DE GRUYTER

José Miguel Palacios-Jaraquemada

PLACENTAL ADHESIVE DISORDERS

HOT TOPICS IN PERINATAL MEDICINE

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Hot Topics in Perinatal Medicine

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Edited by
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Volume 1

José Miguel Palacios-Jaraquemada

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Preface

In 1989, while I was an emergency surgeon in a hospital in Argentina, one of the hospital's obstetricians told me about the death of a mother as the result of a placenta accreta. The mother, who was herself a physician, died as the result of rebleeding occurring hours after a hysterectomy done for her condition. I was astonished to hear this because the hospital in which her death occurred was fully equipped with modern instrumentation and facilities for obstetrics and other medical specializations, and I asked why it had not been possible to stop the patient's bleeding. When the obstetrician replied that the bleeding in such cases was "completely out of control," I decided to find out how to stop such bleeding with the goal of reducing its attendant morbidity and mortality.

As a first step toward this, I conducted the research for my doctoral thesis on the collateral circulation of the pelvis, with work done on fresh and embalmed corpses and in innumerable cadaveric arteriographies. Understanding the behavior of this collateral pelvic vascular network was essential to my subsequent study and practice.

Intensive retrospective analysis of the case that had prompted my commitment revealed no prenatal diagnosis of abnormal placentation by ultrasonography. Because the sonographic signs of this may not have been recognized or familiar at the time of the patient's hysterectomy, I decided to begin a complete study of placental adherence through nuclear magnetic resonance imaging (MRI). Because it addressed a new indication for such imaging, the study initially encountered substantial difficulty stemming from its multiple technical components, but was able to continue through the help of colleagues in Argentina and internationally. At first, our research group tried to analyze the diagnostic differences found with MRI as opposed to ultrasound imaging in disorders of placental adhesion. Soon thereafter, however, we focused particular attention on the morphologic and topographic features of placental invasion of the uterine wall to determine the relationship of these features to the surgical approaches used to treat this condition. The first part of this work was published in 2005, and is among the most important research efforts on the relationship between the findings in this condition and its MRI.

After continued anatomic study, the knowledge acquired from this research led to the development of a new surgical technique that solved the problem of placental adherence and its technical complications through a single, one-step conservative surgical procedure which, although technically complex, restores a primitive uterine–bladder anatomy, minimizing blood loss and providing an excellent reproductive and maternal outcome in patients with such adherence.

In the research leading to this procedure, my attention was drawn to the surgical observation of a number of circulatory phenomena that followed hemostasis or embolization in the uterine circulation, and of which no clarification exists in

the anatomic literature or other scientific literature. After 10 years of study, this led to the discovery of a new anastomotic component of the uterine circulation. Among other topics, this book explains how the uterus maintains its vitality after occlusion of the uterine arteries, even after the concurrent occlusion of both uterine arteries and the superior ovarian anastomotic pedicle.

Participation in research, discussion, and clinical procedures for clinicians, intensivists, hematologists, urologists, and other physicians who manage the clinical aspects of placental adhesive disorders was mandatory to its comprehensive understanding.

Although it was not easy to introduce changes in the surgical protocols that had previously been used for placental adhesive disorders, I received the continuous support of my family, friends, colleagues, assistants and patients to bring about changes in treating these disorders that had initially seemed impossible. Argentina, the country in which I live, has a population with high parity and high rates of cesarean section, which provide an ideal circumstance in which to study placental adhesive disorders. During the 22 years of my study of these conditions, I have operated on hundreds of patients and believe that I have learned something from each of them. Abnormal placental adherence is a major medical disorder, and although I am familiar with the best current means of treating it, I can never relax my guard when confronting it.

Because of the simultaneous growth in the rates of of cesarean births and abnormal placentation, in addition to their potentially deleterious consequences, I believe that the association between these two conditions must be broken within the near future. This goal has led to a bi-national research initiative at the University Hospital of the Centro de Education Medica e Investigaciones Clinicas in Buenos Aires, Argentina, and the Centre Hospitalier Universitaire in Liege, Belgium, for investigating the primary process of abnormal placental adherence. Preliminary results of this initiative have shown that the primary etiology of abnormal placentation can be modified regardless of the number of a patient's cesarean deliveries. If these results are confirmed, the solution to abnormal placentation could be simple, inexpensive, and widely available to obstetricians on a worldwide basis.

Because of the lack of specific clinical images relating to some aspects of abnormal placental adherence, I determined to include in this book the largest series of single graphic and video illustrations in a book in this field. Most of these were collected from patients whom I have treated personally, with others provided by different specialists and friends around the world.

Beyond my own efforts, I want to acknowledge the continuous support of colleagues around the world who continue to enhance my knowledge every day, and the help of other obstetricians and clinical specialists who have made it possible for me to share this knowledge with the worldwide medical community.

Buenos Aires, June 2012 José Miguel Palacios-Jaraquemada

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I want to especially thank the doctors and friends who provided me with their personal images and videos for inclusion in this book.

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Contents

1 General knowledge

1.1 Introduction

Disorders involving abnormal placental adhesion are a group of conditions that cause substantial problems at delivery, the most important of which is hemorrhage. The blood loss from such hemorrhage is typically severe, rapid, and extremely difficult to stop. Particular features, such as neovascularization, distorted anatomy, and a high rate of blood flow can turn a cesarean delivery into a nightmare in a matter of seconds. Deficits in formation of the endometrium during placentation result in strong attachment of the placenta to the myometrium and in some cases to the surrounding tissues. The solution to each of these problems requires specific tactics and strategies. Some approaches seek to provide an early solution to these problems and others a later one. Both alternatives require experienced surgical and clinical management because of the many factors needed to obtain good results. Because its management requires multiple resources, abnormal placentation is a condition with high rates of mortality and morbidity.

To those unfamiliar with the situation, a rapid hysterectomy may seem to be an easy solution under these conditions, but is in fact not simple at all. The surgical treatment of placental abnormalities is extremely complex both because of local features and hemodynamic and hemostatic problems. It is useless to remove the uterus and leave the patient in a critical clinical state, and for this reason, a multidisciplinary approach to placental abnormalities is probably the best option for managing them.

At the beginning of the twentieth century, only a few cases of abnormal placentation had been described, especially after abortions that required intensive curettage and were followed by prolonged uterine infection. At that time, the diagnosis of this condition was made at delivery, and the patient often died within a few minutes thereafter. But at some point something changed, and abnormal placentation began to be more frequent.

At the end of the nineteenth century, the German gynecologist Ferdinand Adolf Kehrer successfully used a lower uterine incision for cesarean section (Kehrer, 1882). At that time, Sänger's conservative cesarean technique was very popular because it prevented the need for hysterectomy after cesarean delivery (Sänger, 1882), and the Kehrer technique passed unnoticed. In 1882, Kehrer and Sänger introduced the practice of cesarean section with silver-wire sutures, which represented a major change from cesarean hysterectomy (Porro, 1876). During the years after this, a number of gynecologists introduced variations of the extraperitoneal approach, but in 1912 the German gynecologist Bernhard Krönig (1863–1918) stated that good results came with the use of a segmental incision rather than an extraperitoneal approach.

In 1921, John Munro Kerr of the University of Glasgow rediscovered Kehrer's technique, and in 1926 the technique of transverse lower segmental incision for

cesarean section was presented to the American Gynecological Society (Munro Kerr, 1926). This variation made it possible to significantly reduce the morbidity and mortality rates in conventional cesarean delivery. After the introduction of this technical variation, the frequencies of the most important causes of mortality and morbidity related to cesarean delivery, such as uterine rupture, hemorrhage, and infection dropped immediately. However, Munro Kerr described certain histologic characteristics of the uterine segment that could be counterproductive in cases of iterative cesarean delivery. The high percentage of collagen in the uterine scars following repeated cesarean section could turn the elastic tissue of a uterine segment into inelastic scar tissue susceptible to rupture.

In the 1960s, the rate of birth through cesarean section in the State of California in the United States was from 3 %–5 % (Petitt et al., 1979), and has been rising in California since 1965. To date, only a few cases of abnormal adhesive placentation (almost 300 cases) in women who have delivered in this way have been published (Sumawong et al., 1966). Today, the rate of cesarean section has increased in almost all countries and varies from 25 %–70 %, with even higher maximum rates in some countries. The exponential increase in the use of cesarean delivery has occurred for multiple reasons, including cesarean delivery after a primary cesarean delivery, fewer forceps deliveries, cesarean delivery in breech presentations, fear of malpractice lawsuits, the use of cesarean section in cases of fetal distress, and cesarean delivery on maternal demand. However, the increasing rate of cesarean delivery since 1965 has also been accompanied by a subsequent increase in morbidity secondary to cesarean delivery, such as from abnormalities of placental adherence (Silver et al., 2006).

By 1960, the estimated frequency of disorders of placental adherence was 1 in 30,000 births; today it is between 1 in 500 and 1 in 2,500 births (Timmermans et al., 2007). These figures clearly indicate a morbid association between cesarean delivery and disorders of placental adherence.

It has been a little more than three decades since abdominal ultrasonography (US) became the first diagnostic method used to detect disorders of placental adhesion. During this time, transvaginal ultrasonography (TVUS), Doppler ultrasonography, power Doppler ultrasonography, placental magnetic resonance imaging (pMRI), and three-dimensional (3D) Doppler ultrasonography have been incorporated gradually into diagnostic practice in obstetrics and gynecology. In some cases, these methods have allowed improved diagnosis and in others have enhanced the visualization of uterine and placental topography.

Improvements in the diagnosis of disorders of placental adhesion facilitated a major change in the presurgical approach to these conditions because that the awareness of placental invasion is a first step toward avoiding its complications. However, advances in the diagnosis of abnormal placental adhesion did not correlate with substantial progress in surgical techniques for its correction.

The use of interventional radiology initially appeared to provide a complete solution to the avoidance of bleeding in disorders of placental adhesion, but after

initial complications even with this technique, it became clear that intensive study of the uterine blood supply was needed to achieve this goal. Technical details of the means for preventing excessive bleeding were different for the uterus as opposed to other organs and tissues, although new solutions to resolving this seem to appear almost every day. One problem is how to occlude an abnormal placental blood supply without causing unwanted damage to other organs. The solution to this is in no way easy because of the extensive network of newly formed vessels that develops with placentation. This collateral system links the uterine blood supply to that of the bladder, ovary and vagina. Occluding it so as to avoid bleeding during surgery but without causing unwanted secondary tissue damage is a skill reserved only to an extremely well trained radiologist. Uterine hemostasis in abnormal placental invasion is not a synonym for uterine arterial embolization. Additional technical and anatomic knowledge is needed for accurate embolization of the uterine artery under such circumstances.

Both reported and unreported complications mandate that obstetricians and gynecologists work in groups and share their information and experience in order to improve treatments for abnormalities of placental adhesion. Currently, members of both specializations are gathering valuable information about placental adhesive disorders; their next step may be to break the association of these disorders specifically with cesarean delivery and teach more about their global experience with these disorders.

1.2 Epidemiology

The reported incidence of placenta accreta has increased from approximately 0.8 per 1,000 deliveries in the 1980s to 3 per 1,000 deliveries in the past decade (Flood et al., 2009; Silver et al., 2006). However, the incidence of a condition also depends on the criteria used to calculate it. In the past, the only diagnostic criteria for placenta accreta were histologic, yet papers on the pathology of the condition indicate that there were some differences between the clinical findings and those of microscopic examination in cases of the condition. For this reason, some authors have included broad criteria for the diagnosis of disorders of placental adhesion, such as findings on clinical diagnosis; findings on pathologic diagnosis; difficult manual piecemeal removal of the placenta if there is no separation after 20 minutes despite active management of the third stage of labor; and heavy continued bleeding from the implantation site of a well-contracted uterus after removal of the placenta during cesarean delivery (Wu et al., 2005). With these criteria, Wu and colleagues reported an incidence placenta accreta of 1 in 533 over a 20-year period ending in 2002.

In some countries in which the rate of cesarean delivery has continued rising until reaching alarming levels, and in others with historically low rates of such delivery, the situation has shown a tendency toward change. In the United States

for example, if the rates of primary and secondary cesarean delivery continue to rise as they have in recent years, the rate of cesarean delivery will reach 56.2% by 2020, and there will be an additional 6,236 cases of placenta previa, 4,504 cases of placenta accreta, and 130 maternal deaths annually. The increase in these complications will lag behind that in cesarean section by approximately 6 years (Solheim et al., 2011.

1.3 Risk factors

An important risk factor for placenta accreta is placenta previa in the presence of uterine scar (Clark et al, 1985a; Miller et al., 1997; Usta et al., 2005), and the most common cause of myometrial damage and uterine scar formation is iterative cesarean delivery (Rosen, 2008).

Although repeated cesarean delivery is a common cause of uterine damage through scar formation, it is the association of such delivery with placenta previa that is among the well-described risk factors for abnormal placentation, with an increasing rate of this in relation to the number of cesarean deliveries (Miller et al., 1997; Silver et al., 2006; Wu et al., 2005). In other words, when uterine scarring is associated with placenta previa, the possibility of placental infiltration of such scars is high.

Other causes of myometrial–endometrial damage are placenta previa in an area of previous uterine curettage, especially in cases of abrasion, especially in patients who have had repeated procedures, which have a close association with placenta accreta. There is also a close relation between the number of cesarean sections performed on a patient and the possibility of the patient's developing a disorder of placental adhesion. The equation seems to be very simple: the greater damage, the higher the possibility of developing abnormal placentation. The rise in the frequency of cesarean deliveries has brought about an exponential increase in adherent placentation, an occurrence that is being universally proven.

Other types of myometrial damage, such as from uterine surgeries, endometritis, and endometrial ablation or radiation, are also related to adherent placentation, although less frequently. Post-cesarean uterine curettage increases the risk of uterine damage over the scar of the cesarean section, as well as the risk of placenta accreta in a subsequent pregnancy. This is not a well-known risk factor, but is a frequent cause of placental adhesive disorders. After delivery, an intensive collagenolysis occurs with the purpose of restoring nonpregnant uterine volume. If during this period curettage causes damage in a recent cesarean scar, normal uterine healing is modified.

A recently published paper reported a markedly higher incidence of abnormal placental adherence in pregnancies induced by in vitro fertilization (IVF) (Esh-Broder et al., 2011). However, larger studies were suggested to confirm this finding

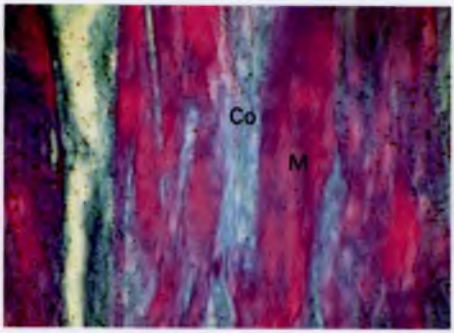

Fig. 1.1: Histology of normal uterine segment at cesarean section. M: Muscle fibers, Co: Collagen.

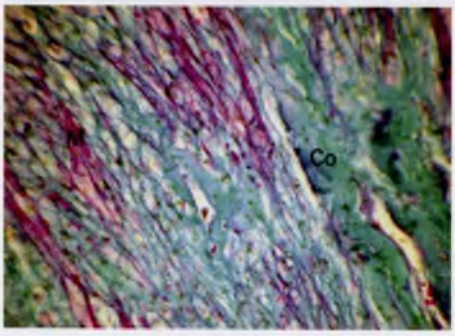

Fig. 1.2: Histology of uterine segment in a second cesarean section. There are a few muscular tissues and a high percentage of collagen. M: Muscle fibers, Co: Collagen.

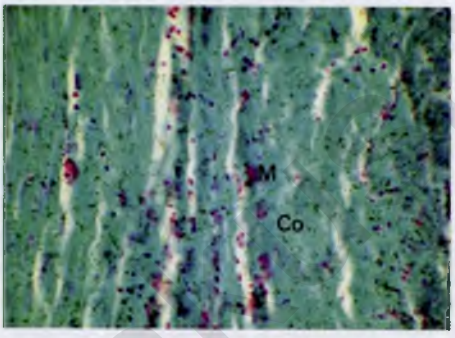

Fig. 1.3: Histology of uterine segment in a third cesarean: Muscular tissue is almost nonexistent; collagens are highly abundant and cover the visual field.

and elucidate its causes. Nevertheless, pregnancies induced by IVF should be considered a risk factor for placenta accreta (Esh-Broder et al., 2011).

In countries in which abortion is illegal, it may be done by skilled or unskilled persons. In general, the safety of nonlegal abortion depends on the costs entailed by its performance. Some alternative means of abortion include access to sterilized material, the costs of control with US and anesthesiology, and other resources for a safe procedure. However, a large group of practitioners of abortion are habitually unskilled and engage in it without minimally necessary surgical precautions. As a result, infection, persistent hemorrhage, and endometrial adhesions are frequent

Most frequent	Frequent	Infrequent	Non-specific
Previous cesarean and placenta previa	Anterior placenta and previous iterative cesarean sections	Endometrial thermo-ablation	Tabaquism
Multiple and abrasive d&c	Placental insertion in area of previous uterine surgery	Radiation	Age over 35 years
Placenta previa, cesarean and d&c	Endometrial infection after abortion	Placenta previa and assisted conception techniques	

Tab. 1.1: Risk factors for placental adhesive disorders (placenta accreta, placenta increta, and placenta percreta).

in the populations these practitioners treat. This situation is commonly hidden during anamnesis because the procedure is illegal, because of the presence of the patient's husband, or simply for reasons of shame. For this reason, physicians need to be alert when a patient shows evidence on US of a placental adhesive disorder in the complete absence of known clinical reasons for its occurrence. Patience and detailed dialogue with the patient are the keys to detecting the reasons for such evidence.

The uterus is formed mainly of a group of muscular fibers and collagens, although in very different proportions according to the different parts of the organ. In the uterine body the proportion of muscular fibers in relation to collagen is higher, a situation that reverses at the level of the uterine segment and cervix. This distribution bears a direct association with the function of the uterus, since the uterine body requires a substantial muscular mass in order to facilitate delivery. The cervix acts as a sphincter that withholds the fetus until the moment of delivery. By this phase of gestation, the enzymatic action of collagenases particularly modifies the collagen of the uterine wall, unfolding it and producing cervical dilation.

When a cesarean section is performed, the segmental incision heals, producing an inextensible collagen structure. With a new pregnancy, traction and countertraction are exerted over the scar, deforming it. Consequently, the enzymatic action of collagenases leads to the los of more collagenous tissue, which in turn weakens the uterine scar left by the previous segmental incision. Repeated cesarean sectioning noticeably reduces the number of muscular fibers in the affected uterine segment, which are replaced by collagen tissue (Figs. 1.1, 1.2, and 1.3).

In mammals, exposure of uterine collagen alters decidualization. In human beings, both collagen III and collagen I are present in a higher proportion in the uterine lower segment, in the septum (when there is one), and in the cornual region, areas which, after the uterine lower segment, have the highest occurrence of adherent placentation (Tab. 1.1).

1.4 Terminology

Placental adhesive disorders are commonly called placenta accreta. However, although placenta accreta is habitually used to describe a wide group of placental adhesive disorders (Belfort 2010), abnormal placental adhesion is, from a strictly histologic point of view, defined when all or part of the placenta attaches abnormally to the myometrium. Three degrees of abnormal placental attachment are defined according to the depth of invasion; these are: (1) placenta accreta, defined as occurring when chorionic villi attach to the myometrium rather than being limited within the decidua basalis; (2) placenta increta, when chorionic villi invade the myometrium; and (3) placenta percreta, when chorionic villi invade through the myometrium to the level of the serosa or beyond it.

Histologic examination shows a clear difference among placenta accreta, increta, and percreta. Nevertheless, and from a clinical point of view, the three degrees of myometrial invasion corresponding to these conditions may coexist at a single site and cause both diagnostic and therapeutic confusion. Therefore, the generic name "placenta accreta" is habitually used to refer to all three types of abnormal placental adherence, regardless of the histologic classification of their degree of invasiveness.

Contrary to what usually occurs in other kinds of anatomopathologic determinations, histologic examination does not constitute a diagnostic "gold standard" in placenta accreta. Because the surgical fragment of the involved uterus is very large, random sampling goes against an accurate diagnosis. Thus, a massive invasion of the placenta may be clinically evident but not be shown in the anatomopathologic report.

Although the histologic definition of placenta percreta is clear, the placenta can reach the serosa to cause this condition by means of two completely different mechanisms: the first is via myometrial rupture and subsequent placental advancement, and the second by vascular invasion. Although both situations would fulfill the histologic diagnostic criteria for placenta percreta, their clinical behavior in terms of surgical correction is completely different.

The technique of in block resection has been helpful for detecting myometrial fibers in placental tissue. In a study reported in 2001, extensive and selective sampling of the placental basal plate revealed a much higher incidence of myometrial fibers than had previously been reported (Khong and Werger, 2001). A review of clinical charts supported the concept that the presence of these fibers can confirm but does not necessarily correlate with a clinical diagnosis of mild placenta accreta. Less severe cases of placenta accreta are frequently associated with previous uterine surgery and multiparity, and although not uncommon, are often not suspected clinically. Placental examination is useful in making the diagnosis of placenta accreta in cases not requiring hysterectomy, particularly if the basal plate is well sampled (Jacques et al., 1996). The histologic finding of myometrial fibers in the basal plate without intervening decidua (occult placenta accreta) in spontaneously

delivered placentas, even in clinically asymptomatic cases of placenta accreta, is not infrequent (Stanek, and Drummond 2007).

This apparent contradiction in histologic nomenclature as opposed to clinical findings suggests the need for a clinical surgical classification of disorders of placental adhesion that can relate the area of invasion to the possibility of bleeding and other complications.

1.5 Types of placental invasion

Although the information that follows has not previously been published, it is the result of 22 years of the continued investigation of placental abnormalities and the findings in more than 450 personally treated cases (Palacios-Jaraquemada, 2011).

Classically, three types of abnormal placental adherence have been described. As noted earlier, these are placenta accreta, placenta increta, and placenta percreta. However, this classification is retrospective, histopathologic, and of doubtful utility during surgery, although some authors have tried to find a close association between the imagenologic findings in these conditions and this histologic classification. Some cases of abnormal placentation may contradict the protocols recommended for managing these conditions, as in cases of placenta percreta amenable to very simple surgical procedures and extremely difficult cases of localized placenta accreta marked by bleeding and requiring extensive surgery. For this reason, the histologic classification of disorders of placental adherence does not maintain a close relationship with the biologic and surgical behavior of these disorders, but has nevertheless been used continuously for years. In contrast, their topographic

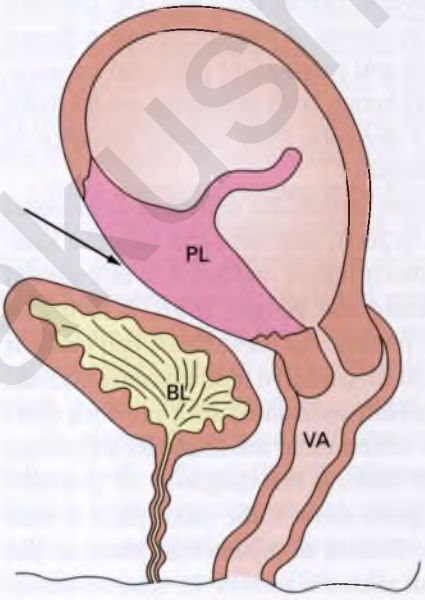

Fig. 1.4: Placental invasion of Type 1: Sagittal slice. The anterior uterine segment is markedly thinner and the placenta reaches the peritoneal surface; but no placental–vesical or vesicouterine NFV are identified. There is a lax dissection plane between the posterior bladder wall and the uterine segment.

classification bears a close relationship to the possibility of their bleeding and their surgical complexity, and also makes it possible to know which vessels and other components of a particular disorder should be identified or controlled and how to do this.

From a surgical and morphologic viewpoint, three major types of anterior placental invasion may be distinguished. In Type 1, the anterior segment of the uterus is noticeably thinner and the placenta reaches the peritoneal surface, but no newly formed placental–vesical vessels or vesicouterine vessels are identified, and there is a lax dissection plane between the posterior bladder wall and the uterine segment (Fig. 1.4). In Type 2 invasion, both the uterine segment and the posterior vesical wall are noticeably thinner and are connected by a fibrous scar, and newly formed placental–vesical or vesicouterine vessels are observed (Fig. 1.5). In Type 3 invasion the uterine segment is thinner, the vesical wall is of variable thickness, the vesicouterine plane has fibrous adherence, and there are variable placental-vesical-uterine NVF (Fig. 1.6).

This intrasurgical classification establishes the type of approach advisable for each of these conditions. Anterior placental invasion of Type 1 is usually labeled diagnostically as representing a false-positive result. In this type of invasion, both ultrasound and placental magnetic resonance imaging (pMRI) show myometrial dehiscence with placental advancement; therefore, and from the histologic point of view, these represent cases of placenta percreta (Fig. 1.7). However, once the fetus is removed from the uterus, placental delivery can be performed without difficulty. There is no bleeding, and on direct examination no remaining adherent placenta is observed. Previous uterine cesarean scar remains in the form of a thin sac, folded and hidden behind the bladder by the action of oxytocin (Fig. 1.8).

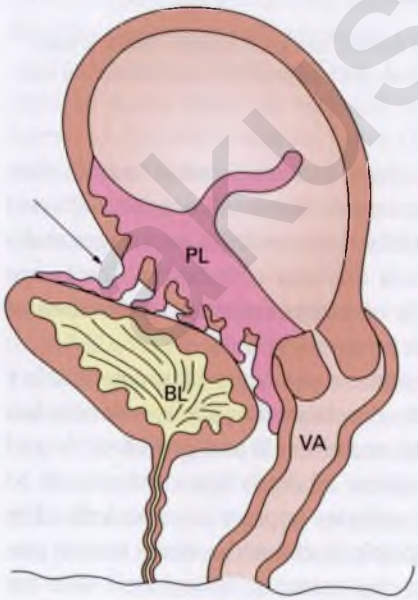

Fig. 1.5: Placental invasion of Type 2: Sagittal slice. The uterine segment and the posterior vesical wall are markedly thinned, and a fibrous scar connects the bladder and invaded area of myometrium. Placental–vesical or vesicouterine NFV are observed.

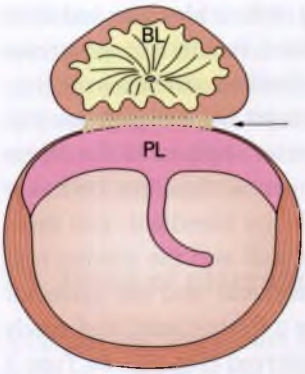

Fig. 1.6: Placental invasion of Type 3: Axial slice.The uterine segment is thinner, the vesical wall is of variable thickness, and placental–vesical–uterine and vesicouterine NFV could be present or not. There is a fibrous-tissue adherence between the bladder and the uterus.

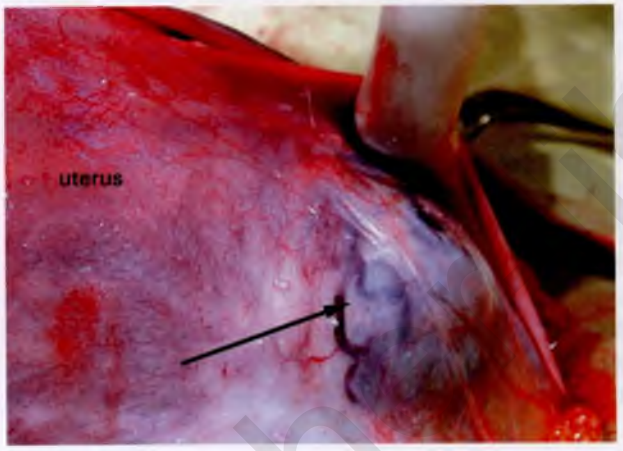

Fig. 1.7: The placenta reaches to the uterine serosa but without vascular invasion (black arrow). From a histologic point of view, this is a placenta percreta, but not from the viewpoint of its clinical behavior.

However, after dissection of the retrovesical space, a more detailed examination would show a circular area of myometrial dehiscence. Placental invasion of Type 1 can be better identified with pMRI than with US because multiplanar images make it easy to see a lack of newly formed vessels in the area of invasion. The image acquired is a complete slice plane and readily allows comparison of the area of invasion with normal tissues, especially in the retrovesical area.

Type 2 invasion of the uterine segment provides a sensation of apparent safety after initial opening of the vesicouterine pouch; the dissection plane is narrow but manageable. During dissection, there begin to occur small and persistent foci of hemorrhage that are difficult to control. The plane of dissection continues to be identifiable until the vesical mucosa or Foley catheter appears unexpectedly (Fig. 1.9). Access to the bladder is almost imperceptible because the dissection is performed on a thin fibrotic plane that connects the posterior vesical wall with the

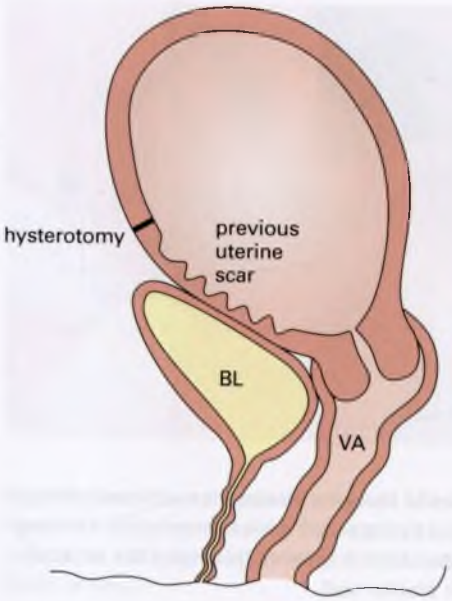

Fig. 1.8: The previous uterine scar remains behind the bladder. After use of a oxytocic agent, the redundant sac is folded. Because of the absence of bleeding, these cases are usually interpreted as false positive diagnoses.

uterine scar as a single structure. If during dissection the anterior side of the invasive placental tissue is injured, there will occur a serious high-pressure placental hemorrhage, aggravated by the fetal content of the placenta. Efforts at hemostasis or suturing of the uterine gap junction usually aggravate the blood loss, making it essential to evacuate the uterus (fetus) immediately so as to subsequently inhibit the placental hemorrhage. After delivery of the fetus, the uterus is brought outside the abdomen for manual compression of the uterine segment. This maneuver is a temporary means for achieving placental hemostasis until proximal vascular control is achieved.

In placenta accreta of Type 3, the newly-formed vascular component of the involved tissue must be released between double ligatures in order to provide access to the vesico-uterine space (Fig. 1.10); the procedure used for the ligature must always leave the main segment of the ligated vessel on the uterine side because this permits ready clamping of the ligated vessel with a hemostat if the ligature is cut or released. If this happens over the vesical sector of the treatment area, it is convenient to perform vascular suturing with synthetic absorbable 000 suture material, and to include the muscle layer of the bladder, which will then be provided with mechanical support at the time of adjustment of the suture (Fig. 1.11).

In cases of fibrosis of the vesico-uterine plane, it is advisable to open the anterior side of the parametrium and to dissect the cervico-vesical space medially (Fig. 1.12). This plane is rarely invaded, but if it is, the posterior bladder wall can be dissected through stepwise vascular ligature and vascular sectioning. Once this space has been inhfiltrated by placenta, the index fingers can be introduced laterally (Pelosi maneuver) to dissect and ligate all NFV (toward the cephalic sector of the vesico-uterine fold), liberating the area of vesico-uterine fibrosis (Fig. 1.13).

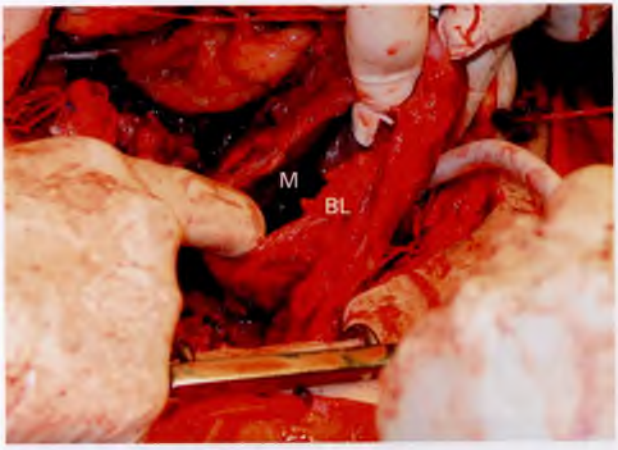

Fig. 1.9: Opening of the bladder is common in cases of abnormal placentation with dense fibrous tissues between the bladder and the uterine serosa. During repair it is common to see a thinned posterior vesical wall, because during pregnancy the uterus is growing, pulling on the adherent fibrous area and opening the muscular layer of the bladder wall.

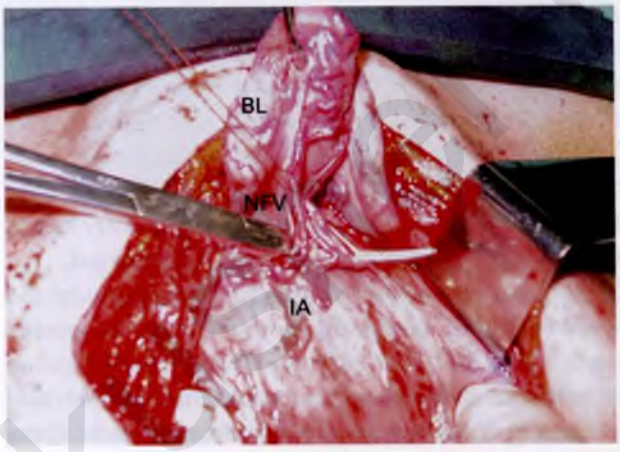

Fig. 1.10: Ligation of NFV is needed when hysterectomy or conservative procedures with tissue resection and repair are done. Access to the upper part of the vagina and cervix is mandatory to having access to a colpo-uterine neovascular component. BL: Bladder, NFV: Newly-formed vessels, IA: Invaded area.

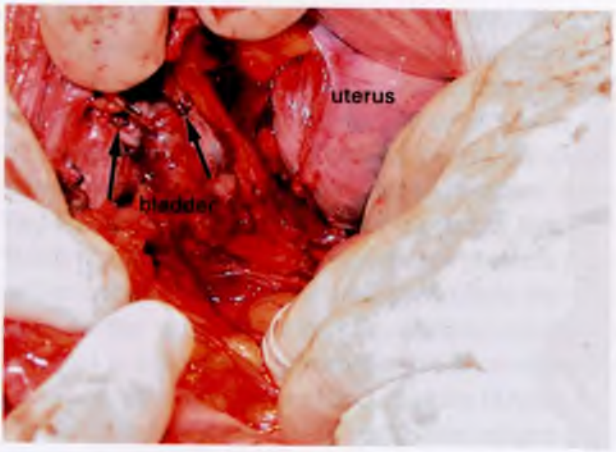

Fig. 1.11: Bleeding can happen during and after bladder mobilization. Because of the fragile structure of NFV it is recommended that ligatures or stitches be used to control bleeding. Stitches that include the thick part of the vesical muscular area are recommended for providing extra support (black arrows).

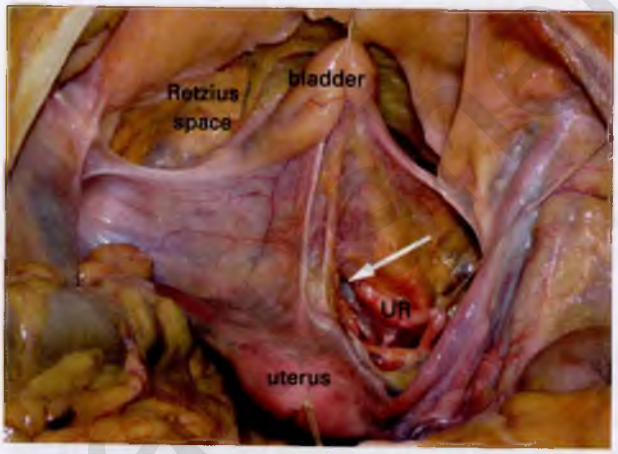

Fig. 1.12: Dissection of a fresh corpse: Opening of the anterior layer of parametrium provides access to the vesico-cervical space (white arrow). This area is almost never invaded by placenta and allows the insertion of a finger for dissecting strong localized adhesions between the bladder and invasive placenta.

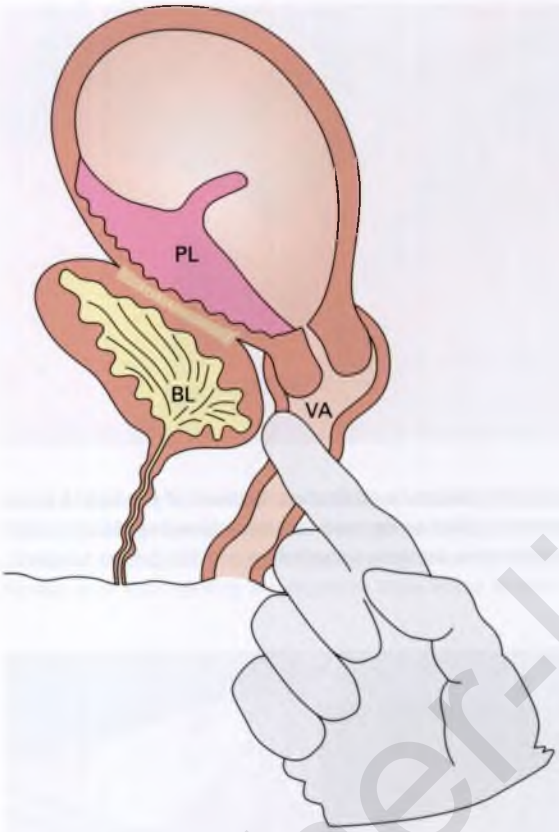

Fig. 1.13: Sagittal scheme of Pelosi's maneuver. Access to vesico-cervical space allows continuous dissection for an area without adhesion.

1.6 Etiology

To date, the specific etiology of disorders of placental adhesion remains unidentified. In normal gestation, the chorionic villi implant into the spongeous layer of the uterine decidua. This normal cleavage plane allows early detachment of the placenta after delivery, as a consequence of uterine contraction. Myometrial shrinkage after delivery compresses the vascular supply to the placental bed, and hemostasis is completed. Abnormalities at any of these steps can contribute to imperfect implantation, failure of placental separation, myometrial contraction and subsequent hemorrhage.

Absence of Nitabuch's layer was for years considered a primary etiology of abnormal placental adhesion. This concept was a consequence of thesis research by the German physician Raissa Nitabuch at the end of the nineteenth century (Nitabuch, 1887). Nitabuch postulated that the fibrinoid layer separated the chorion from the deeper decidua, and acted as a natural limit to trophoblast invasion.

Deficiency of this layer would therefore result in abnormalities of placental adhesion. Recent studies have confirmed that this fibrinoid-layer border effectively limits trophoblast invasion of the endometrium (Pijnenborg and Verycrusse, 2008), but also refuted the concept that Nitabuch's layer has a role in the pathogenesis of abnormal placentation. Problems during decidualization may contribute to the development of abnormal placentation, perhaps through excessive invasion of the decidua because of a deficiency of regulatory factors that limit implantation. Additional histologic findings suggest that in placenta accreta there is a defective interaction between maternal tissues, particularly the decidua, and migratory trophoblast in the early stages of placentation, resulting in undue adherence of the placenta or placental penetration into the uterus accompanied by the development of an abnormal uteroplacental circulation (Khong and Robertson, 1987).

Another hypothesis, which correlates oxygen tension with uterine scar formation, postulates that oxygen tension may contribute to the development of abnormal placentation. Work done in vitro suggests that because the human embryo develops in a relatively hypoxic environment, differences in oxygen tension determine whether cytotrophoblast cells proliferate or invade the uterine wall (Genbacev et al., 1997).

Recently, it has been suggested that placenta increta and percreta, the more severe forms of placenta accreta, are due to dehiscence of a scar, allowing penetration of the uterine wall by chorionic villi and access to the deeper myometrium by extravillous trophoblasts (Tantbirojn et al., 2008).

