip) (see Fig.14-2)
  - Push hard and fast at 100/min
  - Allow full chest recoil
  - 30 compressions:2 rescue breaths ratio
- Defibrillation
  - Remove fetal and uterine monitors
- Empty uterus if no response after 4 minutes

Management and Technique of CPR in Pregnancy

Early detection of CRA, rapid intervention with proper attention to maternal position and technique of CPR, and timely delivery in case of no response to resuscitation efforts are key to reducing adverse maternal and perinatal outcomes during maternal resuscitation. The first step in management is to confirm the presence of a pulseless state and immediately call for help including the need for a delivery pack or tray. A detailed stepwise management of CPR in pregnancy is described in Figure 14-1. In addition, it is important to keep in mind the differences in technique of CPR that are unique to pregnancy (see Table 14-3 and Fig.14-2). Finally, timing of delivery within 5 minutes is potentially lifesaving for both mother and fetus as described following.

Timing of Delivery Is Essential to Provision of CPR in Pregnancy

- Secondary to increased oxygen consumption and reduced functional residual capacity, pregnant women become hypoxic more quickly than nonpregnant individuals, who can suffer irreversible brain injury after only 4 minutes. There are potentially two patients to be saved, and outcome for the neonate is improved by delivery within 5 minutes. Delivery after 10 minutes is associated with reduced survival and the potential for neonatal brain injury.
- Perimortem cesarean section at 4 to 5 minutes: Emptying of the uterus is important to save the life of not only the fetus, but also the mother.
  - For patients ≥24 weeks' gestation (four fingerbreadths above the umbilicus) delivery will relieve aortocaval compression and improve thoracic compliance, which results in improved efficacy of compressions and ease of ventilation. This is even more important in multiple fetal gestations in which compression is exaggerated.
    - Timing is valuable
      - Perform cesarean section at site of CPR
      - No anesthetic is needed
      - Midline abdominal incision
      - Classical uterine incision
      - Any available scalpel or knife
    - Continue CPR during and after delivery

If CPR is successful, then transfer the patient to an operating room to continue the surgery and to continue with the secondary ABCD survey (Table 14-4) of CPR as recommended by the American Heart Association.

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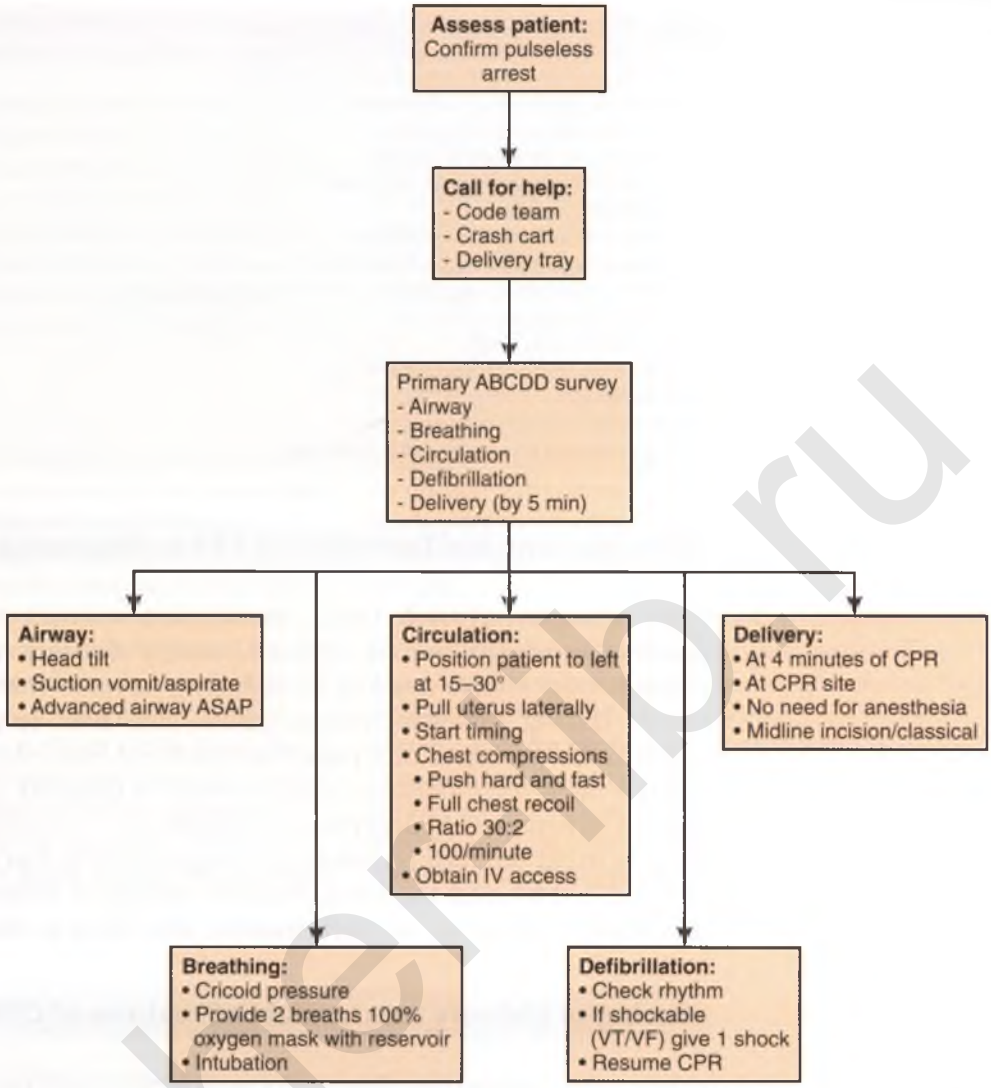

Figure 14-1 Algorithm for primary management of CPR in pregnancy.

Table 14-4 Secondary ABCD Survey

A. Airway
   - Secure advanced airway as soon as possible
   - Rapid sequence intubation with cricoid pressure

B. Breathing
   - Esophageal detector device may not be reliable in predicting correct placement
   - Ventilator support should be tailored to produce effective oxygenation and ventilation

C. Circulation
   - Establish IV access and infuse IVF and/or blood as appropriate
   - Medications may not reach maternal heart until delivery of fetus, therefore, femoral or lower extremity sites should not be used
   - Adhere to standard ACLS recommendations for resuscitation medications
   - Consider causes of arrest and treat accordingly

D. Defibrillation if needed

(Figure 14-1 and Table 14-4 adapted from Cardiac Arrest Associated with Pregnancy. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2005;112:IV-150–IV-153.)

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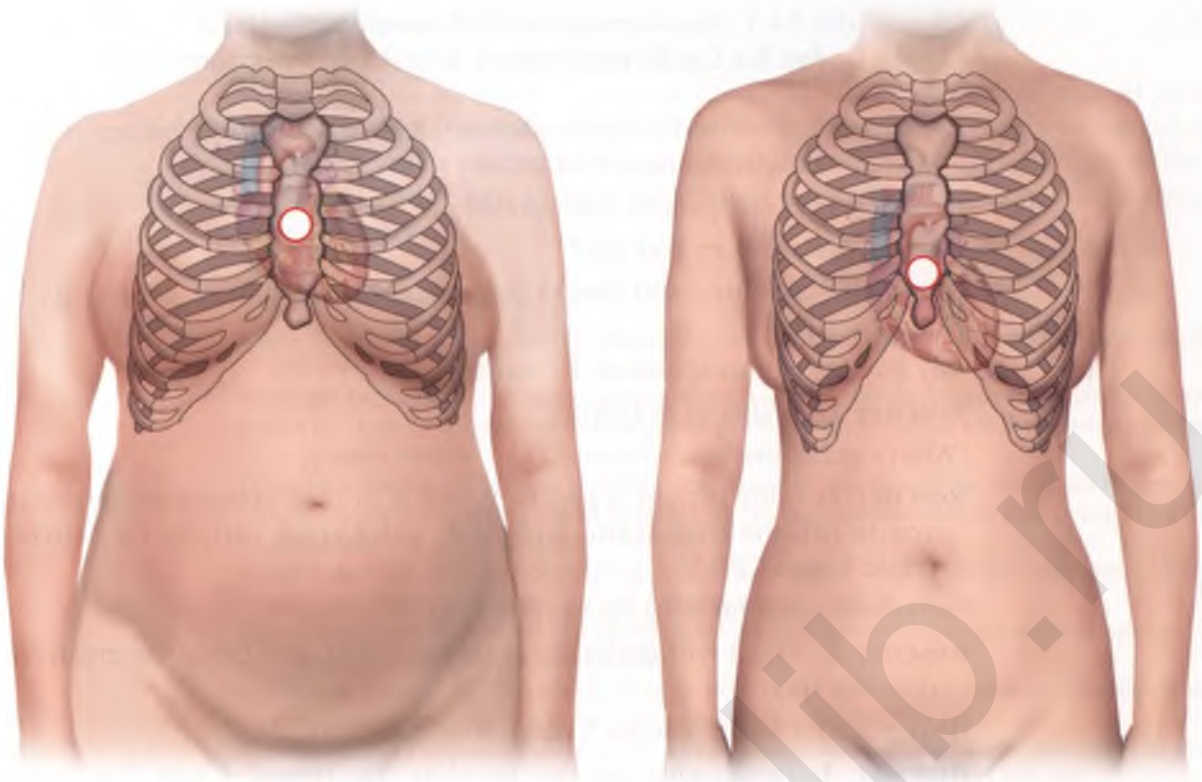
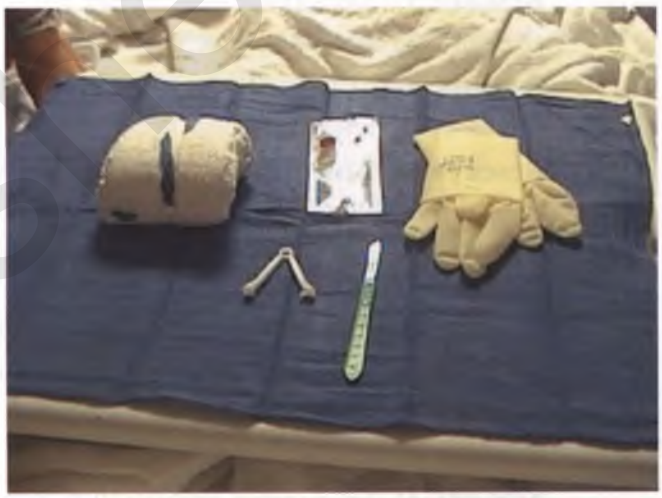


Figure 14-2 Differences in CPR technique between pregnant and nonpregnant women.

Figure 14-3 Recommended delivery pack to be used during CPR.



Conclusion

Because CRA is an extremely rare event during pregnancy and postpartum, it is important that all obstetric units develop training and drills for their staff as described in Appendix 14-1. It is also advisable to have a delivery pack on all resuscitation carts in case of emergencies (see Fig. 14-3). This pack should be available in emergency departments as well as in intensive care units.

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Appendix 14-1: Recommended CPR Script to Be Used for Training/ Mock Codes for Cardiorespiratory Arrest in a Pregnant Patient

Participants: Rescuer 1 finds patient; Rescuer 2 joins rescuer 1 to help; Anesthesia; Attending OB surgeon

Rescuer 1: (finds patient unresponsive)

"Ma'am, Ma'am—are you OK?"

(listens for breathing and checks pulse, pushes help button or calls for help in hall)

"My patient is unresponsive, I need help."

Rescuer 2: (walks in to help)

"What's going on?"

Rescuer 1: "This patient is pregnant and is in cardiorespiratory arrest. I need you to call the code team, notify OB, get a crash cart, and a delivery tray while I start CPR."

(simultaneously hooking up oxygen bag)

Rescuer 2: "OK, I will notify the code team, OB, and bring the crash cart and delivery tray."

(leaves scene while rescuer 1 stays with patient)

Rescuer 1: (establishes airway by chin tilt, provides two rescue breaths, initiates chest compressions, and notes time)

"It is now 10:42."

(Rescuer 2 returns with crash cart and delivery tray)

(At end of compressions, Rescuers 1 and 2 reposition patient to left side)

Rescuer 1: "Let's place her on her left side."

(Anesthesia arrives to room)

Anesthesia: "I am with anesthesia, what is going on?"

(takes over airway)

Rescuer 1: "This patient is 30 weeks' pregnant, and we have been providing CPR now for 2 minutes."

(directs Anesthesia to manage airway and directs Rescuer 2 to resume compressions and steps back)

"You manage her airway and you resume compressions."

(after round of CPR, Rescuer 2 checks pulse)

Rescuer 1: "Check her pulse."