Because the lower segment of the uterus, proximal to the cervical canal has relatively poorer decidualization than the rest of the uterine cavity (Khong 2008), placental adhesive disorders may be consequences of decidual deficiency in a previously damaged area.

In summary, the etiology of abnormal placentation may result in a complex process that includes myometrial and endometrial damage with defective decidualization and trophoblast invasion.

1.7 Intrinsic problems

The prenatal diagnosis of disorders of placental adhesion was a problem in the past, but today, with the accurate knowledge of risk factors and ultrasonographic signs of these disorders, antenatally undiagnosed cases of such disorders are an exception. Nevertheless, their rapid medical presentation or inexperienced or rapid ultrasound investigation can be sources of error in their management.

Abnormal placentation in the lower uterus are more common than tose in the upper segment, and this is a main cause of surgical complexity in the management of disorders of placental adhesion. Narrow spacing, distorted anatomy, a high blood supply, and difficulty in surgical access are habitual problems in their surgical management (Borekci et al., 2008).

Enlargement of invisible microscopic anastomoses by placental growth factors significantly modifies the collateral uteroplacental blood supply, and organs such as the bladder are sometimos completely unrecognizable.

It is common in disorders of placental adhesion that the larger radial and arcuate arteries show pregnancy-induced changes. Usually, such changes in morphology are confined to the smaller spiral arteries (Khong and Robertson, 1987; Tantbirojn et al., 2008) and result in loss of muscular and elastic tissue from their walls. Such changes in the larger radial and arcuate arteries would render these vessels unresponsive to vasospasm and to oxytocic drugs, and would lead to torrencial hemorrhage with attempted removal of the adherent placenta because these arteries are of a large diameter and conduct a far larger volume of blood than the smaller-diameter spiral arteries (Khong, 2008).

The uterine anastomotic system located in the lower part of the uterus (stemming mainly from vaginal arteries) usually provides the main supplementary blood supply to the invasive placenta in cases of lower abnormal placentation. However, access to this component is very limited without previous vesico-uterine separation, a maneuver that can cause extreme blood loss if done without sufficient experience or accurate proximal vascular control.

Lack of myometrial support, from a thinned and distorted uterine scar, eliminates the possibility of hemostasis by myometrial contraction after delivery. The absence of myometrial tissue in the weakened uterine scar facilitates uterine invasion by the newly formed vessels (NFV) in cases of placenta accreta and percreta. Because of the close location of the scar from a cesarean section to the posterior wall of the bladder, this organ is commonly affected in lower placental invasion.

The extreme difficulty in dissecting the tissues in cases of abnormal placental adhesion, the presence of thick and fragile vessels, and the increased blood flow at term often make it impossible to perform both a safe and rapid hysterectomy in such conditions. And if a rapid hysterectomy is possible, it is generally a cause of overt hemodynamic and hemostatic failure.

Deficiency of resources or of trained personnel is frequent in peripheral medical facilities, and it is a serious mistake to think that personal skills are sufficient to solve the problems inherent in abnormalities of placental adhesion. And the lack of essential knowledge of these disorders and their management can mean a big difference in outcome for the patient.

Hemorrhage at a rate of blood flow of 500–800 mL/min is almost impossible to control without a clear knowledge of how to do so or accurate proximal vascular control. In cases of placental adhesive disorder in which there is intrasurgical diagnostic doubt, it is preferable to be conservative and avoid touching or detaching the placenta without adequate resources and a qualified treatment team.

2 Diagnosis

2.1 Presurgical stage

Even though there is no ideal presurgical means of diagnosis for placenta accreta, the sum of the different types of auxiliary diagnoses will provide a fairly accurate picture, useful for decisión-making and for proposing treatment alternatives. Although studies reported by specialists reduce the possibility of false-positive and false-negative results, it is always necessary to be alert to various diagnostic posibilities until direct surgical confirmation is made of a patient's condition. Underestimating or overestimating a clinical situation on the basis of imaging results may lead to irreparable errors and torrential bleeding, as well as to unnecessary hysterectomy. In this respect, it is strongly recommended that special attention be given to cases in which there is strong suspicion of a placental adhesive disorder in a patient without recognizable risk factors for this. It should be remembered that enlarged cervico-segmentary vessels can simulate placenta percreta. In such and other cases of diagnostic uncertainty, both placental MRI and 3D Doppler US can make a differential diagnosis, especially in patients not at risk of near-term complications. If for any reason a presurgical diagnosis is not conclusive, detailed surgical examination is needed before a definitive decision is made about diagnosis and treatment.

2.2 Clinical suspicion

The primary diagnosis in cases of adherent placentation is usually clinical, because the presence of placenta previa in a woman with a history of one or multiple cesarean sections or of any other type of myometrial damage leads immediately to the possibility of placenta accreta. The importance of this step should not be underestimated, since it allows the physician to ask the echographer to look specifically for direct and indirect signs of abnormal placental adherence (Palacios-Jaraquemada et al., 2007). In this regard, it should be remembered that the reliability of US imaging is closely related to the operator's image-acquisition skill and also to the technical limitations of the equipment and method being used. Negative studies in patients with known risk factors for adherent placentation must be re-explored by another trained operator or with an alternative imaging technique. Although they are not a common problem with trained operators, false-negative results are posible in US. Because of the possibly catastrophic consequences of diagnostic mistakes in cases of adherent placentation, it is the treating physician's responsibility to make a best effort toward reaching an accurate presurgical diagnosis.

2.3 Auxiliary diagnosis

Auxiliary diagnosis is needed for patients with known risk factors for abnormal placentation. Within this group, US is the most available, inexpensive, and accurate diagnostic method. Both abdominal US and TVUS are useful in different cases. Initially, US allowed only a retrospective diagnosis (Pasto et al., 1983) of abnormal placentation, and the first recognizable finding was the lack of a sonolucent retroplacental area. Almost ten years later, another report described additional sonographic signs of placenta accreta (Hoffman-Tretin et al., 1992) that included prominent, large or multiple placental venous lakes and periuterine vascularity, progressive thinning and disappearance of the retroplacental hypoechoic zone, and loss of a normal venous flow pattern on Doppler US; however, the experience they described was very limited. In the same year, a first prospective study was published of patients with placenta previa and a history of one or more cesarean sections. The sonographic criteria used included loss of the normal hypoechoic retroplacental myometrial zone, thinning or disruption of the hyperechoic interface between the uterine serosa and bladder, and the presence of focal exophytic masses (Finberg and Williams, 1992). This work was the basis for the echographic diagnosis of placental adhesive disorders; thereafter it is posible to find many publications that assign different values to the reported signs of these disorders.

In the same year of 1992 in which the foregoing studies were published, the first published report of Doppler US in the diagnosis of abnormal placentation was performed (Chou et al., 1992). Doppler imaging showed an unusually intense blood flow within the placental sonolucent spaces and revealed the hypervascularization patterns within both the placental and nonplacental tissues. Highly pulsatile venous flow patterns were detected within the placental blood lakes and the subplacental venous complex. However, Doppler US was used as a complementary technique to the diagnostic use of conventional US. A few years later Chou et al. (Chou et al., 1997), introduced the use of power Doppler US in the diagnosis of abnormal placentation. They described, apart from the typical findings on Doppler US, low-impedance arterial blood flow detected within the uterine serosa–posterior bladder wall, in the boundary zone. Power Doppler US was also used to detect placental adhesive disorders in the first trimester (Shih et al., 2002), and in this study it was observed that the pathologic involvement of large myometrial arteries apparently occurs even before the changes in intraplacental vasculature (lacunar flow), which can be shown by B-mode and color Doppler US in the second trimester. Later, Doppler US allowed the control of placental involution in cases in which the placenta was left in situ (Merz et al., 2009; Dueñas-Garcia et al., 2011). After many years of its use, it is not clear whether Doppler US improves the diagnosis or treatment of placental adhesive disorders over that with US (Palacios-Jaraquemada, 2002; Woodring et al., 2011).

As reported by Merz in 2009, monitoring of placental involution through the use of color Doppler US and human placental lactogen was recommended for pre-

dicting the degree of placental involution and establishing the occurrence of spontaneous placental expulsion within 10 days (Zepiridis et al., 2009). Through a similar approach, 3D angiography was used to determine the proper timing for surgical removal of the placenta in placenta previa accreta (Most et al., 2008).

Slightly more than 20 years ago, the first use of MRI in patients with third-trimester bleeding was reported in the literature (Kay and Spritzer, 1991) and 1 year later, the first diagnosis of placenta percreta was made with MRI (Thorp et al., 1992). At that time US was unable to detect bladder invasion in disorders of placental adhesion. The new method of MRI had some disadvantages with respect to US, such as a high cost, lack of mobile equipment, and limited experience in its use. But the possibility of having multiplanar imaging and total image acquisition was a major advantage of MRI. A few years later, a study was done that compared the efficacy of US, Doppler US, and MRI (Levine et al., 1997). This study showed few differences among these auxiliary diagnostic methods, but showed some advantage of MRI for cases of posterior placenta accreta. As was the case with US, signs of the condition were identified retrospectively with MRI (Maldjian et al., 1999), and a later comparison of MRI and US in relation to their identification of a distorted retroplacental myometrial zone, disrupted uterine–bladder interface, focal exophytic masses, and presence of vascular placental lacunae found that both imaging techniques had poor predictive value in the diagnosis of placenta accreta (Lam et al., 2002). In the early stages of placenta accreta, gadolinium (MRI contrast) was necessary to enhance placental tissue relative to the surrounding tissues (Palacios-Jaraquemada and Bruno, 2000), especially with regard to the depth of placental invasion. Later technical advances in image acquisition and in equipment speed made the use of gadolinium necessary only in a few specific cases. During the years following this, multiple studies were done to compare the sensitivity and specificity of MRI and US. However, a study with a different focus and involving a large series of patients worldwide, concluded that pMRI is essential for defining the topography and area of placental invasión in placenta accreta (Palacios-Jaraquemada and Bruno, 2005). New findings on placental MRI have modified the surgical approach and technique in placenta accreta, reducing morbidity below historical values and significantly increasing the use of conservative surgical techniques. These findings on placental MRI correlate the relationship between the invasive topography of placenta accreta, the arterial pedicles in the condition, and the difficulty of surgery for its treatment. At first, this correlative approach was used in cases of inconclusive findings on US, but later became an accurate method for delineating the invasive topography of the condition. Because placental MRI is the only method that identifies parametrial invasion, its utility is significant in resective surgery for placenta accreta with or without uterine conservation, and is reduced when the decision is made to leave the placenta in situ. The decision about whether to treat posterior placenta accreta depends to a great extent on the utility of MRI and experience in its use (Mazouni et al., 2009).

Additional identification of placenta accreta through the use of biologic markers in maternal blood has been proposed, but the techniques for this are expensive and not as practical as screening.

2.3.1 Ultrasound, Doppler, Three-dimensional Doppler

Ultrasound is the diagnostic method par excellence to detect placenta accreta. Its low cost, widespread use and comfort make ultrasound our first choice screening method for placental adhesive disorders.

Although the advantages of US in conditions of abnormal placentation are evident, some individual maternal and fetal chataceristics can disadvantageously affect the quality of US images. Thus, for example, studies with transabdominal US may be difficult in women who are overweight or in patients with previous abdominal or pelvic surgeries. Transvaginal US eliminates the disadvantages described with transabdominal US, but its most important limitation is the difficulty in evaluating the upper and posterior part of the uterine segment. Transvaginal US provides better imaging of the cervix and lower uterus, especially in lower abnormal implantations without the additional risk of bleeding (Oyelese et al., 2006; Timor-Tritsch and Yunis, 1993). Deficient or excessive bladder filling can cause errors in US images caused by excessive bladder pressure on the uterus. Uterine anteversion produces a posterior shadow in the lower uterine segment, an acoustic effect that is exacerbated in the presence of scars or uterine benign tumors such as myomas. Uterine contractions also modify the anatomy of the lower third of the uterus, making the diagnosis of disorders of placentation difficult in this region and possibly producing false-negative results or confusion with myomas. If the head of the fetus is engaged, a posterior acoustic shadow is generally produced, and this may cause a diagnostic problem. Additionally, a posterior placenta is frequently difficult to evaluate with transabdominal US, and for this reason alternative methods, such as MRI, must be considered in such cases (Levine et al., 1997).

A US study for disorders of placental adhesion should begin with a survey for the risk factors for these conditions. The patient's characteristics and placental location will determine the type of US study (abdominal or transvaginal) to be used. Although none of the signs of abnormal placentation is completely diagnostic, the following discussion pays special attention to patients with known risk factors for it, such as repeated cesarean sectioning and placenta previa, or previous myometrial or endometrial damage. The US grey scale includes different signs that can indicate abnormal placentation. Among the most important are placental lagoons, which, as opposed to the lagoons seen in normal pregnancies, are visible from the beginning of the second trimester to term. These are usually large, irregularly shaped, confluent, and present in large numbers. Their etiology is unknown, and there is no close relation between their number and appearance and the

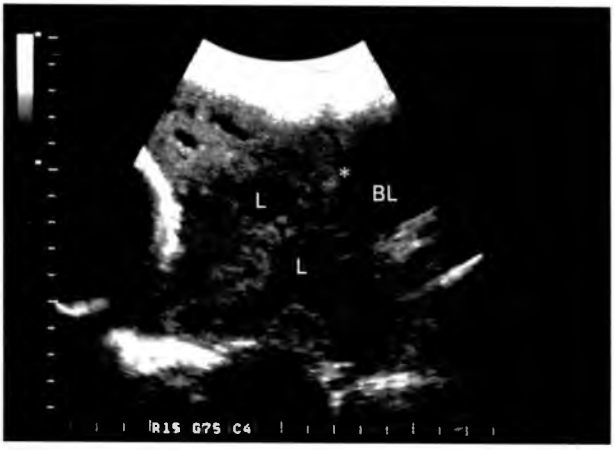

Fig. 2.1: Abdominal US: Presence of multiple lagoons. L: Lagoons, BL: Bladder.

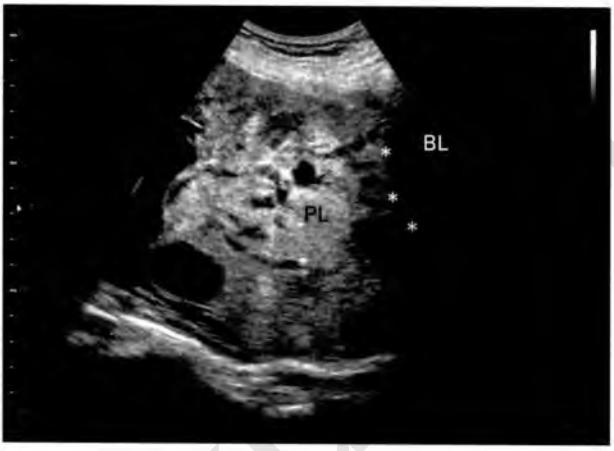

Fig. 2.2: US: Thinning of retroplacental uterine wall (asterisk). BL: Bladder, PL: Placenta.

degree of abnormal placentation (Fig. 2.1). In a substantial proportion of placental lagoons, turbulent flow can be observed, even without US in the Doppler mode. The absence of lagoons does not exclude the presence of abnormal placentation, but their presence is one of its clear risk indicators. The sensitivity of this sign on US is 79% and its predictive value is 92% when it is found between weeks 15 and 40 of gestation (Oyelese et al., 2006).

Another direct sign of abnormal placentation on US is loss of the retroplacental hypoechogenic area, which represents absence of the retroplacental vascular bed, a deficiency of basal decidua, and placental advancement over the myometrium (Fig. 2.2). The presence of this sign is strongly linked to abnormal placentation,

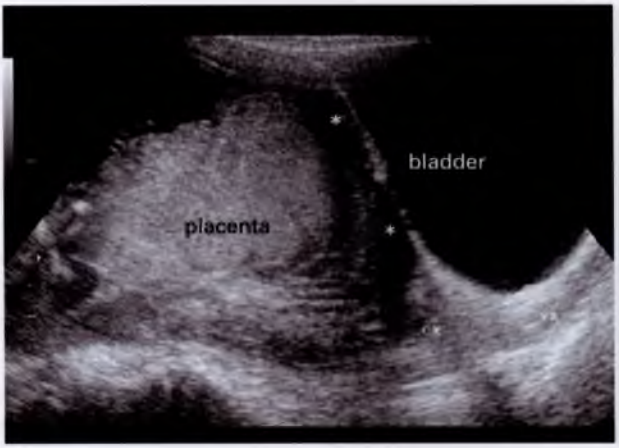

Fig. 2.3: A conventional US examination at 21 weeks of gestation shows total placenta previa with a sonolucent space between the placenta and bladder (asterisk).

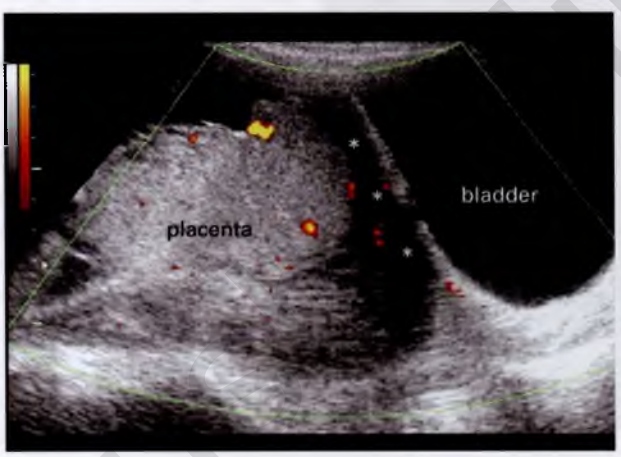

Fig. 2.4: Color Doppler US imaging shows normal placental flow pattern (21 weeks of gestation; total placenta previa). A sonolucent space is seen between the placenta and bladder (asterisk).

but it can also be an on US of a normal placenta. Consequently, the operator must be cautious when a retroplacental hypoechogenic area appears as an isolated marker in a US study. Some authors do not consider this sign important because of its low diagnostic sensitivity and predictivity, with a 48% frequency of false-positive results. In cases of placenta previa without adhesive invasion, retroplacental hypoechogenic area is clearly visible by both abdominal US and Doppler US (Figs. 2.3 and 2.4).

Another US marker of abnormal placentation is progressive thinning of the retroplacental uterine segment (Figs. 2.5 and 2.6). This indicates proximity of the

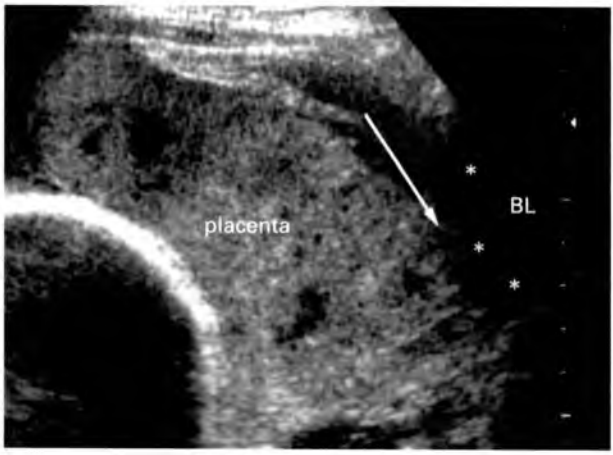

Fig. 2.5: Ultrasound image: Thinning of retroplacental uterine wall (asterisk) and absence of retroplacental hypoecogenic area (white arrow). BL: Bladder.

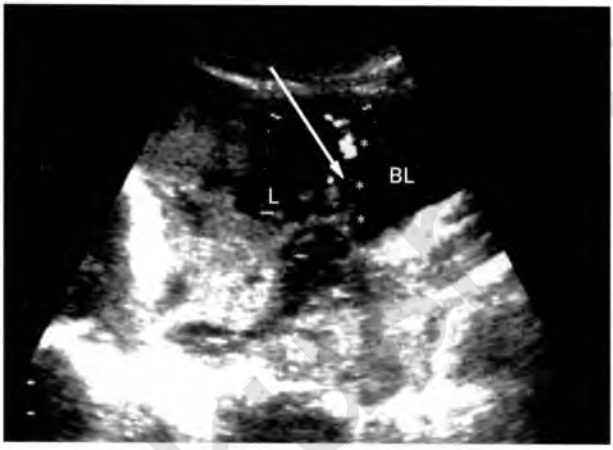

Fig. 2.6: Ultrasound image: Thinning of retroplacental uterine wall (asterisk) and absence of retroplacental hypoecogenic area (white arrow). L: Lagoons, BL: Bladder.

placental tissue to the visceral peritoneum or to neighboring organs, whether it is the result of placental advancement over the myometrium or because the underlying myometrium is extremely thin or nonexistent. It has been suggested that a retroplacental segment that is less than 1 mm thick is a sign of abnormal placentation with 93% sensitivity, 79% specificity, and a 73% positive predictive value (Hudon, 1998). This sign is difficult to identify and must be specifically sought, but in combination with other signs on US it increases the diagnostic reliability of abnormal placentation (Fig. 2.7).

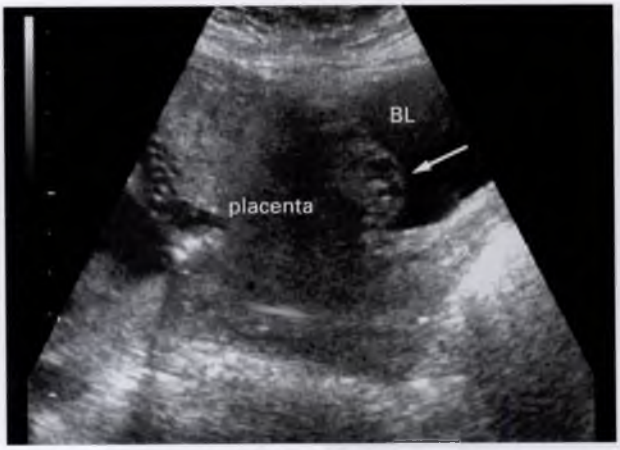

Fig. 2.7: A conventional US image at 21 weeks of gestation shows sonographic signs of placenta percreta with bulging of the uterine serosa–bladder interface and focal mass-like elevation of placental tissue beyond the uterine serosa. Bl: Bladder, White arrow: Placental tissue is seen protruding into the bladder.

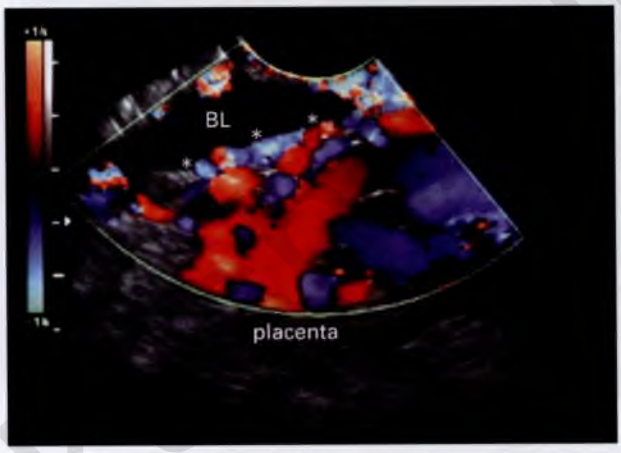

Fig. 2.8: Transvaginal color Doppler US image in a case of massive anterior placenta percreta. Thinning of retroplacentary uterine wall is seen (asterisk). BL: Bladder

Thinning or disruption of the uterine–vesical serosa results from a lack of myometrial support, and exposes the muscular laye of the vesical wall. It is not easy for operators who routinely use US in other than obstetric or gynecology settings to distinguish irregularities of the vesical wall, which are usually the result of multiple cesarean sections, from abnormal placentation. Alhough uncommon, the visualization of extrauterine placental tissue is a diagnostic confirmation of placenta percreta (Fig. 2.8).

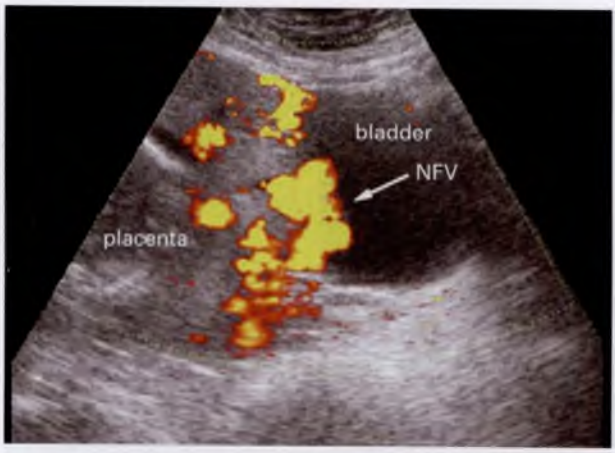

Fig. 2.9: Abdominal color Doppler imaging shows bladder-uterine serosa hypervascularity with aberrant vessels extending into bladder.

Grey-scale US is insufficient for the diagnosis of abnormal placentation, having an 87.5 % sensitivity, 98 % specificity, 93.3 % predictive positive value, and 97.6 % predictive negative value (Haratz-Rubinstein, 2002).

No sign of abnormal placental adhesion on US is 100 % sensitive by itself, but the most reliable sign of placenta accreta is the presence of irregular vascular spaces with arterial flow (Sentilhes et al., 2010; Comstock, 2011).

The finding on US of a sac implanted low over an anterior uterine scar, or of multiple irregular spaces, is an early suggestion of abnormal placentation in the first trimester (Comstock, 2011). Because the embryo is habitually implanted in a fundus, a stable and unmoving sac in the lower uterine segment should raise the suspicion of placenta accreta.

Use of the Doppler mode in US makes it possible to confirm the presence of rich turbulent blood flow in most placental lagoons with abnormal placentation. (Figs. 2.9–2.12). Conversely, the absence of a Doppler signal in the retroplacental bed strengthens the suggestion of loss of the retroplacental hypoechogenic area. This finding is frequent in localized abnormal placentation, being less serious but more usual in such cases than in those of diffuse invasion, and is also associated with the placental lagoons described earlier. Both signs are useful, but must not be regarded as "gold standards" for abnormal placentation because they may in some cases represent false-positives or false-negative results.

It is less common to visualize anomalous vascular tissue reaching organs and structures neighboring the uterus, mainly the vesical wall. The appearance of perpendicularly distributed vessels indicates placental vessels extending toward the myometrium or other neighboring tissues, which is usually associated with abnormal placentation.

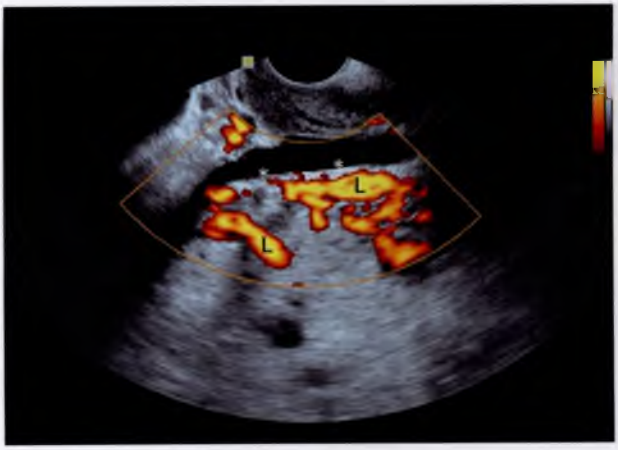

Fig. 2.10: Transvaginal color Doppler US image in a case of anterior placenta percreta. Thinning of retroplacental uterine wall and presence of confluent lagoons are seen.

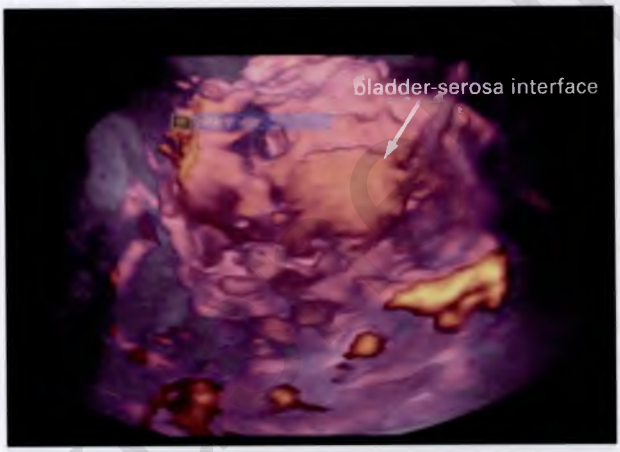

Fig. 2.11: Three-dimensional power Doppler US image of a placenta accreta at 37 weeks of gestation. In the lateral view of this volume-rendered image, the interphase between the bladder and serosa was filled with a prominent vascular layer. In contrast to the scanty intervillous circulation in the normal placenta, the intervillous flow here was rather prominent, and even fused into a vascular lake. The lateral view also showed a hypervascularity inside the placenta.

Several authors agree that Doppler US can be an auxiliary mode of US in the diagnosis of placental adhesive disorders (Woodring et al., 2011; Comstock, 2011), as well as for increasing the diagnostic performance of two-dimensional (2D) US (Sentilhes et al., 2010). In power mode, Doppler US permits the early detection of abnormal placentation in the first trimester.

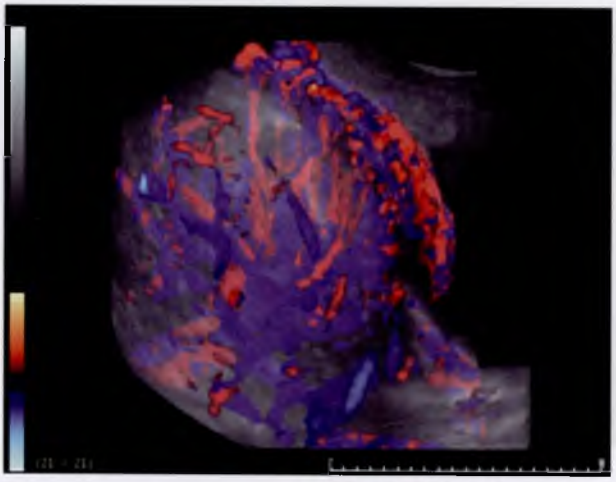

Fig. 2.12: Front view of bladder–serosa interphase, showing dense vascular confluences at the uterine surface. In placenta previa without accreta, the same region would have only discrete vessels. This image also reflects the striking appearance of the anatomotic vascular network seen during cesarean section.

A possible complication of abnormal adhesive placentation left in situ is hemorrhage, and another is the persistence of trophoblastic tissue. Theoretically, the reabsorption or elimination of residual placental material minimizes the possibility of infection and also of hemorrhage. When Doppler studies were done to determine the potential relationship of vascular placental activity to the likelihood of placental elimination, persistente, or curettage, serial Doppler examinations revealed cessation of blood flow to adherent placental tissue from 9–13 weeks postoperatively, followed by the complete reabsorption or expulsion of placental fragments (one case each), and in two cases of placenta previa percreta, uterine curettage was performed without major complications after negative findings for blood flow (Merz et al., 2009).

Color Doppler US was also used in association with the measurement of human placental lactogen (hPL) to predict the expulsion of adherent retained placenta. In both of two described cases of this, a full placenta was expelled at 12 weeks after delivery and from 7–10 days after a decrease in the blood level of hPL, and when color Doppler activity was negative (Zepiridis at al., 2009)

However, it is not clear in the studies done by Merz et al. or Zepiridis et al. how color Doppler US may have modified the natural history of the retained placenta, because placental expulsion as predicted by Doppler US is normally expected within the same time period in which it occurred spontaneously. It is also confusing why uterine curettage was performed in cases of placenta percreta, in which the absence of a uterine anterior wall is to be expected.

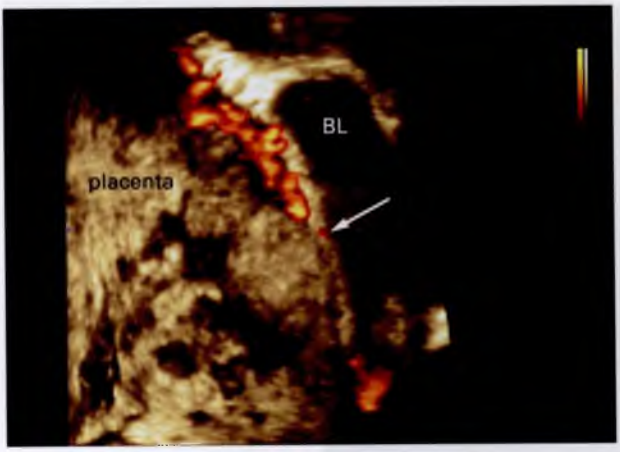

Fig. 2.13: Three-dimensional Doppler US image. BL: Bladder, AV: Abnormal vessels, White arrow: Utero-placental vessel extending into muscular layer of bladder.

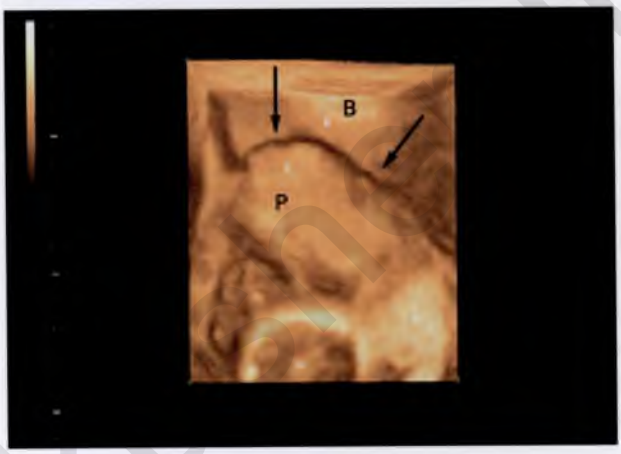

Fig. 2.14: Four-dimensional image of reconstruction shows a loss of the echolucent space between the bladder and the placenta and interruption and irregularity of the uterine serosa–bladder line. A focal, infiltrating, exophytic placental mass is seen projecting into the bladder in coronal scans (black arrows).

Video 2.1: Transvaginal Doppler ultrasonography in placenta percreta.

Intense turbulent flow is seen the vessels of the placental–vesical interface. Note the lack of a signal from myometrium in contact with the bladder.

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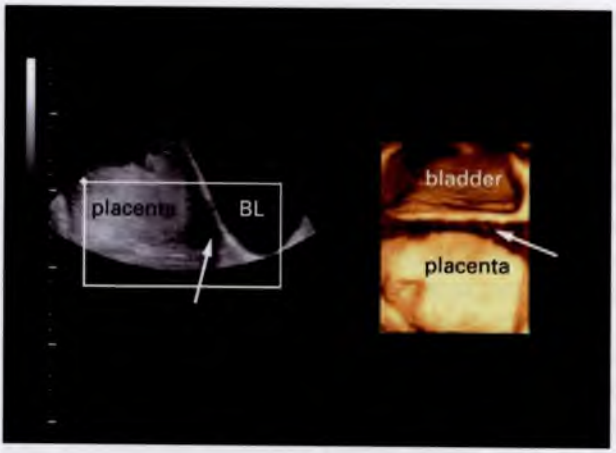

Fig. 2.15: Three-dimensional US image shows a normal echolucent space between the bladder and placenta in this case of simple uncomplicated total placenta previa. White arrows: Sonolucent space between placenta and bladder.

Today, 3D power Doppler US can be used as a complementary technique to make or exclude the diagnosis of placenta accreta (Shih et al., 2009) (Figs. 2.13 and 2.14). It is also used to produce a flow chart for the diagnostic differentiation of placenta accreta and placenta previa without the risk of overdiagnosis (Fig. 2.15). In brief, placenta accreta can be confidently diagnosed if multiple characteristics of its existence are present in a 3D US scan. Three-dimensional US can also be a useful adjunctive tool in refining 2D ultrasonographic techniques for the purpose of identifying the extent and degree of placental invasion of the bladder. Its advantages include: (1) a multiplanar image display that allows the viewing of sections in the sagittal, coronal, and axial planes at the same time, thereby more accurately revealing the location and extent of placental invasion; (2) manipulation of the viewing planes of the spatial angioarchitectural network to better delineate aberrant vessels protruding into the bladder; and (3) the clear display of reconstructed images by live 3D in rotation mode, for a better illustrative effect (Chou et al., 2009).

Video 2.2: Three-dimensional Doppler ultrasonography in placenta percreta (a).

The US scan shows a 3D image of invasive placenta in relation to the bladder

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Video 2.3: Three-dimensional Doppler ultrasonography in placenta percreta (b).

The US scan shows another sequence of 3D images of the case of invasive placenta shown in Video 2.2.

http://dx.doi.org/10.1515/9783110282382_v2.3

2.3.2 Placental magnetic resonance imaging (pMRI)

After initial experience with its use in the third trimester of pregnancy (Kay and Spritzer, 1991), MRI was used for the antenatal diagnosis of placenta percreta (Thorp et al., 1992). Thereafter, some authors made an effort to determine whether placental MRI showed greater diagnostic sensitivity and specificity than US in detecting abnormal placentation. In 1997, for example, the first prospective comparison of the two methods, in a study of 18 patients, was published. The findings did not establish significant differences between the two methods, but it did indicate a theoretically greater sensitivity of placental MRI for identifying abnormal posterior placentation. This was explained by the greater definition provided by placental MRI in the deep plane as compared with US (Ensayes et al., 2009).

The most extensive prospective serial comparison of placental MRI and US so far conducted was published in 2005, which related abnormal placentation and placental MRI. This study did not assess the two methods' diagnostic efficacies, but attempted to determine whether placental MRI provided new information, and sought to determine its value for surgical planning (Palacios-Jaraquemada and Bruno, 2005).

In this study, placental MRI provided new information in 90 % of the cases in which it was used, especially for establishing the surgical topography of placental invasion. This is essential, in that the uterine vascular distribution by region determines the risk of bleeding and also of complications. Additionally, the specific area of invasion is closely related to the likelihood of technical difficulties. This study was also the only complementary study to determine the presence of parametrial invasion, a finding essential to surgical planning for the treatment of abnormal placentation because the presence of placental tissue in the lower pelvis increases the possibility of ureteral damage.

The primary objective in using placental MRI for identifying potentially abnormal placentation is to obtain a well-defined image of the uterine–placental interface and accurate topography of the invasion. Although abnormal placentation stimulates angiogenesis, the NFV are not well developed in the vascular middle (muscular) layer and are highly collapsible, and placental MRI must therefore be performed according to specific technical criteria. Overdistension or collapse of the bladder can cause false-negative or false-positive results. For the specific case of an anterior placenta or placenta previa, a 500- to 600-mL fluid intake at 45 minutes before the study will be essential for producing a semi-full bladder (Palacios-Jaraquemada and Bruno, 2005).

Some signs of abnormal placentation on placental MRI are the absence of a placental–myometrial interface, the presence of confluent lagoons, and exuberant neovascularization, which are similar to the signs of this condition on US. Other signs, such as uterine bulging, a heterogeneous placenta, and hypointensive placental bands, are specific for abnormal placentation (Baughman et al., 2008)

Obstetric MRI is somewhat different from placental MRI in that it focuses on the fetus, rather than putting a diagnostic focus on the uterine–placental interface.

The image field in pMRI will be smaller than that in obstetric MRI because the operator of the imaging device will place the coil in a low position so as to obtain the best image resolution.

Although different techniques can be used in pMRI, ultrafast T2-weighted techniques are recommended. Their technical names vary with different instrument manufacturers, who use their own specific names for different imaging protocols. Nevertheless, ultrafast sequences are commonly used in abnormal placentation. The T2-weighted mode is the first recommended technique for placental MRI because it provides a natural white background – in this case the bladder – for the contrast medium (Palacios-Jaraquemada et al., 2007). This feature makes it possible to efficiently outline the uterine–placental interface (Teo et al., 2009). Because the imaging is done with a rapid sequence, the effect of fetal movement is reduced and a reliable and rapid study can be performed in a simple way. To obtain clear images with minimum artifact, MRI can be used with respiratory gating or with the breath-holding technique. The gating device reveals respiratory movements and acquires images of them; the breath-holding technique obtains images during periods of 15 seconds of maternal apnea.

Sagittal slices in placental MRI allow the classification of placental invasion according to the topography of the S1 and S2 segments of the uterus (detailed explanation in 3.2 section). A placental MRI view in the coronal plane is used to outline invasion, particularly in the parametrium. A view in the axial plane, perpendicular to the posterior bladder wall, is also quite important in providing information about bladder invasion. At this point, the radiologist must modify all of the technical parameters to enhance image precision in the area invaded by the placenta, even at the risk of modifying or producing minor artifacts in the peripheral sector of the scan. The axial plane also provides additional and detailed information about lateral invasion into the parametrium.

When abnormal placentation is suspected in the posterior wall of the uterus (e.g., as the result of abrasive or multiple dilatation and curettage or myomectomy), the white contrast produced by water in the bladder in a T2-weighted scan is not seen. As a result, the image of the placenta may join with that of the posterior myometrium, abdominal viscera, and the vertebral plane, making it very difficult to reach an accurate diagnosis, especially in the lower section of the uterus. In such cases it is advisable to use a gadolinium-based contrast agent and to obtain a placental MRI sequence in the sagittal plane from 60 to 90 seconds after injecting a paramagnetic contrast medium (Thorp et al., 1992). The placental contrast achieved with this will make it readily possible to diagnose posterior abnormal placentation and to establish the limits of the uterine–placental interface.

To date, gadolinium has not shown collateral effects when used for imaging during pregnancy, and there are no reports of its toxicity or of its causing subsequent damage to the fetus (Marcos et al., 1997; Katzberg and McGahan, 2011, Mühler et al., 2011). At present, the use of gadolinium is generally recommended

only for cases in which placental invasion has not been identified through other methods or in which there is a high possibility of maternal risk.

Placental MRI is usually indicated when there is clinical suspicion of abnormal placentation with nonconclusive US study (Warshak et al., 2006; Dwyer et al., 2008). Its main indication, however, is to estimate the extent of invasion and ureteroplacental topography for conservative or resective treatment (Maldjian et al., 1999; Palacios-Jaraquemada and Bruno, 2005; Mazouni et al., 2007), and also to confirm or rule out parametrial invasion. Placental MRI is requested at week 30 when abnormal placentation is suspected; beyond this point, there are no significant changes in the morphology of invasion. A study conducted in 2008 concluded that pelvic US examination is highly reliable means of diagnosing or excluding the presence of a placental adhesive disorder, and found that placental MRI is an excellent tool for the staging and topographic evaluation of these conditions (Maselli et al., 2008).

2.3.3 Placental magnetic resonance imaging (pMRI) gallery

Sagittal series: (Figs. 2.16–2.21), Coronal series: (Figs. 2.22–2.25), Axial series: (Figs. 2.26–2.30)

2.3.4 Serological diagnosis

Advances in biology have allowed prenatal screening for placenta accreta, with the identification of biologic markers for the condition in maternal blood, including cell-free fetal DNA, placental mRNA, and DNA sequences identified by microarray analysis (Mazouni et al., 2007). Some biologic factors, such as creatine kinase levels (Ophir et al., 1999) or elevated levels of alpha-fetoprotein (Kupferminc et al., 1993; Zelop et al., 1992), have in the past been described as reflecting placental dysfunction, but have not been confirmed in this role. Increases in maternal serum levels of alpha-fetoprotein (+2.5) were described in cases of placenta increta and percreta (Hung et al., 1999). At present, no laboratory analysis is considered an essential component of the diagnosis for suspected placenta accreta. Elevated maternal serum levels of alpha-fetoprotein in the second-trimester have been associated with placenta accreta, and a direct relationship between the extent of placental invasion and the increase in the serum concentration of this protein has been suggested. However, none of the markers named here has been evaluated prospectively as a potential component of optimal screening for or of diagnostic thresholds for placenta accreta.

More recent studies have confirmed the abnormal expression of some types of placental mRNA in maternal blood in placenta accreta and some other conditions affecting pregnancy (Miura, 2008). A recent study (El Behery, 2010) suggests an improvement in the results of US for placenta accreta through the simultaneous

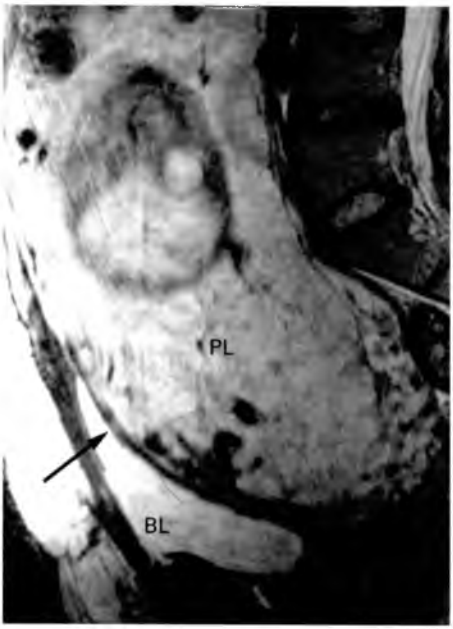

Fig. 2.16: Placental MRI: Sagittal slice with the T2 technique shows a totally occlusive placenta in a patient with three previous cesarean sections. In the circle are multiple a confluent lagoons typical of abnormal placentation. Black arrow: Conserved line between bladder and uterus. PL: Placenta, BL: Bladder.

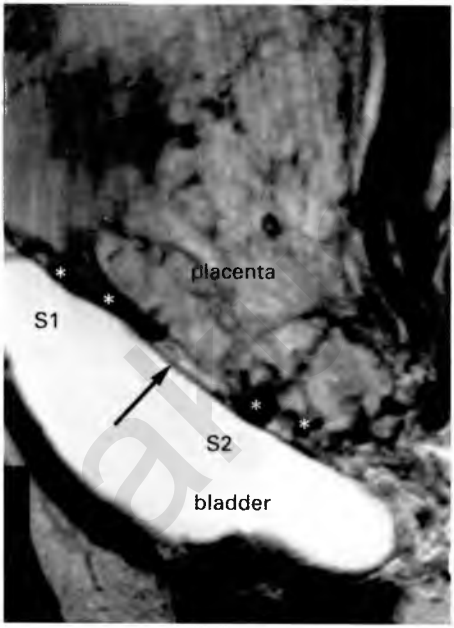

Fig. 2.17: Placental MRI: Sagittal slice with the T2 technique shows a close focus on the invaded area. The arrow shows the only identifiable area of anterior myometrium. Asterisk: Placental invasion in S1 and S2 areas. Newly-formed vessels connect widely placental and vesical circulations typical of placenta percreta with bladder invasion.

assay of cell-free placental mRNA in maternal plasma. However, beyond the measurement of mRNA for human placental lactogen (hPL), no specific mRNA species has been associated with placenta accreta (Simonazzi et al., 2011).

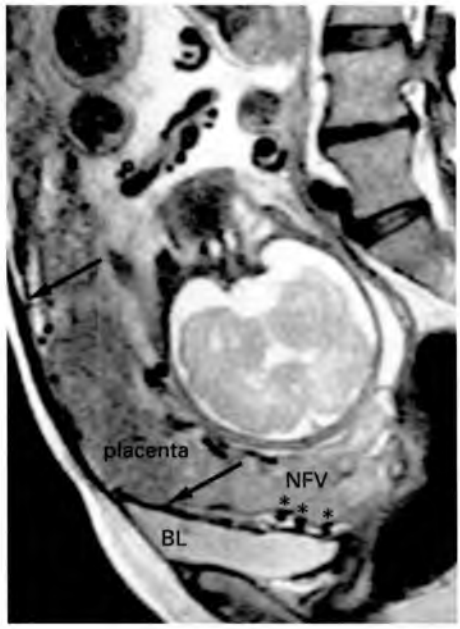

Fig. 2.18: Placental MRI: Sagittal slice with the T2 technique in a patient with complete placenta previa and antecedents of two cesarean sections and curettage after cesarean section by retained trophoblastic tissue. There is no identifiable myometrium between the black arrows. Asterisk marks NFV between placenta and bladder. Note in particular a conserved line between the bladder and placenta, which is optimal for one-step conservative surgery. PL: Placenta, BL: Bladder.

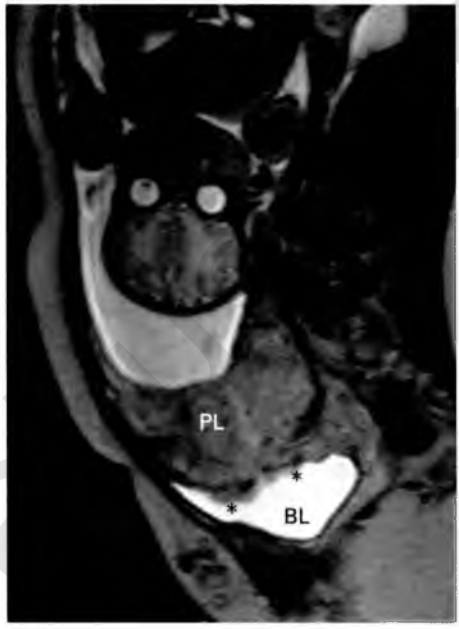

Fig. 2.19: Placental MRI: Sagittal slice with the T2 technique showing a case diagnosed as placenta percreta through US examination. Asterisk shows evident placental tissue inside bladder, but there is no evidence of NFV. Surgical exploration shows a partial rupture of a previous cesarean scar with placental hernia over vesical tissue. One-step conservative surgery was performed and a partial muscular bladder defect was corrected by resection of deformed scar tissue and simple suture.

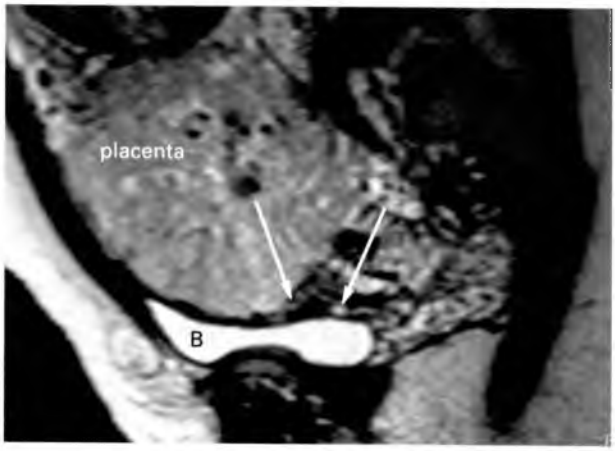

Fig. 2.20: Placental MRI: Sagittal slice with the T2 technique shows an S2 placental invasion with a concentrated area of NFV. This image is almost pathognomonic of trigonal vascular hyperplasia secondary to placental invasion. In such cases a hypertrophic colpouterine anastomotic component grows between the cervix and the trigone. The thickening of the fibrous fascia between these structures makes dissection practically impossible.

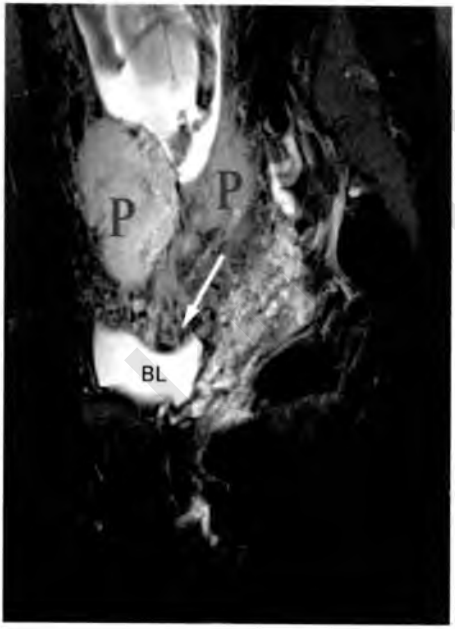

Fig. 2.21: Placental MRI: Sagital slice with the T2 technique shows a massive placental invasion behind the bladder (BL). White arrow show multiple images of NFV. There is no visible signal of normal myometrium in the invasion area.

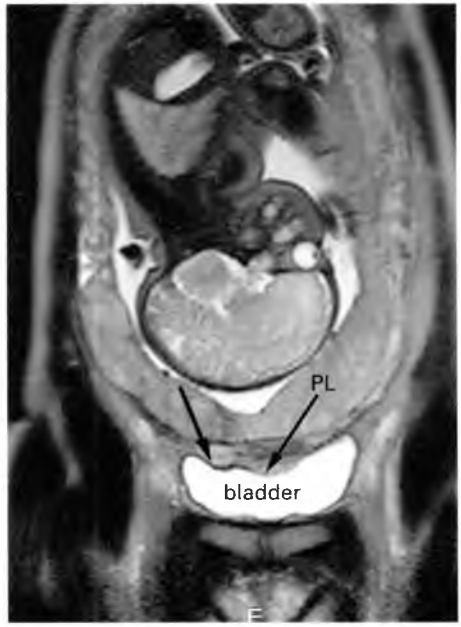

Fig. 2.22: Placental MRI: Coronal slice with the T2 technique in a patient with complete placenta previa and no antecedent risk factors. Ultrasound examinations were positive for possible placenta accreta. Black arrow (left) shows a completely normal interface between the placenta and bladder and black arrow (right) shows a localized placenta percreta. This image is rare in patients without previous antecedent factors; a deep medical interrogation revealed an illegal abortion done 5 years earlier. One-step surgery was performed and the myometrial defect was corrected. PL: Placenta.

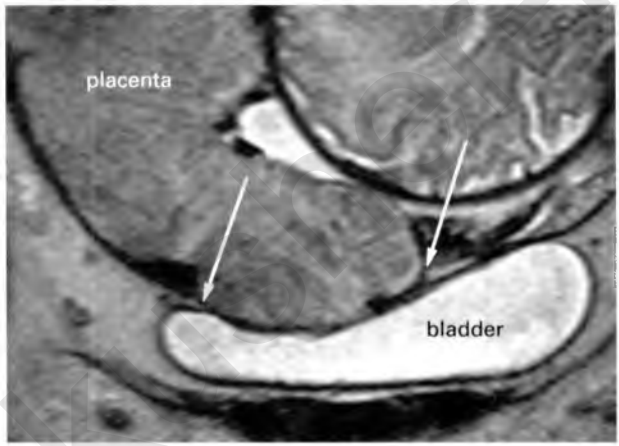

Fig. 2.23: Placental MRI: Coronal slice with the T2 technique in a patient with one previous cesarean section and a US-based diagnosis of placenta previa–accreta–percreta. White arrows show limits of ruptured previous scar; notice the absence of NFV in the "apparent" invasion area. Cesarean section though a Pfannenstiel incision was performed and the defect was corrected.

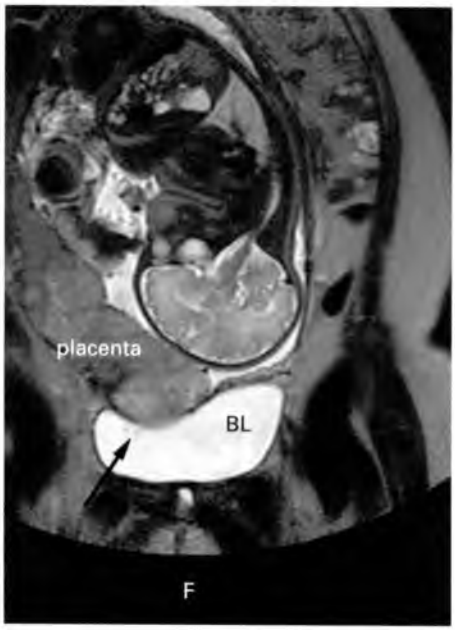

Fig. 2.24: Placental magnetic resonance imaging (pMRI): Coronal slice with the T2 technique in a patient with one previous pregnancy and a spontaneous abortion at 18 weeks. One week after this a D&C was done to remove infected retained trophoblastic tissue. Placenta previa was diagnosed in the current pregnancy through US imaging. Placental MRI shows a myometrial defect with placental advancement without evidence of (black arrow) NFV. Surgical exploration showed a partial dehiscence of the uterine segment. Both borders were cut and uterine repair was performed.

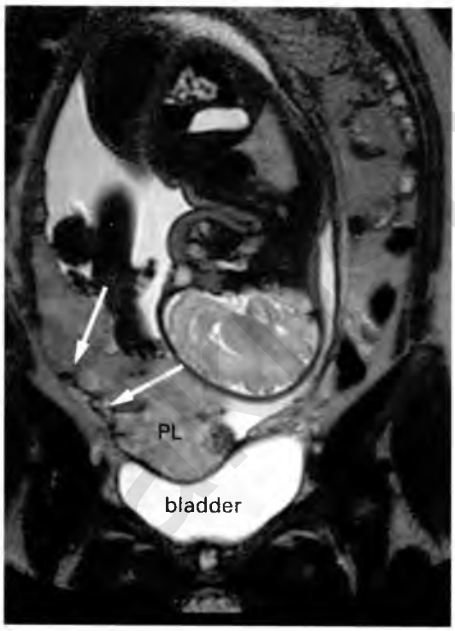

Fig. 2.25: Placental MRI: Coronal slice with the T2 technique in a study requested after doubt about a US diagnosis of placenta previa accreta in patient without recognizable antecedents (first pregnancy). White arrows show an evident area of abnormal placentation. An extensive private interview after study of the patient's case revealed three previous abortions. Placenta percreta was confirmed in the operating room and uterine repair was done in the same procedure.

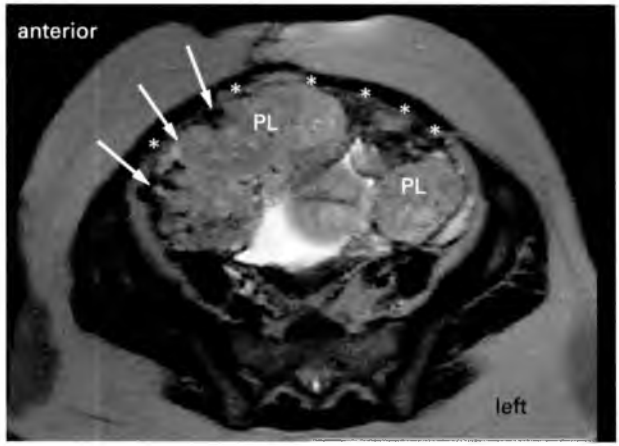

Fig. 2.26: Placental MRI: Axial slice with the T2 technique showing massive anterior and parametrial invasion (white arrows). Asterisks show multiples areas without any evidence of myometrium. Resective surgery in these cases is dangerous, and is recommended only with the availability of all necessary resources. It is important to remember that MRI is the only imaging method that can provide a diagnosis of parametrial invasion, and for this reason its use is strongly recommended in cases of one-step conservative surgery when hysterectomy is a possible treatment. PL: Placenta.

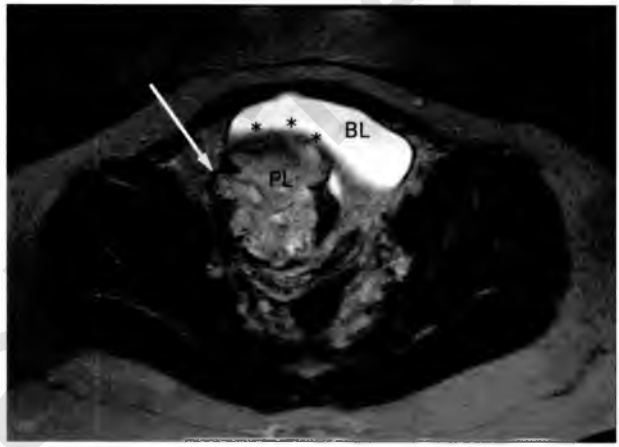

Fig. 2.27: Placental MRI: Coronal slice with the T2 technique in a patient with placenta previa acreta diagnosed by US. Placental MRI was done because of the patient's persistent low abdominal pain. Asterisks show an absence of myometrium behind the bladder, which was found in coincidence with the diagnosis of placenta accreta. However, placental MRI was localized to an area of parametrial invasion (white arrow: placenta percreta) and surgery was scheduled for the next day. The diagnosis of parametrial invasion was confirmed during surgery.

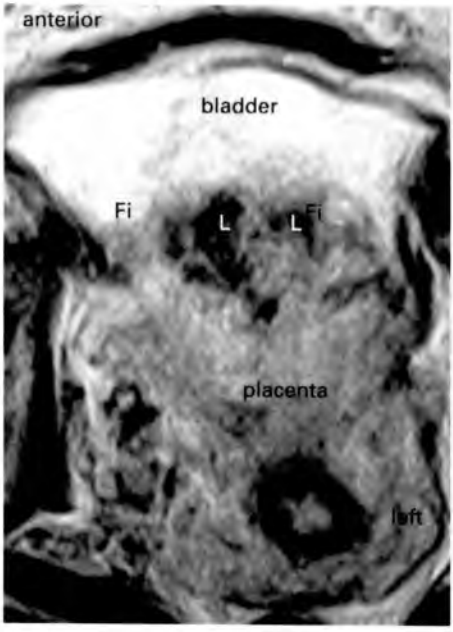

Fig. 2.28: Placental MRI: Axial slice with the T2 technique showing seemingly fibrous tissue (Fi) between the bladder and placentally invaded tissue. Placental lagoons are evident in some invaded areas (L). This type of fibrosis is infrequent but very difficult to manage when total hysterectomy is planned. Technically, one-step surgery can be performed with restrictions, but by coincidence or not, the only case (1 in 82 cases) of recurrence after one-step conservative surgery was seen in a patient with the locally extended fibrosis seen here. Hysterectomy is usually extremely difficult in this setting because the dissection it entails can cause serious damage in the lower part of the bladder. One possible solution is to remove the placenta manually and leave part of the uterine segment and cervix in place. Placement of uterine circular hemostatic knots around the lower circumference of the uterus, which includes vessels within the myometrium, is needed to avoid late bleeding. The knots produce hemostasis through simple tissue compression without the need for further measures.

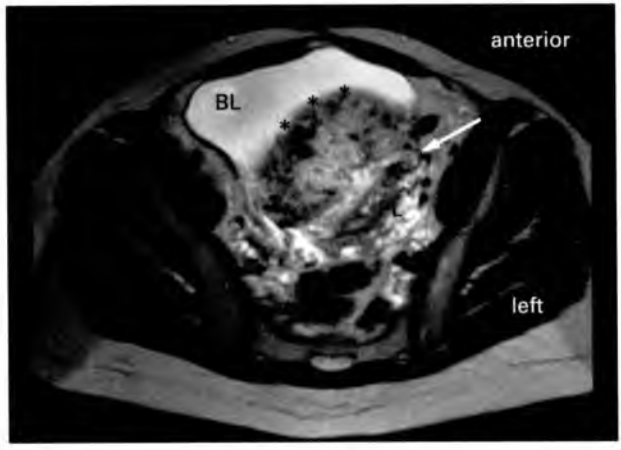

Fig. 2.29: Placental MRI: Low axial slice with the T2 technique: is the image shows one of the complex combinations in abnormal placentation: Parametrial invasion (PI) with invasion of the bladder and trigone (asterisk). The white arrow shows placental tissue outside of the uterus, without any recognizable myometrial tissue. Bladder invasion, shown by the asterisk, covers all the lower area of the bladder (trigone). This area contains many NFV. When resective surgery is planned such (e.g., hysterectomy), the use of aortic vascular control is strongly recommended to avoid bleeding from the pelvic–subperitoneal vessels. Accurate vascular control improves safety during dissection and allows the recognition of structures such as blood vessels and the ureter.

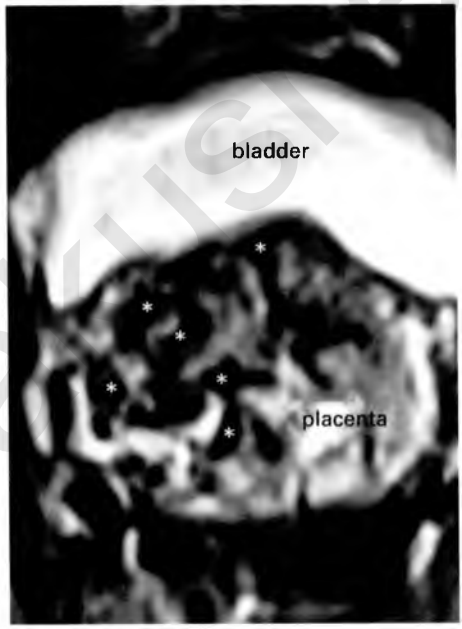

Fig. 2.30: Placental MRI: Axial slice with the T2 technique. Although it can also be true with other methods, the results of placental MRI are operator-dependent. This image shows a massive case of anterior placenta percreta originally reported as "normal." The asterisk shows multiple NFV and lagoons inside the placenta. During the surgery done in this case, it was found that most of the muscular layer of the uterus was replaced by multiple NFV, which made it almost impossible to recognize the bladder tissue even for the senior urologist. Correlation between auxiliary diagnostic methods (US, MRI) and surgical images is strongly recommended as background training for the radiologist.

Clinical suspicion	Ultrasound	Doppler	3d Doppler	Placental MRI (pMRI)	Surgical exploration	Hystological study
Placenta previa+ previous cesarean	At-risk patients, us examination is positive by 18 weeks	Does not improve diagnostic accuracy	Differential diagnosis with placenta previa	Interrupted myometrial line between placenta and uterus	Topography and extension of invasion	Not useful as gold standard diagnosis
Previous multiple cesarean	Echogenic bladder line is absent	Color Doppler us accentuates vascular spaces	Diagnosis of vesical invasion	Presence of confluent lagoons	Presence of parametrial or bladder invasion	Retrospective and not valuable to clinico-surgical decision
Previous cesarean and curettage	Long, narrow vascular channels	Postoperative control of placental involution in in situ placenta		Presence of perpendicular newly-formed vessels between bladder and uterus	Tissue thinning, presence of fibrosis	It can be different according to the specific origin of the sample
Abrasive uterine curettage	Four to six irregular lacunae or more present in 100 % of cases,			Lateral rupture of myometrium by placenta. Parametrial invasion	Anatomy of newly-formed vessels	
Any kind of uterine surgery with myometrial injury	Loss of the clear space between the placenta and uterus is the most important cause of false positives			Topography and extension of area invaded by placenta S1–S2 areas and bladder invasion		
Endometrial damage caused by thermo-ablation or pelvic radiation				Differential diagnosis with placenta previa. thick parallel vessels		
				To optimize diagnostic accuracy.		

Tab. 2.1: Diagnosis of abnormal placentation.

Ultrasound	Doppler and power Doppler ultrasonography	Placental MRI
Multiple vascular lacunae	Intense blood flow within sonolucent placental spaces	Multiple vascular lacunae (Dark intraplacental bands on T2-weighted image)
Loss of normal hypo-echoic retroplacental area	Hypervascularization patterns within the placental and nonplacental tissues	Presence of newly formed vessels connectinguterus, placenta, and sorrounding tissues
Retroplacental myo-metrial thickness less than 1 mm	Highly pulsatile venous flow patterns in: placental blood lakes and sub-placental venous complex	Myometrial thinning with placental advancement
Vessels or placental tissue crossing uterine surface or myometrium–bladder interface	Prominent low-impedance arterial blood flow within the uterine serosa and posterior bladder wall	Lower uterine bulging
		Rupture of myometrial layer by placental tissue

Tab. 2.2: Auxiliary diagnosis signs of abnormal placentation.

3 Surgical anatomy

As surgical techniques have become more sophisticated, it has become clear that new, focused anatomic knowledge is hended for the surgical management of abnormal placentation. The study of normal reproductive anatomy is generally included in the syllabus for the initial years of medical study. Schools of medicine have two main ways of teaching anatomy, one of which treats it as a subject integrated with embryology and histology, and the other as a full-year study. Both methods have advantages and disadvantages. It is very practical to introduce an integrated subject in the general study of body structure, tissue organization, and physiology. This simplifies the understanding of the overall configuration of the body, but it is incomplete. Often, models are used to explain anatomic structures. A one-year study of anatomy typically examines systems and organs in detail, and the use of embalmed dissections is usual for teaching. However, analysis has demonstrated a low level of residual anatomic knowledge in the years following this, even in students showing a high level of performance in such initial study. For this reason, some medical schools have included additional anatomic knowledge in the initial teaching of various medical specialties.

During postgraduate training and residency, surgical anatomy is reviewed both theoretically and practically during surgical procedures (Macchi et al., 2003). However, this may be insufficient to practice in terms of providing practice experience in some procedures or in conditions in which normal anatomy is distorted, such as placental adhesive disorders.

3.1 Anatomic and surgical problems

From an anatomic point of view, access to the subperitoneal sector of the female pelvis requires knowledge of certain details related to the blood supply of this region and surgical management of the ureter and bladder.

Irrigation of the genital organs below the uterine segment depends on a series of pedicles stemming from the internal pudendal and internal iliac arteries (Fig. 3.1) (Palacios-Jaraquemada et al., 2007). Except for the lower vaginal artery (internal pudendal artery), the arterial branches in this region are of middle size or small diameter, which makes their identification and specific hemostasis difficult. In addition to this problem, anatomic access is limited by the narrowing of the pelvis, which limits the surgeon's maneuverability.

Despite the space-specific anatomic restrictions of the pelvic region, adequate dissection of the fasciae and subperitoneal pelvic fat spaces permits solving a significant number of the problems described. However, reducing the morbidity of low pelvic dissection makes it almost essential to identify and move the ureter and bladder (Fig. 3.2).

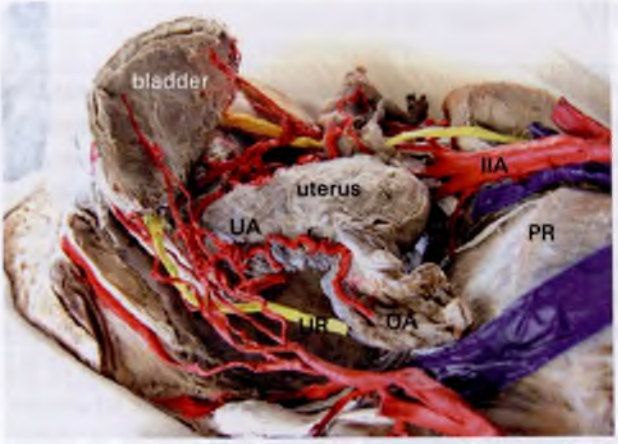

Fig. 3.1: Embalmed anatomic specimen of female pelvis (pelvic irrigation) showing the blood-supply system (superior and right lateral view). In the midline are seen the bladder, uterus, and PR: Promontory, IIA: Internal iliac artery, UR: Ureter, UA: Uterine artery, OA: Ovarian artery. Note the wide anastomotic component behind the bladder and around the lower ureter, among the uterus, vagina, and bladder.

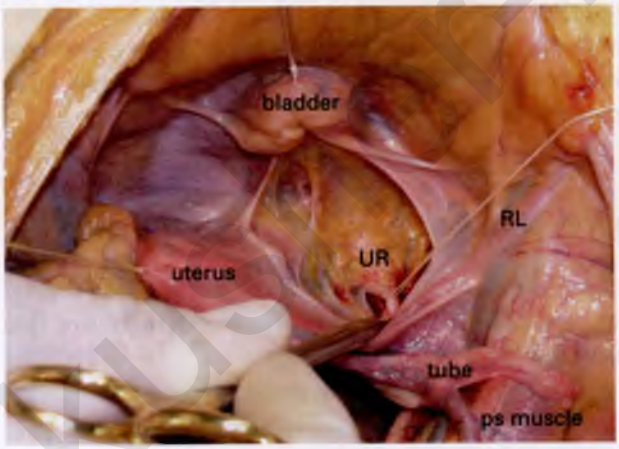

Fig. 3.2: Dissected fresh corpse. View of the pelvic–subperitoneal space and ureteral dissection. PS Muscle: Psoas iliac muscle, UR: Ureter, RL: Round ligament.

Irrigation of the pelvis is provided by an extense network of widely intercommunicating vessels, which immediately replace interrupted flow in any of their branches. Arterial irrigation is mainly provided by the internal iliac artery and its branches; however, the pelvis also receives an anastomotic collateral circulation depending on the abdominal aorta, external iliac artery, and femoral artery (Palacios-Jaraquemada, 1997).

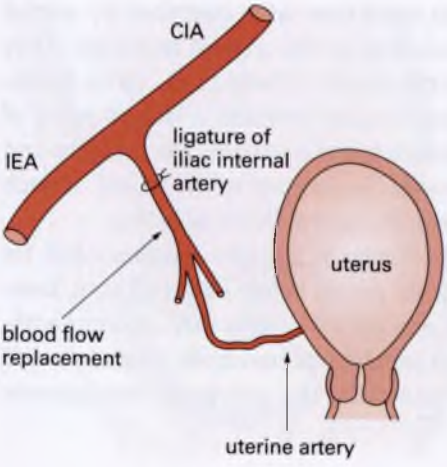

Fig. 3.3: Scheme of anastomotic revascularization after ligation of the iliac internal artery. Arterial pressure after ligation decreases by 70 % but blood flow by only 50 %. Collateral vessels from the internal iliac artery, external iliac artery, femoral artery, and aorta are numerous, and for this reason return of blood flow such ligation is immediate.

Because of the multiple origins of the anastomotic components of the pelvic circulation, it is important to inhibit pelvic bleeding by ligation of its main trunks.

The first ligature of the trunk of the internal iliac artery was probably done in Montevideo, Uruguay, in 1812. At that time the procedure was used as a coadjuvant treatment to resect an aneurysm in the thigh. However, the best known reference to ligature of the trunk of the internal iliac artery might be one published in 1894 in Baltimore (Kelly, 1894) in the bulletin of the Johns Hopkins Hospital, which described this ligature as the most daring procedure for controlling pelvic bleeding. In the particular case described, this arterial ligature was used to inhibit an otherwise unstoppable hemorrhage in a patient with advanced cervical cancer.

Since then, several authors have used this procedure with the goal of reducing hemorrhage in gynecologic and obstetric surgeries. Rubovitz, in 1940, reported the first use of ligature of the internal iliac artery in obstetrics, to inhibit massive bleeding following an episiotomy. Some authors used this technique to improve the hemostatic and general conditions during surgery or as prophylaxis in procedures in which there was the possibility of serious bleeding. Some years later, ligature of the ovarian artery was added as an extension of the basic procedure, although this modification was not altogether successful (Siegel and Mengert, 1961).

After several controversies about its actual value, a series of experiments were conducted with the goal of showing the arterial physiologic changes following ligation of the internal iliac artery. These studies provide a basis for understanding why such ligation fails in more than 50 % of cases. They explain that after ligation of the internal iliac artery, pulse pressure distal to the ligation is reduced by 80 %, although arterial blood flow remains constant to within 50 % because of extensive collateral blood flow. This phenomenon would be the result of an instantaneous deviation of blood flow through the pelvic anastomotic collateral vessels (Burchell, 1964, 1968; Burchell and Olson, 1966) (Fig. 3.3).

Ligation of the internal iliact artery has since then been described by several authors who have in turn used multiple variations to the original technique. They have reported differing and even contradictory results (Chelli et al., 2010; Evans, 1985), but a retrospective review of their experiences indicates a lack of safety of ligation of the internal iliac artery as a common factor caused by the constant and high number of anastomotic collateral vessels, in contrast to the safety of such methods as proximal arterial control in cases of massive pelvic bleeding.

The use of guidelines for alternative treatment is strongly recommended for unexpected complications occurring during the period before surgery. Labor, hemorrhage, possible rupture, and the need for emergency surgery may all occur without the customarily required treatment team members or resources. Guidelines and periodic workshops are recommended for improving the emergency management skills of the available team members.

3.2 Uterine blood supply

No new study of the uterine arterial blood supply has been done for many years. During the nineteenth century, numerous anatomic descriptions referred to branching variations and anastomoses in the uterine, ovarian, and vaginal blood supplies (Bergman et al., 2011). In the middle of the twentieth century, a complete arteriographic study of the uterine artery was done in relation to the arteriographic patterns observed in different gynecologic and obstetric diseases (Fernström, 1955; Borel and Fernstrom, 1953), and one of the most comprehensive anatomic descriptions of the human arterial system was published a few years before this (Belou, 1934). Although it is today almost unknown, this work contains a complete graphic study of the genital arterial system, and full descriptions of its anastomoses with adjacent systems. Anatomic study of the uterine artery has since then apparently become less interesting, most probably because of a lack of its practical application. However, the introduction of selective procedures for uterine devascularization, such as embolization or surgical arterial ligation, has raised a number of questions about the variations in and anastomoses among the uterine, ovarian, and vaginal arterial pedicles.

Classically, two bilateral uterine pedicles have been described. The major pair of these pedicles is that of the uterine arteries and the second, upper pair of pedicles is that of the ovarian arteries, with these pedicles carrying 90% and 10%, respectively, of the blood flow to the uterus. A third pair of pedicles, which is not always present, has been described as an accessory pair; these supplementary vessels are provided by the arteries of the round ligament, which arise from the epigastric artery. With the advent of embolization techniques, it was thought that the upper pedicles maintained uterine blood flow after bilateral occlusion of the uterine arteries. However, this assumption lost credibility after studies of controlled ischemia performed for use of the Flostat™ clamp (Vascular Control Systems, San

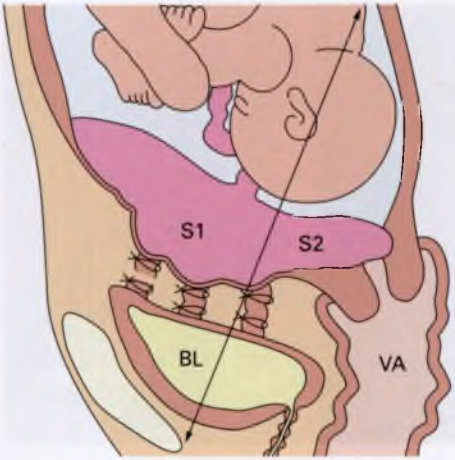

Fig. 3.4: Sagittal scheme showing the division of the S1 and S2 genital vascular regions.

Juan Capistrano, CA). This device occludes both uterine arteries and the adjacent myometrium until the blood flow through both uterine arteries, as seen by Doppler US, is stopped. However, the finding of uterine ischemia within 6 hours despite complete patency of the upper pedicles (ovarian artery and round-ligament artery) excluded the classical theory of revascularization through the upper pedicle (Wranning, 2005). Because compression with Flostat clamping leaves the upper pedicle open, a uterine blood supply is, according to classic theory, guaranteed in case of bilateral occlusion of the uterine arteries. Yet not withstanding this, uterine ischemia appeared after 6 hours, demonstrating the absence of a supplementary blood supply through the upper pedicle. Not until 2007 did an anatomic study show the presence of a thick lower uterine anastomotic component of the uterine blood supply (Palacios-Jaraquemada, 2007). This supplementary system, stemming from the vaginal arterial system, is of greater diameter than the uterine artery itself, and can restore uterine blood flow after occlusion of its two main arterial sources, the uterine arteries. This replacement was also reported angiographically in vivo, and the entire blood flow to the uterus could be visualized through the injection of contrast agents solely into the lower vaginal artery. When the Flostat™ device is applied,the pedicles of both the uterine and vaginal arteries are simultaneously occluded. Consequently the main supplementary system of blood supply to the uterus, provided by the vaginal arterial pedicles, is blocked, causing the uterine ischemia that was observed within 6 hours.