Rescuer 2: "Still no pulse."

Rescuer 1: "Let's check her rhythm; she is in aFib. Prepare to defibrillate."

Rescuer 2: (applies paddles) "I'm clear, you're clear, we're all clear. Shock."

Rescuer 1: "Resume CPR."

(OB Attending enters room)

OB Attending: "This is my patient. How long has she been without a pulse?"

Rescuer 1: "It has now been 4 minutes. Hold CPR and recheck her rhythm. She is still in aFib."

OB Attending: "We must deliver the baby now. I need the scalpel!"

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Acknowledgements

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Indications and Techniques for Transcervical and Abdominal Cerclage

15

John O'Brien M.D.
Lynlee Wolfe M.D.
C. David Adair M.D.
Stephen DePasquale M.D.

Videos Clips on DVD

15-1 Techniques for Performance of McDonald Cervical Cerclage

15-2 Robotic Abdominal Cerclage Placement

Cervical insufficiency is traditionally defined as structural weakness of the cervix leading to the inability of the cervix to sustain an intrauterine pregnancy. The incidence is highly variable depending on the criteria for diagnosis. Most cases are associated with congenital shortening, surgical amputation, or stromal damage secondary to unhealed lacerations. Risk factors for cervical insufficiency are depicted in Table 15-1.

Cervical Cerclage

The use of a surgical strategy to reduce the rate of previable or preterm birth dates to Shirodkar's intervention with transplanted fascia lata using a homograft to improve cervical function. For decades, patients with repeated second-trimester losses have been candidates for history-indicated prophylactic cerclage when the mechanical strength of the tissue is presumed insufficient to resist protrusion of the uterine contents. Cervical performance reflects the tissue's stromal characteristics to limit deformation forces until the appropriately timed onset of labor as well as the capability of the endocervical canal to resist migration of ascending microorganisms or other inflammatory mediators.

Unfortunately if this surgical intervention is limited to women with multiple second-trimester pregnancy losses as originally advanced by Shirodkar, few patients would benefit from a cerclage procedure. However, overly enthusiastic application of this surgical approach has led to unnecessary intervention and an inability to positively define its benefit. After several randomized trials have appropriately raised concern regarding the value of this surgical intervention to prevent preterm birth, evidence has affirmed the concept that contemporaneous findings in the index pregnancy can provide an evidence-based indication for cervical cerclage.

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Table 15-1 Risk Factors for Cervical Insufficiency

- History of spontaneous midtrimester loss
- History of spontaneous preterm birth
- Uterine malformations
  - Mullerian anomalies
  - In utero diethylstilbestrol (DES) exposure
- Cervical trauma
  - Lacerations
  - Conization/LEEP
- Biochemical deficiency (e.g., Ehlers-Danlos syndrome)
  - Decreased elastin, relaxin, collagen

Case 1: Cervical Cerclage

A 28-year-old G3P2 is 20 weeks' gestation. Her obstetric history is remarkable for a first pregnancy complicated by cervical insufficiency and intrauterine fetal demise. Cervical surveillance was performed in her second pregnancy and cerclage was offered when a transvaginal sonogram demonstrated an intact length of 2 cm after the application of fundal pressure for 20 seconds. Funneling to the level of the cerclage was seen after placement of the suture and she delivered preterm at 35 weeks' gestation. A prophylactic intervention was offered for her third pregnancy.

When performed in well-selected patients with premature cervical shortening (Fig. 15-1) and a history of preterm birth, cervical cerclage can improve outcomes. Following insertion of cervical cerclage, the cervix resumes normal length (Fig. 15-2). Cervical cerclage has been shown to improve pregnancy outcomes in a well-designed phase III randomized trial. This trial was performed by the Maternal Fetal Medicine Unit of the National Institutes of Health (NIH) and had as its source population women undergoing cervical surveillance due to a history of prior preterm birth less than 34 weeks' gestation. From this population of approximately 1000 women, the cohort with a cervical length less than 25 mm was offered randomization of surgical intervention or standard management (n = 302 women). Owen and colleagues documented that in the subpopulation of those with the shortest cervical lengths (<15 mm), cerclage significantly lowered the rate of preterm birth less than 35 weeks' gestation, adjusted odds ratio 0.23 (0.08 to 0.66), which was the primary outcome. No other medical or surgical intervention for the prevention of preterm birth tested in a phase III trial has demonstrated such a remarkable benefit in either the study population or a subpopulation.

Despite such a remarkable finding in this subpopulation, when the intervention was assessed in all randomized cases with a cervical length less than 25 mm and a history of preterm birth, only a nonsignificant difference in the rate of early preterm birth was observed—32% versus 42%. An interaction was identified between the length of the cervix and the efficacy of the procedure for the primary outcome. Yet other secondary outcomes were significantly improved at the less than 25 mm cutoff, most important, perinatal death rates, potentially justifying the intervention at the 15- to 24-mm range.

The optimal surgical methodology for cervical cerclage also has been questioned and debated. The two most commonly used procedures, the McDonald and the Shirodkar cerclage, have been compared by several retrospective studies without identification of clear superiority. However, such comparisons are remarkably limited by sample size and a tendency not to investigate the clinical circumstance, which would allow differentiation in method to become discernible, and prospective trials are lacking. Given the absence of clear superiority, surgeons have justified their approach based on their comfort or training for the operation. However, several basic questions need to be asked. Is a Shirodkar cerclage with its need for more extensive cervical dissection the optimal approach for a woman with profound premature cervical shortening, marked thinning of the cervical stroma, and minimal substrate for dissection? Intuitively, the answer is no. Shirodkar developed his technique as an elective procedure prior to the onset of marked cervical change. In contrast, should women undergo a McDonald cerclage with minimal intravaginal cervix due to prior cervical procedures? Again, intuitively, the answer is no. No single procedure is optimal for all circumstances, and operators should be familiar with an array of procedures to best care for patients or make referrals to those with such understanding.

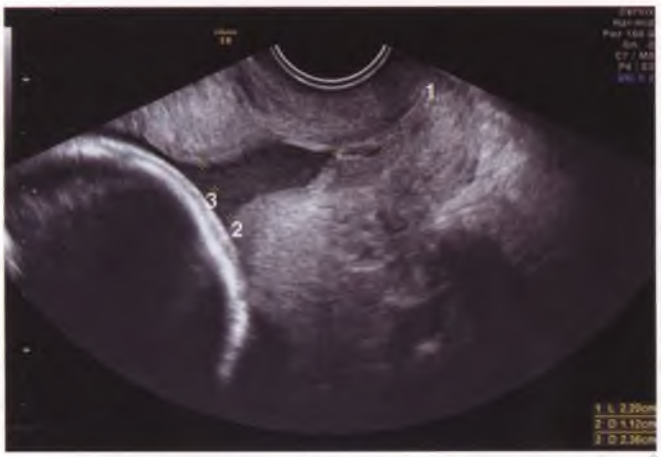

Figure 15-1 Ultrasound of the cervix demonstrating shortening with funneling.

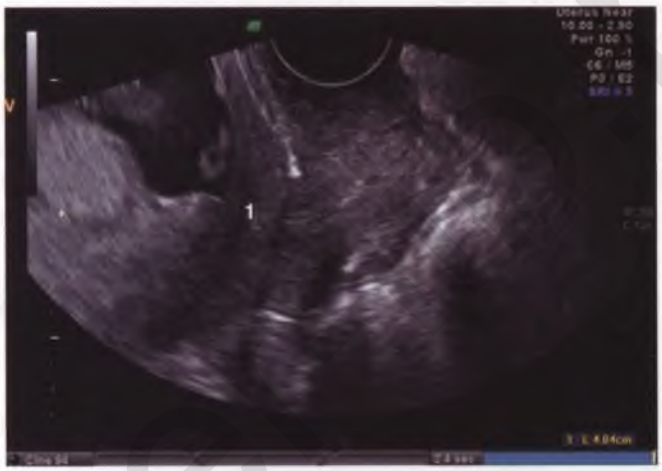

Figure 15-2 Ultrasound of the cervix after cerclage placement showing suture in place with cervical lengths becoming normal.

Indications for Cervical Cerclage

History-Indicated Cerclage

Cerclage is performed at approximately 12 to 14 weeks' gestation for a history of multiple prior previable deliveries (original articles justified intervention with three or more such losses) characterized by painless cervical change. Well performed obstetrical history may allow for identification of candidates for the intervention compared with objective testing; however, generalizing the findings from trials that use subjective assessments alone as an indication for therapy may be limited due to confounding presence in other health care systems. Given the rarity of remarkable cervical change occurring prior to 14 weeks, these interventions have been appropriately described as prophylactic procedures.

Physical Exam–Indicated Cerclage

This cerclage is indicated at a previable gestational age regardless of obstetric history for women with visible fetal membranes and by necessity, some cervical dilation so long as chorioamnionitis is not identified. Observational studies and randomized trials have identified a reduction in mortality with cerclage in this setting. Amniocentesis with amnioreduction has been justified in this

circumstance for both diagnostic and therapeutic purposes. Of note, intra-amniotic inflammation is not uncommonly observed in this population but intra-amniotic microbial seeding is less frequent. Intra-amniotic infection is a contraindication for proceeding with this surgical intervention. Given the exposure of the membranes to the external environment, these procedures have been termed as emergency or rescue operations. Although this terminology appropriately describes the severity of the circumstance, such terms should be reserved to an intact cervical length of near zero with visible membranes.

Ultrasound-Indicated Cerclage

In women with a history of preterm birth, transvaginal sonographic surveillance of cervical length can reduce the likelihood for recurrent preterm birth. Both medical and surgical interventions may be indicated for this population depending on the degree of premature cervical shortening. The exact cervical length cutoff to optimize outcomes and provide an indication for when to proceed with cerclage is unknown, but evidence suggests a benefit at less than 25 mm. Given that a treatment is being indicated for an ongoing depiction of cervical deformation with ultrasound-indicated cerclage, these procedures have been appropriately termed therapeutic cerclage.

Surgical Techniques of Cervical Cerclage

McDonald Cerclage

The McDonald cerclage was not the first procedure described to address the issue of cervical insufficiency; however, it is clearly the simplest intervention. The patient is positioned in dorsolithotomy and a weighted speculum is used to expose the cervix. The anterior and posterior lips of the cervix are grasped with sponge forceps or a tenaculum. The first point of introduction of the cerclage suture is important because the goal is to maximize postoperative cervical length, but not to enter the bladder, which can descend on the upper intravaginal portion of the cervix. The change in mucosal character from the smooth cervical surface to the folds of the vagina has been used as a safe demarcation site for initial placement of the stitch. However, a slightly more aggressive approach in most patients will place the first bite of the cerclage 3 to 5 mm distal to this interface depending on the individual anatomy.

Most operators use a 5-mm Mersilene tape (Ethicon, Inc., Somerville, NJ) and bury the material into the cervical stroma starting at the 12 o'clock position carrying it counterclockwise to the 10 o'clock position. Mersilene is the preferred material in the opinion of the author because it has a tendency to tear through the cervical stroma either at the time of cinching the knot or with the unfortunate complication of preterm labor. Using a pursestring-type stitch, the cervix is circumnavigated until the 12 o'clock position is again reached. Avoiding exit sites at the 9 o'clock and 3 o'clock positions is advantageous to avoid the cervical vasculature and unnecessary excess bleeding. The most important, and perhaps the most difficult, aspect of the operation is to maintain an optimal distance from the external os as the cervix is circumnavigated. The tendency, which should be avoided, is to lose residual cervical length particularly as the suture material is placed in and out of the posterior lip. The intravaginal segment of the posterior lip is typically shorter than the anterior lip.

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In addition, the redundancy of the mucosa is greater in the posterior fossa in part to accommodate the need for a change in cervical position that occurs with labor. Therefore, care is necessary to ensure a sufficient depth to the needle pass is obtained to reach the cervical stroma. Despite the apparent simplicity of the technique, performing the intervention well does require a thoughtful approach. See Figures 15-1 to 15-4 for ultrasound pictures of the cervix before and after placement of McDonald cerclage. (See the DVD for video demonstration of placement of McDonald cervical cerclage.)

The procedure illustrated in the video is performed in a patient with a history of two preterm births. In this example, note that the Mersilene tape is reintroduced into the cervical stroma adjacent the exit point, so that the cervical mucosa can more easily heal over the mucosal defects, and thereby minimize access points for vaginal flora to enter the cervical stroma potentially soliciting an inflammatory response. Furthermore, the needle must be passed into the cervical stroma and not merely in the loose connective tissue plane between the cervical mucosa and cervical stroma. A cerclage that occupies the space between the cervical stroma and mucosa will regrettably "slide off" the end of the cervix when intracervical pressure increases.