Additional anatomic studies have shown that the female reproductive system has two distinguishable vascular areas (Palacios-Jaraquemada et al., 2005, 2007). If in a sagittal slice of pelvis an imaginary line is drawn perpendicular to the middle sector of the posterior bladder wall, there can be identified a topographic region that comprises the uterine body, labeled S1, and a lower topographic region, corresponding to the lower uterine segment, cervix, and upper part of the vagina, labeled S2 (Fig. 3.4). The S1 region is supplied with blood by collateral vessels of

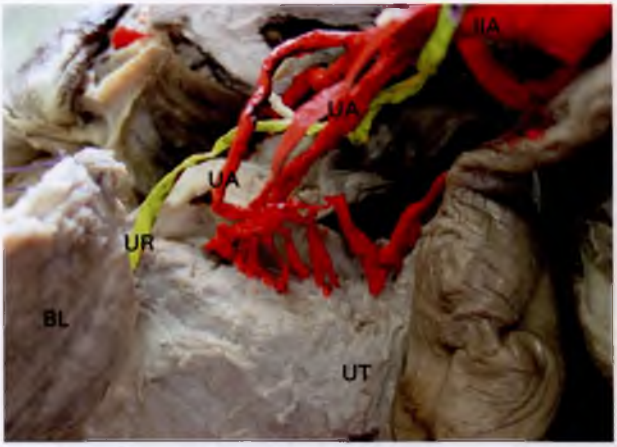

Fig. 3.5: Superior view of the right uterine pedicles in an embalmed corpse. A double right uterine artery (UA) was seen to arise from the anterior division of the internal iliac artery (IIA); both right uterine arteries are seen relation to the ureter (UR). BL: Bladder, UT: Uterus.

the uterine artery and, to a lesser extent, by the ovarian artery; consequently, methods for occluding, ligating, or compressing the branches of these vessels will efficiently stop bleeding in this region. On the other hand, the S2 region is supplied by five subperitoneal vascular pedicles — those of the upper, middle, and lower vaginal arteries, the inferior vesical artery, and the internal pudendal artery — and for this reason the hemostatic mechanisms applied in the S1 region (for the uterine artery or its branches) are usually ineffective for controlling bleeding in the S2 region; instead, specific hemostatic procedures must be applied in the S2 region to achieve effective hemostasis. It is an interesting point that hemostasis in each of the two regions depends on the origin of its blood supply regardless of the primary condition affecting the uterus.

Few variations in the uterine artery have been described (Fig. 3.5). There have been two phases in the study of this artery. The first was conducted by anatomists in the nineteenth century and the second by radiologists in the past decade. Apart from having different points of origin, the uterine artery does not have a complex anatomy and is relatively easy to capture in radiologic studies. On the contrary, the vascular pedicles in the S2 region described above are more complex as the result of having multiple origins (Fig. 3.6). The upper and middle pedicles of the vaginal artery usually arise from the internal iliac artery, the uterine artery, or the lower vesical artery, but the lower vaginal artery stems almost exclusively from the internal pudendal artery. This is a very important point because the lower vaginal artery is a thick anastomotic pedicle, usually one-third thicker than the uterine artery (Fig. 3.7). The internal pudendal artery is the anterior trunk of the posterior division of the iliac internal artery. This pedicle can therefore be a source of blood loss in cases of lower uterine or vaginal bleeding. In such cases, radiologic explora-

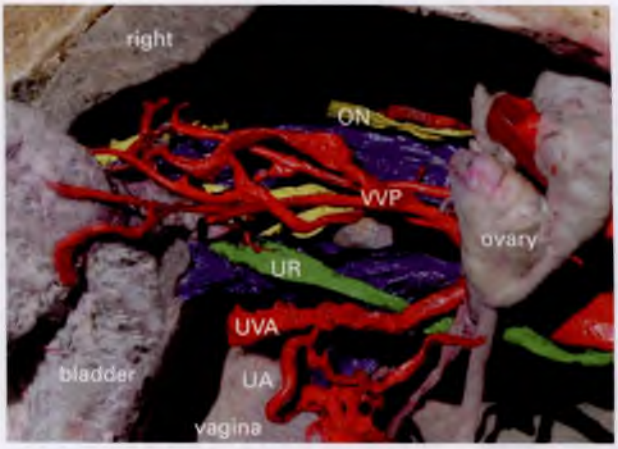

Fig. 3.6: Embalmed corpse: Superior view of right hemipelvis. The technical complexity of treatment in the S2 region is due to the great number of vessels in a narrow space. UA: Uterine artery, UVA: Upper vaginal artery, UR: Ureter, VVP: Vesico–vaginal pedicle, ON: Obturator nerve. In yellow and behind of the vesico-vaginal pedicle is the vesical autonomic branch of hypogastric plexus.

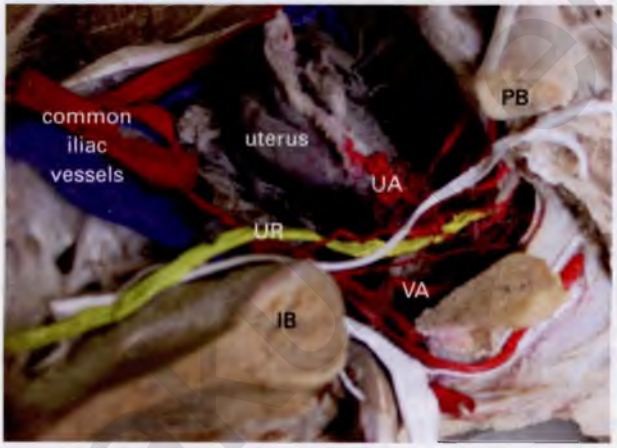

Fig. 3.7: Embalmed corpse: Right lateral view of parametrium after partial resection of iliac bone (IB) and pubis (PB). Right uterine (UA) and vaginal artery (VA) are separated by the ureter (UR). Notice the caliber of the vaginal artery (internal pudendal–posterior branch of the internal iliac artery) with respect to the uterine artery (internal iliac–anterior division).

tion of the anterior branch of the internal iliac artery and the uterine artery can yield negative results, even with active bleeding. In some cases of conservative treatment with removal of the placenta, uterine arterial embolization, and compressive sutures over the invaded area, there may be silent hypotension without

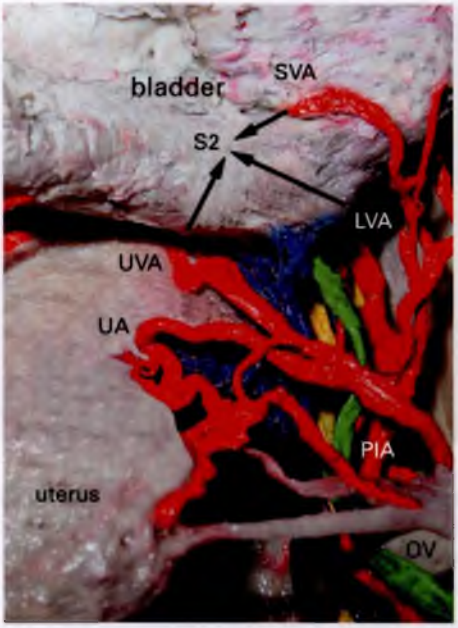

Fig. 3.8: Embalmed corpse: Superior view of right hemipelvis (anteroposterior view): The lower vaginal artery (LVA) arises from the internal pudendal artery (PIA) and is thicker than the uterine artery (UA). The upper (UVA) and lower vaginal artery provide the main collateral blood supply to the S2 region in abnormal placentation. Black arrows show the main collateral blood supply in S2 invasion. UR: Ureter, OV: Ovary, SVA: Superior vesical artery.

any evidence of bleeding. If after conservative or resective surgery for abnormal placentation a patient inexplicably develops hypotension, without evidence of pelvic drainage or vaginal bleeding, negative findings on US, and a negative anterior iliac angiogram, a retroperitoneal hematoma should immediately be considered.

Because the main blood supply to the S2 region is pelvic and subperitoneal, bleeding in this region can be clinically occult. In such cases CT or surgical exploration is needed, and the interventional radiologist must explore the entire arterial tree below the site at which the renal arteries arises. Branches of the lower, middle, and upper vaginal arteries usually provide accessory blood flow in cases of abnormal anterior placentation. This collateral blood flow reaches the uterus in two major ways: through the vesical arteries and through the utero–vaginal arterial anastomosis (Fig. 3.8). This explains why uterine artery embolization accounts for most failures in the treatment of postpartum hemorrhage, in which it can fail in about 40 % of cases (Chou et al., 2004). Study of the normal anatomy of the region shows that there are no significant pedicles between the uterus and the bladder, yet it is well known that in cases of abnormal placentation there is extensive circulation between these two organs. This apparent contradiction can be explained by embryology. During fetal angiogenesis, vascularization produces a network of interconnected vessels in the pelvis (Neas, 2011). After tissue development, specific vessels become enlarged and evolve into definitive organ pedicles; on the other hand, there occur microanastomoses among vessels in the early vascular tree that can be seen microscopically. In cases of abnormal invasion, the placenta produces a high level of angiogenic factors, which are responsible for the enlargement of

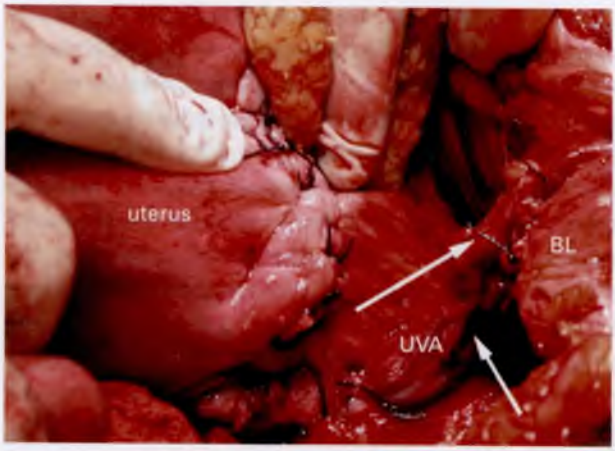

Fig. 3.9: Vesico–uterine space after one-step conservative surgery. All of the placentally invaded area and the entire placenta were removed. The utero–vaginal anastomotic pedicle (UVA) is seen with a vaginal branch (white arrow) and a direct anastomotic vessel to the bladder (black arrow). If embolic material is put into this vessel, it may cause unwanted bladder embolization.

undetectable microanastomoses and vascular remodeling. Vascular endothelial growth factor (VEGF) has shown significantly higher levels in placenta accreta than in normal pregnancies (Tseng et al., 2006). As a result of stimulation by VEGF, an array of newly formed vessels provides added blood flow to the area of invasion. These vessels have a nonclassical anatomic pattern and their structure is very different from that of normal vessels. The absence or minimal development of the tunica media reveals its rapid development as the result of stimulation by vascular growth factors.

This type of circulation must be considered as anterior placenta percreta, especially when it is used for arterial embolization. The passage of small embolic particles through newly formed vessels may be the cause of unwanted or nontargeted embolization, which is known to cause extrauterine tissue necrosis (Fig. 3.9).

3.3 Induced vascular neoformation

Twenty-two years of study of disorders of placental adhesion and direct familiarity with personally treated cases of this disorder have shown that neoformed vessels (NFV) are usually associated with disorders such as placenta accreta and placenta percreta (Palacios-Jaraquemada, 2011). This anastomotic blood supply links the placenta, the uterus, the bladder, and their adjacent tissues. These NFV are vessels with a large axial diameter and allow an adequate vascular blood supply to the placenta. Following the establishment of abnormal placental adherence, the proliferation of angiogenic factors induces the development of NFV to increase the

volume of placental blood flow. However, this change increases the fragility of these vessels through underdevelopment of their tunica media. At first sight, these vessels have a disordered pattern, but this group of arterial and venous vessels can be organized into the following three anastomotic levels: (1) a vesicouterine system (VUS); (2) a placental–vesical system (PVS); and (3) a colpouterine system (CUS). The VUS habitually involves vessels that connect the uterine arteries with the posterior-superior bladder wall and also with the contralateral uterine artery through a transmedial interuterine anastomosis (TIA). This low transverse uterine anastomosis has an axial caliber equivalent to that of the uterine artery, is always located in the anterior cervical–isthmic vascular confluence, and widely connects the upper, middle, and lower arterial pedicles.

The VUS is formed by superficial vessels, and can be observed through the vesicouterine fold and perpendicularly from the uterus to the bladder. The presence of direct anastomoses of considerable size between the uterine arteries and the bladder must be taken into consideration, especially during endovascular occlusion, because these direct communications can lead to undesired embolization in the vesical parenchyma.

The placental–vesical system is probably the best known of the three systems of NFV seen in cases of abnormal placental adherence. It establishes a rich connection between the placental vasculature and that of the muscular layer of the bladder; it is morphologically perpendicular to the vesicouterine plane, unlike the exuberant myometrial vascularization of placenta previa, in which the vessels are parallel to the myometrium. There is no morphology that excludes a PVS, and the envolved vessels have been observed as thin and numerous anastomotic vessels as well as thick, easily identifiable cords. The PVS can send vessels to and receive vessels from the entire surface of the posterior bladder wall, and can have extensive connections with branches of the upper and lower vesical arteries (internal iliac and internal pudendal arteries), although it frequently has such connections with the upper vesical pedicle.

The CUS is anatomically the least known and most hidden of the thres systems of NFV associated with disorders of placental adherence, yet probably the most physiologically important of these systems. The CUS is located in the tic region of the anterior vaginal wall and is parallel to its long axis. Consequently, this system can be identified only through deep dissection of the retrovesical space. Occasionally, however, the CUS may not initially be visible with such dissection because its macroscopic visualization depends on the degree of anastomotic vascular development in this system, which may vary from imperceptible cords to replacement of the muscular tunic of the vaginal wall for a noticeably developed vascular plexus (vascular pseudometaplasia). The CUS connects the lower, middle, and upper vaginal pedicles to the caudal branches of the uterine artery, as well as to the intrauterine arcade of anastomoses with the ovarian artery, and is located anatomically between the muscular fibers of the uterine wall. The azygos artery of the vagina is probably one of the best well-known components of the CUS to gynecologists.

Technically, blood flow in the vesicouterine and placental–vesical anastomotic pedicles is likely to be controlled by endovascular occlusion of the uterine arteries (via transanastomotic flow) or by occlusion of the anterior branches of the internal iliac artery. However, hemostasis at the colpouterine pedicle can be very difficult or nearly impossible to achieve, since it would require arresting flow through a high-flow anastomotic arcade between the vaginal and uterine arterial pedicles. Selective endovascular occlusion may be performed through the selective use of microcatheters. On the other hand, hemostasis in the CUS can be achieved in a simple, efficient, and safe way with compression with square sutures (Cho et al., 2000).

Video 3.1: Flow replacement after uterine artery embolization.

Experimental studies have demonstrated that the upper uterine anastomotic pedicle (ovarian artery) cannot maintain uterine vitality after bilateral occlusion of the uterine artery. The lower uterine anastomotic system is provided by the vaginal arteries, which replace uterine blood flow after bilateral arterial embolization. Simultaneous occlusion of both the uterine and vaginal arterial systems creates a high risk of uterine necrosis.

http://dx.doi.org/10.1515/9783110282382_v3.1

Video 3.2: Anastomotic embolization.

Development of NFV increases the degree of microscopic anastomosis of vessels of the uterus, bladder, and vagina in abnormal placentation. Unwanted organ embolization can occur if embolic particles are derived from these vessels. Because descriptions of normal anatomy do not include vesical branches of the uterine artery, their presence must be anticipated in order to avoid nontargeted embolization, which can result in unwanted organ ischemia or necrosis.

http://dx.doi.org/10.1515/9783110282382_v3.2

3.4 Bladder dissection

Dissection of the bladder and effective management of the ureter are very important in the surgical management of abnormal placentation. Both of these structures are typically involved in anterior placental invasion or during surgery for it. An abnormal blood supply, adhesions, a narrow operating space, and lack of dissection planes may make this surgery exceedingly difficult for the obstetrician and urologist (Abbas et al., 2000; Parva et al., 2010) (Figs. 3.10 and 3.11). One option for preventing this is to avoid touching the tissues invaded by the placenta and leaving the placenta in situ, but on some occasions it is necessary to perform a hysterectomy, and in such cases urologic and bladder dissection are mandatory for avoiding unwanted damage.

In some countries, partial resection of the bladder is usual or considered almost necessary during the surgical treatment of anterior abnormal placentation,

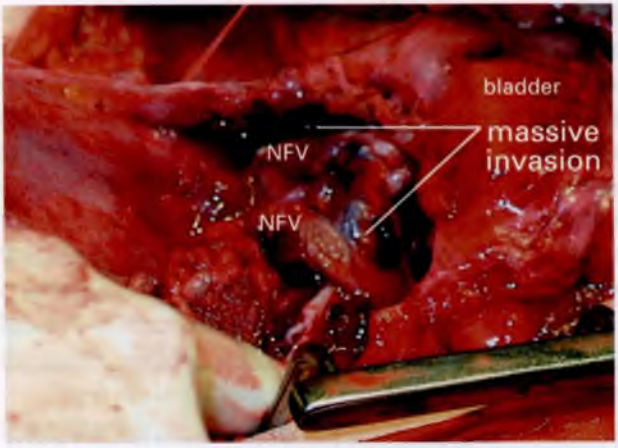

Fig. 3.10: Massive lower vesical and trigonal invasion. The NFV are dense and connect the invaded area with a vesico–vaginal anastomotic pedicle. Dissection of this area without accurate proximal vascular control is almost impossible.

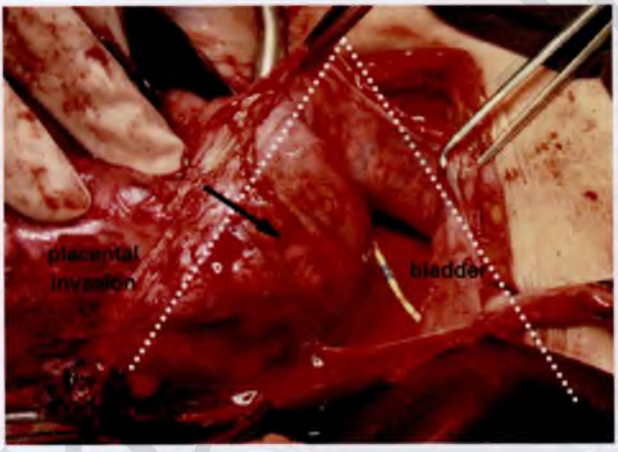

Fig. 3.11: Intraoperative photograph showing abnormally adherent placental invasion through the full wall of the bladder, with a thin intact mucosa. A ureteral stent can be visualized in the open bladder trigone.

especially in cases of placenta percreta (Konijeti at al., 2009), in which excision or reconstruction of the bladder may be necessary, depending on the severity of hemorrhage and the depth of invasion of the placenta into the bladder (Washecka and Behling, 2002; Matsubara, 2010). Although some authors have elected to use this procedure, augmentation cystoplasty after cesarean hysterectomy for placental invasion of the bladder may cause morbidity in young women with a urinary bladder of small capacity (Bakri et al., 1995). Nevertheless, careful dissection allows

separation of the bladder from the uterus and placenta. In contrast to the situation with neoplasia, complete removal of tissue is not mandatory because the placenta is not an invasive tumor; the only purpose of placental invasion and the NFV that accompany it is the provision of an additional blood supply to compensate for lack of myometrial support. Technically, the advantage of partial resection of vesical tissues is to minimize blood loss and also to facilitate the dissection, and although reduction of bladder capacity can produce serious morbidity in young women, it may in benign processes be avoidable with adequate measures.

The bladder has two sets of bilateral pedicles, which enter through the superior and inferior lateral vesical wall, respectively. The superior vesical artery arises from the internal iliac artery and the inferior vesical artery arises from either the internal pudendal artery or internal iliac artery (Standring, 2008). Two bilateral pedicles are widely anastomosed inside the muscular (detrusor) layer of the bladder wall and also anastomose with the vaginal pedicles. The resulting vascular network provides wide anastomotic vascularization, which allows the use of any type of knot or suture over the muscular layer without compromising irrigation, although the network can be obstructed by small particles during embolization, and for years it was thought that unwanted bladder necrosis was the result of reflux of particles into the iliac arterial system. However, in cases of abnormal placentation, growth of the anastomotic vascular network between bladder and the uterus is common, and represent a direct link between two arteries. This network is not described in textbooks, but was recently documented in necropsies and surgeries (Palacios-Jaraquemada et al., 2007).

Retrovesical dissection is not a common practice in standard obstetric training, and for this reason fear of performing it is common. Unwanted vesical damage, the possibility of fistula, and unexpected bleeding are the most common problems in such dissection. However, safe retrovesical dissection is feasible and with the use of simple preventive measures can be accomplished without common complications.

In general, an initial view of anterior abnormal placentation might suggest that bladder dissection in this condition is almost impossible. However, attachment and adherence of the placenta to the posterior bladder is not a mandatory technical limitation to the dissection of these two structures. The first measure in its performance is to identify the correct dissection space, and in order to facilitate this it is essential to apply traction to pull the bladder with two Allis clamps. The bladder should be thickly gripped with the clamps to avoid damage and to simplify dissection. The dissection should begin inside the round ligament and a small buttonhole should be made to allow the passage of a dissection clamp. Double ligatures should be used in the peritoneum and neoformed vessels, after which cuts can be made between the ligatures. The poor muscular layer of the NFV in anterior abnormal placentation leads to their collapse in the manner of veins, and often results in their being missed during maneuvers involving traction. Their inadvertent

cutting or treatment with coagulation by electrocautery can cause postoperative rebleeding (Palacios-Jaraquemada et al., 2004).

The next stage in retrovesical dissection requires care and is usually slow, and dissection of the NFV produced by the invasive process must be made meticulously, since these vessels are fragile and carry a high blood flow. In some cases the tissue between the two ligatures made in the procedure is insubstantial; if substantial tissue is encountered, a cut on the vesical side is preferred. If the ligature moves or if bleeding occurs, a simple insertion of sutures that include the muscular layer of the bladder wall can be done. In certain cases dissection may be obstructed by dense tissue fibrosis, making it difficult to identify a well-defined anatomic plane. In these circumstances dissecting the surgical plane in the bladder permits the creation of a tunnel between the cervix and the bladder, after which upward traction can be applied to seprate the bladder from the placentally invaded area. This maneuver, described as retrovesical bypass, is very useful and allows access to the posterior and lower region of the bladder (Pelosi and Pelosi, 1999).

Retrovesical dissection is complete when all of the NFV between the bladder, uterus, and placenta have been ligated and there is clear access to the cephalic sector of the vagina. At this point movement of the bladder allows access to the upper part of the vagina. In this situation, single-step conservative surgery or hysterectomy can be performed without the problems that usually occur in this type of placental invasion. After dissection, it is common to see defects in the muscular layer of the bladder. Some of these defects are produced by traction in areas of adhesion between uterine scar tissue and the bladder, and others by holes produced by thick NFV; both are easily repaired with absorbable 000 sutures. At this point or after closure of the abdomen, it is strongly advisable to examine for any additional points of bleeding in the posterior wall of the bladder. In cases of extensive bladder repair, infusion of methylene blue through a Foley catheter reveals invisible holes in the bladder wall (Palacios-Jaraquemada et al., 2004).

Some authors advocate the use of different types of resection of vesical tissues in cases of anterior abnormal placentation (Matsubara, 2010). The primary goal of this is to avoid bleeding through NFV; although retrovesical bypass is very good for this, resection of vesical tissue may produce secondary morbidity in young women through the resulting reduction in bladder capacity. In our experience and after having performed retrovesical dissection in more than 450 cases, we have never had to resect bladder tissue. The dissection of tissue in this procedure requires time and patience, but this is probably the best way in which to avoid bladder resection.

In some cases the bladder can open even after an accurate dissection. In such cases it is recommended that Allis clamps be used to gather the healthy vesical edges and to continue the dissection until healthy bladder tissue is reached. It is not mandatory to cut the borders, although it is advisable if they are thin. The suture used must be absorbable, and can include all of the layers of the bladder

wall. A silicone or standard Foley catheter must be inserted and left in place for 7–10 days to complete the primary healing process. If during this process the catheter becomes blocked with clot, it should preferably be replaced immediately rather than flushed, because the pressure used for flushing may increase the bladder pressure and cause intra-abdominal leakage.

Abnormal placental invasion with gross hematuria requires special attention because it commonly causes massive intraoperative hemorrhage and disseminated intravascular coagulation, either of which can represent a life-threatening emergency (Abbas et al., 2000; Shawish et al., 2007).

An abundant collateral blood supply in a narrow space, and the hemostatic consequences of continuous gross hematuria, are factors that prevent safe and bloodless surgery for retrovesical dissection. Such cases are therefore commonly treated with a conservative approach. In general, the placenta is left in situ and one or more attempts, as may be needed, are made at vascular embolization. The main idea is to wait until vascular invasion decreases so as to obtain a better chance of performing safe surgery at a later time (Lee et al., 2008; Gauthier et al., 2011). Efforts at supracervical hysterectomy and bilateral ligation of the iliac artery in such cases have been unsuccessful. If bleeding cannot be stopped, a packing of laparotomy pads can be used to promote hemodynamic stabilization, with the original problem solved via a secondary procedure or by surgery (Caliskan et al., 2003).

Placental invasion of the trigone of the bladder is uncommon, although some cases of this are probably inadequately identified. In certain cases, a CUS anastomotic system is significantly enlarged, especially between the trigone and the cervix. This anastomotic system develops in the anterior region of the uterine cervix and connects vessels with the vaginal system through the inferior vesical arteries. Because these NFV develop beneath the external connective tissue layer of the cervix, their safe dissection is almost impossible. Identifying this vascular behavior is possible in sagittal MRI slices, but the invasion is not truly histologic but rather mimics such invasion.

Because the vaginal and lower vesical arteries arise from the internal pudendal artery, endovascular hemostasis is not always possible through the uterine arteries or the anterior division of the internal iliac artery. Accurate vascular control, such as by infrarenal aortic cross-clamping or the insertion of endovascular balloons in the common iliac arteries or aorta, is needed before a posterior retrovesical dissection is begun. Since these procedures are not available at most centers, Section 6.2, on hyterectomy, presents an alternative solution.

Some years ago, I used cystoscopy as a routine method of presurgical diagnosis in cases of suspected anterior placenta accreta and percreta. Although the study was not comfortable, the seeming likelihood of its providing useful information justified the discomfort. However, experience with its use in a hundred cases demonstrated the lack of a relationship among the findings on cystoscopy, degree of placental invasion, and difficulty of the corrective surgical procedure. In theory,

bladder distention (necessary for the cystoscopic study) collapses the NFV in anterior placenta accreta and percreta. Moreover, there is no logical reason for such distention because these vessels are visible inside the bladder. As mentioned earlier, the purpose of NFV in abnormal placentation is to provide the placenta with additional blood, and these vessels are located in the muscular layer and not in the mucosa of the bladder wall. However, some vascular anastomotic pattern can be seen during cystoscopy in placenta accreta and percreta, although it is not specific. It is also uncommon to find placental tissue inside the bladder (Mashiah et al., 1988).

3.5 Ureteral dissection

Knowledge of ureteral anatomy within the pelvis is needed to avoid damage during certain gynecologic and obstetric surgeries. The ureter is about 30 cm long, of which half is in the abdomen and half in the pelvis. In entering the pelvis, the ureter passes across the internal and external iliac vessels. At this point the pelvic half of the ureter is inside the infundibulo-ovarian ligament, a landmark that facilitates surgical recognition because it is covered only by the posterior peritoneum. In the pararectal space, the pelvic ureter is 2 cm inside the ovarian pedicle or 2 cm outside the uterosacral ligament. Because ureteral irrigation is metameric, its dissection carries some risk of causing devascularization. In this regard it is important to know that the abdominal ureter receives its pedicles from the medial side and the pelvic ureter from the lateral side. For this reason, medial dissection of the pelvic ureter is recommended for avoiding ureteral devascularization. When the pelvic ureter must be recognized after pararectal exposure, its course should be followed by means of medial dissection. Because identification of the anterior ureter is typically difficult, it is suggested that a transparametrial tunnel be made around the ureter. With this simple maneuver, the anterior pelvic ureter can be recognized inside the pelvic–subperitoneal fat. Applying a rubber band or a silk #5 to #7 suture allows moving the ureter until dissection of the bladder is completed. Although anterior dissection can entail some difficulty because of the presence of the uterine artery and its branches, it is probably the best way to avoid unwanted damage under conditions (e.g., parametrial invasions) that hinder safe dissection.

Ureteral damage resulting from abnormal placentation is a complex problem whose manageent depends on its nature and on the point at which it is recognized. Ureteral invasion is the result of exuberant NFV induced by parametrial placental invasion, and is not caused by placental tissue itself. This type of involvement is uncommon but difficult to resolve (Caliskan et al., 2003). Its conservative treatment with the placenta in situ is a distinct issue because the surgery for this is done on the upper part of the uterus, far from the usual sites of placental invasion and the ureteral damage it causes.

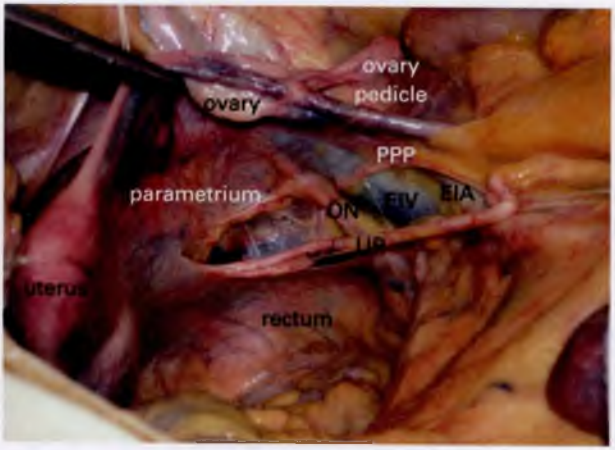

Fig. 3.12: Dissected fresh corpse (superior view of the right pelvis). The ureter (UR) is 2 cm from and parallel to the ovarian artery pedicle. It crosses the external iliac artery and vein and the obturator nerve (EIA–EIV–ON). After ureteral dissection beyond the internal iliac crossing, the creation of a tunnel above the ureter that crosses the lower border of the parametrium facilitates identification of the ureter in the lateral-cervical space. PPP: Primitive posterior peritoneum.

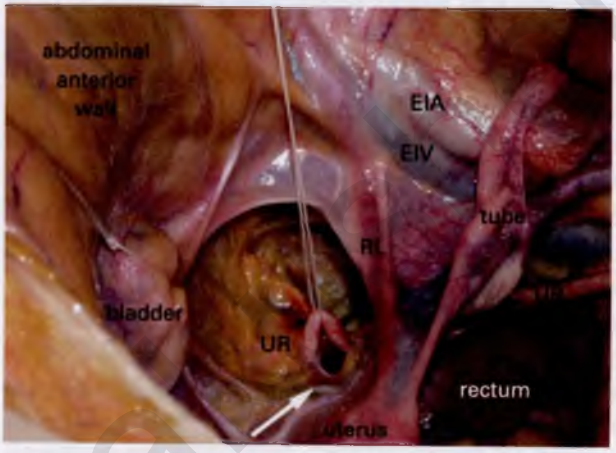

Fig. 3.13: Dissected fresh corpse (superior view of right pelvis). After the vesico–uterine fold is opened, the ureter can be dissected in the pelvic–subperitoneal fat. UR: Ureter, EIA: External iliac artery, EIV: External iliac vein, RL: Round ligament, White arrow: Uterine artery.

It should be noted that single-step conservative surgery and hysterectomy for placental invasion carry the risk of causing ureteral damage. If measures such as catheterization fail to prevent the effects of damage done by placental invasion, surgical identification of the damage is mandatory (Figs. 3.12 and 3.13) because ligation or sectioning may be unintentionally performed, especially in cases of

parametrial placental invasion. In general, parametrial invasion covers the ureter though forward advancement, although in some cases such invasion passes behind the ureter. With invasion, the ureter is pushed to a medial position. This change in its position can be very dangerous because the ureter may be confused with NFV and be inadvertently ligated.

In the case of placental adhesive disorders involving the S2 segment of the uterus and treated with extensive dissection, vessel ligation, and the removal of invading placental tissue, it is uncommon to identify ureteral damage (i.e., caused by ligation or sectioning) during such surgery. However, if it is identified, the best posible option for its treatment involves immediately calling a urologist for consultation.

If the ureter has been ligated, it is recommended that the ligation be removed and the local condition of the ureter be evaluated. If the ligature is not very tight and the ureter is apparently healthy, the surgical procedure being done on the uterus should be completed and the ureter should be monitored if necessary. However, if ureteral damage is evident through altered coloration and the absence of muscular ureteral activity, a simple alternative is to resect the damaged area, insert a double J catheter in the ureter, and insert three stitches with size 5/0 absorbable suture material to approximate the two sections of the ureter. The purpose of resecting the damaged area of ureter is to avoid a possible urinary fistula or a late obstruction by fibrosis.

It is also recommended that a double-J catheter be inserted in cases of ureteral damage inflicted by cutting. If the defect is minimal (e.g., 1 or 2 mm long), a double J catheter can be inserted through the site of damage and the procedure can be completed. In cases involving major defects in the ureter it is preferable to resect the damaged area and to proceed in the same way as with ureteral ligature.

Inadvertent ligation of the ureter after hysterectomy usually presents with acute back pain, which generally appears 1 or 2 days after the procedure. Obstetricians must be alert to this symptom because it can be hidden by postoperatively administered analgesic agents. Even if it shows partial ureteral dilation, a US scan may be insufficient to differentiate this from physiologic excretory urinary dilation in the gravid state caused by uterine compression. When there is doubt about the source of the dilation, CT urography is helpful, and is necessary to exclude a diagnosis of ureteral damage after difficult hysterectomy, especially in the case of damage caused by abnormal placentation. In cases of ureteral opbstruction, retrograde cystourethrography is advisable. This study can confirm the diagnosis and in other cases reveals extrinsic compression, usually by a hematoma. In such cases the endoscopic placement of a double J catheter can solve the problem until the external compression is relieved.

When ureteral damage is discovered by radiologic or endoscopic studies done after the treatment of placental invasion, its primary repair is not possible, and the most common solution for damage to the lower ureter is ureteral reimplanta-

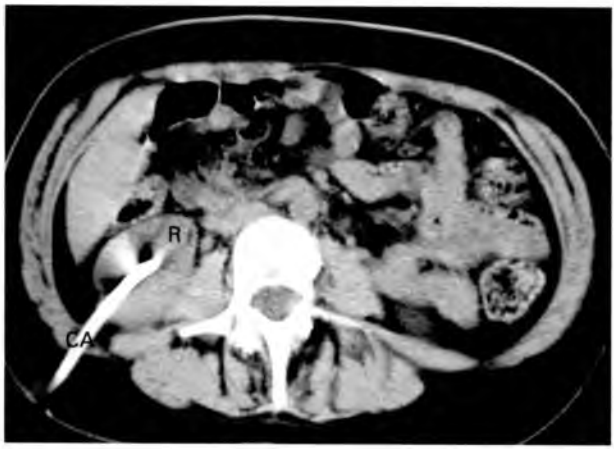

Fig. 3.14: Computed tomography, axial slice: Percutaneous urinary drainage is used in cases of inadvertent ureteral ligature until definitive repair can be accomplished. R: Right kidney, CA: Percutaneous nephostomy catheter.

tion. This procedure must be performed by a specialist and consists of insertion of the healthy ureter into a muscular vesical tunnel, which acts to prevent reflux. Although there may be differences among different specialists, a ligature or sectioning of the ureter that is identified within 1 week can generally be treated through reconstructive surgery performed after a percutaneous nephrostomy (Fig. 3.14). If the ureteral damage is discovered later than this, a period of 2 months should be allowed to pass so that the usual process of fibrosis can diminish.

Video 3.3: Proximal ureter (fresh corpse).

The infundibulo-ovarian ligament is one of the most reliable landmarks by which to identify a pelvic ureter. When only the posterior peritoneum is cut 2 cm inside of the infundibulo-ovarian ligament, the ureter is easily identifiable. Dissection through the medial side of the pelvic ureter is recommended because the ureteral vessels enter the lateral side of the ureter, and as a result, dissection of the pelvic ureter on its medial side is safe because it does not affect the ureteral bllod supply. Dissection through a posterior approach is also possible until the parametrium is reached. The creation of a periureteral tunnel is recommended to identify the ureter in the paravesical space. This space contains many vessels and is filled with fat below the peritoneal reflection, both of which factors make its identification difficult. If an instrument is passed through the parametrial tunnel above the ureter, this element can be detected by touch, allowing identification of the ureter. This maneuver is simple to perform and allows achúrate identification of the ureter even in the presence of hemorrhage or a hematoma.

http://dx.doi.org/10.1515/9783110282382_v3.3

Video 3.4: Distal ureteral dissection (fresh corpse).

Opening of the anterior parametrium allows access to the pelvic–subperitoneal space. After blunt dissection of fat and recognition of the ureter, a rubber band or silk #5 suture is inserted to move the ureter during traction. Ureteral dissection is done at the medial border and the uterine artery is separated from the ureter via gentle maneuvering, after which ureteral dissection continues until the bladder is reached.

http://dx.doi.org/10.1515/9783110282382_v3.4

Video 3.5: Ureteral catheterization in placenta percreta.

Ureteral catheterization is recommended in cases of parametrial invasion of the uterus or cases in which total hysterectomy is planned because of abnormal placentation. Although ureteral catheterization is a rapid procedure when done by a trained urologist, it can be difficult in some situations. Extensive parametrial invasion can make it difficult to pass a catheter into the renal pelvis. One of the most difficult situations in which to accomplish catheterization is vascular hyperplasia between the trigone of the bladder and the uterine cervix. The hyperplastic tissue usually rotates and modifies the spatial location of the ureteral ostium, making catheterization a difficult procedure. It is strongly recommended thnat ureteral catheterization be done carefully, and that if macroscopic hematuria appears, the catheterization should be stopped immediately to avoid the risk of massive and uncontrollable hemorrhage.

http://dx.doi.org/10.1515/9783110282382_v3.5

Video 3.6: Cystoscopy in placenta percreta.

The initial view shows an aspect of placenta percreta after laparotomy. Cystoscopy then shows an aspect of the NFV occurring in the process of placental invasion as seen through the mucosa. Notice that there is no pathognomonic image of placental invasion. One ureteral meatus is congested, but without bleeding. Both macroscopic hematuria and the visualization of placental tissue by cystoscopy are uncommon. The placental tissue uses an additional blood supply from the muscular layer of the bladder, for which reason endoscopic visualization of the vessels involved in the process is not common as is their vizualization by surgical exploration. The bladder distension necessary for endoscopy compresses a newly formed vessel and makes it difficult to visualize by endoscopy.

http://dx.doi.org/10.1515/9783110282382_v3.6

Video 3.7: Ureteral dissection (live).

In some cases of parametrial invasion, either the ureter cannot be catheterized or catheterization is unavailable. The video shows a live identification of the ureter during surgery. As noted previously, posterior ureteral identification is easier than anterior ureteral identification. Following this, the ureter can be gently pulled with a rubber band to allow the dissection to con tinue on its medial side until the lateral cervical space is reached.

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3.6 Pelvic access spaces

The female pelvis is a complex, three-dimensional anatomic space in which the reproductive, urologic, and gastrointestinal systems join. These systems are maintained by bones, muscles, and ligaments, as well as by extensive vascular and neurologic systems. Understanding of the inherent relationships between different structures in the female pelvis will help ensure successful surgeries in this region (Dietrich et al., 2008).

The transverse lower abdominal Pfannenstiel incision was first described in 1900 (Meeks, 1996), and is today perhaps the most commonly used transverse incision in obstetrics and gynecology. The incision is usually made two finger-breaths above the pubic symphysis, and gives excellent access to the pelvis. Some variations of the original Pfannenstiel technique were introduced to provide greater exposure (Maylard, 1907; Cherney, 1941). These techniques include cutting or detaching the rectal muscle and creating a transverse opening into the peritoneum. Even though these techniques had both advantages and disadvantages, they are not now commonly used, probably because they are technically more complex and the expansion opening they provide is not significant.

Pelvic–subperitoneal spaces are typically avascular and exist because the pelvic viscera are derived from different embryologic structures. In the midline, the pelvic spaces include the retropubic space, the vesicovaginal space, the rectovaginal space, and the retrorectal space. Laterally, two spaces, the paravesical space and pararectal space, are present and are separated from one another by the cardinal ligament (Dietrich, 2008). Access to pelvic spaces is the key to the practice of safe surgery in abnormal placentation because it allows the avoidance of unwanted damage to urinary and vascular structures. Access to prevesical, retrovesical, and pararectal spaces is mandatory to arresting some types of obstetric hemorrhage, especially in the S2 area. More than 90 % of the vessels that supply the the genital organs in the S2 area are pelvic–subperitoneal. Conditions such as placenta accreta, placenta percreta, cervicosegmentary pregnancy, lower uterine rupture, upper vaginal tears, and pelvic puerperal hematomas habitually require an accurate anatomic and surgical knowledge of this extraperitoneal space.

Accurate management of the retrovesical, paravesical, and pararectal spaces is needed in the surgery required for the most common disorders of placentation. Opening of the retrovesical space is a critical step in performing hysterectomy or one-step surgery in cases of placenta accreta or percreta. Although NFV might seem to make this maneuver almost impossible, it is not complex with the use of a simple tactic. This involves first gripping the bladder gently with two Allis clamps and then pulling it to expand the areolar tissue between the posterior bladder wall and the anterior uterine surface (Fig. 3.15). The vesicouterine fold is then identified and cut near the uterine surface. After minor dissection, a surgical forceps is used to grasp part of the peritoneal vesicouterine fold that includes both evident and hidden NFV. This collateral blood supply between the bladder and invaded

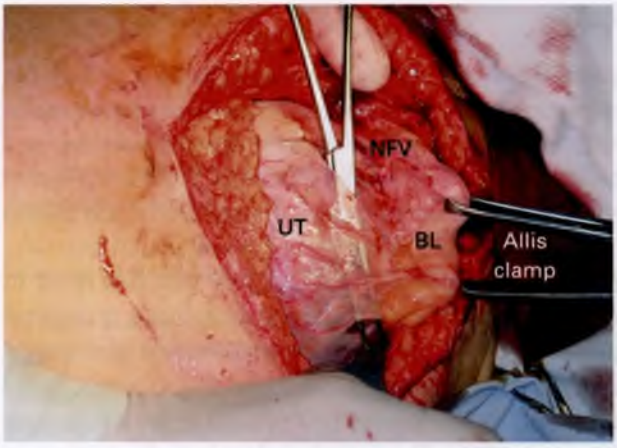

Fig. 3.15: Allis clamp used to pull out the bladder to provide space for ligating NFV between the bladder and the uterus. BL: Bladder, UT: Uterus.

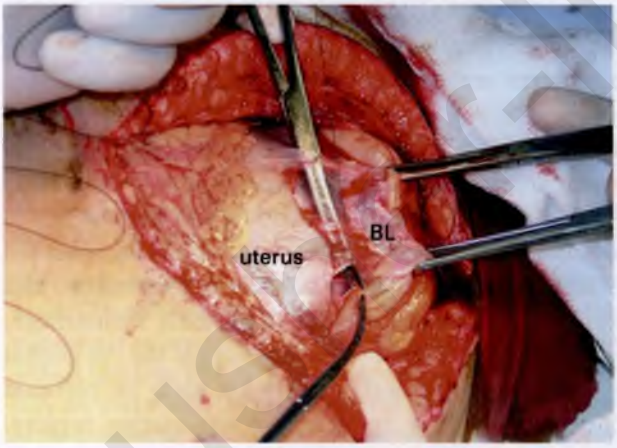

Fig. 3.16: Ligation of NFV is recommended because of the lack of collagen and muscle in these vessels. Use of electrocautery could produce late bleeding.

myometrium is ligated with double sutures and then cut (Figs. 3.16 and 3.17). It is recommended that the procedure be repeated until the dissection between the two uterine edges is complete. It is then possible to see the two layers of parametrium separately, as well as the fatty tissue of the pelvic-subperitoneal space, This maneuver allows direct access to the uterine arteries as well as permitting the pelvic ureter to be moved to within the fatty tissue of the lateral pelvic wall. The Allis clamps are then reapplied in the middle part of the bladder to maintain the angle between the bladder and uterus in order to continue the procedure until the cervix or upper vagina is reached (Fig. 3.18). This procedure is recommended for

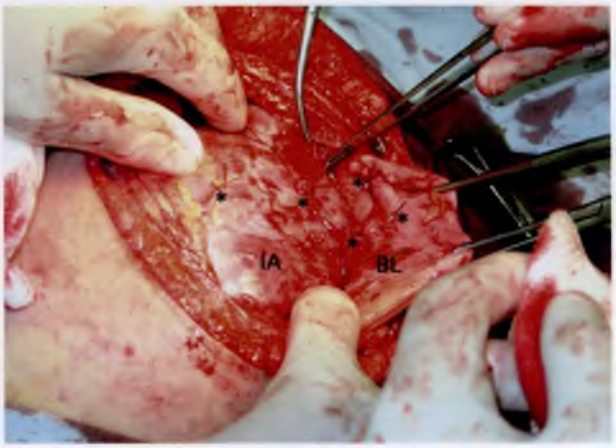

Fig. 3.17: Vesico–uterine view during dissection and cutting of NFV (in asterisks). BL: Bladder, IA: Invaded area.

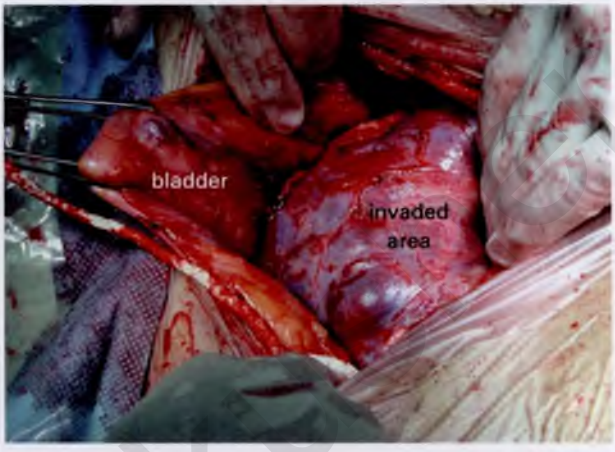

Fig. 3.18: Vesico–uterine dissection is finished when the upper vagina and cervix are reached. The vesico–uterine plane (white arrows) should be free of NFV connections.

placenta accreta, placenta percreta, or cervicosegmentary pregnancy. Regardless of whether the procedure finally chosen is hysterectomy or a conservative procedure, retrovesical dissection provides excellent visualization of the area of placental invasion and the pedicles within it.

Access to the paravesical space is strongly suggested in cases of parametrial placental invasion. The most important problem in such cases is direct visualization of the ureter within the placental tissue and exuberant NFV in a narrow and deep space. Because of the complex relationship among the ureter, vessels such as the inferior vesical artery and vaginal arteries, the placenta, and surrounding

tissues, it is more useful to identify the ureter first and to then move it away from the other structures. Access to the laterorectal space for identifying the ureter is described above.

Video 3.8: Retroperitoneal spaces (fresh corpse).

The pelvic–subperitoneal space communicates widely with the retroperitoneal space. This video shows easy dissection and communication of the pelvic space with the retroperitoneum. Subtotal hysterectomy in abnormal S2 placentation can cause rebleeding and undetected retroperitoneal hemorrhage. There are reports of large retroperitoneal hematomas undetected by US as a result of scanning only of the pelvis. Retrospective analysis has shown that US scanning must include a complete abdominal scan to be suitable for detecting retroperitoneal hematomas. Postoperative hypotension without vaginal or abdominal bleeding in patients who have subtotal hysterectomy or conservative treatment for invasions of S2 should suggest retroperitoneal bleeding. If exploration with US is negative and hypotension persists, urgent laparotomy or CT must be done.

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3.7 Proximal vascular control

Dissection is difficult but necessary in resective conservative and extirpative procedures on the uterus, and active bleeding clearly makes it almost impossible to safely perform this surgical procedure.

Hemorrhage in abnormal placentation is the source of a number serious complications, including shock, coagulopathy, multiple organ failure, and death. Running the risk of uncontrollable hemorrhage is a poor option in abnormal placentation, even for a technically brilliant operator, because of its high possibility of loss. Placental blood flow at term is about 500–700 mL/min, and accurate control of blood flow at this rate allows a better possibility of success. Because the pelvis has a wide network of intercommunicating vessels, it is very important to understand how this collateral blood supply works in order to choose the best available means of vascular control.

3.7.1 Aortic

The infrarenal abdominal aorta is a portion of the aorta between the origin of the renal arteries and the aortic bifurcation (Fig. 3.19). The entire course of this segment of the aorta is over the spine, with the aorta covered by the posterior peritoneum. In this segment, the infrarenal aorta gives rise to collateral vessels, the ovarian arteries, the inferior mesenteric artery, and the lumbar arteries. These last four pairs of vessels provide blood to the lumbar muscles and also to the spinal cord. Below the fourth lumbar artery and above the aortic bifurcation is a posterior area of the aorta without branching vessels that is frequently used at a site at

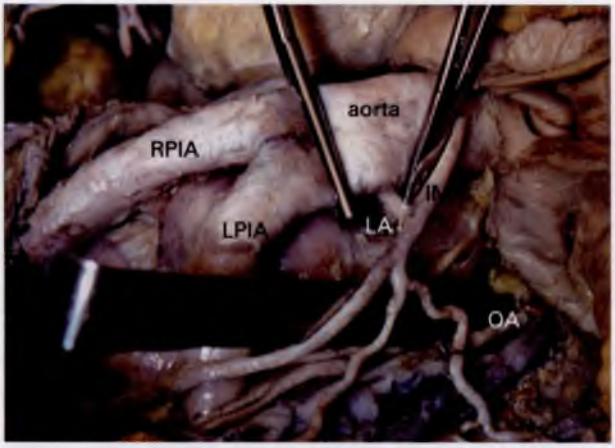

Fig. 3.19: Dissection of an embalmed corpse (superior view of the infrarenal aorta). The illustration shows the aortic division into the right and left primitive or common iliac arteries (RPIA–LPIA) was seen during the dissection. Aortic cross-clamping at this level allows blood flow via the inferior mesenteric artery (IMA), lumbar arteries (LA), ovarian artery (OA), and intrarachideal system.

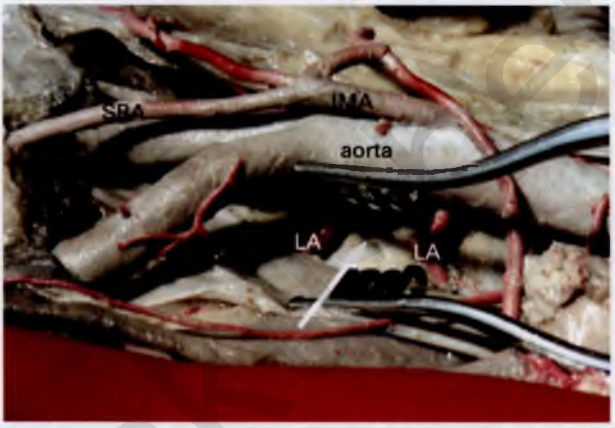

Fig. 3.20: Dissection of an embalmed corpse dissection with vascular injection of latex (superior and left lateral view of the infrarenal aorta). The white arrow shows a clear space between the third and four lumbar arteries, which is habitually used as the site for a #7 silk suture loop or an aortic clamp. LA: Lumbar arteries, IMA: Inferior mesenteric artery, SRA: Superior rectal artery.

which to apply a clamp (e.g., Satinsky clamp) or other device such as a #5 silk suture or a rubber band to stop aortic blood flow (Fig. 3.20). Because most of the anastomotic vasculature to the pelvis arises below this level, infrarenal aortic occlusion or clamping is a rational means of proximal vascular control of blood flow to the pelvis. Because such vascular control is applied below the origin of the

Fig. 3.21: Bernstein's latex balloon used for endovascular aortic occlusion.

lumbar arteries, it does not modify the blood supply to the spinal cord. Aortic vascular control can be achieved by external, internal, or endovascular means. External compression may be applied with the hands or with a specific device. Procedures performed in pregnant women have shown that bimanual compression at the level of the umbilicus can significantly reduce blood flow through the femoral arteries (Riley and Burgess, 1994). It has been determined that an external abdominal pressure equivalent of about 40 kg/cm^2 or about 90 pounds is necessary to occlude 100% of femoral arterial blood flow (Soltan et al., 2009). A simple maneuver can be used to occlude the aorta during cesarean section: After the uterus is pulled out of the pelvis, the sigmoid colon is displaced to the left, revealing the posterior peritoneum over the aorta in the promontory. Simple aortic pressure over an osseous plane then readily produces vascular compression.

Infrarenal cross-clamping should be performed by a specialist or a well-trained surgeon because it implies accurate dissection of the aorto–caval space. This vascular dissection must be performed after cutting of the aortic adventitia, and overlooking this detail can be a source of serious complications through inadvertent damage to the inferior vena cava. After dissection of the adventitia, a double loop of #7 silk suture is placed around the infrarenal aorta. If immediate hemostasis is necessary, the loop is tightened in a simple manner. This protocol has proven useful as a simple surgical means of preventing severe bleeding in abnormal placentation (Palacios-Jaraquemada, 2001) and is as effective as instrumental cross-clamping (Chou et al., 2010).

The infrarenal abdominal aorta can also be occluded by means of an endovascular balloon (F3.21). It should be done by an experienced interventional radiologist, and its use for the control of massive obstetric hemorrhage was first described in 1995 (Paull et al., 1995), and the balloon technique has some advantages with regard to bilateral occlusion of the iliac vessels because apart from its versatility for controlling the most important anastomotic pathways, aortic occlusion represents a single procedure in a thick vessel, the abdominal aorta, rather than two techniques in the common iliac arteries. This means a shorter procedural time, greater simplic-

ity and ease of manipulation, and a lower risk of thrombosis. The balloon is passed through the femoral artery and then introduced into and passed through the aorta to the aortic bifurcation. One in the infrarenal aorta, the balloon is inflated with saline solution to occlude the aorta. Absent femoral pulses are verified bilaterally to ensure the efficacy of the aortic occlusion. Experience suggests that the prophylactic insertion of a balloon catheter for aortic occlusion is a safe and an effective method for controlling anticipated bleeding in caesarean hysterectomy in a parturient with placenta accreta (Andoh et al., 2011; Masamoto, 2009). Other conditions associated with massive pelvic bleeding have recently also been treated with this method (Martinelli et al., 2010; Tang et al., 2010).

Some authors have reservations about the use of infrarenal occlusion because of its possibly compromising the blood supply to the spinal cord. However, in contrast to infrarenal cross-clamping for the treatment of aneurysmal disease, the intraaortic balloon occlusion used in obstetrics is done immediately above the aortic bifurcation, thus leaving open all lumbar and metameric branches of the aorta to the spinal cord or cauda equina.

Some collateral blood flow remains even after infrarenal aortic cross-clamping or occlusion. Extra-aortic routes of blood supply to the pelvis include the ovarian artery, internal mammary artery, inferior epigastric artery, intrarachideal system, and inferior mesenteric.

Video 3.9: Aortic dissection (fresh corpse).

General view of the retroperitoneal space. The posterior peritoneum is open and the anterior aorta is exposed. Aortic dissection starts with cutting of the aortic adventitia anterior to the aorta and continues along both borders in order to separate the aorta from the inferior vena cava. Primary opening of the aorto–caval space can be dangerous in unskilled hands because of the possibility of unwanted damage to the inferior vena cava. When aortic balloon catheterization is unavailable, temporary ligation or cross-clamping of the infrarenal aorta (above the aortic bifurcation) is an alternative means of achieving vascular control. For nonspecialist operators, intrasurgical or external compression of this vessel (against the promontory) is also efficient for this purpose.

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Video 3.10: Aortic balloon (live).

Use of an aortic balloon in the infrarenal aorta is one of the most accurate means of achieving proximal vascular control of the blood supply to the pelvis. Such control is highly suitable in the case of a preoperative diagnosis of placental invasion of S2 with the decision to treat this through total hysterectomy, because the aortic balloon occludes all collateral anastomotic routes of blood flow to this area. The aortic balloon is inserted through a femoral artery and is sited immediately before the aortic bifurcation. With the balloon in place, the interventional radiologist measures the amount of balloon fluid needed to completely stop aortic blood flow, and the balloon is immediately deflated to guarantee a fetal blood flow. In case of a need for acute hemostasis, such as in hysterectomy, the interventional radiologist inflates the balloon with a predetermined amount of fluid and aortic blood flow is stopped instantly.

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3.7.2 Common iliac

Because of differing results in occlusion of the internal iliac artery, some authors consider extending vascular control to the pelvis by means of an endovascular balloon inserted in both common iliac arteries. Experience with bilateral occlusion of the common iliac artery in patients with placenta percreta and also in those with cervical pregnancy has been favorable (Shih et al., 2005; Yang et al., 2007). This type of vascular control is efficient because it occludes the blood flow provided by anastomosis with the pudendal arterial circulation through blocking of the posterior branch of the internal iliac artery, and also occludes an anastomotic femoral-arterial component of blood flow to the pelvis. The safe occlusion time allowed for both common iliac arteries is about 90 minutes, which is determined by the ischemic survival of the skeletal muscle they supply. A bilateral approach can increase the risk of local complications, but no large serial study has been conducted to affirm this. However, prophylactic arterial balloon occlusion may be associated with risks unique to pregnant women (Sewell et al., 2006).

Bilateral balloon catheterization of the internal iliac arteries may be insufficient or ineffective in some cases of posterior placenta accreta or placenta percreta, and also in some cases of variation in typical arterial anatomy. Cases of posterior placental invasion are not as common as those of anterior invasion (more than 90 %). Posterior placental accreta is typically associated with multiple abortions with endometrial and myometrial damage. In such cases the CPU branch of the superior rectal artery may be unusually enlarged. Because this artery originates above the site of balloon occlusion of the common iliac arteries, it may be the source of severe bleeding after posterior percretal detachment or puerperal hysterectomy.

Although occlusion of the common iliac artery provides accurate proximal vascular control of pelvic blood flow, unexpected embryologic variation can substantially modify its efficacy or produce serious complications (Figs. 3.22 and 3.23). A persistent sciatic artery is a rare anatomic variant in which the internal iliac artery and the axial artery of the embryo provide the major blood supply of the pelvis and lower limb. The persistent sciatic artery typically arises from the abdominal aorta and replaces the branches and the path of the internal iliac artery. When this vessel is present, the superficial femoral artery is usually poorly developed or absent (Paraskevas et al., 2004; van Hooft et al., 2009). An apparent contradiction that may occur in cases of persistent sciatic artery during bilateral occlusion of the common iliac arteries is the absence of a pulse in the femoral artery despite a finding of 100 % oxygenation with oximetry in the lower limb. A persistent sciatic artery usually continues along the course of the internal iliac artery and surrounds the head of the femur from behind. Recognizing this variation is crucial in cases of arterial embolization, because if the persistent sciatic artery is confused with the internal iliac artery, therapeutic embolization can result in unwanted necrosis

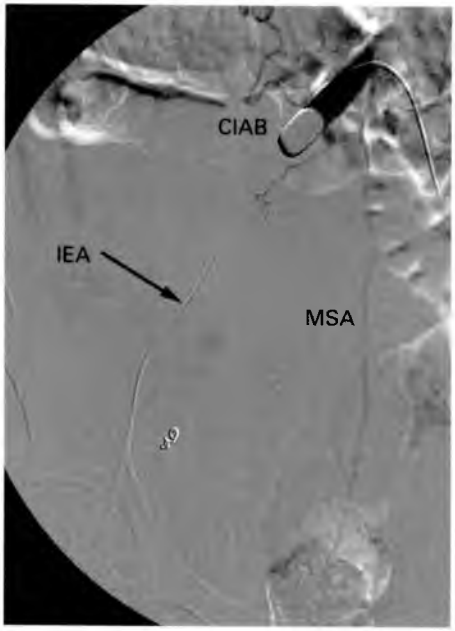

Fig. 3.22: Subtraction pelvic arteriography showing an inflated common iliac arterial balloon (CIAB). Although the femoral pulse was zero, lower limb oximetry showed nearly 100 % oxygen saturation. The only visible collateral vessel in this vascular control view is the medial sacral artery (MSA), which cannot provide an adequate supplementary blood supply to the lower limb. Black arrow shows a wire inside the external iliac artery (IEA).

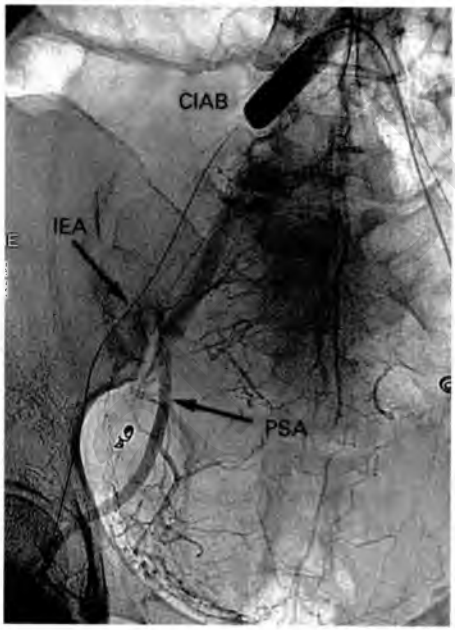

Fig. 3.23: Subtraction pelvic arteriography: Injection of contrast material above the common iliac balloon shows a thick, persistent sciatic artery (PSA) that passes behind the femoral neck to provide continuous blood flow to the lower limb. Although this variation is not very common, it must be known by the interventional radiologist to avoid confusing the PSA with the iliac internal artery. Inadvertent embolization of this branch can result in unwanted lower-limb ischemia or necrosis.

in the lower limb. Meticulous arteriographic technique may prevent inadvertent embolization of this rare anatomic variant (Hsu et al., 2005).

3.7.3 Internal iliac

Ligation of the iliac internal artery is probably the oldest procedure used for proximal vascular control in the pelvis. The first written report of its use is found at the beginning of the nineteenth century, after which it was used in multiple hemorrhagic conditions in gynecology, obstetrics, and traumatology. However, the results of its use in different situations were controversial, with its sometimes proving useful and sometimes not. This was clearly revealed by a series of papers about the physiology of ligature of the internal iliac artery by Burchell (1964, 1966, 1968) at the University of Illinois at Chicago. He found that the interrupted blood flow beyond the point of ligation of the iliac internal artery was replaced immediately by an extensive network of collateral vessels. This work changed the use of ligature of the internal iliac as artery for proximal vascular control in the pelvis. A recent publication (Iwata, 2010) examined the blood loss in different variants of placenta accreta managed with ligation of the internal iliac artery, and whether the different pathologic findings in these variants would affect blood loss during cesarean hysterectomy. It found no significant difference between the mean blood loss and pathologic findings in cases of placental adhesive disorders managed with such ligation.

After the introduction of endovascular occlusion of pelvic vessels (embolization), the idea of controlling massive pelvic bleeding by occluding the internal iliac artery was revived (Dubois et al., 1997). At the time, internal occlusion and embolization were done in a single procedure. A few years later, however, the first experience was described of the use of balloon occlusion of the anterior iliac artery as a unique method of vascular control with historic cases of abnormal placentation used as controls (Levine et al., 1999). This study found that of the use of balloon catheterization to occlude the pelvic artery in patients requiring cesarean hysterectomy for placenta accreta did not improve surgical outcomes as compared with management of the condition without such catheterization. The efficacy of bilateral occlusion of the internal iliac artery is random and low (Clark et al., 1985), and for this reason it is important that balloon catheters not be used instead of but rather in conjunction with pelvic embolization. This allows the avoidance of systemic heparinization for iliac-catheter placement, which would exacerbate ongoing hemorrhage during balloon inflation. An alternative to this is to infuse heparinized saline solution through the catheters and to remove them immediately when hemostasis is achieved (Greenberg et al., 2007). It is understandable that ligation or simple endovascular occlusion of the internal iliac arteries would have similar results in terms of hemodynamic physiology. A recent analysis of Doppler US patterns after ligation of the internal iliac arteries suggests that there were no significant differences in the waveforms of Doppler flow velocity in the uterine, arcuate, and ovarian arteries of women who underwent bilateral ligation of the internal iliac arteries and women who did not (Yildirim et al., 2009). These findings

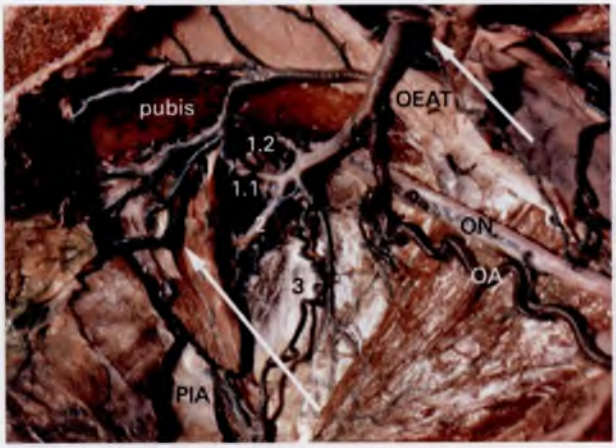

Fig. 3.24: Dissection of an embalmed corpse with vascular injection of latex (superior and right view of obturator fossae). Detailed anastomotic connections among the femoral, anterior internal, and posterior internal iliac trunks are seen. Right arrow shows the direction of the anterior branch of internal iliac artery, and the left, arrow shows the direction of branches of the internal pudendal artery (posterior trunk of the internal iliac artery). Between the two systems, an obturator–epigastric anastomotic trunk (OEAT) links the external iliac system with the obturator artery (OA), with the femoral system via subpubic branches (1.1 and 1.2), with the inferior gluteal artery (2), and with the internal pudendal artery (PIA), which is marked by the vessels numbered 3. ON: Obturator nerve.

could indicate that such ligation does not affect the reproductive organs, and also suggests a minimal hemostatic effect of the procedure itself. These findings are in accord with the physiologic studies conducted by Burchell and mentioned earlier. It must therefore be asked whether authors who have reported good results of ligation have reported them as consequences of the procedure itself or as the effect of multiple therapeutic measures taken at the same time, such as surgery, embolization, and the administration of procoagulants, among others. The collateral blood supply immediately restores blood flow after ligation of the iliac internal arteries (Mayer et al., 1975; Burchell, 1968). The anatomy of the supplementary pelvic vascular system is exuberant and makes blood flow immediately available after acute ligation or occlusion of the internal iliac arteries (Palacios-Jaraquemada et al., 2007).

The collateral blood supply network consisting of the obturator, internal pudendal, inferior gluteal, vaginal, lumbar, ovarian, epigastric, middle and lateral sacral, and deep femoral arteries is a rich network of visible vessels (Fig. 3.24). In young women, this network can instantly replace blood flow to the pelvic region after ligation or occlusion of the internal iliac arteries in the absence of previous or prolonged partial blockade. Development or enlargement of newly anastomotic vessles into the pelvis is seen only in elderly patients with extensive atherosclerosis because of the occlusion of their main vessels.

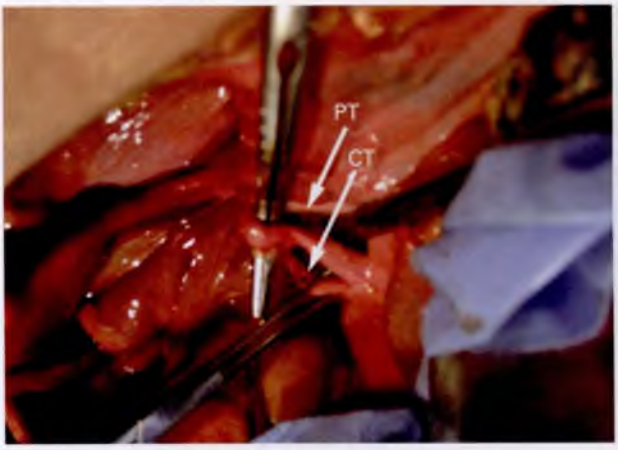

Fig. 3.25: Dissection of a fresh corpse (upper view of right pelvis). The common trunk of the internal iliac artery is the most proximal vessel (CT); the posterior trunk (PT) is only evident after dissection and intentional mobilization. Both trunks, the common and anterior, are near the ureter.

It is very difficult to ascertain precisely why internal ligation or occlusion of the internal iliac arteries can succeed or fail, but an anatomic and physiologic analysis of these methods showed that they are not optimal, safe, or effective for controlling massive pelvic bleeding (Dilauro et al., 2012). The pelvic anastomotic system is mainly vertical, and this disposition requires bilateral occlusion or ligature of the internal iliac artery as a minimal hemostatic procedure. Both occlusion and ligation of the iliac internal artery are procedures done on the anterior division of the artery, and thus exclude the first trunk of the posterior division and the internal pudendal artery. Because the internal pudendal artery provides more than 90 % of the blood flow to the genital region below the peritoneal reflection, occlusion or ligature of the anterior internal iliac artery is not a procedure capable of controlling genital bleeding in the S2 region (Palacios-Jaraquemada et al., 2007).

In order to avoid vascular complications, ligation of the anterior trunk of the internal iliac artery is recommended; however, identification of the posterior trunk of the artery is not at all easy (Fig. 3.25), and most simulation videos available on the Internet show a ligation of the common trunk of the artery. A good understanding of retroperitoneal anatomy is needed to reduce the risk of serious intraoperative and postoperative complications of ligating the internal iliac artery (Camuzcuoglu et al., 2010). Despite their very low incidence, complications of such ligation include ureteral injury; a compromised blood supply to the gluteal region (from ligature of the posterior trunk of the internal iliac artery); inadvertent injury to the external iliac artery resulting in ischemic damage to the lower extremities; and injury to the internal iliac vein resulting in further hemorrhage in unstable patients (Yildirim et al., 2009).

Another consideration in ligation of the internal iliac artery is the specific maneuver needed for ligation of the arterial trunk, because inadequate dissection of the arterial adventitia can do serious damage to the iliac internal vein. Accidental injury to the internal iliac vein during dissection of the internal iliac artery, although uncommon, can cause massive pelvic hemorrhage in a patient with previous pelvic bleeding. This complication may herald a rapidly fatal outcome. Only a few descriptions of complications of damage to the internal iliac vein have been published (Rao et al., 1978), and their existence is more widely known from surgical anecdotes. However, an idea of the seriousness of such complications is provided by the published report of the case of a patient who needed 16 liters and ligation of the internal iliac artery for blood loss caused by venous damage after ligation of the internal iliac artery to achieve hemostasis after an open prostatectomy (Korusić et al., 2009).

Video 3.11: Ligation of the internal iliac artery (live).

The video shows a simulation of ligation of the internal iliac artery during cesarean section. Approach through the lateral-rectal space allows identification of the ureter, with the internal iliac artery seen immediately beside the ureter. However, the artery identified in the video is a common internal iliac trunk. Dissection of the artery from the vein can be a dangerous maneuver, because the arterial adventitia must be dissected before such dissection of the two vessels from one another. Damage to the iliac internal vein during the ligation procedure is very difficult to repair and can therefore be disastrous. If this happens in a patient with shock and hemorrhage, the possibility of survival is extremely low. Although the procedure for ligation of the internal iliac artery is the object of extensive training and workshops in some locations, these do not guarantee its success, and ligation of the artery fails in more than 60 % of cases, especially in cases of lower abnormal placentation. It should be kept in mind that bilateral ligation is a minimal procedural requirement because of the vertical rather than horizontal orientation of the collateral pelvic vascular system. A further problem is identification of the posterior trunk of the internal iliac artery because it passes between two veins. Ligation of the main trunk of the internal iliac artery is associated with ischemic complications and makes subsequent embolization almost impossible even when needed.

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Video 3.12: Dissection of the internal iliac artery (fresh corpse).

A pelvic ureter is identified 2 cm closer to the midline of the body, enclosed within the ovarian vascular pedicle; mobilization of the ureter reveals the internal iliac artery. Note should be taken of the proximity of the internal iliac artery to the external iliac artery, which in some cases has been confused with the anterior division of the internal iliac artery. Following this, the video shows dissection and identification of the posterior trunk showoff the internal iliac artery. It may be dangerous and ineffective to use these maneuvers bilaterally in a patient with severe bleeding, and this should be taken into account before using this procedure.

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Pseudoaneurysm is an unusual but another possible complication of ligation of the internal iliac artery. An Indian study suggests the need for follow-up of patients who have undergone such ligation (Nanjundan et al., 2011). Pseudoaneurysm of the uterine artery, a rare cause of secondary postpartum hemorrhage, can be managed with ambolization of this artery (Raba, 2009).

Besides the foregoing complications, embolization of the pelvic blood supply after ligation of the internal iliac artery can result in the unwanted occlusion of collateral vessels in this widely interconnected although anatomically and physiologically limited vascular network, causing necrosis (Zanati et al., 2010).

Of further note is that ligation of the internal iliac artery makes it almost impossible to perform a subsequent embolization because it closes the main arterial pathway to the pelvis. In such cases the only way to enter the pelvis is through the route consisting of the external iliac artery, femoral artery, deep femoral artery, internal circumflex artery, transobturator anastomotic artery and obturator artery. There is a report of this being done to stop vaginal bleeding after the ligation of both internal iliac arteries. The procedure lasted several hours and the fluid and blood replenishment needs were huge, but the patient survived, probably because of the endovascular surgeon's dexterity and accurate clinical management.

3.7.4 Uterine

The ability to arrest bleeding in the female genital system is directly related to the management of specific arterial pedicles. As for any other organ in the body, knowledge of these arterial pedicles is the key to controlling hemorrhage in the female genital system and to managing devascularizations and partial resections of the uterus. As a medial organ, the uterus has two major bilateral pedicles: those of the uterine arteries (Fig. 3.26) and the ovarian arteries, but is also supplied with blood through anastomotic vessels from the vagina or adjacent organs. In cases of abnormal placentation, knowledge of these other pedicles can be very important for achieving hemostasis. This knowledge may be essential in the case of conservative treatment because for resective procedures, the origin of NFV is unimportant in resective procedures since the resection includes all of them.

In disorders of placental adhesion in the S1 region, the uterus can be excluded vascularly from the circulation in a very simple way. In this technique, two openings are made surgically, in the lower part of the parametrium and in the avascular area immediately beside the uterine pedicle, after which a rubber tourniquet can be applied around the lower segment of the uterus in the form of a loop, which can then be tightened to compress the myometrium and occlude the uterine arteries as well as the lower anastomotic component (vaginal arteries–superior rectal artery). Additionally, two bulldog clamps are applied below the corneal area of the uterus to occlude the ovarian and uterine branches of the ovarian artery. This simple

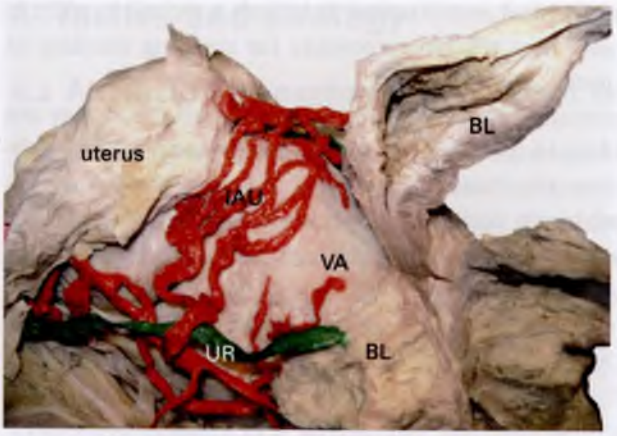

Fig. 3.26: Dissection of an embalmed corpse (anterior and lateral view of the uterine blood supply). The bladder (BL) was cut and divided into two parts to visualize an interuterine anastomotic system (IUA). This anastomotic blood supply communicates with both uterine arteries outside the surface of the myometrium. This system is widely connected with the upper and lower vaginal arteries. Obliteration of this system by embolization might be difficult in cases of anterior abnormal placentation, in which microanastomoses linking the uterus, the vagina, and the bladder are extensive. Application of compression sutures such as Cho's square suture may be easier than obliteration because it works in a specific area, independently of the vessels that provide a supplementary blood supply. UR: Ureter.

technique stops almost all uterine blood flow and allows the resection of adhesive placental tissues and uterine hemostasis and myometrial repair without bleeding.

Placental invasion in the S2 region is very difficult to treat because of the critical need for accurate dissection and exposition of the invaded tissues, which in turn implies accurate vascular control (such as bilateral control of the infrarenal aorta or common iliac arteries) or meticulous dissection and stepwise ligation.

Procedures for achieving uterine hemostasis, such as Cho's (Cho et al., 2000) and Pereira's techniques (Pereira et al., 2005), are better than the B-Lynch (B-Lynch et al., 1997) or Hayman techniques (Hayman et al., 2002) when abnormal placentation is located in the S1 region. Cho's and Pereira's procedures occlude the anastomotic collateral vessels in the posterior uterus that derive from the rectal and upper vaginal arteries and are usually enlarged in posterior abnormal placentation. Bilateral ligation of the uterine arteries has been ineffective in controlling hemorrhage in the S2 region resulting from placenta previa or placenta accreta because the S2 uterine pedicles arise from the pudendal artery and not from the uterine arteries. Selective arterial ligation in cases of abnormal placentation in the S2 region are one option for controlling hemorrhage in this region because they obliterate the pedicles by knots made after their direct identification and dissection. This technique implies wide mobilization of the bladder and identification of the ureters, since the vaginal pedicle is immediately beneath the bladder. Although

Cho's procedure requires wide vesical mobilization, it is from a technical point of view the simplest, fastest, and most efficient procedure for stopping bleeding in S2 region (Palacios-Jaraquemada, 2011).

The use of multiple hemostatic procedures on the uterine circulation may not enhance the control of bleeding in the placental bed in the presence of an established coagulopathy. Nor does the conjunctive use of different procedures (B-Lynch/Cho, Cho/Hayman) produce a noticeable improvement in hemostasis. Stopping the bleeding and restoring adequate clotting status are priorities for obtaining satisfactory results.

4 Tactics and strategy

4.1 Alternative management in different settings

Therapeutic resolution of the most common presentations of placenta accreta can be addressed through illustrative sketches. Because there is no ideal situation for the management of each such presentation, these sketches include variables that depend on the patient, the hospital environment, and the obstetrician's training. Indeed, the behavior of a specific type of invasion may vary even within a particular hospital, depending on the local conditions or available resources. The following sections discuss the most common presentations of placenta accreta and the means for choosing the safest ways of treating them. The primary objective will be to promote delivery in a simple and safe way, minimizing maternal hemorrhage and the possibility of the complications common in each such situation.

Scenario 1

Strong suspicion of placental invasion
Low placentation with a history of cesarean section
+
Labor
Metrorrhagia
+
Team with limited experience
Impossibility of patient transfer
Low availability of resources

On many occasions a patient comes into the emergency care service of a treatment facility during labor, undergoing metrorrhagia and without any previous obstetric controls. Unfortunately, there is not always the time or possibility of performing a US or other complementary examination, although information can always be obtained through questioning. If there is an epidemiologic risk of placenta accreta, such as from low placentation or a history of cesarean section, treatment must be conducted as if there were a definitive diagnosis. Alternatively, there may be a diagnosis but no means or trained personnel for solving the type of invasion defined in the diagnosis. In the presence of metrorrhagia and labor, safe transfer of the patient is usually impossible, and the patient's condition must be resolved on an urgent basis.

Because it is essential not to injure or detach the placenta when removing the fetus, it is advisable to perform an infraumbilical middle incision, in some cases including periumbilical extension. The uterus is next exteriorized from the abdominal cavity, and an anteroposterior fundal incision is made (Fig. 4.1). This incision is preferred to a transverse incision because suturing of the latter incision may occlude the tubal orifice. Once the neonate is born, a solenoid suture is performed, which includes the severed myometrium together with the placental membranes.

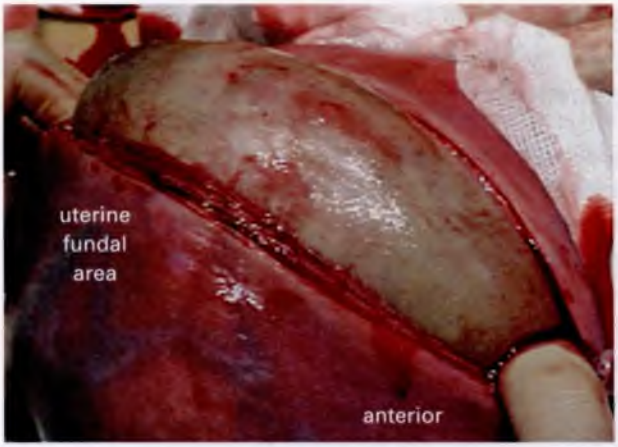

Fig. 4.1: Anteroposterior fundal hysterotomy allows delivering an infant though a noninvaded and safe area of the uterus.