Several complications from this intervention are possible and should be avoided if possible. The cerclage must not traverse the entire width of the cervical stroma and enter the endocervical canal. This complication enhances migration of microorganisms into the uterine cavity. If premature cervical

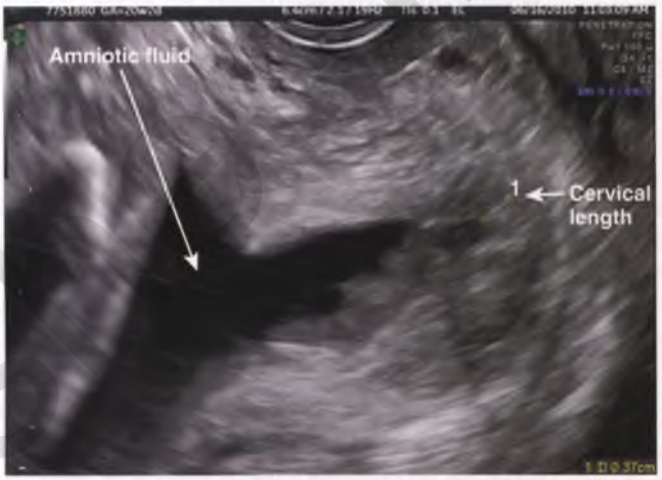

Figure 15-3 Ultrasound of the cervix revealing an extremely short cervix measuring 3.7 mm with funneling.

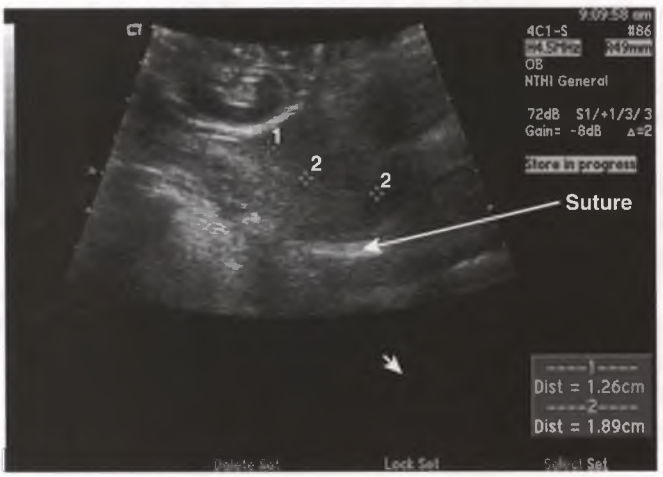

Figure 15-4 Ultrasound of the cervix following cerclage showing the suture in place.

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shortening is already present, such a full-thickness needle-pass may also result in iatrogenic premature rupture of membranes (PROM). The delicate balance to reach a sufficient depth to ensure the suture is within the stroma versus too deep a stitch, which causes ruptured membranes, is a skill developed only with experience. Those with minimal experience should err on a shallower placement with compensation, if needed, in tying the knot more tightly. Remarkable tension is needed for tying any cerclage when Mersilene tape is used; however, undue force should not be used if the operator prefers monofilaments because this will slice the cervical tissue. After tying, the free ends of the tape are cut with sufficient tail to allow for easy identification at a future speculum examination to ease removal.

Several surgical adjuvants have been proposed to aid cerclage placement particularly when performing a rescue cerclage. Amnioreduction has been discussed. Other adjuvants include a Foley catheter balloon or cervical ripening balloon placed against the membranes and ultimately guided up into the endocervical canal into the lower uterine segment. This technique when combined with multiple ring forceps grasping the cervical edges may allow for membrane replacement and minimize damage to the chorion. Damage to the chorion, either prior to surgery by overdistention of the membranes into the distal vagina, or intraoperatively, by manipulation of the membrane during surgery, can be visualized as membrane splitting. Any such defect of the chorion is associated with an extremely poor prognosis. It is therefore important to inspect the membranes for evidence of structural integrity prior to proceeding with intrauterine replacement of prolapsed membranes and care should be used during any manipulation of the membranes.

Another adjuvant technique is bladder filling sufficient to "push" the fundus away from the cervix. The author has seen minimal success with such a strategy, and care should be taken not to overfill the bladder to cause bladder rupture/trauma. Finally, placing several gauze pads in the posterior fornix after the suture has been placed and pulling the cervix over this vaginal "ball valve" creates some ascent of the membranes prior to tying the cerclage suture and may reduce the frequency with which membranes prolapse past the level of the cerclage.

The postoperative management of the patient undergoing cerclage has been questioned. Should individuals undergoing this intervention undergo postoperative sonographic surveillance and what is the utility of this information? Migration of the membranes to the level of the cerclage has been associated with an increased risk for earlier preterm birth. Those patients in whom the membranes have reached or prolapsed past the level of the cerclage may be candidates for increased bedrest, but this adjuvant intervention has not been shown to improve outcomes by prospective trials. Other medicinal adjuvants such as the use of progestins, antibiotics, or antiinflammatory agents have not been adequately investigated by larger observational or randomized trials in these women at highest risk. Evidence from secondary analyses in those undergoing the NIH cerclage trial did not demonstrate benefit for adjuvant progestin treatment, but patient selection was not stratified by residual postoperative cervical status, and further evaluation is needed.

Removal of a McDonald cerclage is relatively easy as traction is placed on the intravaginal remnants of the suture and the loop is cut. This intervention can be performed in the office setting. The most serious longer-term complications of cervical cerclage result from cervical trauma when the cerclage suture is not removed in a timely manner despite the onset of labor. These complications include large cervical tears and fistula formation.

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Modified Shirodkar Cerclage

The Shirodkar procedure was developed for women with the worst reproductive history. The Shirodkar procedure has a theoretic advantage because this methodology results in direct visualization of the cervical stroma, which is the target tissue, and the lack of exposure of suture material to vaginal flora. However, to achieve these ends the procedure requires far more cervical dissection than a McDonald approach.

For the Shirodkar cerclage, the initial surgical steps are with a scalpel. After appropriate positioning and prepping, a semilunar incision is made across the anterior cervical mucosa just proximal to the bladder. This anterior mucosa is dissected off the underlying stroma, and the bladder is pushed intra-abdominally. Displacing the bladder away from the external os provides the potential to increase the residual cervical length after the procedure as the knot can be placed higher. Furthermore, this dissection strategy also makes an intravaginal surgical approach feasible for women who have little remaining intravaginal cervix due to prior cervical surgery. When the cervix is flush with the apex of the vagina, the only options are for intra-abdominal approach or the use of this type of dissection. Unfortunately, if an imprecise anterior cervical incision is made or if prior surgery creates a problem with developing the surgical plane, the bladder can be entered. If this complication is encountered, it is most frequently repaired by a transvaginal two-layered closure and prolonged Foley placement. Once the anterior incision is performed, attention is turned to a similarly placed posterior semilunar incision that allows dissection of the posterior mucosa off the posterior cervical stroma. The result is a partially denuded cervix with exposure of the cervical stroma both anteriorly and posteriorly, which gives the operator direct access to the site of the tissue deficiency.

With the use of a curved Allis clamp, the lateral margins of the anterior and posterior cervical semilunar incisions are approximated on the right. A Mersilene tape is passed anterior to posterior through the cervical stroma medial to the clamp. I then prefer to take a small bite through the posterior cervical stroma as the needle is passed from the right lateral margin of the posterior incision to the opposite side to ensure the material is well anchored in stroma. In a similar manner, the Allis clamp is used to approximate the anterior and posterior cervical incisions on the opposite side. The needle and tape are passed from posterior to anterior through the stroma of the cervix, and again a small bite is made anteriorly to better imbed the suture material into the stroma. The knot is then tied tightly. The cervical incisions are closed with a running suture and the knot is buried beneath the mucosa if the operator desires to stay true to the original surgery.

However, one criticism with the Shirodkar procedure is the difficulty with removing the stitch if a vaginal delivery is pursued because these patients may require a repeat trip to the operating room to dissect free and incise the buried suture. One strategy to minimize this difficulty is to leave the distal tails of Mersilene past the knot exposed to the vagina during closure of the cervical incision allowing removal similar to the McDonald approach.

Despite the best surgical attempts, the transvaginal approach for cerclage placement has inherent limitations given the inability to access the upper cervix. For women with little intravaginal cervix, for those with extensive cervical lacerations, or those who have previously failed a transvaginal approach, an abdominal cerclage may be preferred.

Abdominal Cerclage

In 1965 Benson and Durfee were the first to describe a novel approach using an abdominal placement of a cervicoisthmic cerclage. The abdominal method was ideally suited for patients with an extremely shortened exocervix in which the transvaginal approach was rendered technically impossible or for those with prior unsuccessful transvaginal procedures. Since the introduction of transabdominal cerclage, the reported fetal survival rate has improved from 21% to 89% in this select population.

Case 2: Abdominal Cerclage

A 23-year-old G3P0-2-0-0 was referred to our perinatal unit at 10 weeks' gestation. Her first pregnancy resulted in a 21-week loss secondary to cervical insufficiency. She underwent a prophylactic vaginal cerclage during her second pregnancy in the early second trimester. This pregnancy ended in spontaneous labor and subsequent delivery at 25 weeks with neonatal demise shortly thereafter. For the third pregnancy she underwent early evaluation of the cervix with transvaginal ultrasound. The cervical length was 4 cm; however, on speculum examination, evidence of cervical trauma and concomitant laceration was noted and the recommendation of an abdominal procedure was made. She was counseled on the options of traditional laparotomy and laparoscopic and robotic-assisted laparoscopic techniques. She elected to pursue robotic-assisted laparoscopic surgical placement.

She underwent the procedure at 13 weeks' gestation. Fetal heart tones were confirmed both preoperatively and postoperatively. The operative time was 149 minutes and estimated surgical blood loss was 25 mL. She was discharged on postoperative day 1 without medication. The antepartum course remained event free with stable serial transvaginal ultrasounds with a cervical length of more than 3 cm and negative fetal fibronectin testing. She underwent cesarean section delivery at 38 weeks' gestation due to spontaneous labor. The male infant weighed 7 lb, 1 oz with Apgar scores of 8 and 9. Inspection of the stitch confirmed proper placement and was left in situ for future pregnancy desires.

Discussion

An abdominal cerclage may be placed as an interval procedure or during pregnancy in the early antepartum period. Interval placement is technically easier, but may lead to increased spontaneous abortion and dysmenorrhea and has the potential to delay or impair conception. Another difficulty encountered with the interval placement is in the case of fetal demise or elective termination. The stitch may be occlusive to D&E procedures and may require removal by reexploration, laparoscopy, or by means of a posterior colpotomy.

Techniques of Abdominal Cerclage

Traditionally, the approach to an abdominal cerclage requires a laparotomy and is typically executed between 11 and 13 weeks. The uterus is exposed and a bladder flap is sharply dissected inferiorly to a level below the internal os. The uterus is gently retracted cephalad by an assistant while the surgeon sweeps the uterine vessels lateral. The suture typically used is a 5-mm Mersilene tape (Ethicon, Inc., Somerville, NJ) with either a double or single attached needle placed through an avascular space of the posterior leaf of the broad ligament at the level of the lower uterine segment and cervix—the uterine isthmus. This location allows for secure placement of the suture bounded by the uterus above and the uterosacral ligaments below (Fig. 15-5). The suture can be passed in either the anterior to posterior direction and tied posteriorly or in the posterior to anterior direction and tied anteriorly. Either direction is acceptable. It may be technically easier if the knot is tied on the anterior side. This configuration, however, may lead to a potential complication of bladder erosion. The original description calls for the suture to be tied in a single throw-knot with the free ends of the cut suture approximated and affixed to the band with a nonabsorbable

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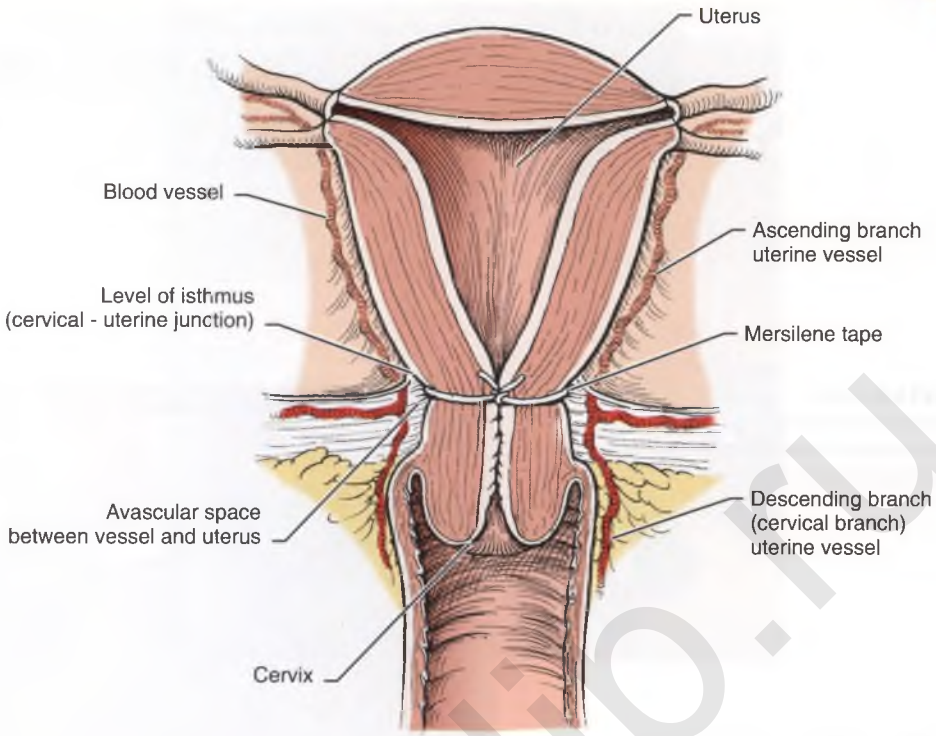

Figure 15-5 Placement of abdominal cerclage. Surgical placement of circumferential Mersilene tape around the uterine isthmus and median to uterine vessels. Knot is tied anteriorly. Posterior knot placement is also acceptable. (From Gabbe SG, Niebyl JR, Simpson JL: Obstetrics: Normal and Problem Pregnancies, 5th ed. Philadelphia, Elsevier, 2007, p 660.)

suture such as silk or fine Prolene. An alternative technique described by Ludmir and Owen consists of passing a right-angle clamp through the avascular space and through the posterior leaf of the broad ligament grasping the suture and pulling it anteriorly. The same is done on the opposite side and the suture is tied anteriorly. The purported advantage with this approach is the absence of a sharp needle.

The laparotomy approach carries significant morbidity and it requires two major laparotomy procedures each with a prolonged recovery period. Recent literature has described the use of a laparoscopic approach. Although this approach may improve the recovery period, it requires significant surgical skill and experience in comparison to a traditional open approach. The potential significant benefits to the patient are presented by comparison in Table 15-2. Several techniques for a laparoscopic approach have been described and are largely based on the previously described abdominal approach. Under general anesthesia the patient is placed in the dorsal lithotomy position. Pneumoperitoneum is established either by an open laparoscopic approach or with a Veress needle. Intraabdominal pressure is maintained at ≤12 mm Hg. The patient is placed in Trendelenburg and accessory port sites are placed under direct visualization. Typically two 5-mm lower-quadrant ports and a 10- or 12-mm suprapubic port are sufficient. Vaginal manipulation varies by surgeon and it includes no manipulation to a modified atraumatic gauze ball or gauze on a ring forceps.

The vesicouterine peritoneum is identified and dissected transversely as in the traditional approach. Both harmonic shears and laparoscopic scissors have been described for this purpose. Dissection is carried down below the level of the uterine isthmus making sure to expose the uterine vessels. Cho and colleagues described dissecting the posterior leaf of the broad ligament to gain better visualization of the uterine arteries. Ghomi's description suggests this dissection in not necessary but makes an effort to drive the needle a few

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Table 15-2 Postoperative Comparison of Laparotomy Versus Laparoscopic Surgery

	Laparotomy	Laparoscopic
Hospitalization	3-7 days	Same day or 23-hr observation
Postoperative pain	Moderate-severe	Minimal
Recovery time	4-6 wk	2 wk
Adhesions	±	Minimal

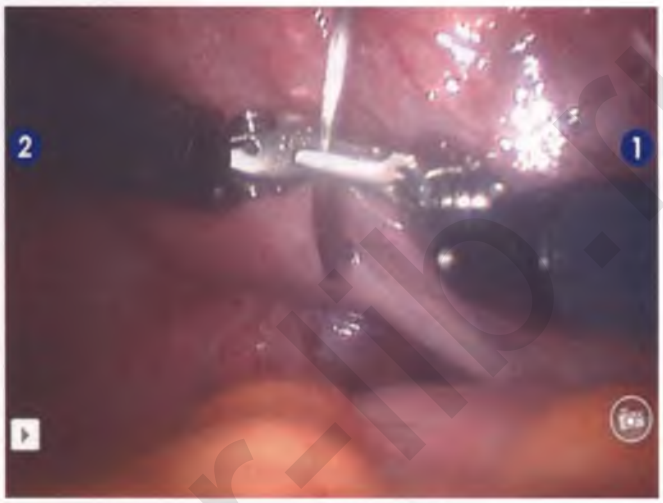

Figure 15-6 Needle placement medial to the uterine vessels and superior to the uterosacral ligament.

millimeters into the cervical tissue to ensure freedom from the uterine arteries. We agree with this placement and furthermore believe it provides for better stabilization of the suture material.

The 5-mm Mersilene ligature is typically used either by partially straightening the prepackaged needle at the curve of the needle or by attaching a free needle that will accommodate the port being used for the suture material. The ligature is then placed medial to the vessels and lateral to the uterus at the level of the uterine isthmus and tied anteriorly (Fig. 15-6). This can be accomplished by passing the needle first anterior to posterior, bringing it around posteriorly and passing it posterior to anterior or by using a double needle suture and passing the needle anteriorly both times. Both intracorporeal and extracorporeal knot tying have been described. An alternative technique described by Lesser and associates consists of creating a window in the broad ligament bilaterally at the level of the internal os and feeding the Mersilene tape through each side, then tied anteriorly.

One of the greatest challenges to this approach is the manipulation of the gravid uterus. Instruments such as the Diamond-Flex triangular retractor (Snowden Pencer, Atlanta, GA) and a large-shaped fan retractor have been reported as useful. A single gauze ball on ring forceps and digital vaginal manipulation have also been used for aid in the dissection.

The technically easier interval laparoscopic procedure is performed in a similar fashion. The nonpregnant uterus is easily manipulated and less vascular. Most described methods involve using a sharp instrument such as an Endo Close device (Auto Suture Company, Norwalk, CT) to pierce the avascular space alongside the isthmus and then pull the suture through anteriorly. The firm

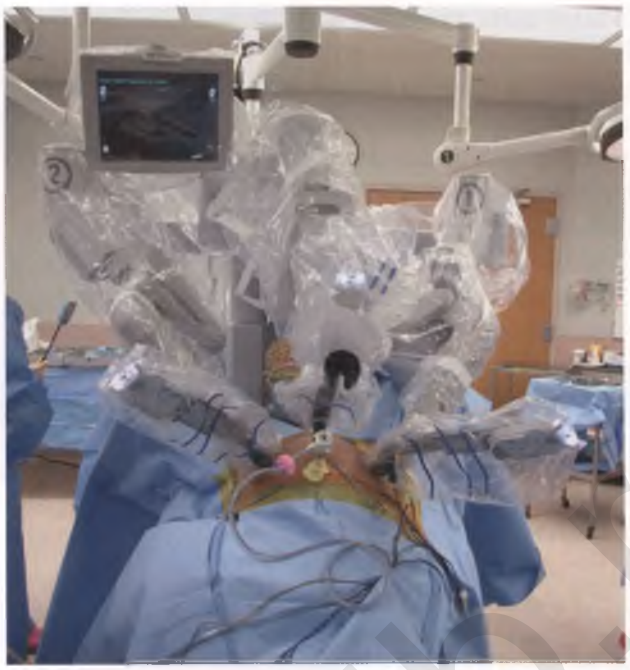

Figure 15-7 Anterior view of the daVinci S surgical system (Intuitive Surgical) after docking.

nature of the nonpregnant uterus allows for demarcation of the uterine isthmus in contrast to the difficultly in delineating the isthmus in the softened pregnant uterus with multiple vessels.

All laparoscopic approaches require a highly skilled laparoscopic surgeon. Traditional laparoscopic surgery is limited by loss of depth perception and absence of dexterity. This makes procedures performed during pregnancy technically difficult and nearly impossible in some patients, such as those who are obese or who had multiple previous abdominal surgeries. Recently robotic-assisted laparoscopic surgery (RALS) has been used to perform an abdominal cerclage in the interval and pregnant state. RALS combines three-dimensional vision and more than 500 degrees of motion returning the open feeling to a laparoscopic case.

Technically these surgeries are performed similar to traditional laparoscopic placement. The open technique is used to place the first laparoscopic trocar approximately 8 cm above the umbilicus, which is then used for the zero-degree endoscope. The abdominal cavity is then insufflated with CO_2 gas to an intraabdominal pressure of 12 mm Hg. Four other trocar sites are then chosen and placed with laparoscopic visualization. Two 8-mm ports are typically placed 10 cm lateral to the umbilicus on the left and the right and used for the robotic arms. Two 10-mm accessory ports are placed to the left and right of the camera port. The laparoscopic robot system is then docked to the patient (Fig. 15-7). The Maryland bipolar forceps and monopolar curved scissors, both Endo Wrist instruments (Intuitive Surgical, Inc, Sunnyvale, CA), are used in the robotic arms. Retractors as described in the laparoscopic section are used in the accessory ports.

In order to gain adequate exposure in the pregnant uterus, the round ligaments may be sacrificed bilaterally. A bladder flap is then created and taken down to the level below the internal os. Using two laparoscopic fans, the uterus is gently mobilized in either direction, so that the 5-mm Mersilene stitch can be thrown at the level of the internal os. We typically place the stitch first posterior to anterior through the paracervical tissue medial to the vessels, wrapping it around the cervix to the opposite side, where it is thrown anterior to posterior

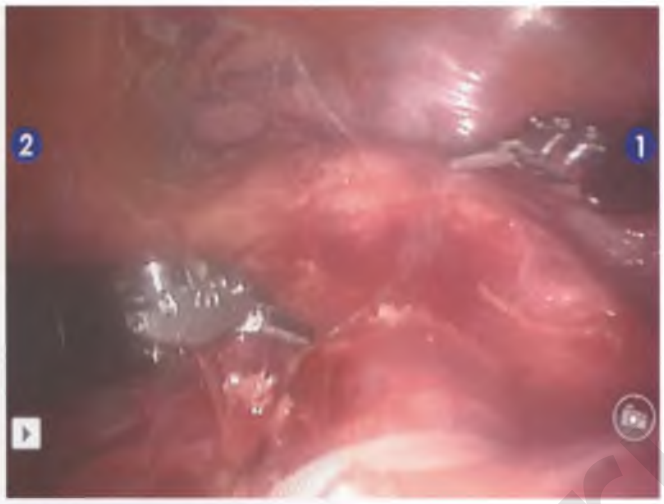

Figure 15-8 The stitch is visualized at the cervicoisthmic junction.

Figure 15-9 The knot is firmly secured in place with the robotic arms while the fan retractor is used to elevate the uterus.

(Fig. 15-8). The suture is tied with the robotic arms in the midline on the posterior aspect of the uterus (Fig. 15-9).

The added visual advantages may provide more precision and accuracy for needle placement and the strength of the robotic arms allow for firm secure intracorporeal knot tying as opposed to the traditional laparoscopy. RALS is a potentially emerging procedure limited by the availability of a robotic system and appropriately trained surgeons. (See the DVD for video demonstration of robotic-assisted laparoscopic abdominal cerclage.)

Complications

The most significant intraoperative complication of abdominal cerclage placement is rupture of membranes. Manipulation of the pregnant uterus may also hypothetically stimulate contractions, leading to spontaneous abortion. Excellent visualization as well as minimal uterine manipulation may prevent these complications. The RALS approach is uniquely positioned to assist in minimizing these complications. Suture displacement is less of a concern with an abdominal cerclage as the suture is bounded by the ligament below and the

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uterus above. Another concern is the management after fetal demise or pregnancy complication. If the stitch has adequate placement and the patient desires future childbearing, a cesarean delivery with stitch preservation may be followed. If no further childbearing is desired, the stitch may be removed by traditional laparotomy with concomitant caesarean delivery and sterilization. If the stitch has suspect placement one may alternatively use techniques such as laparoscopic removal or a posterior colpotomy to facilitate removal followed by vaginal delivery of the conception. Tocolytics and antibiotic prophylaxis are not routinely used for postoperative care.