The umbilical cord is ligated near the placental insertion, and both the hysterotomy and laparotomy wounds are closed with sutures. It is essential to understand that no attempt be made to detach the placenta because the resulting hemorrhage will not be stopped either by compression or suturing. If hysterectomy is necessary, it must be total and blood will almost inevitably be needed, as will hemoderivatives and skills for managing the bladder and ureter.

Leaving the placenta in situ after the neonate is removed results in minimal or no bleeding. If the site of adherence of the placenta is beside NFV, it will remain in place; if on the contrary the case is one of placenta accreta due to a parietal deficit, the placenta may become detached and be evacuated vaginally without additional bleeding. In the case of partial evacuation, it must be considered that exerting traction to the placenta can cause massive bleeding, and a full treatment team must be prepared for managing such hemorrhage before such evacuation is done. In both cases the patient's coagulation status must be controlled and intravenous antibiotics must be given earlier to prevent infection in retained tissues. After the initial approach, conservative management or hysterectomy can be indicated, depending on the case. Although the existence of a period of safety in the absence of bleeding by retained placenta has not been ascertained, no hemorrhagic complications have been reported sooner than 72 hours after a fundal cesarean approach. This period is usually adequate for transferring the patient to a higher-level treatment center or for gathering a qualified surgical team for the patient's treatment.

Today, the presurgical diagnosis of placenta accreta has a high rate of reliability. Both US and pMRI make it possible to establish an accurate correlation of what is deduced through images with what is actually observed during surgery in a particular case of the disorder. However, and as has been previously stated, definitive therapeutic decisions must be made once the surgical stratification of the

Video 4.1: Fundal hysterotomy (live).

This procedure is highly recommendable for treating abnormal placentation in cases of emergency, lack of resources, or specific training. The hysterotomy is performed in a safe area and far from the uterine site of abnormal placentation. After the neonate is delivered, the umbilical cord is ligated and the uterus closed with the placenta inside. A golden rule is to avoid detaching the placenta, in order to prevent unmanageable bleeding.

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Scenario 2

Established diagnosis
No wish for future pregnancy
+
Tissue destruction over 50 % of the uterine axial circumference
Untreatable hemorrhage
Disseminated intravascular coagulation
+
Team with qualified experience
Availability of hospital resources

patient has been completed. This has double significance, in that: (1) different laws justify hysterectomy after all possibilities for conservation of the uterus have been exhausted; and (2) the use of endovascular occlusion after the fetus has been removed may limit the use of hemostatic techniques if the initial diagnosis yields a false-positive result.

A positive presurgical diagnosis of placenta accreta without the patient's wish for future pregnancy does not necessarily simplify matters. Hysterectomy, which is total if there is invasion of the S2 region, has a high percentage of complications. Local factors, such as tissue destruction over 50 % of the uterine axial surface, the presence of disseminated intravascular coagulation (DIC), and uncontrolled hemorrhage are all indications for hysterectomy. Nevertheless, certain measures reduce morbidity and mortality in these circumstances. However, even though DIC and uncontrolled hemorrhage are indications for hysterectomy, it has been said that once the existence of these conditions has been established, the decision to undertake surgery has been made too late. Surgeons who have operated on these patients know that performing a hysterectomy can be fatal or produce serious morbidity from massive bleeding. This is because obstetric hysterectomy produces an estimated loss of 2 L–3 L of blood (Henrich et al., 2008), a volume that in case of shock or serious coagulopathy can be extremely dangerous. If the strategy is reversed and effective vascular control is applied before hysterectomy, everything changes. Applying an Esmarch bandage from the uterine fundus toward the cervix immediately stops any such hemorrhage and at the same time restores volume in the intravascular space by facilitating blood flow into this space from the

intramyometrial tissues. Because of the high pressure produced by the Esmarch bandage, encircling the uterus twice with the bandage reduces the uterine axial volume by half. This provides time in which to take hemodynamic and hemostatic compensatory measures without uterine bleeding. Under these conditions, and with a trained treatment team, hysterectomy can be performed under the best local and clinical conditions, which is likely to reduce the risk of intrinsic complications (Palacios-Jaraquemada and Fiorillo, 2010).

It is not easy to perform a hysterectomy on a patient with placenta accreta; the possibilities of parametrial, ureteral, and vesical invasion increase the risk of complications, and the procedure must be done by trained surgeons in an environment with the necessary technical resources. Absent this, compression of the infrarenal aorta produces sufficient hemostasis to permit the procedure without active bleeding. In this respect it should be remembered remembered that ligation of the internal iliac artery does not provide acceptable pelvic hemostasis because of the great number of anastomotic collateral vessels in this region; if hysterectomy is done, the minimal necessary procedure is bilateral ligature of the internal iliac artery, which implies a longer operating time and carries the possibility of complications. If, as has been previously mentioned, tearing of the trunk of the the hypogastric vein during the procedure may cause a fatal hemorrhage in the absence of the praxis or specialized assistance needed to prevent this. In the case of aortic compression, sustained and adequate pressure is needed only over the sacral promontory, without additional precautions.

The topographic division of the uterine vasculature into the regions designated S1 and S2 indicates the type of hysterectomy that is usually necessary in a particular case of placenta accreta. If the invasion is located in S1 (uterine body), hysterectomy can be subtotal. In contrast, a total hysterectomy must be done in cases of invasion involving the S2 region. This is supported by the very high percentage of episodes of recurrent bleeding after subtotal hysterectomy for invasion of the S2 region (Torreblanca Neve et al., 1993). When reoperation is done under these conditions, usually in the presence of an established coagulopathy and shock, the surgical risk and possibility of associated complications increase.

Vascular control is essential to preventing shock, hemorrhage, and coagulopathy, and is therefore a cornerstone in the treatment and prevention of complications arising from hysterectomy for placental invasion. Preventing hemorrhage permits correction of the patient's hemodynamic and hemostatis status, although this requires time and proper clinical management. Overlooking these facts and thinking that a hemorrhagic problem arises only from a bleeding uterus leads to serious clinical errors that increase maternal morbidity and mortality.

Most publications on the management of placenta accreta relate to patients in the group defined by the characteristics of the scenario presented here. The increase in cesarean sections and surgeries for placenta accreta in various groups of young patients with reproductive capacity has led to conservative treatments for

Scenario 3

Established diagnosis
Wish for future pregnancy
+
Tissue destruction under 50 % of uterine axial circumference
Minor coagulation disorders
+
Team with qualified experience
Availability of hospital resources

it. Today, such conservative treatment comprises two main approaches: leaving the placenta in situ or performing a one-step surgery for its removal. The purpose of leaving the placenta in situ is to prevent maternal bleeding during delivery while preserving fertility. However, one-step surgery was designed to be an integral solution to abnormal placentation. The one-step procedure prevents bleeding through the surgical control of NFV, facilitates resection of the placenta and of all invaded tissue, and allows accurate reconstruction of the uterine wall.

Scenario 4

Placenta percreta with parametrial invasion
(Intrasurgical diagnosis)

As noted earlier in this book, placental MRI is the only diagnostic method that allows the diagnosis of placental invasion of the parametrium (Fig. 4.2). However, only the experienced radiologist can identify and confirm this, through placental MRI with coronal and axial slices. In most cases, however, the diagnosis of parametrial invasion is intrasurgical. If neither a qualified team nor hospital support is available, access should be gained through the uterine fundus, and the placenta must be left in situ. However, this type of invasion can also be a major challenge even for a highly trained treatment team. The invasion occurs in a narrow space, beside the ureter, with NFV and an exuberant blood supply. In some cases it is useful to insert a simple transvesical ureteral catheter, but this cannot always be done because the invasion often compresses and obliterates the ureter by external compression. Forcing the procedure may lead to cataclysmic hemorrhage, necessitating removal of the infant and hemostasis through aortic compression that must be performed within seconds.

Parametrial invasion can significantly distort the normal pelvic anatomy. On occasion the placenta invades the pelvic floor and moves the ureter medially and anteriorly. In this and other situations that shift its normal anatomic relationships, it may be confused with NFV and be ligated on the posteromedial side of bladder. The ureter may also lose the typical anatomic position in which it is normally located within and more medially than the path of the infundibulopelvic ligament. Occasionally, parametrial invasion is huge and moves the ureter outside the ovarian pedicle (which it is normally inside). Finding the ureter, and following and

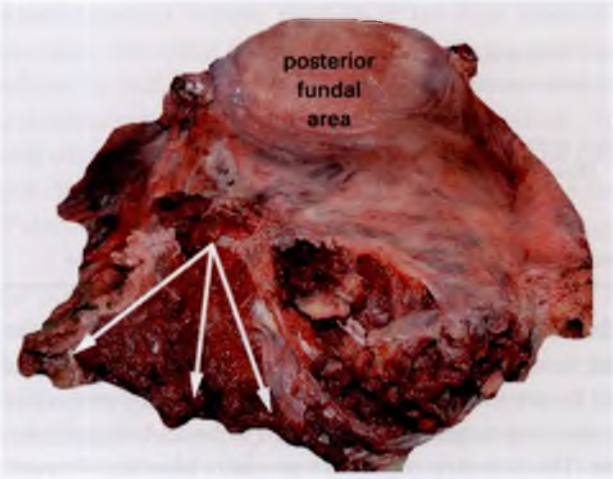

Fig. 4.2: Massive posterior and left parametrial invasion (white arrows). Because of a lack of healthy tissue below the area of invasion, it is completely impossible to perform conservative reconstructive surgery in a case such as this. Optimal vascular control is mandatory, as is also accurate ureteral identification.

dissecting it in a bleeding vascular magma can cause great concern, even to a highly experienced treatment team.

! **Scenario 5**

Posterior placenta accreta or percreta
Pre- or intrasurgical diagnosis

Posterior placenta accreta or percreta in the S2 region behaves like parametrial invasion, whereas invasion in the S1 region can usually be resolved simply if certain rules are followed. Generally, posterior placenta accreta following myomectomy or D&C does not alter fetal extraction. Once delivery is performed, the uterus will be exteriorized outside the pelvic cavity. If the placenta has not invaded the uterine wall to cause placenta percreta, it will be removed by hand and the bed to which it was attached will be curettaged. Following this, one or two square (Cho) stitches will be inserted over the area of invasion. This procedure is highly superior to any other type of vascular control because it not only includes the collateral vessels of the uterine and ovarian arteries, but also the anastomotic branches of the upper rectal artery, which enter through the posterior peritoneal reflection.

In the case of placenta percreta, temporary double clamping of the uterine and ovarian arteries (with Bulldog clamps) or the placement of a pericervical elastic loop are preferred for hemostasis prior to resection of the area of placental invasion. Following this the myometrium is sutured, clamping is applied, and square

stitches are made if necessary. For placenta percreta in other locations, control of clotting with firbinogen is unavoidable after removal of the placenta.

Video 4.2: Posterior placenta percreta (live).

A case of posterior placenta percreta consecutive to laparoscopic myomectomy. Because the abnormal placental attachment is in the S1 region, surgical resolution of the condition is simple. After the infant in this case was delivered, a rubber band was placed around the uterus to occlude both uterine arteries. A simple resection of the area of invasion with the attached placenta was done with a scalpel and the defect was repaired in two planes. Invasions in the S2 region must typically be treated through hysterectomy.

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4.2 Scheduled surgery

All preventive measures in abnormal placentation are taken to reduce the risk of hemorrhage and its related complications. When the condition has been clinically suspected and confirmed with imagenologic analysis, the treatment team needs to decide the strategy for its treatment and make a plan for resolving possible complications (Palacios-Jaraquemada, 2007). There is no optimal gestational age at which to schedule surgery for the treatment of abnormal placentation, but conventionally, programmed surgery is preferred even if fetal maturity is not completely guaranteed. Striking an optimal balance between maternal and fetal risk is a challenge in selecting an optimal date for surgery in abnormal placentation. Although the ideal time at which to perform a cesarean section is controversial, there is a tendency to operate between weeks 36 and 37 of gestation in cases of placenta accreta and at week 35 in placenta percreta. A study published in 2010 (Robinson and Grobman, 2010) found that a scheduled delivery at 34 weeks of gestation was the best strategy in cases of placenta previa and placenta accreta and resulted in the highest quality-adjusted life years under the base case expectations. Approaches involving delay for awaiting the confirmation of fetal lung maturity failed to yield better outcomes than strategies for delivery at the corresponding gestational age without amniocentesis. After sensitivity analyses, delivery at 37 weeks of gestation without amniocentesis was selected as the preferred strategy in limited situations, and delivery at 39 weeks of gestation was chosen as the preferred strategy only in unlikely situations (Robinson and Grobman, 2010).

Even with the best possible management, the blood loss associated with placenta accreta can resemble that in major trauma. For this reason, guidance of transfusion through the use of protocols for damage control resuscitation may reduce morbidity and mortality in this condition. Careful preparation and close communication between anesthesiology, obstetrics, interventional radiology, gynecology, oncology, blood banking, and specialized surgical teams are essential in the care of patients with placenta accreta (Snegovskikh et al., 2011).

It should be kept in mind that maternal morbidity is reduced in women with placenta accreta who deliver in a tertiary-care hospital with a multidisciplinary care team, rather than in a standard obstetric center (Eller et al., 2011).

4.3 Emergency

Emergencies related to abnormal placentation can be divided into those occurring in cases in which there is a presurgical diagnosis or suspicion or abnormality and those in which there is intrasurgical doubt. A presurgical diagnosis of abnormal placentation is ideal because it allows preparation of the treatment team and resources, which may or may not have limitations, as well as providing guidelines for alternative treatment. Moreover, while some centers have good equipment and facilities for emergency prenatal US and blood testing, others do not, and in some areas with large populations and low levels of birth control, it is common for a multiparous mother, sometimes with a history of multiple cesarean sections, to enter a hospital near term and with hemorrhage, in labor, or with other conditions that may compromise fetal well-being.

When cesarean section is needed urgently (e.g., as in serious fetal distress), all clinical and laboratory controls must await completion of the procedure. However, if exuberant and prominent vascularization is seen during laparotomy in the uterine segment involved in a case of abnormal placentation, especially in patients with previous cesarean sections, it is necessary to avoid hysterotomy for placenta previa or placenta accreta. It is preferable to extend the incision and incise the uterus through a safe area (Wax et al., 2004). In other cases, in which there is doubt about whether or not the placenta is firmly attached to the uterine wall, it should be remembered that hysterotomy can be performed far from the uterine location of placental attachment because if hysterectomy is done in a wrong location, it can produce a rapidly fatal hemorrhage. Obstetricians should be aware of the risk of uterine scarring and abnormal placentation in women who have undergone D&C, since it may lead to a life-threatening obstetric hemorrhage (Zwart et al., 2007).

Spontaneous uterine rupture can be a consequence of placenta percreta. Although placenta percreta is uncommon, the emergency physician should be aware of it because of its propensity to cause uterine rupture and catastrophic bleeding (Innes and Rosen, 1985). This possibility must be considered in patients with acute abdominal pain and shock during pregnancy and a previous diagnosis of abnormal placentation or known risk factors for it. Nevertheless, uterine rupture and bleeding are not mandatory reasons for hysterectomy, and in some cases can be treated conservatively (Filardo and Nagey, 1990).

The prognosis in a case of uterine rupture depends on the site of rupture, its size, its proximity or damage to the uterine pedicle, the location of abnormal placentation (S1 or S2), and the ability to achieve accurate vascular control as a

first step. A stable clot, appropriate hemodynamic status of the patient, and good technique are the keys to resolving this sudden problem. As mentioned earlier, subtotal hysterectomy or repair (for small ruptures) can be a solution to placental invasions in the S1 area. However, ruptures by placental invasions in the S2 area usually require total hysterectomy. The need for subtotal hysterectomy in cases of S2 rupture should be carefully weighed beforehand, because they are likely to represent rebleeding through the retroperitoneum, especially in patients in whom pelvic packing has been used or the parietal peritoneum has been closed over the vaginal cuff.

4.4 Additional resources

Some technical resources are available in cases of expected pelvic hemorrhage, although the relationships among their safety, advantages, and cost remain as controversial topics in obstetric practice. However, use of these resources can improve the treatment of projected postpartum hemorrhage, such as that in some types of abnormal placentation.

Cell salvage in obstetrics has significantly increased in the last few years (Allam et al., 2008; Louage and Van de Velde, 2010). However, there is poor evidence for its use in terms of economic justification or a reduced need for transfusion. Nor do any criteria now exist for use of a cell saver machine in the management of abnormal placentation because the prediction of hemorrhage in this setting is difficult, and initiating cell salvage when it is not needed is an obvious waste of resources. To minimize wastage of disposables, the reservoir of the cells saver can be set up at the beginning of a treatment procedure and the centrifuge system turned on only if the patient bleeds heavily (Peacock and Clark, 2011).

Intraoperative blood-cell salvage is used in obstetrics for women at significant risk of hemorrhage during cesarean section, such as those with placenta accreta, placenta percreta, and placenta previa. It can also be used for patients who refuse allogeneic blood transfusion. During cesarean section, the blood for cell salvage is collected with a suction catheter before incision of the uterus and after collection of the amniotic fluid. The blood is first processed and then transfused back into the patient, either during or after the operation (Kessack and Hawkins, 2010). A leukocyte-depletion filter is used to reduce the number of leukocytes in the transfused blood. The filter also reduces the volume of amniotic fluid and number of fetal blood cells in the transfused blood. The safety of blood-cell salvage in obstetrics has come under question because of concerns about the risks of contamination of recovered blood with amniotic fluid and of maternal–fetal alloimmunization.

Although data on the outcomes of patients treated with blood-cell salvage and its allogeneic blood-saving effect are still limited, current cell-saver machines remove most particulate contaminants from the blood, provide leukocyte-reduction filtration, and provide further safety measures. Furthermore, amniotic fluid

embolism is no longer considered an embolic disease but is instead proposed to be a rare anaphylactic reaction to fetal antigen. Consequently, intraoperative cell salvage in obstetrics should be considered in patients at high risk of hemorrhage or in cases in which allogeneic blood transfusion is difficult or impossible (Liumbruno et al., 2011).

4.5 Training

Accurate knowledge and precise skills are necessary for the management of abnormal placentation. Like many other pathologies, placenta accreta and placenta percreta have many faces, which can be seen before, during, or after surgery done for their treatment. In general, there is the need for decisions made about their management to show clear consequences of efficacy and error. The only problem is that these disorders can modify prior decisions within seconds. For this reason, obstetricians, clinicians, surgeons, and intensivists believe that the best option in their management is to reach a treatment plan and proceed with it, and under these circumstances training is mandatory.

Training in the managament of abnormalities of placentation can be divided into theoretical knowledge and surgical skills, both of which are necessary to achieve the best results. The first useful information in the management of these disorders is to know what not to do, because their management generally leaves no time in which to correct mistaken decisions. The obstetrician must always have in mind the concept of multidisciplinary management, but must also know that this is not possible in all centers. For this reason, it is necessary for the obstetrician to have a complete technical and clinical understanding of the problems inherent in the management of abnormalities of placentation.

Hemostatic and hemodynamic management are essential before, during, and after surgery. Although common tests exist for surgical patients, abnormalities of placentation hide particular problems. Knowing and managing them can be the difference between success and an unexpected outcome.

Surgical problems in the management of abnormal placentation involve clinical-surgical decisions and the surgical technique itself. The author has for years taught the complex anatomy of the pelvic–subperitoneal spaces and organ dissection in the pelvic region, but the accurate management of disorders in this region requires a personal, knowledge of its anatomy, in three dimensions .

Standard training in obstetrics and gynecology does not habitually include techniques for optimal management of the pelvic–subperitoneal spaces, although the training for some subspecialties, such as gynecologic oncology, does include this. The lack of familiarity with and practice of these techniques causes fear and insecurity in the performance of dissections in these spaces, which is probably why the average obstetrician avoids them. Yet without a 3D image of pelvic anatomy, surgery for disorders of placental adhesion can be a nightmare.

Experience with 2D images and prosected corpses has demonstrated their lack of utility for the in vivo management of clinical abnormalities. Perhaps the best means for acquiring a utile and practical knowledge of such management is to create one's own 3D image of pelvic–subperitoneal anatomy through active dissection. Fresh corpses can be a good resource in this respect, but are expensive, limited to use by a small number of participants, and do not reflect the complete reality of the living body. The author has for a number of years given courses in the surgical pelvic anatomy of live structures and tissues. These courses begin with a review of the pelvic anatomy in 2D based on surgical images and video presentations. The course participants then perform a series of predetermined procedures, which include dissection of the pelvic fascia and the opening and recognition of pelvic structures under close senior supervision. The procedures require 10 to 15 minutes in each case, and are previously approved by the Ethical Committee of the institution in which the courses are given. The main advantages of this approach are its low cost, availability, the participant's acquisition of knowledge and technique in 3D, and the use of living tissue. Other training includes learning in the operating theater, and may comprise the assessment of trainees, goal setting, and instructional methodologies.

5 Clinical problems

Abnormal placental adherence is not a problem restricted to the uterus and the surrounding organs; it also includes the management of possible complications, most of them clinical ones. Hemorrhage and its immediate and late consequences, such as shock or multiorgan failure, can determine the outcome for a particular patient. Although clinical management is not a direct responsibility of the obstetrician, the practice of obstetrics does entail an essential knowledge of most of the common clinical disorders affecting the female reproductive system and of their specific management.

5.1 Hemostatic problems

Common errors in the management of bleeding associated with placenta accreta are a failure to recognize the severity of the bleeding, insufficient administration of fluid during resuscitation, and delay in stopping the bleeding (Lombaard and Pattinson, 2009). Because abnormal placentation can be a cause of exsanguinating hemorrhage, it is recommended that the preparation for its surgical treatment include 4 bags of packed, crossmatched red blood cells (RBC); 4 bags of fresh-frozen plasma (FFP); and additional reserves of packed RBC, FFP, cryoprecipitate, and platelets (Snegovskikh et al., 2011). Additionally, two large-bore catheters (French catheter size 14–16) should be emplaced for the rapid restoration of fluid volume (blood and infusions) and that a central catheter be inserted for measuring central venous pressure (CVP) (Mayer et al., 2004). It is also recommended that a catheter be inserted in the radial artery for monitoring of the median arterial pressure, and that samples be taken regularly to check the patient's acid–base status and arterial oxygen pressure. Blood loss must be corrected with crystalloids, blood, and hemoderivates. For this, it is advisable to have one or two safe sites of venous access. Crystalloids or Ringer's lactate solution should be administered in a 3:1 ratio to the estimated hemorrhagic volume. The replenishment of volume acts to maintain peripheral oxygenation, protect the microcirculation, and avoid multisystemic damage. The administration of fluids must be done at an early point in accord with the patient's basic clinical signs. The accurate management of hemostasis and avoidance of hemorrhage whenever possible are essential because all continuous bleeding ends in coagulopathy.

General treatment guidelines for placenta accreta include hemorrhage control, volume correction, replacement of coagulation factors or platelets (if necessary), and correction of hypothermia or acidosis. Continuous monitoring during surgery is recommended, especially of hemodynamic and hemostatic parameters, which can change quickly after massive hemorrhage. This is also why emergency laboratory services can be important during surgery and recovery. Although continuous

monitoring guarantees the availability of volemic and other vital parameters, it is not always available, and in some institutions entails unacceptable delays. Despite the difficult problems entailed in anatomic access, dissection of NFV, and organ adherence, they are far smaller than the crisis of uncontrollable hemorrhage. Unmanageable bleeding leads to shock, coagulopathy, and death, and for this reason its prevention is a vital primary skill. Moreover, although the management of coagulation and shock may be very difficult without accurate laboratory control, the delay of at least 1 hour delay between the drawing of a blood or other sample and the results of its laboratory assay render these results historical as opposed to immediate, especially in the case of active bleeding. Consequently, the clinical experience and management provided by a skilled anesthetist are, with the exception of acid–base testing, more important than the hemostatic emergency test, which includes the fibrinogen level, platelet count activated partial thromboplastin time (aPTT), prothrombin time, and a hemogram (complete blood count [CBC]).

Furthermore, although abnormal placentation represents an obstetric and anatomic problem, it also represents a clinical problem. The sinuous placental implantation over damaged ureteral tissue that occurs in this abnormality, accompanied by both dynamic and passive traction, can promote microscopic tissue damage. As a response to this injury, a clot is formed and then reabsorbed through fibrinolysis. If this cycle is perpetuated, the plasma level of fibrinogen declines insensibly, without any clinical manifestation of this decline. During delivery, excess fibrinogen, induced by pregnancy, is deposited in the placental bed, creating a stable clot. But if the plasma level of fibrinogen is low or near 200 mg% at the time of placental detachment, the formation of a stable clot is almost impossible. The cycle that promotes clot formation and fibrinolysis results in immediate coagulopathy, hemorrhage, and subsequent hypotension, and shock and multiple organ failure if the disorder progresses or the resuscitative procedure is incorrect. So far, no clinical correlation has been made between the size, topography, or type of placental invasion in a given case with the likelihood of its causing abnormal fibrinolysis. For this reason, it is recommended that the levels of a patient's plasma fibrinogen and fibrin degradation products (FDP) be measured both 2 weeks and 24 hours before surgery for an abnormality of placentation. A plasma fibrinogen level below 250 mg/dL mandates the availability of 1 U of defrosted cryoprecipitate per 10 kg/weight before the patient's skin is incised (Palacios-Jaraquemada et al., 2004).

When accompanied by a coagulopathy or when more than 1 U of blood has been lost and intractable bleeding continues, the management of massive hemorrhage requires the use of plasma products. The coagulopathy in such cases results from hemorrhagic shock, hypothermia, and the activation, consumption, and dilution of coagulation factors. Plasma products have a critical role in maintaining sufficient levels of coagulation proteins to ensure hemostasis. Fresh-frozen plasma is a source of all coagulation proteins and is required when the prothrombin time (PT) and activated partial thromboplastin time (aPTT) exceed 1.5 times their normal

control values. Cryoprecipitate is the plasma product that has the highest percentage of fibrinogen. It is the most critical coagulation protein, and is required rapidly, and it is needed at levels > 1 g/L to maintain a stable clot. Prothrombin complex concentrates, monocomponent factor therapy, and fibrin sealants each have a role in specific clinical settings (Erber and Perry, 2006).

In a study of women experiencing postpartum hemorrhage, the plasma fibrinogen level was the hematologic parameter that best correlated with blood loss by hemorrhage and was the most useful marker of developing hemostatic impairment. Use of coagulation products that contain greater amounts of fibrinogen, such as cryoprecipitate or fibrinogen concentrate, may be more appropriate than whole blood or fresh-frozen plasma, especially if the plasma level of fibrinogen is very low or decreasing rapidly (de Lloyd et al., 2011).

The use of procoagulants such as recombinant factor VIIa (rFVIIa) has been described in the surgical treatment of abnormal placental adherence, and the literature contains some recommendations on the use of rFVIIa in massive postpartum hemorrhage, especially in cases refractory to other hemostatic measures. However, these recommendations must be interpreted with caution because they are based on few and uncontrolled studies. Further evidence is needed to know the real efficacy, optimal dose, and safety of rFVIIa in managing critical postpartum hemorrhage (Franchini et al., 2008; Searle et al., 2008).

5.2 Hemodynamic management

The goal in the hemodynamic management of abnormal adhesive disorders is to avoid hemorrhagic shock, which is defined as a syndrome resulting from the acute loss of circulating volume accompanied by a cardiorespiratory deficit and low tissue availability of oxygen. Changes occurring in pregnancy, which include an increase in blood volume in 30 %–50 % of cases, allow pregnant women to tolerate blood loss until they have lost 15 % of their blood volume before the onset of tachycardia or hypotension in the decubitus position. Because mortality is associated with the duration of shock rather than with volume loss, it is crucial to accurately measure a patient's hemodynamic parameters during every stage of surgery for abnormal placentation. An increase in the serum lactate level, increased peripheral extraction of oxygen, and a decrease in the plasma level of bicarbonate are indices of compromise in the systemic oxygenation. In the setting of hemorrhagic shock, capillary lactate levels correlate with the lactate levels in arterial blood. These preliminary findings suggest that the micromethod for quantitating blood lactate could be a bedside means of assessing the severity of hemorrhagic shock and guiding its treatment. The device for measuring lactate in capillary blood is easy to use, cost effective, and could be used for point-of-care monitoring in the hospital as well as in pre-hospital settings (Collange et al., 2010).

Decreased tissue level of oxygen is inversely related to the possibility of survival after acute hemorrhage. A decrease in venous pressure or oxygen saturation implies that peripheral utilization of oxygen exceeds oxygen delivery by blood flow (shock). Arterial hypotension at a diastolic blood pressure below 90 mm Hg in a patient in the decubitus position is a sign for alarm and can develop in massive obstetric hemorrhage without appropriate treatment. Blood loss above 30 % of the total blood volume exceeds physiologic reserves and makes tissue oxygen availability critical. Because a decrease in diastolic blood pressure to less than 70 mm Hg for more than 30 minutes is associated with mortality in more than 80 % of cases, proximal vascular control is critical in every instance of acute hemorrhage. Evidence shows that puerperal hysterectomy as a surgical method of hemostasis could be replaced by other, less invasive methods. Puerperal hysterectomy in the presence of hypovolem ic shock is associated with high morbidity and mortality. Although uterine bleeding can be stopped, its hemodynamic consequences can culminate in multiorgan dysfunction and death.

5.3 Anesthesia

Although spinal or epidural anesthesia is associated with fewer complications than is total anesthesia, the eventual presence of shock in a patient with sympathetic blockade induced by spinal anesthesia is not always well managed in cases of massive hemorrhage. In the case of some hemostatic disorders diagnosed before surgery, there is the high possibility of a compressive hematoma developing at the site of puncture for spinal anesthesia, which can grow and cause ischemia of the spinal cord. Similarly, spinal puncture associated with surgical DIC or deep coagulopathy, could have the same consequences as described earlier. If there is a significant risk of massive bleeding, substantial hypotension, or coagulopathy, general anesthesia is usually indicated in the surgical management of placenta accreta (Snegovskikh et al., 2011). However, regional anesthesia may be a good choice if there is accurate control of bleeding (Palacios-Jaraquemada et al., 2004) and in selected patients (Murata et al., 2009). A combination of regional and general anesthesia is also suitable in cases of conservative management in which general anesthesia is used for hysterectomy after delivery.

As with other surgical and clinical considerations in abnormal placentation, the key to effective anesthesia is the avoidance of bleeding. As mentioned earlier, massive hemorhage with a loss of 30 %–40 % of blood volume within a period of minutes can be extremely difficult to manage. Here once again the need for collaboration in the management team is evident. Medical resources, group experience, the ability to achieve accurate vascular control, and the ability to manage hemodynamic and hemostatic problems will determine the best technique for anesthesia in any particular case.

6 Surgical alternatives

Hysterectomy is the most frequent but not the only treatment for disorders of placental adhesion, and one of its main indications (73.3%) is to control the hemorrhage that can accompany such treatment (Christopoulos et al., 2011). This percentage once again reveals that the primary challenge in such treatment is to know how to efficiently stop uterine bleeding. In terms of surgical alternatives, hysterectomy is more widely used to control bleeding than are other means of accurate proximal vascular control. Therefore, emergent obstetric hysterectomy is closely associated with shock, coagulopathy, and high rates of morbidity and mortality, while nonemergent hysterectomy for placenta accreta is associated with significant morbidity in the form of hemorrhage and urinary tract insult (Hoffman et al., 2010). The known and typical complications of hysterectomy led to the consideration of conservative means of treating placental adhesive disorders.

6.1 Surgical approach

During their presurgical evaluation of patients with disorders of placental adhesion, the treatment team analyzes both surgical and clinical variables and possible complications and their solutions. Clinical aspects of the presurgical evaluation include chemical and hemostatic tests, types of anesthesia, means for blood replenishment, and aspects of general management. Decisions relating to surgery may be more complex. First in this regard is the need for a consensus on the best time at which to perform a surgery. As mentioned earlier, there is a general consensus that 35 weeks of gestation is a good time at which to perform surgery for placenta percreta and 36 to 37 weeks is a good time for the surgical treatnment of placenta accreta. However, labor, hemorrhage, hematologic disorders, and fetal problems can all modify these times. The topography of placental invasion in a particular case is very important in planning the tactics and surgical technique for its treatment (Fig. 6.1). The S2 region is the most common site of invasion, but not all adhesive abnormalities in this region present the same level of difficulty (Figs. 6.2–6.5) An intrinsic difficulty in S2 invasion, which involves the parametrium (Figs. 6.6–6.9), comes from the close proximity of this region of the uterus to the ureter within the narrow pelvic space. For this reason, it is highly recommended that some type of ureteral catheterization be used when parametrial invasion has been diagnosed. Moreover, despite the high level of reliability of prenatal diagnostic procedures, there is no certainty about the type or subtype of placental adhesive abnormality in a particular case until the abdomen is opened. Consequently, it is convenient to have a treatment plan based on the surgical findings in a given case, since both under- and overestimation of the patient's pathology can result in inappropriate surgery. Placental MRI provides a high degree of accuracy for

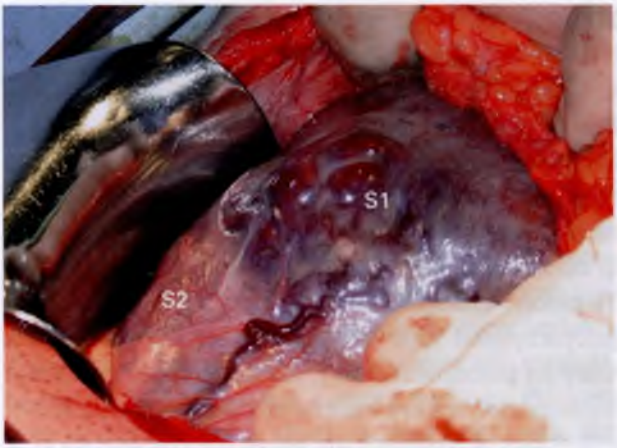

Fig. 6.1: Surgical view of abnormal placentation in the S1 area secondary to laparoscopic myomectomy. A clear S2 area, available for performing a safe hysterotomy, was seen. After delivery, uterine vascular control was accomplished with four clamps, and the entire area of invasion, with the placenta, was removed. One year after reconstruction (one-step conservative surgery), the mother in this case gave birth to a new infant by cesarean section without complications, a recurrence or abnormal placentation, or postpartum hemorrhage.

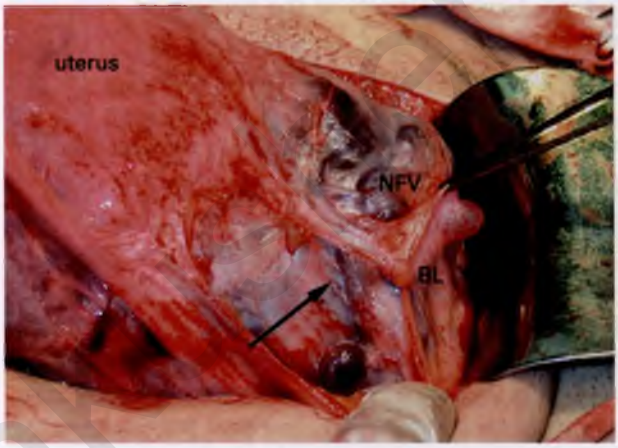

Fig. 6.2: Surgical view of abnormal placentation in the S2 region. Massive development of NFV between the uterus, placenta, and bladder (BL) is evident. Nevertheless, a distinguishable plane is seen between the bladder and uterus (black arrow).

determining the possibility of uterine damage and hence of uterine preservation in a particular case. Cases in which there is diagnostic doubt in relation to the patient's clinical or obstetric background require diagnostic confirmation in the operating room because some types of dehiscence of scars from the surgical treat-

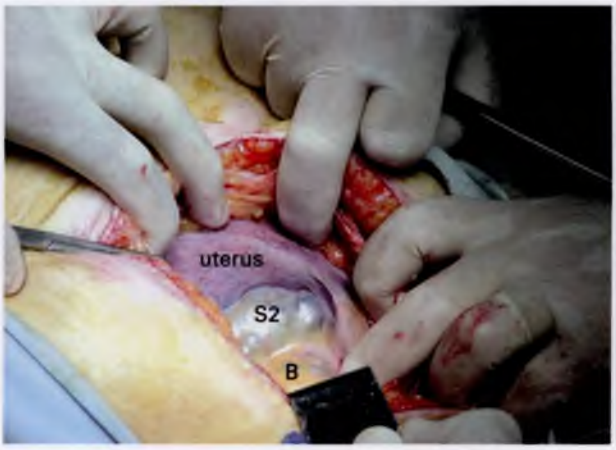

Fig. 6.3: Surgical view of abnormal placentation in the S2 area in a second pregnancy, treated with curettage after cesarean section for a deep, aberrant cotyledon and 10 days of subsequent metrorrhagia. Behind this seemingly slight placental invasion is a serious bladder (B) invasion with partial dehiscence of the uterus. After an accurate dissection, one-step conservative surgery was done. The patient became pregnant again on two occasions (at 14 and 25 months respectively, after her plastic procedure) without recurrence.

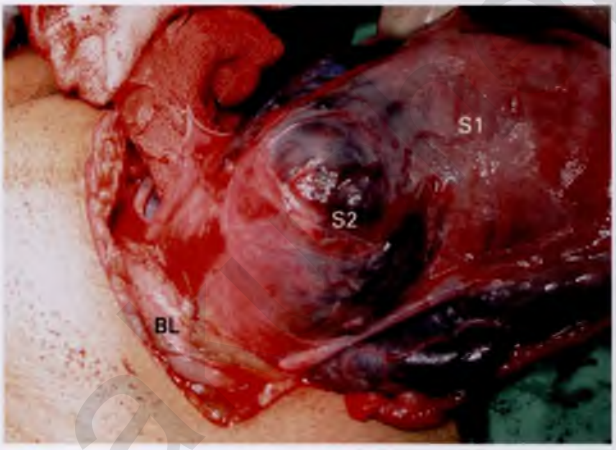

Fig. 6.4: Surgical view of abnormal placentation in the upper S2 area. Previous bladder (BL) dissection allows accurate exposure of the area of invasion. After delivery, the entire area of invasion was removed together with the entire placenta. The presence of normal myometrium below the invaded area is essential for one-step conservative surgery.

ment of placenta previa can mimic serious cases of abnormal placentation (Figs. 6.10 and 6.11). Although all conservative and resective approaches have a risk, a presurgical guide with possibilities of decision is provided.

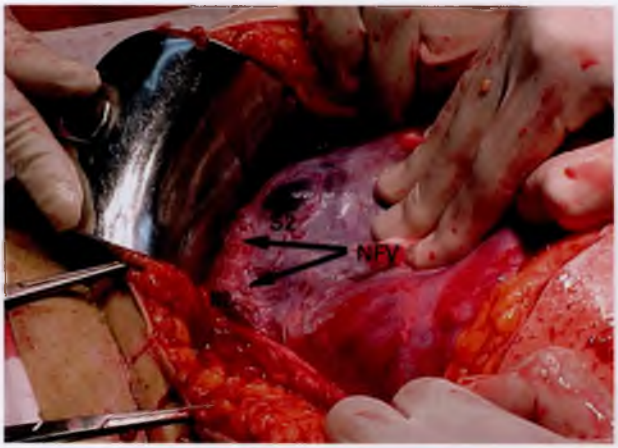

Fig. 6.5: Surgical view of abnormal placentation in the S2 area. In some cases the NFV in such invasion are not very evident at first, and there is a temptation to avoid bladder (BL) dissection and to instead perform an upper hysterotomy.

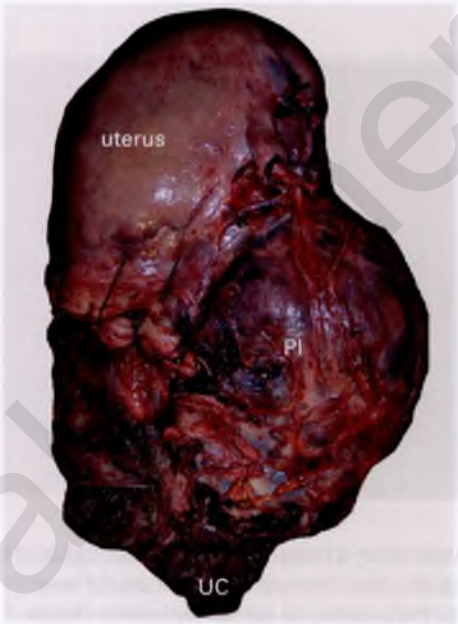

Fig. 6.6: Surgical specimen of total hysterectomy, showing massive left parametrial invasion (PI) extending to the uterine cervix (UC).