Summary

Cervical cerclage can be performed by multiple methodologies. Sufficient evidence exists to state that the procedure can advance gestational age and reduce mortality. The procedure should be reserved to those with a history of prior preterm birth and premature cervical shortening, those with prior preterm birth and evidence of persistent cervical pathology such as extensive cervical laceration, those with visible membranes in the midtrimester without intrauterine infection regardless of obstetric history, or women with a history of multiple losses characterized by painless cervical dilation. The McDonald cerclage is the simplest intervention to perform and as such, may enhance patient safety particularly in patients with marked cervical shortening, but such a hypothesis is not proven by randomized trials in this population. The Shirodkar cerclage has theoretic advantages for enhancing placement of the supportive suture into the stroma due to direct visualization. Surgeons should be familiar with each of these techniques in order to optimize the approach.

There continues to be a place in modern obstetrics for an abdominal approach to cerclage for cervical insufficiency. Although randomized clinical trials have not validated any of these approaches, the laparoscopic approach with less morbidity has been widely accepted when performed by an experienced laparoscopist. We speculate with continued experience and evolving technology, the RALS approach will be the procedure of choice for abdominal cerclage placement for both interval and current pregnancies at 11 to 13 weeks, with traditional laparotomy being reserved for later placements.

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16

Techniques and Complications of Planned and Emergency Cesarean Section

Michael Karram M.D.
Baha M. Sibai M.D.

Video Clips on DVD

16-1 Techniques for Performing Cesarean Section with Discussion of Management of Potential Complications

Cesarean delivery is the most common major surgical procedure performed on women worldwide. The rate of cesarean section continues to rise due to a multitude of causes. As the rate increases, the number of repeat cesarean sections will also increase due to the decreasing rate of vaginal birth after cesarean (VBAC) section. In the United States, the cesarean section rate ranges between 25% to 35%. Most likely this rate will continue to rise due to the changes in patient demographics as well as the changes in obstetrical practice (Table 16-1). Many patients ask for primary elective cesarean section and obstetricians are not being trained to perform operative deliveries, breech deliveries, and vaginal deliveries of multiple gestation. As more and more cesarean sections are performed, we will undoubtedly see an increase in the number of surgical complications such as hemorrhage, infection, and bowel and bladder injuries. These complications are more likely to occur in the presence of one or more of the risk factors listed in Table 16-2. This chapter reviews the appropriate techniques

Case 1: Planned Cesarean Section

The patient is a 32-year-old G2P1 who presents at full term (39 weeks). She had a previous cesarean section at 32 weeks for severe pregnancy-induced hypertension. She has had an uncomplicated pregnancy and desires a repeat cesarean section. The cesarean section was performed without complications. The patient experienced a normal postoperative course and is discharged on her third postoperative day.

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Table 16-1 Factors Associated with Increased Cesarean Section

- Advanced maternal age
- Obesity
- Planned induction of labor
- Preexisting medical disorders
- Increased rates of obstetric disorders
  - Gestational diabetes
  - Fetal macrosomia
  - Gestational hypertension-preeclampsia
  - Placenta previa/abruption
- Multifetal gestation
- Planned cesarean section
  - Avoiding trial of labor with previous cesarean section
  - Maternal request
  - Multiple cesarean section scars

Table 16-2 Risk Factors for Adverse Maternal Outcome During or After Cesarean Section

- Morbid obesity
- Emergency or crash cesarean delivery
- Cesarean section after prolonged induction
- Cesarean section during second stage of labor
  - Prolonged second stage
  - Prolonged pushing efforts
  - Failed instrumental delivery
- Abnormal placentation
  - Central previa/accreta
  - Abruption with disseminated intravascular coagulopathy (DIC)
- Previous abdominal/uterine surgery
  - Multiple cesarean sections
  - Classical or T-incision
- Multifetal gestation
- HELLP (*h*emolysis, *e*levated *l*iver enzymes and *l*ow *p*latelet count) syndrome or acute fatty liver of pregnancy
- Receiving low-molecular-weight heparin

that should be used when performing an elective or emergency cesarean section, with emphasis placed on how to avoid and manage complications related to this procedure.

Technique of Repeat Cesarean Section

The most common incisions used for repeat cesarean section are transverse incisions (Pfannenstiel, Maylard, or Cherney). Vertical incisions are usually used in emergency situations for rapid entry into the abdominal cavity. A Pfannenstiel incision was used in this patient based on the fact that this same incision was used on her previous cesarean section. Once the incision was made, the subcutaneous tissue was opened and bleeding was cauterized with suture ties or electrocautery. The fascial layer was then opened and extended with the scissors or knife. It is then reflected cephalad and posterior. The peritoneum is entered and the gravid uterus is visualized and inspected for any rotation or abnormal anatomy. The visceral peritoneum or bladder flap is taken off the lower uterine segment. The lower uterine segment is incised transversally and extended laterally either bluntly or sharply with scissors. The amniotic sac is ruptured and the fetus is delivered in the vertex presentation. Suctioning of the

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nasopharynx is performed followed by cord clamping and cutting. The placenta is delivered by gentle traction or manually. The uterus is exteriorized through the anterior abdominal wall, and the uterine cavity is explored for any placental remnants. The lower uterine segment is closed in two layers using absorbable suture. The uterus is repositioned back into the abdominal cavity and the gutters are cleaned and irrigated; and when the sponge and needle count is correct, the abdomen is closed. The fascia is closed with an absorbable suture. The subcutaneous tissue is reapproximated and the skin is closed. (See the DVD for video demonstration of a repeat cesarean section.)

Case 2: Emergency Cesarean Section

The patient is a 39-year-old G4P3 with two previous cesarean sections and known low-lying placenta (not a complete placenta previa). The patient declined genetic testing and had an uncomplicated prenatal course. The only problem the patient presented with during her pregnancy was a bleeding episode in her early second trimester. This episode was resolved with conservative management. She presented to labor and delivery at 37 weeks having active contractions and some vaginal bleeding with a nonreassuring fetal heart rate tracing. The patient was taken back for an emergency cesarean section. On opening the abdominal cavity through a previous transverse incision it became obvious that the bladder was attached very high on the lower and upper segments of the uterus. The bladder was taken down sharply to minimize the chance of bladder injury. Once the bladder was completely mobilized and the lower uterine segment was exposed, the lower uterine segment was incised and extended manually. The fetus was delivered by footling breech extraction and the placenta was noted to be low lying and was delivered in the usual fashion. The uterus was exteriorized and explored for any placental remnants. At this point it was noted that there was an extreme amount of bleeding and that the uterus was not contracting down as quickly as expected. Appropriate procedures to manage intrapartum hemorrhage during the time of cesarean section were instituted. Anesthesia was instructed to start another large-bore IV and give 20 units of oxytocin (Pitocin) at a rapid rate. Uterine massage was performed. Also, intramyometrial injection of vasoconstricting agents such as oxytocin or Hemabate may also be used at this point (see the DVD for demonstration of appropriate technique for uterine massage and intramyometrial injection of a vasoconstricting agent). On examining the lower uterine segment it was obvious that a lateral extension had occurred on the patient's left side extending very close to the uterine vessels (Fig. 16-1). Multiple sutures were placed to control the bleeding. Once the bleeding was under control there was concern about possible ureteral obstruction secondary to one of the suture ligations. At this point, the safest and most efficient way to ensure ureteral patency or diagnose ureteral compromise was to go to the extraperitoneal portion of the bladder and make a high advertent cystotomy allowing direct visualization of the ureteral orifice. (See Chapter 17 for the technique for opening and closing the bladder.) The anesthesiologist was instructed to give IV indigo carmine and once the bladder was opened, approximately 4 to 6 minutes later it was obvious that there was spillage of dye from both ureteral orifices, thus assuring no ureteral compromise. The bladder was then closed in two layers using an absorbable 3-0 suture. The first layer was through and through with the second layer being an imbricating stitch (see Chapter 17). At this time it was noted that the uterus was again becoming boggy even after giving IV and intramyometrial vasoconstricting agents. Anesthesia was instructed to send blood for a coagulation profile and it was decided to perform a B-Lynch suture (see the DVD for video demonstration of how to perform B-Lynch suture). On failure of the B-Lynch to control the bleeding, an O'Leary stitch was used to ligate the uterine vessels and another stitch to ligate the utero-ovarian vessels (Fig. 16-2). With failure of the these stitches to control the hemorrhage, a bilateral hypogastric artery ligation was performed (refer to the DVD and figures for techniques of O'Leary stitch and utero-ovarian vessel ligation). After all conservative surgical procedures were used in an attempt to control the hemorrhage, the decision was made to perform a cesarean hysterectomy (see Chapter 4 for technique of cesarean hysterectomy).

Discussion

Numerous studies describe the technical aspects of performing a cesarean section. There is some debate concerning the benefits and risks regarding the techniques for cleansing of the skin, type of skin incision (transverse versus midline), separation of the rectus muscle, opening of the peritoneum (bluntly or sharply), whether to develop a bladder flap, type of uterine incision (low transverse or low vertical), and technique of extension of uterine incision (blunt or by scissors). There is also debate about the closure of the various layers after delivery of the infant. The decision should be based on the clinical situation as well as the expertise of the surgeon with a specific technique. The goal is to use the technique that is associated with the least morbidity to both the mother and neonate.

Case 2: Emergency Cesarean Section—cont'd

As with any surgical procedure, the surgeon should use meticulous aseptic technique for entrance into the abdominal cavity, uterine incision, and closure. In addition, protocols should be in existence for universal administration of prophylactic antibiotics (all cases) or therapeutic antibiotics in case of chorioamnionitis, as well as universal methods to reduce the risk of thromboembolism (sequential compression devices, early ambulation, and prophylactic heparin). Moreover, protocols should be developed for early detection, evaluation, and management of patients with suspected intraabdominal bleeding and infection (pulmonary, urinary, uterine, or wound).

A recent review of the literature focusing on randomized trials, analyzing the technical aspects of cesarean section, suggests that the best outcome is achieved by a transverse incision of the skin, blunt dissection of the subcutaneous tissue, omission of development of the bladder flap, blunt extension of the uterine incision, and leaving the peritoneum open. It also suggests closure of the subcutaneous tissue when the thickness is ≥2 cm. The decision regarding the uterine incision should depend on fetal gestational age, status of lower uterine segment, fetal lie (vertex, breech, or transverse), location of the placenta, presence of fibroids, and other factors. In general, low transverse incision is appropriate in most cases; however, a low vertical or fundal incision may be necessary at very early gestational age when the lower segment is not developed, in cases of prolonged rupture of membranes with breech presentation, in cases of total placenta previa (to avoid cutting through the placenta), and in cases of a transverse lie with the back down. If a low-transverse incision is performed, blunt expansion with the fingers of the uterine incision in a cephalocaudal direction is associated with lower blood loss as compared to a transverse extension. If needed, the incision can be extended as a J or U type or even in the form of a reverse T. On rare occasions when the lower segment is very thick without labor, extension of the uterine incision may require the use of a bandage scissors. These latter types of uterine incision are associated with increased rates of uterine rupture in subsequent pregnancies, and require the performance of planned repeat cesarean section for subsequent pregnancies.

There is general agreement that the uterine incision should be closed with a continuous closure of all layers but not including the endometrium. Studies also suggest that closure of the incision in two layers is associated with lower rates of uterine dehiscence or rupture in subsequent pregnancy if a VBAC is elected.

In the discussed cases, it is apparent that a cesarean section can proceed uneventfully or in certain cases it can be associated with serious complications. Therefore, all surgeons should be trained to avoid or minimize these complications and detect them promptly. In case of previous cesarean section and multiple adhesions, the bladder or bowel can be adherent to anterior abdominal wall, peritoneum, or uterine fundus. Therefore, it is important to anticipate these problems while entering the peritoneal cavity. All layers should be identified and incised in a stepwise manner, and all adhesions should be dissected prior to any uterine incision. If the bladder flap is attached high above the lower segment, the flap should be developed to avoid injury to the bladder. In addition, in case of extension of the uterine incision laterally to the uterine vessels or downward toward the cervix, attention should be paid to avoid compromising the ureters with suturing the bleeding vessels. If multiple sutures are needed to control bleeding, bladder and ureteral patency should be ensured prior to closure of the abdomen. The ureters could be transected, ligated, or partially kinked (refer to Chapter 17 for various techniques to identify and manage bladder and ureteral injuries). In some situations, it is important to call for help and consult with a surgeon with more expertise in managing pelvic hemorrhage or urologic injuries.

In the postoperative period, all patients should have close monitoring of vital signs including vaginal bleeding or bleeding of the incision site. If intraabdominal bleeding is suspected, the patient should receive immediate resuscitative measures, and if necessary taken back for immediate reexploration. Indications for relaparotomy after cesarean section are listed in Table 16-3.

In summary, there are several possible ways to perform cesarean section. It is very important that all surgeons be familiar with the indications and limitations of the various techniques of cutting and suturing of the different layers involved. It is also important that the technique used be tailored to the clinical situation.

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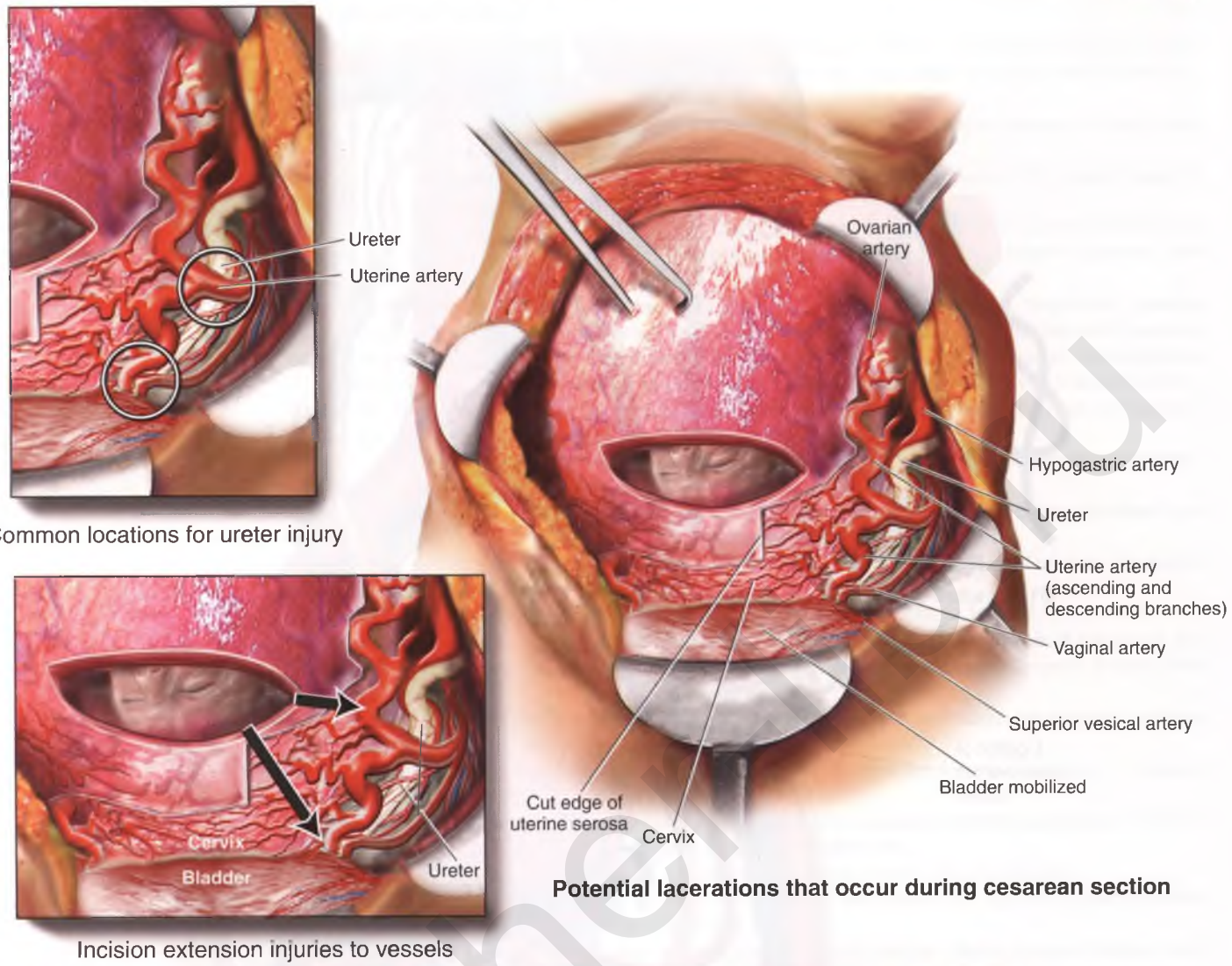

Figure 16-1 Demonstrating anatomy of vessels and sites of laceration at time of cesarean section extending into vessels.

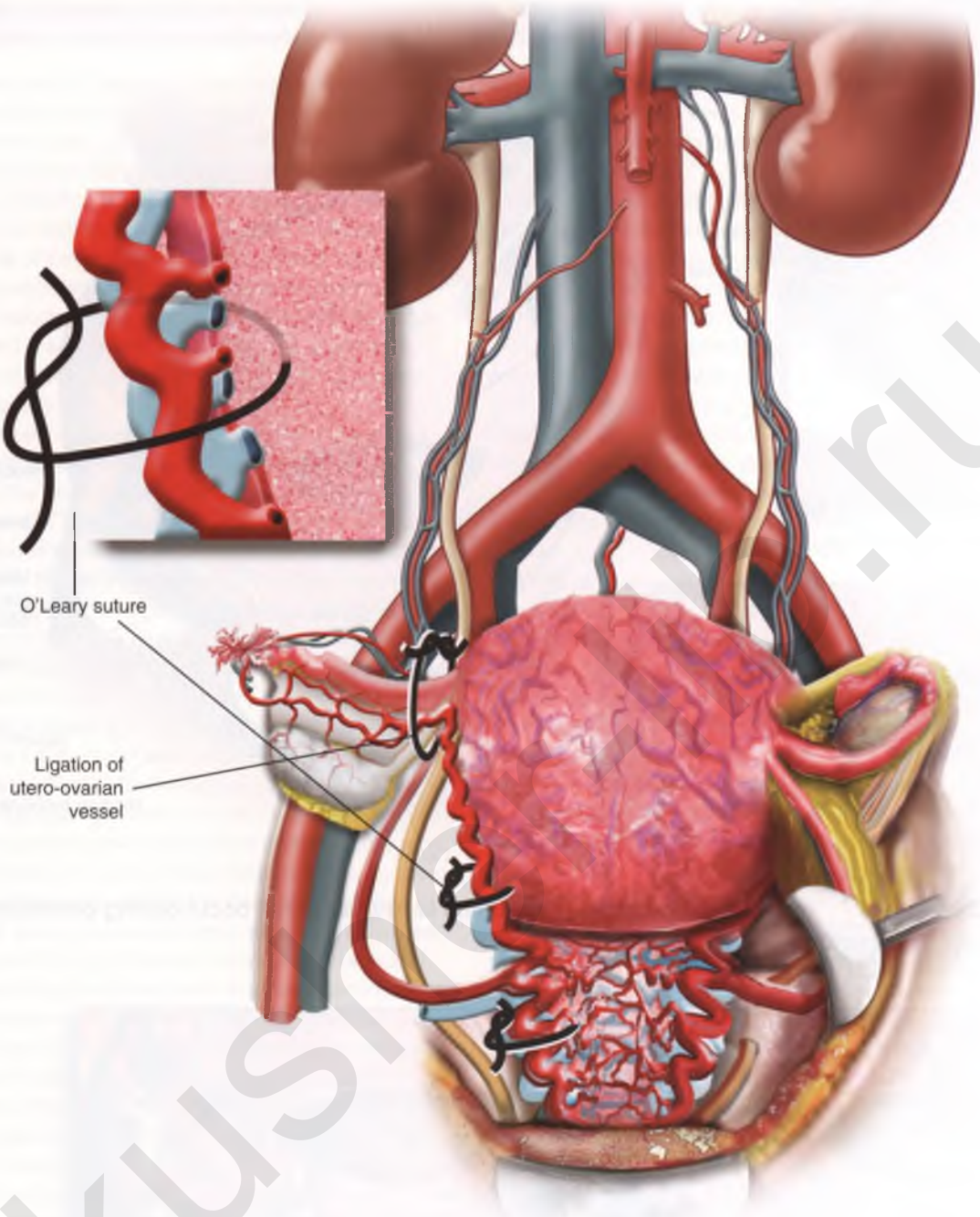

Figure 16-2 Demonstrating ligation of the ascending and descending branches of uterine artery as well as ligation of tubal variant ligament.

Table 16-3 Indications for Relaparotomy After Cesarean Section

- Postpartum hemorrhage
  - Uterine atony
  - Bleeding from uterine incision/lacerations
  - Bleeding from myomectomy site
  - Broad ligament hematoma
- Bladder or ureteral or bowel injury
- Pelvic abscesses
- Fascial dehiscence
- Intraperitoneal bleeding of other etiology
  - DIC
  - Lacerated or ruptured liver or spleen
  - Use of anticoagulant therapy

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17

Avoiding and Managing Lower Urinary Tract Injury During Vaginal and Abdominal Deliveries

Mickey Karram M.D.

Video Clips on DVD

17-1 Cadaveric Dissection Demonstrating Anatomy of the Bladder and Lower Ureter

17-2 How to Perform an Intentional Cystotomy with Visualization and Inspection of the Inside of the Bladder, Passage of Ureteral Stents, and Two-Layered Closure of Cystotomy

Iatrogenic injury to the urinary tract can occur during any vaginal or abdominal surgery. The reported incidence of bladder injury during cesarean delivery ranges from 0.14% to 0.94%. The largest series of 23 bladder injuries reported by Eisenkop and associates in 1982 demonstrated an overall incidence of 0.31% with 0.19% occurring in primary cesarean deliveries and 0.6% occurring in repeat cesarean deliveries.

Nielsen and Hokegard evaluated overall surgical complications in more than 1300 cesarean deliveries. They found most complications occurred during emergency cesarean delivery and that six factors were associated with increased complications. These included station of the presenting part before surgery, labor before surgery, low gestational age of less than 32 weeks, rupture of chorionic membranes prior to surgery, prior cesarean delivery, and skill of surgeon.

Although urethral and bladder injury during vaginal delivery is exceedingly rare, it may occur secondary to large lacerations or rupture of a urethral diverticulum. Cesarean section, which comprises an estimated 25% of deliveries in the United States, has been associated with bladder injury as well as ureteral compromise. Ureteral injury can be secondary to angulation by improperly placed sutures or direct ureteral damage such as a crush injury or a partial or complete transection. As with any surgical complication early recognition of injury and repair during the primary surgery almost always result in less morbidity for the patient with a more successful outcome secondary to increased ease of repair of the involved tissue. When suspected or apparent injury to the bladder or ureter occurs during cesarean section, the primary surgeon or surgical consultant may face several difficulties unique to the cesarean delivery.

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These include the fact that obstetric facilities designated for abdominal deliveries are inadequately equipped with the urologic tools such as cystoscopes and C-arm–compatible tables required for endoscopic and radiographic assessment of the urinary tract. Also the large uterus as well as bleeding from engorged pelvic blood vessels may render surgical dissection of the bladder and distal ureter difficult. Finally, a Pfannenstiel incision, which is usually made by the obstetrician, may be inadequate for appropriate dissection and exposure of the ureters. However, it remains imperative that appropriate management is prompt recognition of the injury because if left unrecognized and not repaired, these injuries may give rise to significant late consequences including renal damage and genital urinary fistula. If the obstetrician is not comfortable addressing the lower urinary tract injury appropriate urologic or urogynecologic consultation should be obtained. (See the DVD for video demonstration of anatomy of the lower ureter and bladder.)

Important steps that should be used in the performance of a cesarean section have been reviewed in Chapter 16. These include appropriate mobilization of the bladder off the lower uterine segment, especially in patients who have had previous cesarean sections as well as the avoidance of blind clamping of the uterine vasculature when lateral extensions occur (Fig. 17-1).

Bladder injuries that occur during cesarean section are usually easily identified and repaired. If the bladder injury is a high extraperitoneal injury such as an inadvertent cystotomy occurring during entrance into the peritoneum, it should be managed with a layered closure of the cystotomy. As this is a high cystotomy in a non-dependent portion of the bladder, it will require minimal postoperative drainage. In contrast to a cystotomy that occurs in the base of the bladder, for example, during the dissection of the bladder off of the lower uterine segment or during an emergency cesarean section when the entire bladder is inadvertently transected as the baby is being delivered (Fig. 17-2). These are intraperitoneal cystotomies in a dependent portion of the bladder requiring drainage for 10 to 14 days and would probably best be served by obtaining a cystogram prior to the removal of the catheter to ensure there is no extravasation of urine. The general concepts of bladder repair are the same as those for vesicovaginal fistula repair. There should be appropriate mobilization of the bladder off any adherent structure to allow a tension-free closure of the injury. Delayed fine absorbable sutures should be used to close the bladder in two layers (Fig. 17-2C). Closure can be in an interrupted or running fashion with the first layer approximating the mucosa and the second layer imbricating the muscularis. (See DVD for video demonstration of two-layered closure of cystotomy.) As mentioned, the length of postoperative drainage depends on the site and extent of the injury.