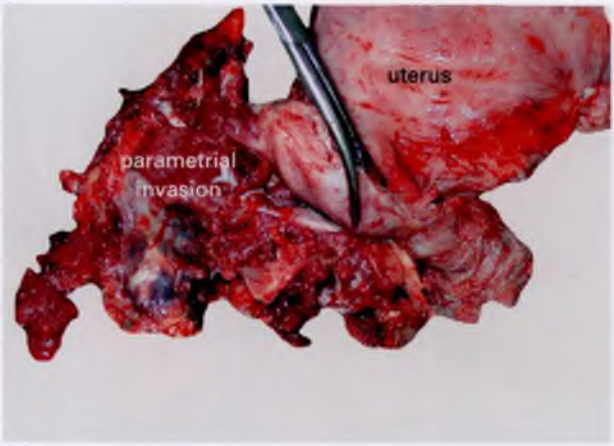

Fig. 6.7: Surgical specimen of total hysterectomy, anterior view, in a patient with two previous cesarean sections and a history of abrasive abortion at 6 months after a late cesarean section. Parametrial invasion was discovered during surgical exploration. A US examination was done for anterior placenta previa; note the absence of classic anterior invasion. Because of limitations in US, pMRI is the only method for detecting parametrial invasion.

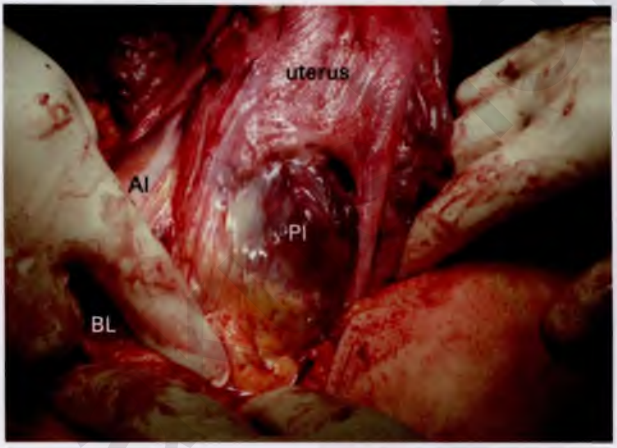

Fig. 6.8: Surgical view of anterior placenta percreta with parametrial invasion. Note the communication between uterine vessels and vesical–uterine NFV (separated by the round ligament). This wide communication consists of a dense population of vessels that makes possible the passage of embolic material between the two vascular systems, increasing the possibility of unwanted embolization of the bladder and vagina. PI: Parametrial invasion, BL: Bladder, AI: Anterior invasion. Destruction of more than 50 % of axial uterine diameter turns impossible a uterus repair.

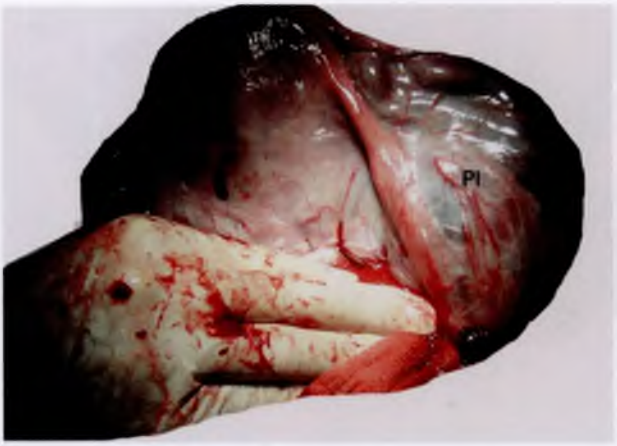

Fig. 6.9: Surgical view of anterior placenta percreta with parametrial invasion: Lateral placental bulging compresses the lateral pelvic wall, making it impossible to introduce a ureteral catheter. Surgical exploration by a urologist was initially unsuccessful, but after a deep dissection, the ureter was found 3 cm outside the ovarian pedicle. In cases of parametrial invasion and inability to find the classical anatomic landmarks, the rotation and movement of anatomic elements should be considered for the purpose of reorganizing the search for their locations.

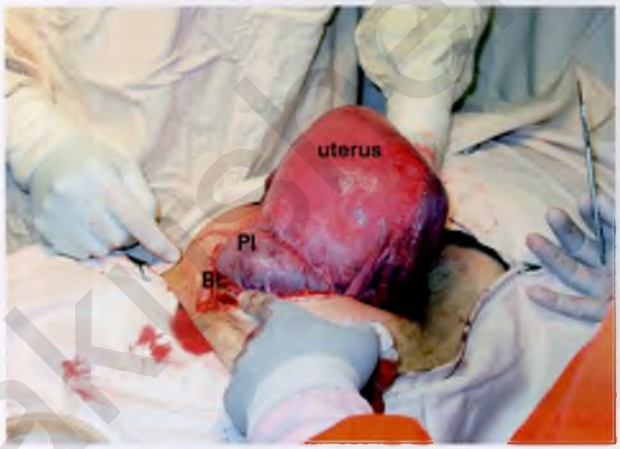

Fig. 6.10: Intraoperative view: A simple bulging of the uterine wall caused by deficient scar tissue or a partial placental hernia (PI) thought to represent the site of a previous hysterotomy can be confused with placenta percreta. In such cases, US typically shows placental tissue extending up to the serosa, but this does not represent true placenta percreta. The absence of additional signs, such as lagoons or NFV, in a patient without evident antecedent factors for placenta previa makes it necessary to carefully explore the area of involvement during surgery, in order to avoid overdiagnosis.

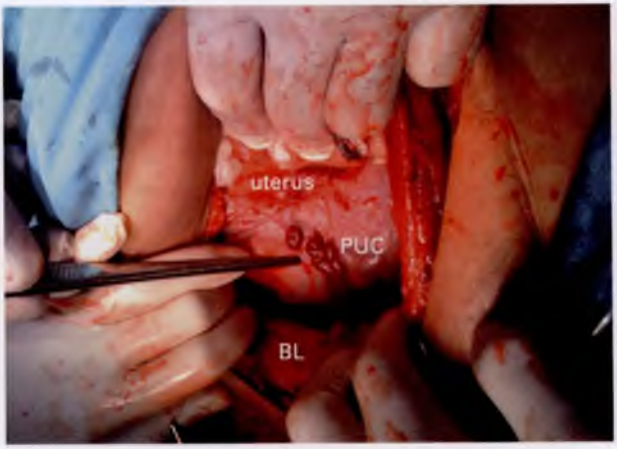

Fig. 6.11: Intraoperative view in a patient with one previous cesarean section and total occlusive placenta previa. Thinning of the myometrium, the presence of NFV, and a placenta in contact with uterine serosa were seen on US examination. Because of the prenatal diagnosis of placenta percreta, a total abdominal hysterectomy was planned. Placental MRI also showed features of placenta previa, but also showed parallel vessel disposition in the uterine surface. This type of circulation is compatible with an additional blood supply commonly seen in cases of placenta previa. Surgical exploration demonstrated an exuberant pericervical and segmental circulation without any abnormal placental adherence, which counteracted an initial proposal of puerperal hysterectomy by simple cesarean section.

6.2 Resective procedure (hysterectomy)

As explained in the discussion of Scenario 2 in Chapter 4, hysterectomy in abnormal placentation is not a minor issue and must be considered as complex surgery. Knowledge of when to use a specific procedure for hysterectomy is indispensable to avoiding serious complications. The risks of uncontrollable hemorrhage and tissue damage exceed the margin for erroneous decisions. Pulling out a neonate through an undamaged area of the uterus, ligating the umbilical cord, and closing the uterus are the best and safest options when resources and skills are limited.

The resources needed for safe and efficient hysterectomy depend on the conditions in which the hysterectomy is performed. Hemostatic hysterectomy, with the goal of preventing uncontrollable bleeding with hypotension, coagulopathy, or peripheral shock, typically carries a high degree of clinical risk and requires close control to avoid immediate and late multiorgan failure. For this reason, the decision to perform hemostatic hysterectomy should include the pre-existence of accurate proximal vascular control to provide time for the restoration of hemodynamic and hemostatic parameters. The circulatory volemic deficiency in patients with sustained shock can cause peripheral hypoperfusion, increased capillary

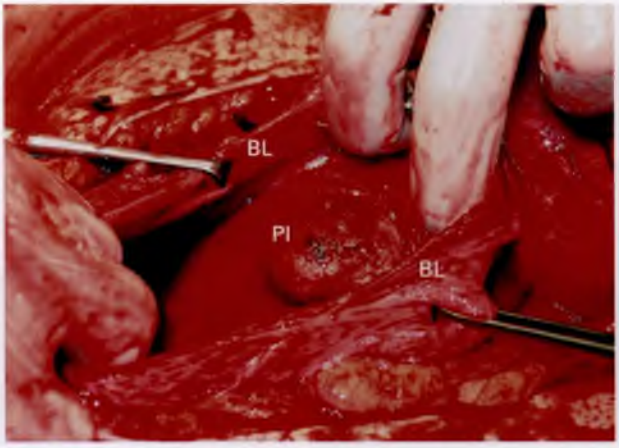

Fig. 6.12: Intraoperative view (bladder is open). Macroscopic bladder invasion was evident. The patient had recurrent episodes of macroscopic hematuria for 2 weeks. Initial electrofulguration by cystoscopy was successful. If hematuria resumes, a second attempt at electrofulguration is strongly contradicted because of the possibility of placental rupture. Arterial embolization was not available.

permeability, and leukocyte diapedesis, among other disorders. Because of these factors, it is necessary to understand that in terms of treatment, hemostatic hysterectomy is an extreme resource in cases of abnormal placentation, and that capable clinical support is essential to its success. Patients undergoing hemostatic hysterectomy also require continuous checking of their acid–base status as an indicator of the systemic inflammatory response syndrome (SIRS), a generalized immune response to peripheral damage caused by shock. Stable vascular perfusion, which differs from normotension, puts the patient in the best possible physiologic state before and during resective uterine surgery.

Elective postpartum hysterectomy in hemodynamically stable patients is also an important procedure, but is usually well tolerated. Postoperative controls are focused on detecting unexpected bleeding and assessing the patient's hemodynamic and hemostatic variables. Total abdominal hysterectomy is recommended for abnormal placentation in the S2 region, and subtotal hysterectomy for that involving the S1 region. Because the surgery needed in cases of S2 invasion is highly complex, many authors have chosen subtotal hysterectomy in this situation. However, this can be extremely dangerous because of a demonstrated high rate of recurrent bleeding with this procedure. The potential involvement of the cervix and other pelvic structures by placental adhesive disorders may change the approach to using hysterectomy rather than subtotal hysterectomy (Wang et al., 1998; Tadesse et al., 2011).

The resection of placentally invaded structures in the lower pelvis, especially the cervix and bladder, can be technically difficult (Figs. 6.12–6.14) and potentially

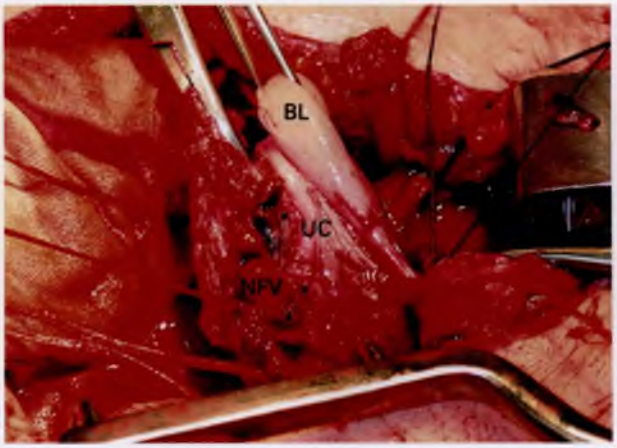

Fig. 6.13: Intraoperative view of cervical–trigonal invasion. Firm, adherent placental tissue was left between the bladder (BL) and the cervix (UC). Notice the dense network of anastomotic NFV (asterisks).

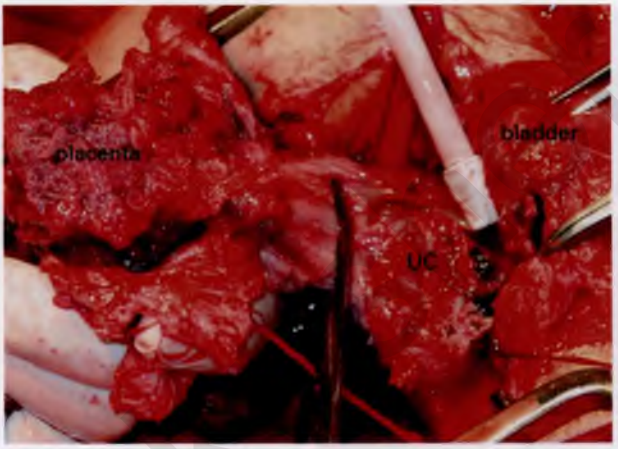

Fig. 6.14: Intraoperative view of cervical–trigonal invasion. Residual placental tissue has been removed, leaving the uterine cervix (UC) clean. The bladder was repaired and the procedure was completed.

dangerous without accurate proximal vascular control. When vascular control is unavailable, circular and consecutive stitches can be placed around the lower part of the uterus before hysterectomy is performed (Fig. 6.15). In the same lower placental invasions, the tissues in the vesico-uterine space are made difficult to dissect by excessive scar connective tissue between the bladder and the invaded myometrium. Evident displacement of the ureter among others may make the dissection almost impossible, even for trained or senior specialists (Fig. 6.16).

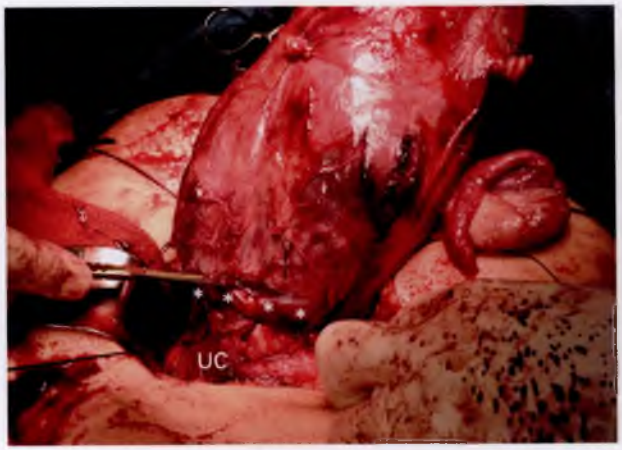

Fig. 6.15: Intraoperative view of cervical–trigonal invasion. Because of the intensive fibrous tissue mixed with NFV, suturing of the uterine cervix (UC) for secure hemostasis may be is one of the safest solutions for treating a case such as this.

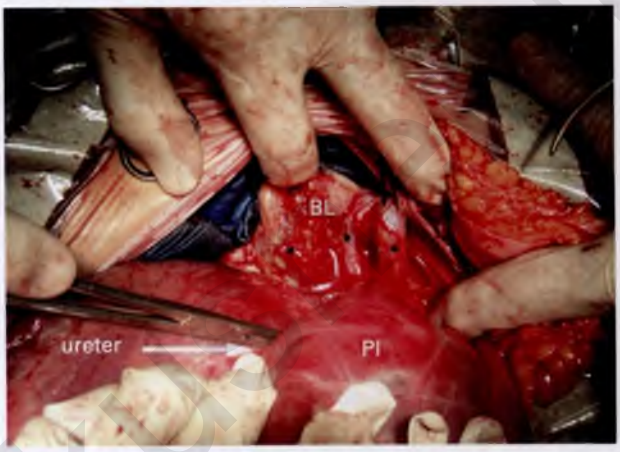

Fig. 6.16: Intraoperative view of massive anterior and parametrial invasion: The dissection plane between the bladder and invaded area is fibrotic and adherent. Newly-formed vessels (asterisk) are not easily identifiable. The ureter was displaced to the midline by placental invasion between anterior and parametrial invasion (white arrow). During initial exploration, the ureter was at first confused with the vesical artery.

The intensivist needs to be alert to the possibility of hypotension in patients undergoing hysterectomy for the treatment of abnormal placental adhesion. The intensivist also needs to understand that complimentary diagnostic procedures are required for the clarification of all clinical signs and symptoms that produce inexplicable hypotension. If there is doubt, CT is preferable to US as a means for explor-

ing all of the abdominal compartments, particularly the retroperitoneum and pelvic–subperitoneal spaces. Closing the peritoneum over the cervical stump can shift blood loss to the retroperitoneum after hysterectomy and delay the identification of this. Abdominal US is usually negative for bleeding because it commonly includes only the hypogastrium. Hypotension after hysterectomy in patients without evidence of bleeding (external or internal) is generally interpreted as a result of hypovolemia. Some obstetricians refuse to re-explore patients with manifestations of possible bleeding, which is understandable in view of the technical problems involved in removing the rest of the uterus (lower segment and cervix) in subtotal hysterectomies or in finding the source of bleeding if it is occurring. A simple option is to order an angiography while remembering that this should also cover both the anterior division of the internal iliac artery and the internal pudendal artery because of their role as origins of the main collateral vessels in the S2 region. The internal pudendal artery is the first branch of the posterior trunk of the internal iliac artery and follows the direction of the trunk of this artery, for which reason many anatomists consider the pudendal artery as a terminal branch of the internal iliac artery. To be completely sure that the catheter for angiography is inside the internal pudendal artery, irrigation of the vulva should be seen after the injection of contrast medium (Palacios-Jaraquemada et al., 2007). However, the exploration for sources of bleeding after hysterectomy, even when done by a well-trained interventional radiologist, can be made difficult or erroneous by surgically induced modifications in regional anatomy and vasoconstriction resulting from hypotension.

If angiography is not available or ineffective, surgical exploration is needed. Because of the complex nature of the dissection required for placental invasion of the S2 region, good proximal vascular control and identification of the ureter are recommended before it is begun. These two measures help to ensure the safety of the dissection. It is very important for the obstetrician to find the source of bleeding, which is generally the cervical stump or ovarian artery. Despite some opinion to the contrary, it is probably not impossible to identify the source of bleeding in a pelvic hematoma. If the hematoma has caused hypotension, the origin of the bleeding must be identified and vascular control, ureteral identification, and patience offer the best possible means for accomplishing this. Although a retroperitoneal hematoma is uncommon, it is a complication that carries the potential for serious morbidity and possible mortality (Ridgway, 1995). A 3D anatomic knowledge of pelvic structures and landmarks is necessary for the safe and effective treatment of such a hematoma; without these skills, anatomic distortion by the hematoma makes it impossible to identify vital structures (Committee on Maternal Welfare, 1966).

The use of pelvic packing after hysterectomy is recommended in the presence of unstable clots or when oozing is evident (Figs. 6.17 and 6.18). Its permanency must be closely controlled in relation to hemodynamic parameters, and care must be taken that its abdominal volume does not hide source of hemorrhage.

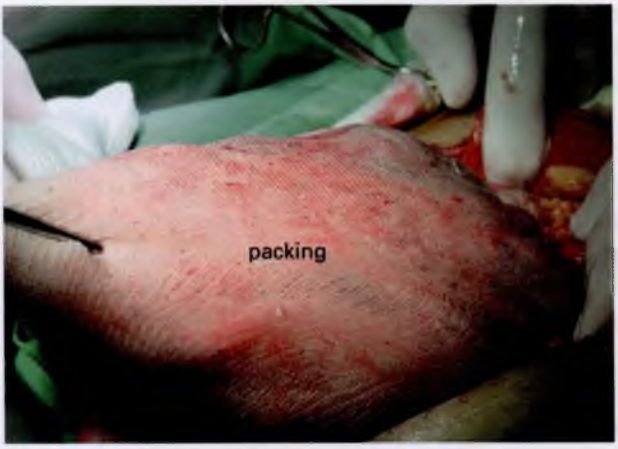

Fig. 6.17: Intrapelvic hemostatic packing: The use of a tubular bandage as a bag allows the insertion of multiple laparotomy pads as a single unit. This type of bag facilitates molding of a packing to fit into the pelvis, including insertion through a Pfannenstiel's incision.

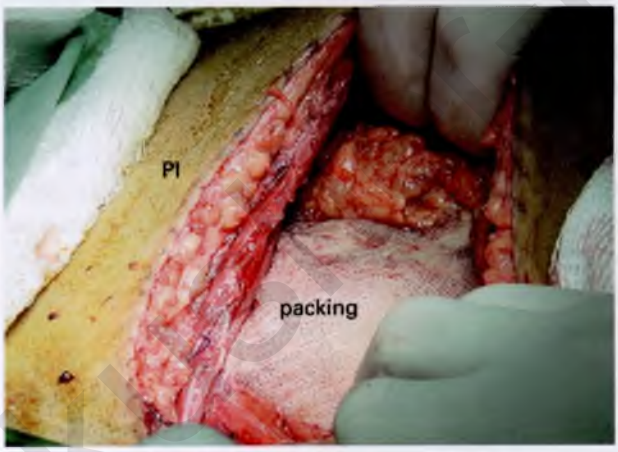

Fig. 6.18: Intrapelvic packing in situ: Placement of pelvic packing as a unit facilitates its removal without the need for attention to the number of laparotomy pads left in place, because both the upper and lower ends of the tubular bandage are knotted.

Lastly, it should be noted that the avoidance of hysterectomy as a primary treatment modality for abnormal placentation in patients under 40 years of age might decrease the possibility of premature ovarian failure, because hysterectomy cuts both the ascending and ovarian branches of the uterine artery, which can decrease ovarian blood flow and function (Wen et al., 2006; Xiangying et al., 2006).

6.3 Conservative procedures

Conservative treatment for abnormal placentation was designed primarily to avoid bleeding and secondarily to preserve fertility. Two procedures are available for this, which differ completely from one another in design. One-step surgery represents an attempt to resolve all of the problems of abnormal placentation, such as hemorrhage, induced NFV, and tissue damage; treatment of the placenta in situ emphasizes the avoidance of hemorrhage. Differences between the procedures for in situ placenta and one-step conservative surgery are especially marked with regard to the rates of recurrence of hemorrhage and other complications in subsequent pregnancies and in the complexity of the follow-up and surgical procedures for treating these complications. Treatment of an in situ placenta carries a greater risk of recurrence of bleeding and infection, and a high rate of recurrence of this condition in a subsequent pregnancy.By comparison, one-step conservative surgery has a minimum risk of complications in relation to previous procedures, although the complexity of the procedure is greater and greater training is needed for its safe performance.

6.3.1 In situ placenta

To prevent anticipated bleeding, conservative treatment in abnormal placental adhesion avoids dissecting tissues in adverse situations (Figs. 6.19 and 6.20). How-

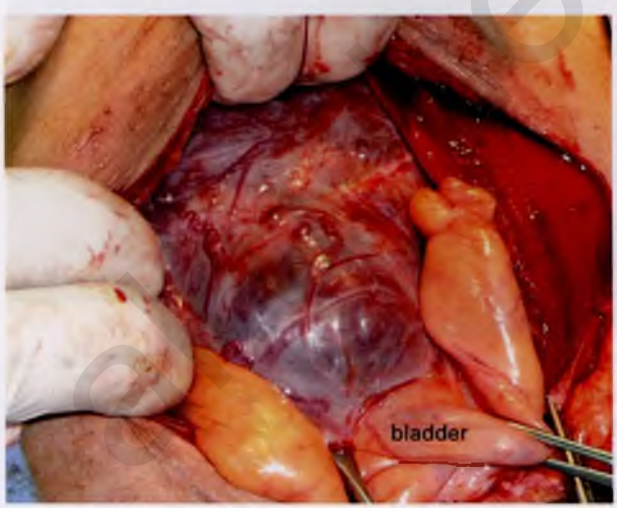

Fig. 6.19: Anterior placenta percreta diagnosed in the operating room in a patient with Type O negative blood and without any previous controls (obstetric or ultrasound) for pregnancy. Lack of resources or training could rapidly transform this problem into a crisis. The infant in this case was delivered by fundal incision and the placenta was left in situ. One week later, the area of placental invasion and the placenta were removed in a single surgical section. The uterus was repaired in two planes (two-step conservative surgery).

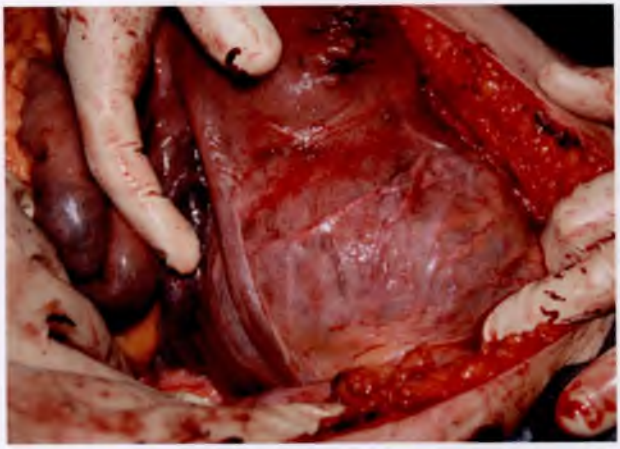

Fig. 6.20: Conservative treatment in a patient with placenta accreta suspected prenatally on the basis of Doppler US and MRI. The illustration shows anterior placenta previa in a patient with a history of two previous cesarean sections and curettage. After intrasurgical evaluation, the patient's condition was considered to be placenta accreta without signs of bladder invasion.

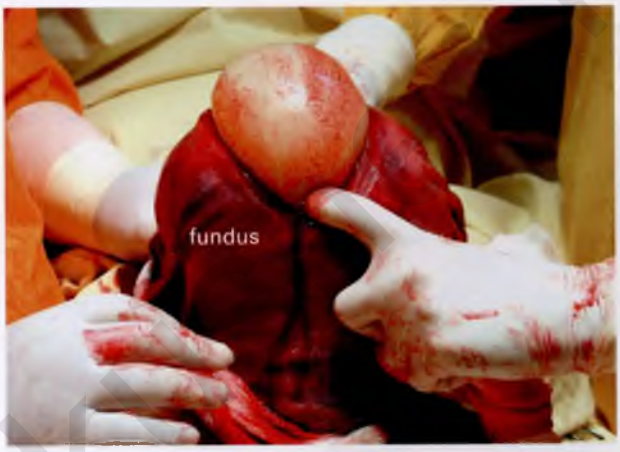

Fig. 6.21: Intraoperative view: Fundal hysterotomy in a case of anterior placental invasion (AI). The infant was delivered in an area without placental tissue.

ever, this is not a new approach to abnormal placentation (Brody, 1963), and its first successful use was described nearly 80 years ago (Capechi, 1933). When it is preferred to leave the placenta in situ, hysterotomy is done from outside the area of placental invasion, the infant is removed, and the umbilical cord is cut near the placenta with carefully controlled hemostasis (Figs. 6.21–6.23). This appears to be a safe alternative to the radical management of abnormal placentation (Kayem et al., 2007), especially when extrauterine organs such as the ileum or rectus are

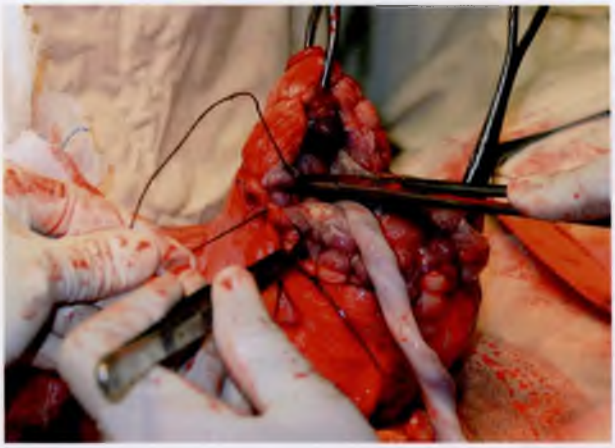

Fig. 6.22: Intraoperative view: After delivery, the umbilical cord was ligated near the insertion of the placental insertion of the cord. The uterine edges were sutured with continuous hemostatic sutures.

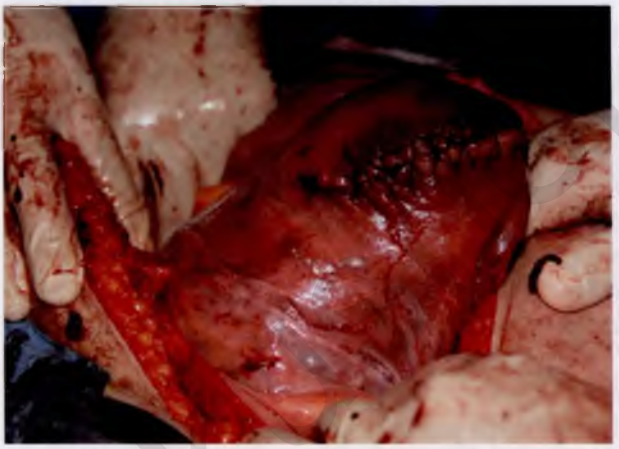

Fig. 6.23: Intraoperative view: Surgical aspect after closure of the uterus.

involved (Lee et al., 1995; Pearl et al., 1996). The invaded area and the placenta are left in situ without any attempt to remove them (Fig. 6.24). Some authors embolize both uterine arteries and partially resect the placenta. This maneuver, which reduces placental mass, clearly reduces initial bleeding and is associated with a reduced rate of infection. However, it leaves the possibility of secondary bleeding, DIC, and septic shock. The role of methotrexate or arterial embolization in improving the safety of the conservative management of abnormal placental adhesion and placental reabsorption requires further study (Musalli et al., 2000). The administration of antibiotics might be effective in preventing uterine infection,

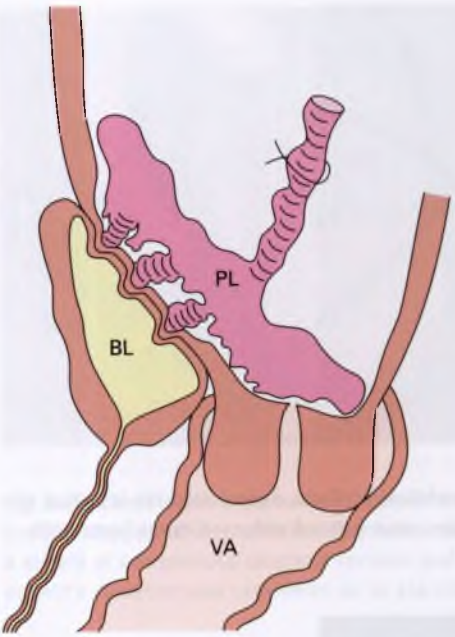

Fig. 6.24: Position of the placenta and area of placental invasion with the placenta is left in situ. PL: Placenta, B: Bladder, VA: Vagina.

but their efficacy remains to be proven (Timmermans et al., 2007). Daily control by laboratory blood testing and assessment of clinical parameters to detect complications such as DIC or infection, the use of antibiotics, preventive arterial embolization, and a prolonged hospital stay imply additional expenses without assuring therapeutic efficacy. Under ideal conditions, the measures described above for the in situ treatment of abnormal placental adhesion result in a decrease in the size of the placenta or in its reabsorption or calcification within weeks or months. Spontaneous placental expulsion has also been reported, with and without hemorrhage.

After the primary episode of adhesion has been resolved and the placenta has been expelled or reabsorbed, there is the possibility of future pregnancy. However, although numerous cases of pregnancy following the conservative management of an in situ placenta have been reported, the reproductive outcomes in such cases have generally shown a high risk of recurrent adhesion in a subsequent pregnancy. In a series of 96 patients or whom follow-up data were available after the conservative treatment of placenta accreta, 8 of the patients had severe intrauterine synechiae and were amenorrheic, 34 had had new pregnancies, 21 had had third-trimester deliveries of healthy infants, 1 had had an ectopic pregnancy, 2 had had elective abortions, and 10 had had miscarriages; 28.6 % of the patients had had recurrences of placenta accreta and 19 % had had postpartum hemorrhage (Sentilhes et al., 2010). However, conservative treatment for placenta accreta can help women avoid hysterectomy and when done in centers with adequate equipment and resources involves a low rate of severe maternal morbidity (Sentilhes b, 2010).

The postoperative care of patients undergoing in situ treatment for abnormal placentation is related to the type of surgery performed. Conservative treatment of an in situ placenta is probably the treatment option that requires the greatest care. Because placental tissue is left in the patient, the most important concern is infection. Prophylaxis and concurrent treatment with two broad-spectrum antibiotics that cover gram-negative, gram-positive, and anaerobic pathogens is needed, but is not a guarantee of success. The most used antibiotic regimen includes the administration of amoxicillin and clavulanic acid (875 mg t.i.d.) for 10 days. If signs of infection appear, a combination of amikacin and metronidazole is usually added. But as sometimes happens in other clinical situations, antibiotics in the presence of infected tissue may be completely ineffective, and because it is well-known that antibiotic treatment can mask incipient sepsis, daily clinical examination and laboratory evaluation of the patient are necessary. Cases of septic shock in the conservative treatment of placenta in situ have been reported (Chiang et al., 2006), as has late infection with placental removal and uterine conservation (Morel et al., 2009). However, obstetricians are aware that unnoticed infection from a lack of close control or a highly virulent pathogen can have unexpected outcomes. The postoperative clinical evaluation of patients undergoing conservative treatment for abnormal placentation must take into account all signs and symptoms of possible infection because the latter can be silent or hidden. Although myometrial tissue is highly resistant to infection, hysterectomy is usually the elective treatment when placental infection cannot be controlled. This is logical, first because uterine repair made over infected tissue is doomed to failure, and second because the patient's recovery and the avoidance of sepsis are priorities in any treatment for abnormal placentation.

A retained placenta can also be a cause of coagulopathy through sepsis or another source of endothelial damage (Letsky, 2001). For this reason, complete testing of the patient's coagulation status is strongly recommended during postoperative management. The most frequently used tests of clotting function are the PT, partial thromboplastin time (PTT), and tests for fibrinogen and fibrin degradation products (FDP), together with diagnostic tests for DIC such as the D-dimer test, which can be early markers of major hemostatic problems. The occurrence of a coagulopathy necessitates the removal either of placental tissue or the uterus. Hysterectomy is the most common treatment in this situation, although removal of the placenta and tissue repair with accurate hematologic treatment may be another option. Proximal vascular control is strongly recommended while hemostatis and hemodynamic parameters are stabilized. This type of vascular control was well tolerated, and permitted preservation of the uterus and less operative time.

Another possible complication of treatment for in situ placenta is bleeding. Postoperative uterine contractions and areas with partial placental detachment can be the sources of such bleeding. Attempts at manual removal of the placenta after methotrexate therapy and occlusion of the internal iliac artery by balloon catheteri-

zation can result in massive hemorrhage (Butt et al., 2002). For this reason, patients being treated for an in situ placenta require measures for preventing such hemorrhage. Evaluation of the blood color and volume of blood loss, frequency of bleeding, and effects of clinical attempts to modify such hemorrhage permit a decision to either wait for it to stop or intervene actively to stop it. Selective arterial embolization or hysterectomy can be effective treatment options. Some protocols include selective and bilateral uterine artery embolization as a measure for preventing secondary bleeding. However, it has not been proved that this last measure avoids or reduces the risk of hemorrhage or, conversely, that it increases the possibility of infection.

Retained placental tissue can remain in place (Davis and Cruz, 1996), be expelled, undergo a reduction in volume, or calcify (Diop et al., 2009). There is no linear correlation between the blood or urine level of chorionic human gonadotropin (hCG) and the size of retained placental tissue because hCG reflects the activity of placental tissue rather than its permanency as inactive tissue in abnormal placentation. Spontaneous delivery of the entire placenta has been described in early stages after conservative treatment for abnormal placentation. In these cases, placental tissue was expelled vaginally, without additional bleeding. The author has seen cases identified as representing abnormal placentation in which placental detachment occurred spontaneously during cesarean section. In these cases a US scan can show evidence of abnormal placentation without a myometrial interface beyond the placenta. This apparent imagenologic–surgical contradiction has been explained by the clear revelation, upon downward displacement of the bladder, of a retrovesical hole in the myometrium through which placental tissue can reach the uterine serosa, but without NFV between the two surfaces. For this reason it is important to stress that the final diagnosis of abnormal placentation is made in the operating room, and that the best option for treating it can be pursued after this (Fig. 6.25). The partial expulsion of placental tissue is also described in the literature (Timmermans et al., 2009), but there is no consensus about whether the placenta should be removed in the postpartum period in such cases, or be left in place to be reabsorbed or be expelled spontaneously. Some reports describe removal of the placenta in the postpartum period at intervals varying from 2 weeks to 2 months after delivery (Doumouchtsis and Arulkumaran, 2010). A combination of oxytocic agents has been successfully used for the expulsion of a retained placenta accreta in two cases (Morgan and Atalla, 2009), but there is a need for care in such cases because some placental tissue may be trapped in the uterus by thick NFV. Manual extraction of the remaining placental tissue in this situation may precipitate torrential bleeding (Teo et al., 2008; Palacios-Jaraquemada and Fiorillo, 2009). However, hemorrhage can also occur late after surgery (postoperative day 44) in patients chosen for the conservative treatment of an in situ placenta (Luo et al., 2005). Patients who opt for conservative medical management should be informed about the possibility of catastrophic bleeding associated with a retained placenta, which would ultimately require blood transfusions and hysterectomy.

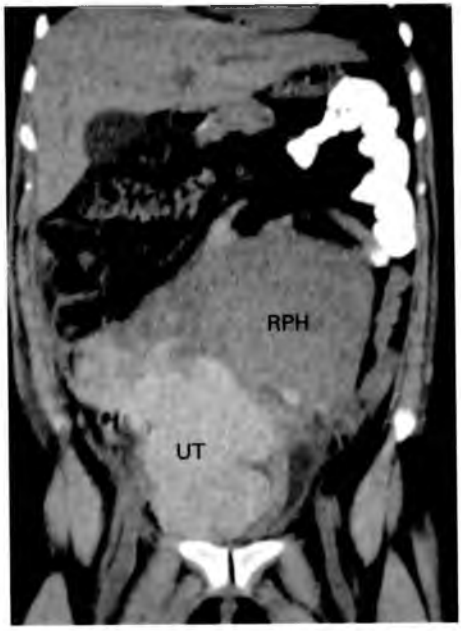

Fig. 6.25: Computed tomography, coronal reconstruction: There has been partial removal of placental tissue during cesarean section because of an adherent aberrant cotyledon. Carbetocin and methilergonovine were added to enhance contraction and hemostasis. Four hours later, the patient developed persistent hypotension. No vaginal bleeding occurred. A US examination showed no free blood and an empty endometrial cavity with some small clots. A CT scan showed a large retroperitoneal hematoma (RPH) with normal uterus (UT). Surgical exploration showed a pelvic–subperitoneal uterine rupture (1 cm). Residual placental tissue was removed and the uterus was repaired in two planes.

In a case of late retained placenta complicated by pelvic pain and menorrhagia unrelieved by sharp curettage, resection of the area of uterine invasion and retained placenta was described as successful (Schnorr et al., 1999), and was more recently described as an effective initial treatment for placenta percreta (Simsek et al., 2010). Another report describes resection of a placentally invaded area of uterus after severe hemorrhage resulting from manual detachment of the placenta (Riggs et al., 2000). After resection, three Foley catheters were inserted into the uterus and it was closed to provide hemostasis, with concomitant treatment with methotrexate (Riggs et al., 2000). Persistence of the myometrial bulging and flaccid area characteristic of anterior abnormal placentation prevents the hemostasis normally produced by myometrial contraction. An accurate resection and new suturing provide good myometrial support and improved hemostasis. The concomitant use of methylergonovine, carbetocin, and other oxytocic agents is not recommended for reducing the area of distorted uterine scar. Although intensive myometrial contraction could reduce the redundant and thin uterine scar. This thera peutic measure is not recommended because when uterine tonus decreases, bleeding occurs a few hours postpartum.