The diagnosis of ureteral injury at the time of cesarean section can be challenging. If ureteral injury is suspected, the obstetrician should go to great lengths to ensure ureteral patency or definitely diagnose the injury at the time of the cesarean delivery. The most common site for ureteral injury during cesarean delivery is at the level of the uterine vessels while the most common site of injury at the time of hysterectomy is the lower portion of the ureter near the uterosacral ligament. The most reliable mechanism to ensure ureteral patency is to pass a ureteral catheter through the orifice of the ureter and up above the point of concern. Because ureteral catheters are not commonly available in the cesarean delivery room, a 5- or 6-French pediatric feeding tube can serve the same function. After making an advertent high extraperitoneal cystotomy (Fig. 17-3), the ureteral orifices can be visualized (Fig. 17-4). The Foley catheter is pulled up through the cystostomy and using a long dressing forceps the

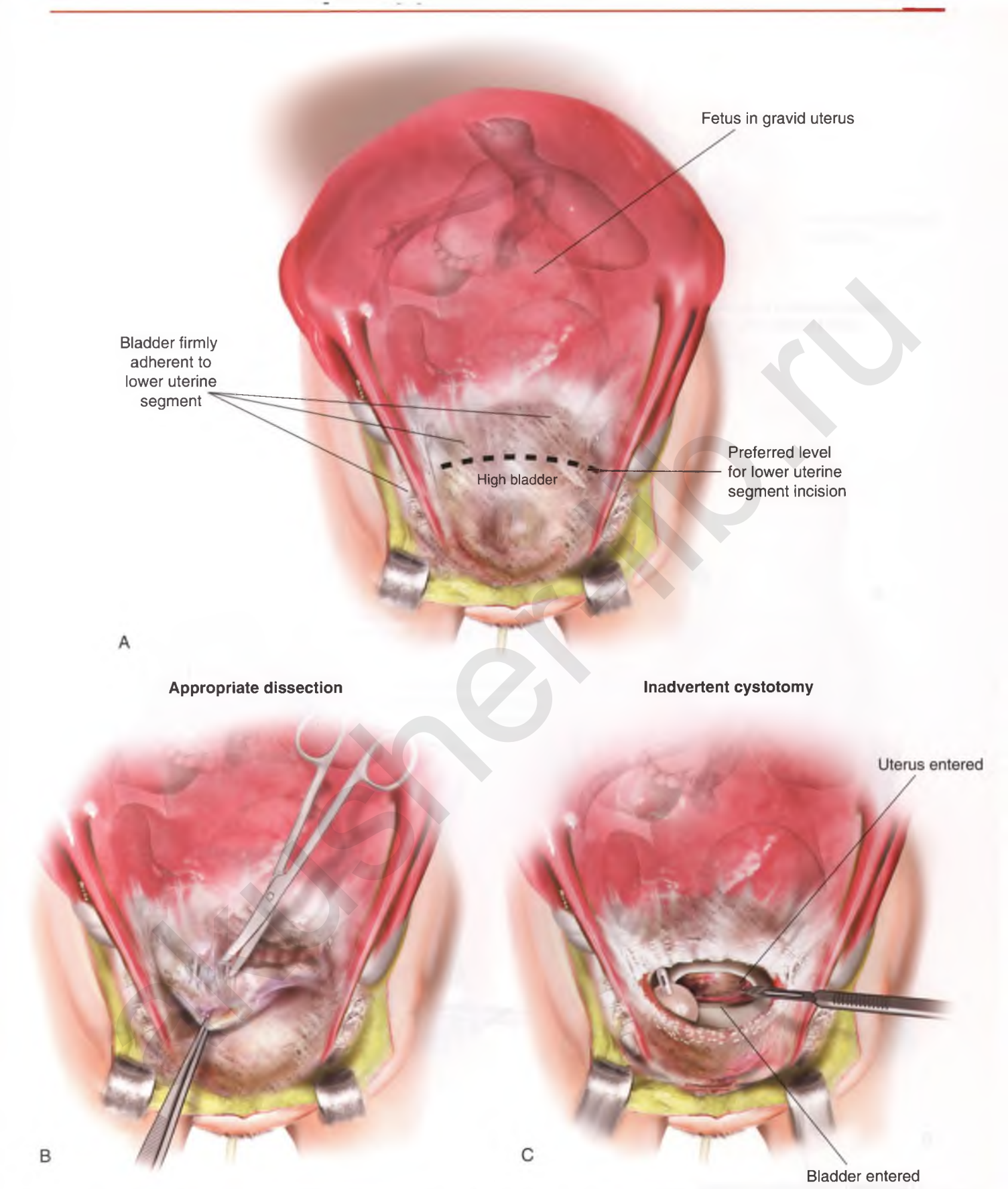

Figure 17-1 Gravid term uterus with scarred bladder flap occupying entire lower uterine segment. **A,** Dotted line depicts level for lower uterine segment incision. **B,** Sharp dissection should be used to mobilize the bladder off the lower uterine segment **(C).** If the bladder is not mobilized, inadvertent *cystotomy* is more likely to occur when performing a lower uterine segment incision.

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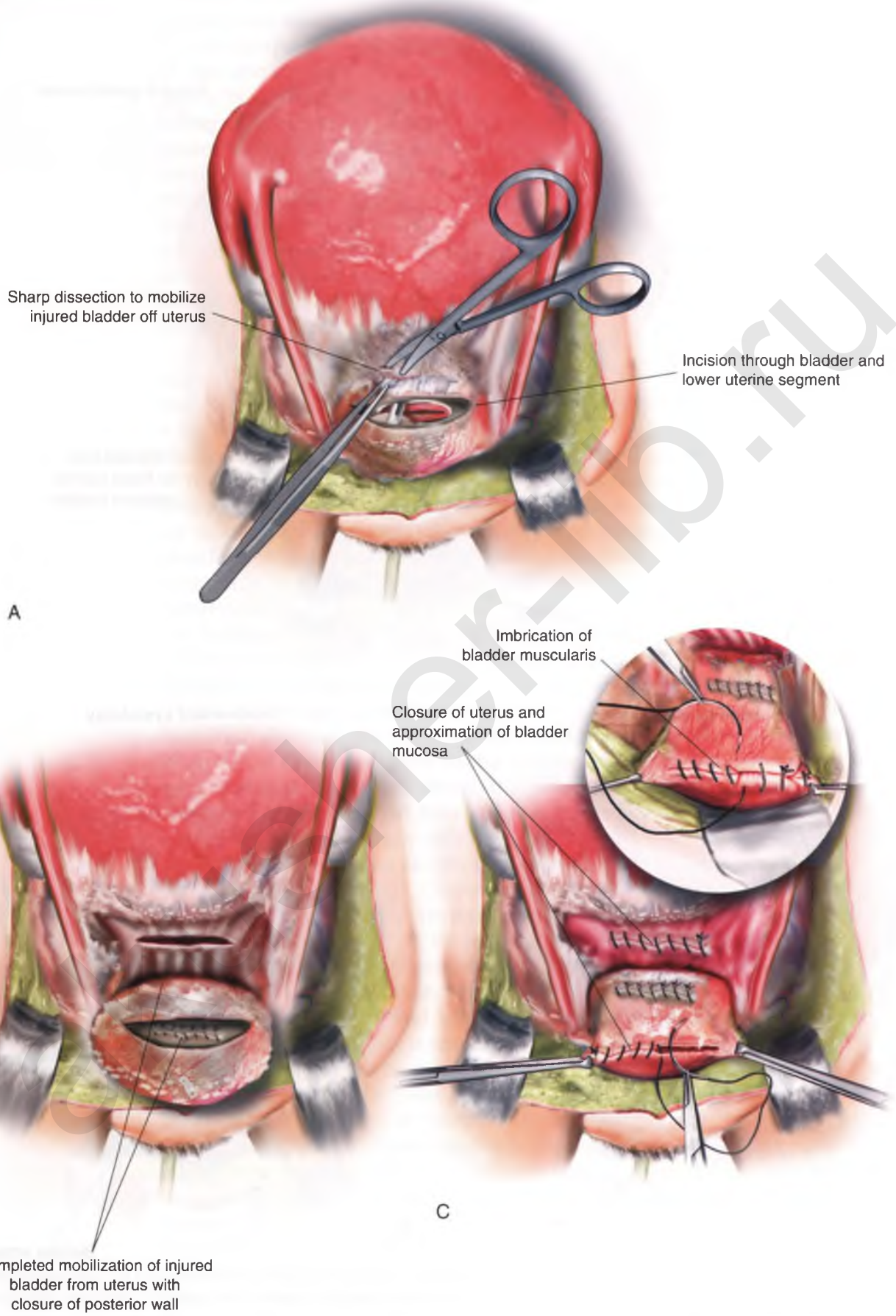

Figure 17-2 Transection of bladder during delivery of baby through a lower uterine segment incision. **A,** Sharp dissection is used to mobilize the injured bladder off the lower uterine segment. **B,** Bladder has been completely mobilized off lower uterine segment and posterior wall of the bladder has been repaired. **C,** Uterine incision has been closed and bladder cystotomies have been closed in two layers.

Figure 17-3 Technique for performing a high extraperitoneal cystotomy. Note Foley ball is mobilized into the extraperitoneal part of the dome of the bladder and monopolar cautery is used to cut through the wall of the bladder.

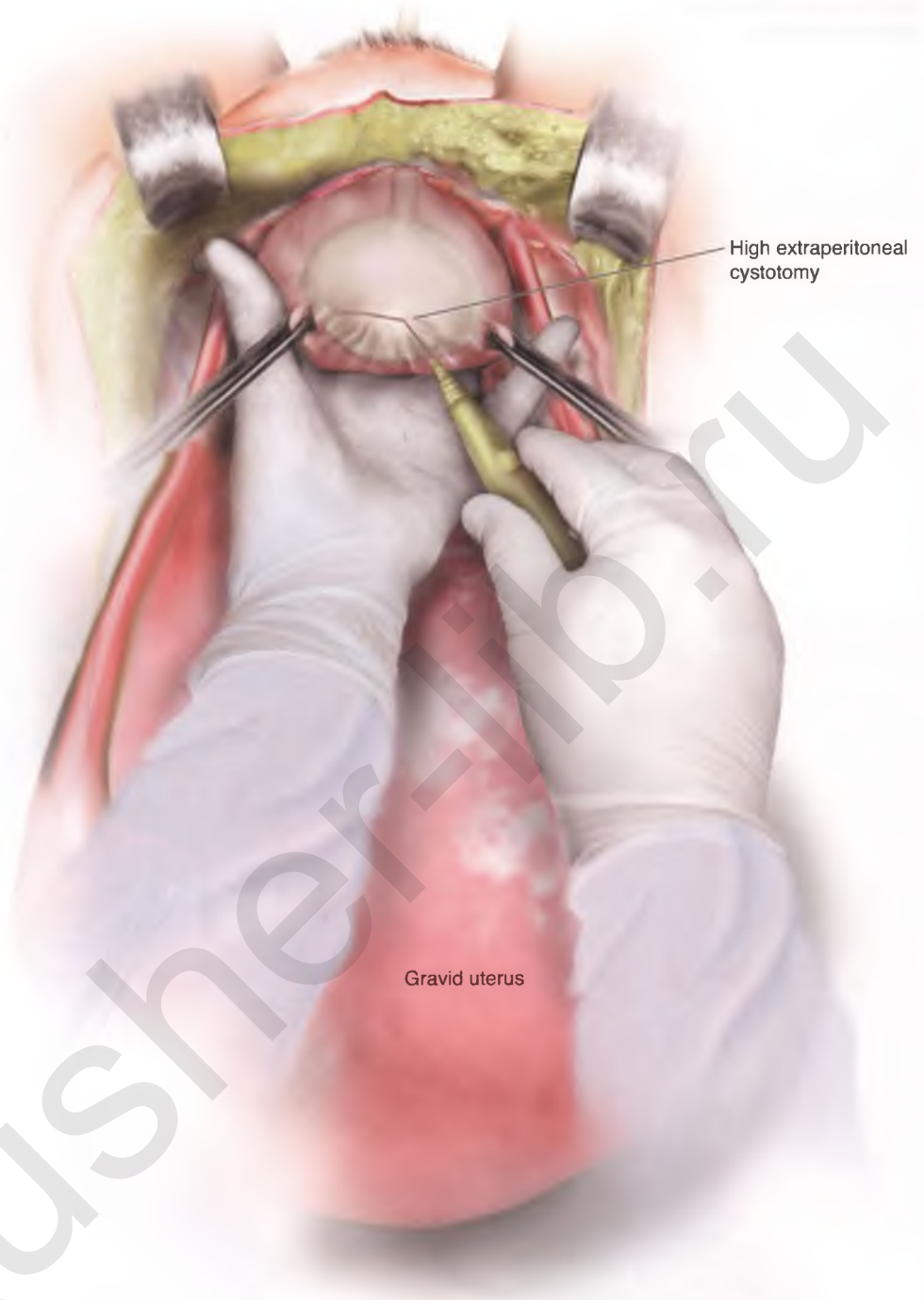

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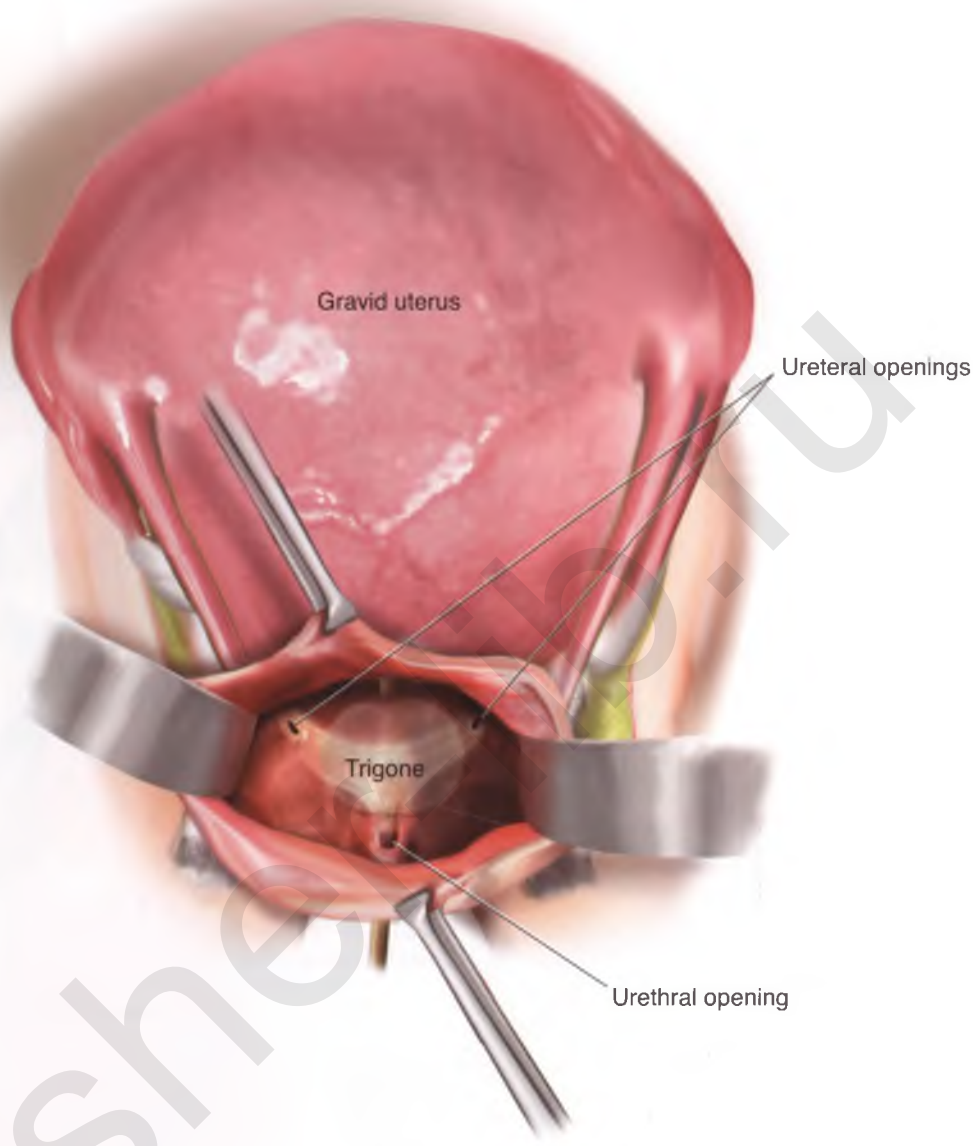
Figure 17-4 Trigone of the bladder as viewed from a high advertent cystotomy.



pediatric feeding tube can be passed in a retrograde fashion up into the ureter. (See the DVD for video demonstrations of advertent cystotomy with passage of pediatric feeding tube into the ureter as well as how to appropriately close the cystotomy. ☜) If it easily passes beyond the site of concern, this ensures ureteral patency. If there is an obstruction, kinking, or outright damage to the ureter (Fig. 17-5), urologic consultation should be obtained. Based on the type of injury, the resolution can be as simple as releasing the ureter from an obstructing suture and passage of a double J stent or as extensive as a ureteral reanastomosis or reimplantation of the ureter into the bladder (see Fig. 17-5). It is extremely important to stress that when these injuries are identified and managed at the time of the cesarean delivery, this decreases the potential of significant long-term morbidity.

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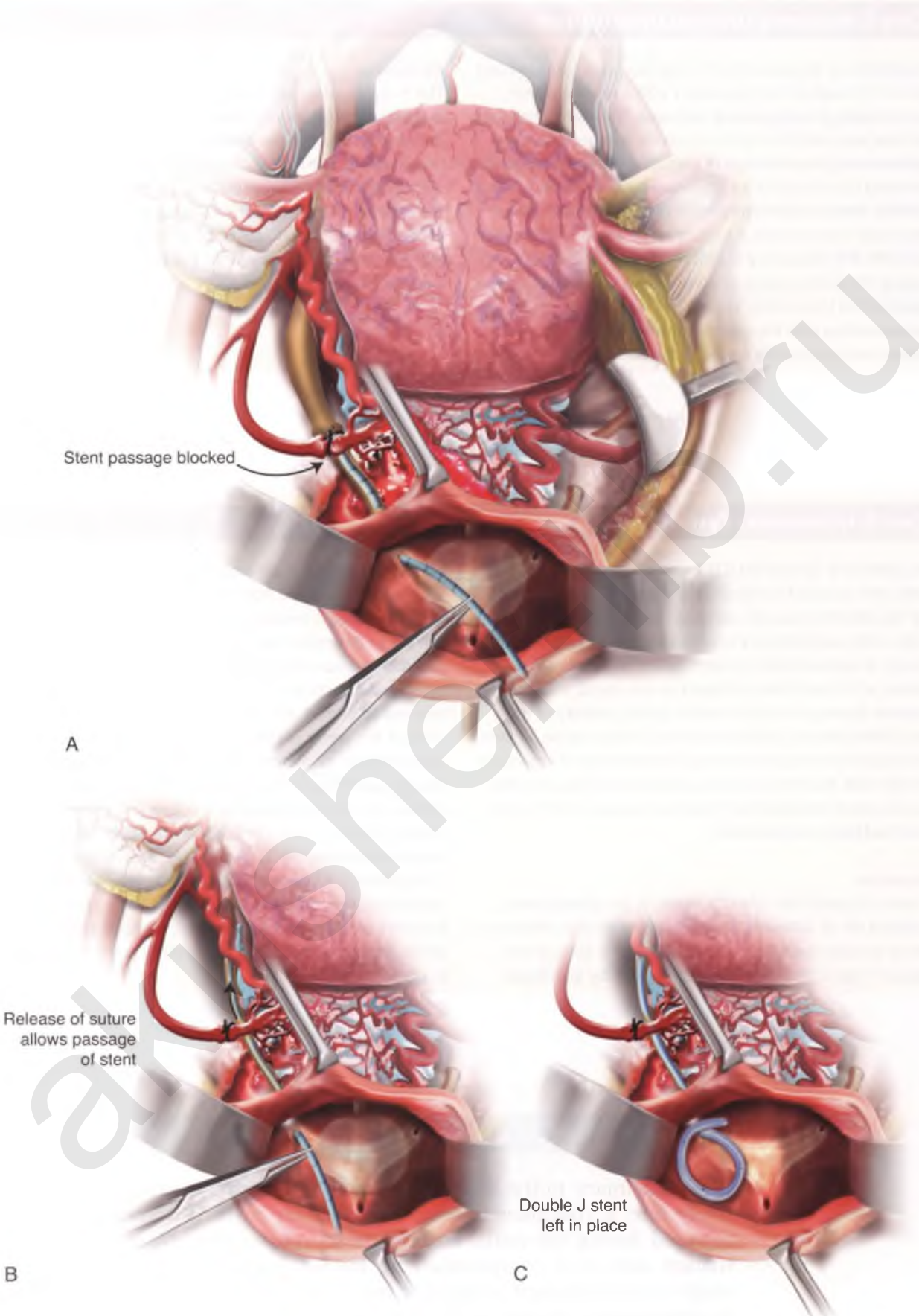

Figure 17-5 **A,** Passage of ureteral stent through high cystotomy. Note complete obstruction of right ureter secondary to a suture used to ligate the uterine artery. **B,** Release of suture allows passage of stent beyond area of injury. **C,** Double J ureteral stent left in place to allow ureter to appropriately heal.

Case 1: Ruptured Urethral Diverticulum

The patient is a 31-year-old G2P1 who has been managed and followed throughout her pregnancy with a relatively large, asymptomatic anterior vaginal wall mass. The obstetrician felt the mass was consistent with a cystocele and because it was asymptomatic, no evaluation or management was needed. She presented in active labor and spontaneously delivered a 9 lb, 5 oz infant over a midline episiotomy. With the delivery of the head, there was an obvious rupture of the anterior vaginal wall mass with the release of a significant amount of purulent material. While attempting to place a Foley catheter it became apparent that the anterior vaginal wall was directly communicating with the urethra. With some difficulty the catheter was successfully placed in the bladder.

Discussion

This is a rare situation in which a urethral diverticulum was misdiagnosed for a cystocele and unfortunately during the delivery of the baby the diverticulum ruptured. Appropriate management should be, if available, immediate consultation with a urologist or urogynecologist. If not available, maintain the catheter in place. Most situations like this would not be addressed until the effects of pregnancy have subsided to allow an appropriate closure of the urethral opening and excision of the diverticular sac.

Case 2: Inadvertent Cystotomy with Ureteral Injury

The patient is a 35-year-old G3P2 who presents in labor at 38 weeks with ruptured membranes and fetal distress. She has had two previous cesarean sections. Due to prolonged fetal bradycardia, an emergency cesarean section was performed through a transverse skin incision. Failure to mobilize the bladder, which was firmly adherent to the uterus, resulted in complete transection of the bladder during delivery of the head. There was also a lateral extension of the uterine incision that resulted in excessive bleeding from the uterine vessels on the right side. Numerous sutures needed to be placed in this area to control the bleeding. There was concern that the right ureter had been compromised.

Discussion

In such a situation the bladder needs to be appropriately mobilized off all adherent structures, most notably the lower uterine segment (see Fig. 17-2) to allow for a tension-free closure; 5 mg of IV indigo carmine is given by anesthesia. Both ureteral orifices are visualized through the cystotomy. If no spillage is noted from one or both ureters, a ureteral stent or pediatric feeding tube is passed in a retrograde fashion to determine the site and extent of injury. Most likely in the case presented simple deligation of the offending sutures and passage of a double J stent may be all that is required to correct the ureteral compromise (see Figs. 17-3 to 17-5). If the ureter is completely transected, an end-to-end reanastomosis or ureteral reimplantation is required. The bladder cystotomy should be closed in two layers with a 3-0 polyglycolic delayed absorbable suture (we prefer chromic catgut) (see Fig. 17-2). The bladder should be drained with a transurethral Foley for 10 to 14 days. Sometimes if the urine is very bloody a suprapubic catheter is also placed to make sure continuous drainage occurs if the Foley catheter becomes blocked with a blood clot. Urologic consultation would determine how long the ureteral stent should be left in place.

Conclusion

Although injury to the bladder and or ureter during cesarean section is relatively rare, it continues to occur on a regular basis. If unrecognized and not repaired during the initial surgery these injuries can give rise to fistula formation and even compromise the kidney leading to a nephrectomy. The single most important prognostic indicator of ultimate patient morbidity due to urinary tract injury is the time of recognition. If necessary, appropriate urologic or urogynecologic consultation should be obtained.

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