Calcification of retained placental tissue is also possible (Diop et al., 2009), but is generally not a significant problem. However, large intrauterine areas pf calcified tissue can act like an intrauterine device and produce infertility. Tissue removal by hysteroscopy usually solves this problem.

Methotrexate, a folate antagonist, it was proposed as an adjuvant treatment for abnormal placental adhesion (Arulkumaran et al., 1986). Methotrexate inhibits

rapidly dividing cells and is known to be effective against proliferating trophoblastic tissue. However, the mitotic activity of the placenta at term is less than 1%, and for this reason the real utility of methotrexate for treating abnormal placental adhesion is unknown. Placental expulsion at 5–18 days after methotrexate therapy has been reported (Timmermans et al., 2007), but there have also been reports of surgery needed in the postpartum period for heavy vaginal bleeding or significant hemorrhage after attempted manual removal of the placenta following methotrexate treatment (Jaffe et al., 1994).

This apparent contradiction in the success and failure of treatment with methotrexate can be explained by the different types of abnormal placental adhesion. Cases of mild adhesion may respond to methotrexate in the same way as untreated cases, whereas those with a high content of NFV do not benefit from treatment with a folate antagonist. These contradictory outcomes and the inherent possibility of clinical complications argue for the discussion of treatment with methotrexate with the patient in each particular case.

6.3.2 One-step conservative surgery

Technical alternatives for the treatment of abnormal placental adherence have remained the same for more than 60 years, with the two options of performing a hysterectomy or leaving the placenta in situ. However, an intensive analysis of the major surgical problems in treating abnormal placentation, of hemostasis and dissection, gave rise to a new technical alternative. This alternative technique was originally designed to solve all of the problems of placenta accreta in a single surgical procedure with the purpose of restoring the original uterine and vesical anatomy. For adherent placentas located in the uterine segment, this implies the: (1) vascular disconnection of NFV and the separation of invaded uterine from

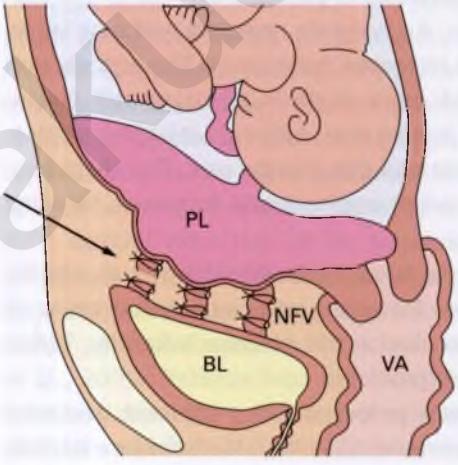

Fig. 6.26: Disconnection of the vesical–uterine (VUS) and colpo-uterine (CUS) anastomotic systems from an area of placental invasion. Black arrows show an extrauterine anastomotic component of the invading placenta.

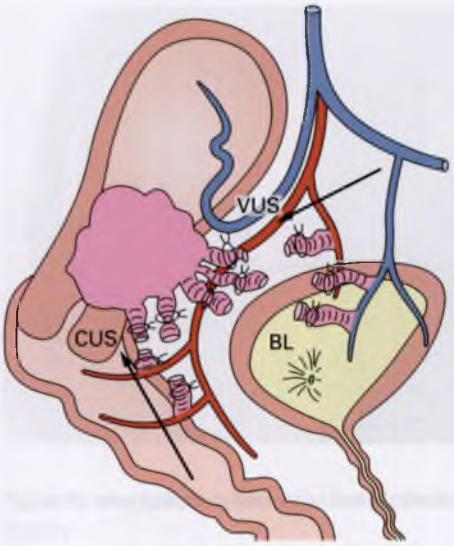

Fig. 6.27: Ligature of vesico-uterine system (VUS) and ligature of colpo-uterine system (CUS) for final hemostasis after complete resection of invaded tissues.

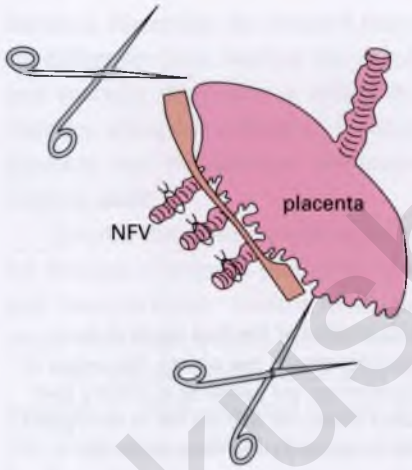

Fig. 6.28: Resection of invaded tissues and placenta.

invaded vesical tissues (Fig. 6.26); (2) performance of an upper-segmental hysterotomy; (3) resection of all invaded tissue and the entire placenta in one piece with previous local vascular control (Figs. 6.27–6.29); (4) use of surgical procedures for hemostasis; (5) myometrial reconstruction in two planes (Figs. 6.30 and 6.31); and (6) bladder repair if necessary (Palacios-Jaraquemada, 2004).

Pfannenstiel incision based on the findings in a placental MRI study is habitually used in the one-step alternative procedure described above, although on some occasions a midline incision is preferred. The ligation of newly formed vessels between the bladder and the invaded tissues reduces the vesicouterine blood flow and consequently the possibiloity of hemorrhage during hysterotomy. This disconnection of NFV between the bladder and invaded tissues is essential at the time

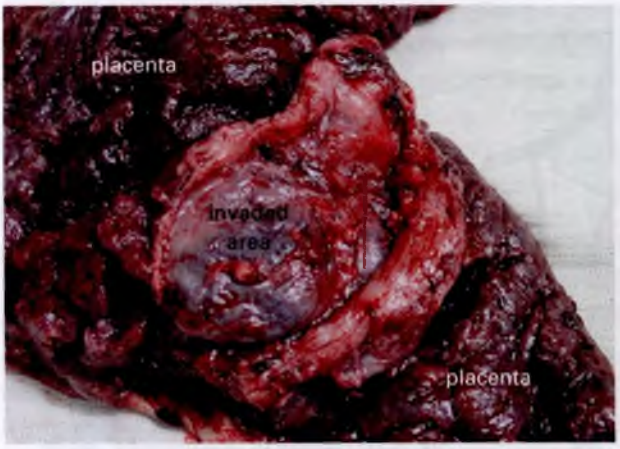

Fig. 6.29: Surgical specimen containing the entire placenta and placentally invaded area of myometrium.

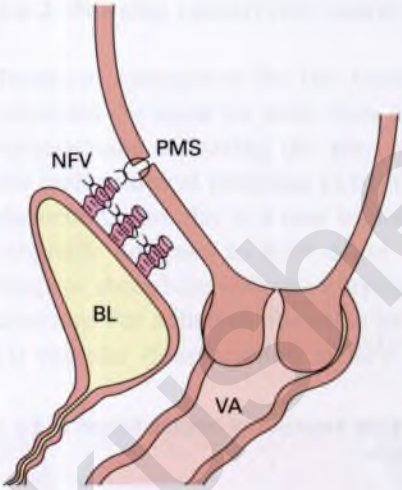

Fig. 6.30: Illustration of the final result of myometrial reconstruction in two planes. Two edges of healthy myometrium are joined by a primary myometrial suture (PMS). All NFV are left in the ligated state in the posterior part of the bladder (B).
VA: Vagina.

of hemostasis of the colpouterine system (CUS). The newly formed vessels contained in the lower anastomotic component are generally responsible for the pericervical bleeding following placental detachment in one-step conservative surgery. Although these vessels can be controlled by selective vascular ligation; however, it is much simpler, more efficient, and practical to accomplish achieve hemostasis with a Cho compression suture. Anterior placenta accreta is usually located in the S2 region, and it is therefore convenient to have some method of achieving efficient uterine vascular control.

An upper segmental incision is made over the cephalic sector of the placenta, but since this interrupts the VUS anastomotic system, the bleeding it produces is

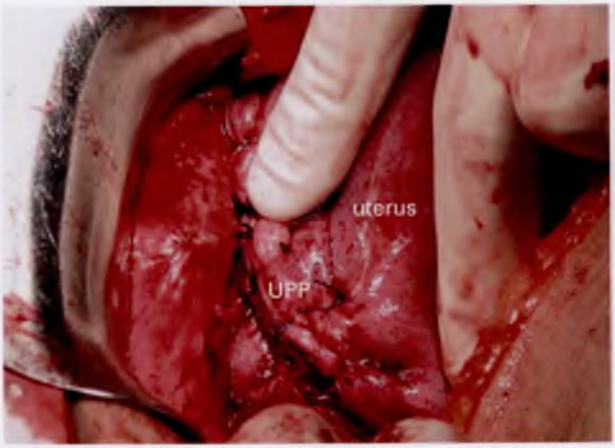

Fig. 6.31: Intraoperative view of a uterine plastic procedure (UPP) after one-step conservative surgery.

minimal. Resecting the invaded tissue together with the placenta represents a radical difference from leaving the placenta in situ. Even though the process of tissue and vascular dissection is longer in the one-step than in the conventional procedure for abnormal adhesion, it rules out the possibility of infection from retained placenta and reconstructs the anterior uterine wall and bladder to almost their original state.

Despite the need for advanced training for performing the one-step procedure for treating abnormal placental adhesion, and the need for thorough hemostatic and hemodynamic control in its performance, it provides a wholly satisfactory result with high-quality uterine and vesical anatomic restitution.

The process of dissection and vesico-placental-uterine separation has made it possible to avoid any kind of vesical resection in more than 450 cases treated with the alternative method. Vesical thinning or vesical muscular hernias consecutive to dissection or vascular ligature are repaired by simple suturing with synthetic resorbable #000 suture (Figs. 6.32–6.34). In order to avoid placental adherence in a subsequent pregnancy, a double layer of regenerated cellulose mesh is applied over the suture line and behind the bladder (Fig. 6.35).

Personal and collegial experience has shown that in pregnancies following use of the one-step procedure for treating abnormal placental adhesion, the state of the repaired area of uterus was exceptional (Palacios-Jaraquemada, unpublished data) (Figs. 6.36–6.38). It is recommended that an MRI scan be done as a control procedure to verify the state of the repaired tissue before any subsequent pregnancy (Fig. 6.39).

A further advantage of the alternative surgical procedure for abnormal placental adhesion is that only two partial recurrences of such adhesion have so far been

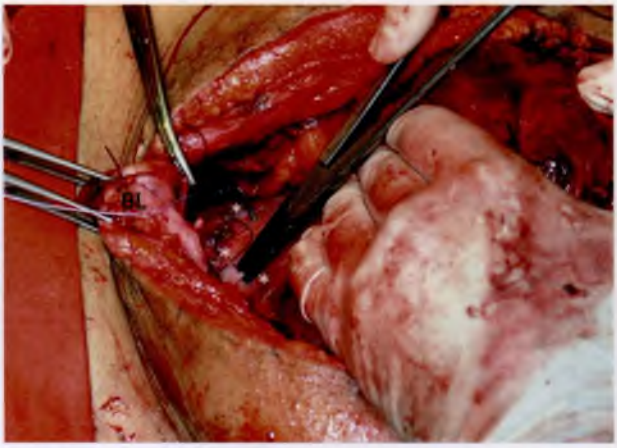

Fig. 6.32: Intraoperative view: Access to the upper part of the vagina through the vesical–uterine space allows hemostatic procedures in the colpo–uterine anastomotic component before plastic uterine reconstruction. Newly formed vessels are ligated to permit access to this area.

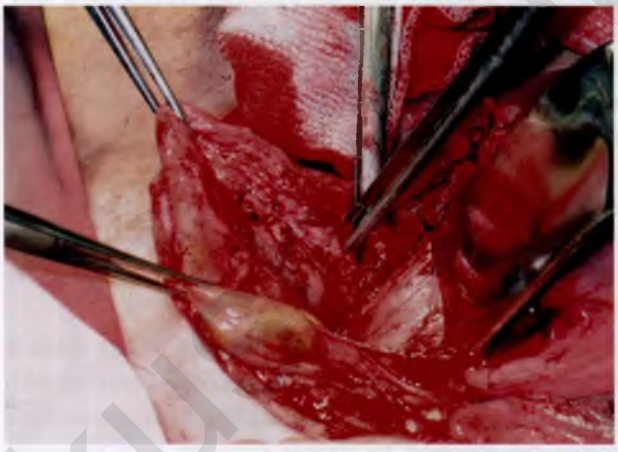

Fig. 6.33: Intraoperative view: In extensive bladder repair it is recommended that proof of the hydrostatic condition of the bladder be demonstrated by the instillation of methylene blue through a urinary catheter.

found in 116 pregnancies following use of the one-step conservative procedure (Palacios-Jaraquemada, personal unpublished data).

In the one-step procedure, the entire area of placental invasion and the entire placenta are removed. This releases a nonmeasurable amount of thromboplastin into the vascular system, which is activates the process of fibrinolysis. To date, no correlation has been established between the surface or type of invaded area and decline in the plasma fibrinogen (Factor 1) concentration after surgery. For this

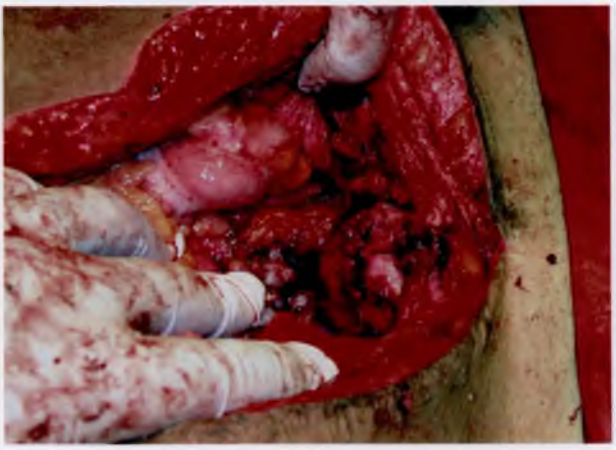

Fig. 6.34: Intraoperative view: Suturing of holes in muscular layer of vesical wall with absorbable #000 suture. The holes are a consequence of muscular tears caused by fibrosis traction or the passage of NFV.

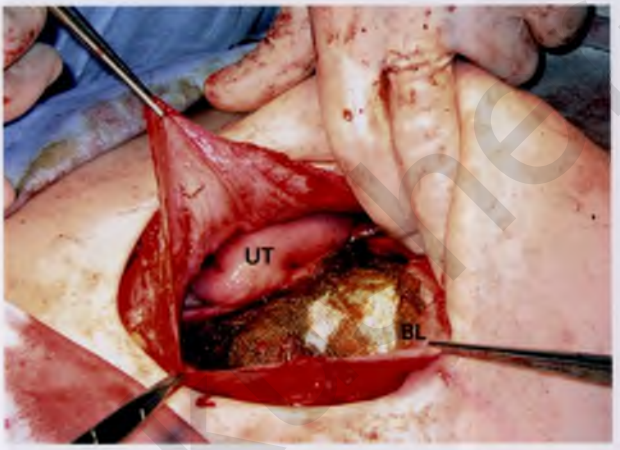

Fig. 6.35: Placement of cellulose regenerated mesh between the bladder and area of uterine repair, done to avoid placental uterine–bladder adherence in future pregnancies.

reason, one of the most important controls in the one-step procedure is the measurement of fibrinogenemia at 15 minutes and again at 1 hour after resection of the placentally invaded area of tissue. Levels of Factor 1 (fibrinogen) approximating 200 mg% must be ensured because the minimum level needed to produce a stable clot in the placental bed is 150 mg%. If postoperative testing shows a level of Factor 1 below 150 mg%, a dose of cryoprecipitate of 1 U/10 kg body weight must be given even in the absence of any evidence of bleeding. A plasma fibrinogen

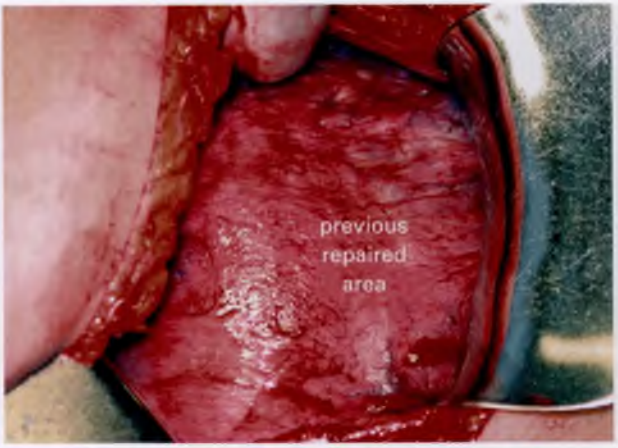

Fig. 6.36: Next subsequent cesarean section in patient at 18 months after repair of anterior S2 placenta percreta.

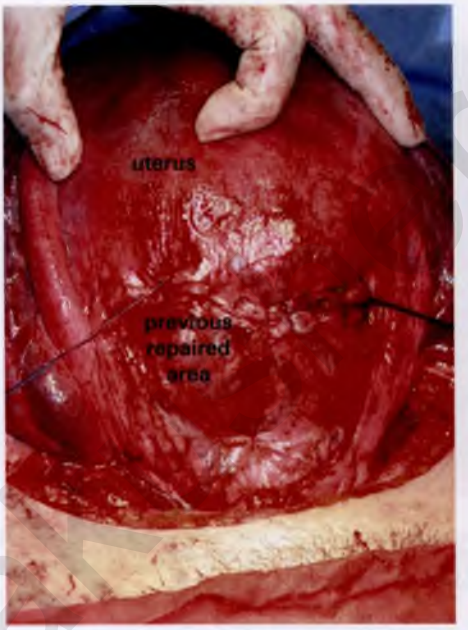

Fig. 6.37: Next subsequent cesarean section in patient with S1 invasion at 13 months after repair of placenta percreta.

below this level causes an unstable clot, and with a high possibility of blood loss after the one-step proicedure (Palacios-Jaraquemada et al., 2004).

Additional controls, such as assessment of the patient's acid–base status, standard hematologic parameters, and clinical variables, are recommended. These

Fig. 6.38: Next subsequent cesarean section in patient with S1 invasion at 22 months after repair of anterior S2 placenta percreta

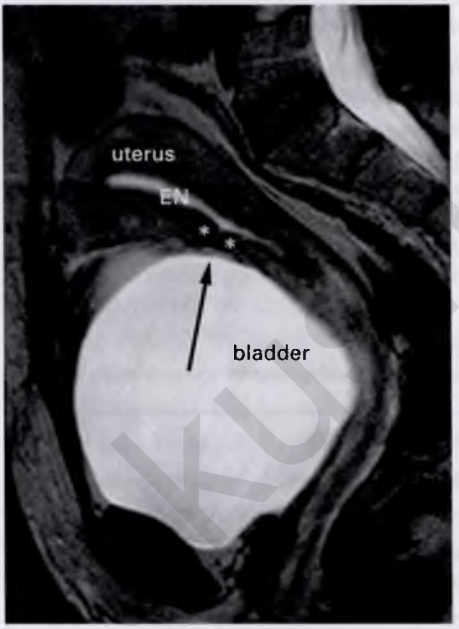

Fig. 6.39: Uterine MRI made as a control after myometrial reconstruction through one-step conservative surgery. A healthy outcome of primary uterine reconstruction is seen between the asterisks. EN: Endometrium.

depend on the facilities available in the hospital in which the one-step procedure is done, and can be performed in the intensive care unit or by a specialized clinical team. Close control of the patient's arterial pressure, urinary output, and peripheral perfusion is necessary, especially during the first 24 hours after surgery.

One-step surgery is usually a complex procedure involving cesarean section, pelvic dissection, vascular ligation, tissue resection, and repair. For this reason is

is recommended that the patient's oral intake after the restoration digestive transit begin with the intake of fluid so as to avoid bowel dilatation. On average, this process takes one or one-and-a-half days.

The one-step procedure for treating abnormal placentation represents a significant difference from conservative alternative treatments for this disorder, and another application of complementary imaging technology, such as placental MRI, for identifying the topography of placental invasion in a given case of such placentation and planning specific hemostasis for it (Mazouni et al., 2009).

Video 6.1: Bladder dissection (one-step conservative surgery).

Dissection of the bladder is the first stage in one-step conservative surgery for abnormal placentation and also a necessary step during hysterectomy for such placentation. Bladder traction with Allis clamps allows finding a correct plane for the dissection between the uterus and the bladder. Ligation of NFV between ligatures provides secure management. Electrocautery is not recommended because of the fragility of these vessels.

http://dx.doi.org/10.1515/9783110282382_v6.1

Video 6.2: Ward technique-modified hysterotomy-(One-step conservative surgery).

Although the Ward technique (Ward, 2002) was designed to reduce bleeding in cases of placenta previa, it is an invaluable tool during one-step conservative surgery. After vesical–placental disconnection, a hysterotomy is performed in an upper part of the bulging area of the uterus. Even though placental tissue is located in this area, it does not produce additional bleeding because the collateral blood supplies provided by vesical arteries were interrupted with the prior ligation and cutting of these arteries. A hand is placed between the myometrium and placenta until the infant is found. The uterus is then moved outside the pelvis with the placenta attached to a lower uterine segment. This maneuver provides control until uterine hemostatic techniques are applied and the area of invasion can be resected.

http://dx.doi.org/10.1515/9783110282382_v6.2

Video 6.3: Invaded area trimming (one-step conservative surgery).

After the infant is delivered, the whole placenta and the area of invasion are completely removed. The video shows trimming of the area of invasion during one-step conservative surgery.

http://dx.doi.org/10.1515/9783110282382_v6.3

Video 6.4: Plastic procedure (one-step conservative surgery).

The video shows myometrial repair after resection of the invaded area of uterus and the whole placenta. This aspect of the repair is similar to that in a cesarean section; the uterus is moved out of the pelvic space to permit suturing in an easy way. During this stage, hemostasis and the patient's fibrinogen level are checked closely. If additional techniques for uterine hemostas\is techniques are necessary, they are performed before the uterus is closed.

http://dx.doi.org/10.1515/9783110282382_v6.4

6.3.3 Two-step conservative surgery

When surgical conditions are not ideal for hysterectomy because of the lack of an experienced team or limited blood or resources, leaving the placenta in situ can be the best option for avoiding immediate and serious complications in the treatment of abnormal placentation. Although this is commonly done in some countries, it is not an option in other areas in which obstetrics is practiced (e.g., Argentina). When initial treatment has been provided and the infant has been safely delivered, there is time in which to plan alternative treatments abnormal placental adherence. Although hysterectomy is the most commonly chosen treatment, the author was consulted about two cases of placenta percreta (involving patients 31 and 36 years old, respectively) for which the patients requested conservative treatment but without the risks and long follow-up that are frequent when the placenta is left in situ. In these two cases the author and his group performed a second surgery at 1–2 weeks after the previous one. The steps in this procedure were the same as in one-step conservative surgery, but the tissues felt completely different. The NFV had collapsed and there was some slight edema between the bladder and uterine surface that facilitated the maneuvers for dissection.

In the two-step procedure, accidental rupture of the anterior area of placental invasion does not have the same significance as during cesarean section, because there is no or only minimal bleeding as a consequence of the relief of intrauterine pressure with deliver of the infant. The posterior bladder wall is more rigid as the result of the edema that is present but is easily manageable by dissection. Moving the bladder downward is mandatory for visually ensuring a healthy myometrium and providing clear access to the upper part of the vagina in the same way as in one-step conservative surgery. Following this, fine hemostasis is undertaken in the posterior wall of the bladder with stitches of #000 synthetic reabsorbable suture. After vesico-uterine dissection is complete, the entire placenta, together with the myometrium that has been invaded, is removed in one piece. Curettage is then done of any residual fragment of placenta. Bleeding through the placental bed comes from one or two thick vessels and also from capillaries. The author and his group used stitches of polyglactin or synthetic absorbable #1 suture material for square Cho sutures as a first option in cases of capillary bleeding.

Resection of the invaded area is needed, and we used two planes to close the uterus. Two sheets of regenerated cellulose (Surgicel™, Ethicon, Inc., Cornelia, GA, USA) were placed between the posterior wall of the bladder and the uterine suture.

Both of the patients with placenta previa who were described above became pregnant again, at 14 and 18 months, repectively, after reconstructive surgery. The location og the placenta was posterior in both cases and cesarean sections were performed at weeks 39 and 39.5, respectively, according to the obstetrician's indication. In both patients, the repaired area was observed to behealthy and the perinatal outcome was normal.

Although experience with the two-step procedure is very limited, its use for the conservative management in abnormal placentation can eliminate the risk of hemorrhage in the first procedure and allows eliminating the risk of sepsis, DIC, secondary haemorrhage, and recurrence in the second procedure. Dissection in two-step surgery is easy and carries less of a risk of bleeding than does one-step dissection. The disadvantage of the two-step procedure is obvious in that it entails two surgeries instead of one, but this alternative procedure may be quite useful in terms of follow up and secondary morbidity with respect to the classical treatment of placenta in situ.

6.3.4 Cesarean scar pregnancy

Disorders of placental adhesion can be localized primarily in the cesarean scar at the site of their occurrence. Pregnancy in a patient with a uterine cesarean scar is an infrequent form of ectopic pregnancy, but in the past decade has been frequently reported, probably because of the increasing rate of cesarean section (Seow et al., 204). Pregnancy in a cesarean scar implies a risk of heavy or life-threatening hemorrhage, uterine rupture, and the need for hysterectomy (Marcus et al., 1999). The most common symptom is vaginal bleeding, but the diagnosis is typically made by US or Doppler US, with MRI usually necessary to provide more details about the tissue characteristics of the affected area of the uterus. Because of the high risk of sudden hemorrhage or rupture (Rotas et al., 2006), anticipatory treatment is not recommended. The aarly diagnosis of cesarean scar pregnancy permits the planning of a conservative approach to its management and helps to preserve fertility through the use different techniques or combination of techniques.

The special presentation of abnormal placentation represented by cesarean scar pregnancy is anatomically and histologically similar to that of placenta percreta, because placental invasion reaches the uterine serosa or beyond it. But in contrast to cases treated at term, the development of NFV is less severe than in placenta percreta. Because the placental invasion is focused on the scar, there is a high possibility of rupture with hemorrhage. In some cases the vascular growth of NFV accords with the placental blood-supply requirement. If the fetal blood supply is guaranteed and sustainable myometrial support is provided, a uterine-scar pregnancy may be able to continue to near-term.

The technical resolution of cesarean scar pregnancy implies solving three main problems: the hemorrhage it entails, anatomic access, and repair of the invaded area. Although optimal treatment has not been established, removal of the fetus and the placental tissue with less severe hemorrhage is usually performed.

The combination of systemic feticide chemotherapy and hysteroscopically guided uterine evacuation has proved to be a successful treatment for cesarean scar pregnancy (Chiang et al., 2011).

Laparoscopic treatment is a good means of removing the embryo and gestational tissues, but definite the ectopic mass was reportedly not easy to dissect

owing to the overlying bladder, a fact that is a disadvantage of laparoscopic surgery. In the reported case in which this was done, surgery was postponed for 1–2 weeks to allow the ectopic mass to grow to more than 3 cm so as to permit its identification during surgery (Lee et al., 2008).

In complicated cases with uterine rupture, treatment with hysteroscopy combined with laparoscopy may be successful. It has been shown in some cases that operative hysteroscopy alone cannot be used in patients with cesarean scar rupture, and it has been suggested that direct laparoscopy or hysteroscopy combined with laparoscopy might be a better choice for treating this condition (Li et al., 2011).

A systemic or local, single-dose, multidose, or combined methotrexate (MTX) regimen can interrupt cesarean scar pregnancy, but while the remaining degenerative gestational tissue is being reabsorbed or discharged spontaneously from the uterine scar, the resulting intermittent vaginal bleeding can continue and is sometimes heavy (Wang et al., 2009)

The locally guided injection of MTX and hysteroscopic removal of cesarean scar pregnancy were reported to be successful in resolving it and preserving fertility, with a rapid return to normal morphology of the uterine cavity (Deans and Abbott, 2010). Combined therapy involving the injection of MTX and embolization was shown to be superior to orally administered MTX in cases of cesarean scar pregnancy with deep implantation of the amniotic sac (Lian et al., 2011).

The author has successfully used one-step conservative surgery in cases of cesarean scar pregnancy (Palacios-Jaraquemada, 2011). The surgical procedure is so similar to that for near-term abnormal placentation. Advantages of this surgical procedure include immediate resolution of the condition without the use of antimetabolic drugs or embolization, the complete removal of invasive and trophoblastic tissues, and accurate repair of the myometrial defect.

Video 6.5: Parametrial ectopic scar pregnancy (one-step conservative surgery).

Resolution of a parametrial ectopic scar pregnancy by open surgery. This type of ectopic pregnancy is also a placenta percreta, and in this case was treated by means of one-step conservative surgery. The key to accurate access in such pregnancy is to move the bladder down; the NFV are small with respect to near term placenta percreta, but delicate hemostasis is necessary to avoid additional bleeding. All invaded tissue and the entire placenta are removed, after which a plastic surgical procedure is performed to repair the uterus.

http://dx.doi.org/10.1515/9783110282382_v6.5

Procedure	Invaded area less than 50 % of axial uterine circumference	Invaded area more than 50 % of axial uterine circumference	Desire for future pregnancy	No desire for future pregnancy Age <40 years	No desire of future pregnancy Age >40 year
Resective hysterectomy	Not recommended	Recommended	Not recommended	Not recommended	Recommended
Conservative one-step surgery	Recommended	Not recommended	Recommended	Recommended	Not recommended
Conservative treatment of in situ placenta	Not recommended	Recommended	Not recommended (high recurrence rate)	Recommended	Recommended

Tab. 6.1: Abnormal placentation: Suggested tgreatment decisions.

Specific problems	In situ placenta	One-step conservative surgery	Two-step conservative surgery*
Hemorrhage reduction	+++++	++++	1ºS +++++ 2ºS ++
Pelvic dissection	No	+++++	+++++
Placental removal	None or partial	Complete	Complete
Invaded area removal	Never	Complete	Complete
Postoperative recovery	2 to 6 weeks	3 to 5 days	Stage 1: 1–2 weeks Stage 2: 3–5 days
Possibility of ureteral damage	No	+	+
Postoperative infection/ sepsis	Mid to high	No	At early stage
Postoperative dic/ hemorrhage	+++	+	At early stage
Recurrence in next pregnancy	High	Extremely low 1/82	Not described 0/2
Specific training	Low	High	High
Abdominal incision	Midline	Pfannenstiel	Depending on previous surgery

*Very limited experience.

Tab. 6.2: General comparison among current conservative possibilities.

6.4 Surgical hemostasis

Surgical hemostasis in the uterus can be achieved by vascular occlusion (external, such as through vessel ligation, or internal, such as through embolization), capillary compression (with uterine compression sutures), or organ resection (as with hysterectomy). Each technique has its indications, advantages, and disadvantages, which depend on the training of the treatment team, equipment, and topographic area of placental invasion.

6.4.1 Arterial ligatures and compression methods

The ligation of uterine vessels has long been used to achieve hemostasis. One of the first published procedures of this type was bilateral uterine ligation (Figs. 6.40 and 6.41), introduced by Waters in 1952 (Waters, 1952). After that, a combination of procedures was added to improve results in complex cases of postpartum bleeding. One of these procedures was called stepwise uterine devascularization (AbdRabbo, 1994), and was immediately accepted by a large group of obstetricians. However, it is not clear how this technique works, because all of the intravascular system of the uterus is interconnected. It was proved that pressure reduction after bilateral uterine ligation stops bleeding through uterine atony. But unfortunately, stepwise devascularization was marked by multiple cases of failure, mainly in abnormal placentation, probably because the technique does not consider the

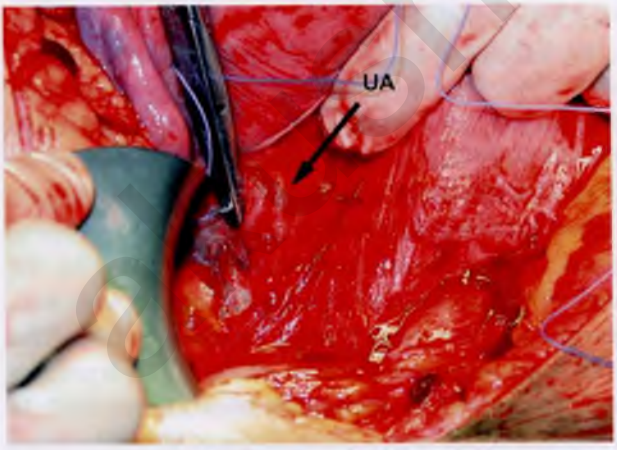

Fig. 6.40: Intraoperative view (ligation of uterine artery) 1: Uterine artery ligation is indicated in cases of bleeding in the S1 area because the branches of the artery are distributed in the uterine body. Ligation of the uterine artery in abnormal placentation has had high rate of failure because most of the area of placental invasion is located in the S2 region, which is irrigated by extrauterine pedicles.

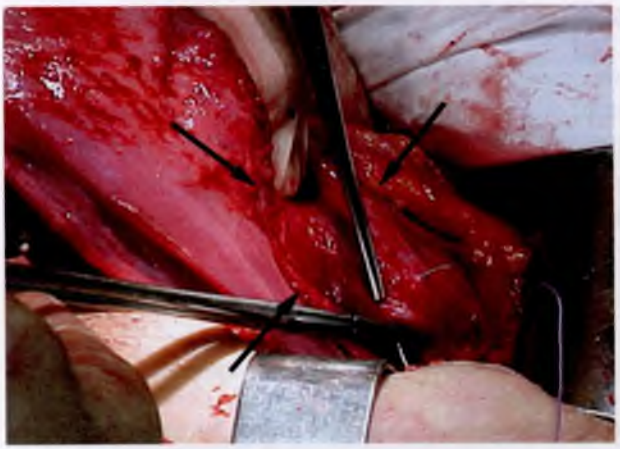

Fig. 6.41: Intraoperative view (ligation of uterine artery) 2: Note that the area within the black arrows corresponds to the posterior wall of the bladder (non-peritonized). The most common technical mistake in the use of this technique is a high ligation. In order to identify and accurately ligate the uterine artery, it is essential to move the bladder down so as to expose the arterial trunk.

lower uterine anastomotic connections or the clotting state at the moment at which the procedure is applied. In 1997, the first series of uterine hemostatic procedures based on myometrial compression was published (B-Lynch et al., 1997), and many variations of this were later introduced (Allam and B-Lynch, 2005). However, these techniques have different efficacies in regard to the area of bleeding and also to the patient's clotting status and topographic of the uterine area in which they are used. A large retrospective series, which includes all kinds of uterine hemorrhages, has demonstrated that there is a specific relationship of technique to the topographic area of the uterus being treated, but not to the primary condition (Palacios-Jaraquemada, 2011). A similar efficiency was seen for bleeding in the body of the uterus for uterine ligation, uterine embolization, and the main uterine compression sutures in cases of uterine atony. However, this can have diminished efficacy in cases of abnormal placentation. This apparent contradiction is caused by the abnormal blood supply through NFV, thatcan arise in ways that are not very well-known. In some cases of abnormal placentation in specific locations in the uterine body (S1), standard measures of hemostasis have not been efficient. This is particularly common in posterior accretas secondary to multiple abortions or D&C. In these cases a branch of the superior rectal artery is the origin of NFV (abnormal placentation area), which are interconnected with uterine vessels. This pedicle does not have primary connections with the uterine pedicles, and its branches are therefore not occluded after uterine ligation or embolization, making local hemostasis very difficult. But some type of compression sutures, such as Cho square sutures (Cho et al., 2000) (Fig. 6.42) or the Pereira procedure (Pereira et al., 2005),

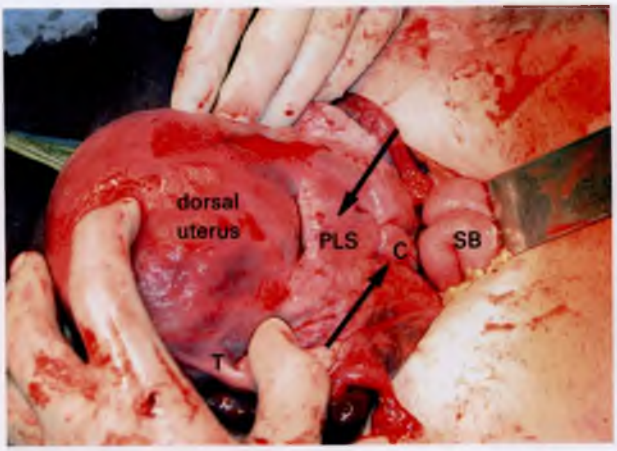

Fig. 6.42: Dorsal view of the uterus with one of two Cho square sutures near the cervix (C) and the second in the posterior lower segment (PLS). T: Tube, SB: Small bowel.

occlude all NFV through direct compression. In other words, it is not essential to specifically know the origins of anastomotic pedicles because the procedure occludes all of them in one step.

Something similar happens in the lower genital area (S2). This sector has multiple pedicles that arise in the pelvic–subperitoneal area. In cases of local resection of abnormal placentation, specific hemostasis may be difficult. Because the pedicles in this sector do not depend on the uterine artery, ligation or occlusion of this vessel is not efficient to achieve hemostasis. However, use of the Cho dquare suture provides accurate control of bleeding in a single procedure. As in the S1 region, the Cho square suture in region S2 occludes all anastomotic vessels in a specific area regardless of their origin. The Cho square technique is particularly useful during one-step conservative surgery or hysterectomy for abnormal placentation in the S2 region because the upper part of the vagina or cervix contains many NFV induced by growth factors (Palacios-Jaraquemada, 2011)

6.4.2 Embolization

Uterine arterial embolization plays an important role in the management of major obstetric hemorrhage; (Fig. 6.43); however, this is not a simple procedure because of the need for an accurate knowledge of the complex pelvic circulation as well as the need for dexterity of the operator in performing selective vascular occlusion, especially in abnormal placentation, and in the use of microcatheters, which requires a high level of skill (Spies, 2004). For all of these reasons, adequate training in female pelvic vascular anatomy is essential for the physician who performs

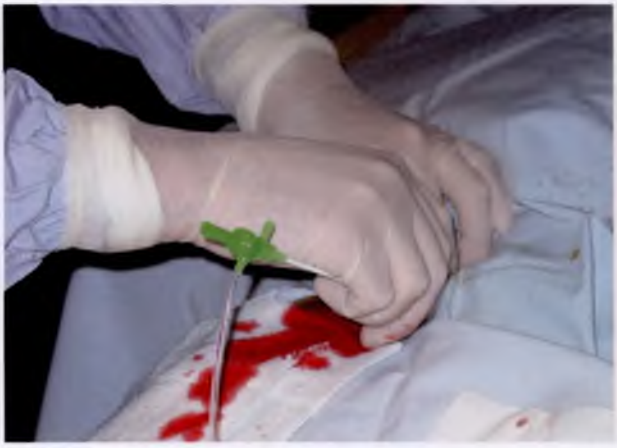

Fig. 6.43: Femoral access prior to the insertion of catheters for pelvic embolization.

uterine embolization, especially for success in the management of postpartum hemorrhage (Saraia et al., 2002; Wi et al., 2009; Wang et al., 2009; Bensalah et al., 2010).

Arterial embolization in disorders of placental adhesion can be done for different reasons, such as direct uteroplacental hemostasis or a reduction in blood flow. It is very important to understand that the procedure must be included as part of the treatment. For this reason, the embolization used in a particular treatment can be different if the planned treatment is a hysterectomy, a resection of the area of placental invasion, or treatment of the placenta in situ. There is extensive experience in the use of prophylactic embolization in cases of abnormal placentation. In such cases both uterine arteries are occluded prior to the hysterectomy. However, it is not clear whether the embolization in this setting reduces hemorrhage while hysterectomy is being performed, because all uterine arterial pedicles are ligated during dissection, and not cut.

Some authors describe failures of uterine arterial embolization, especially in abnormal placentation with deep invasion (Chou et al., 2004; Park et al., 2011; Soyer et al., 2010). These experiences are in accordance with the distribution of vessels in the lower genital system, because the vesical and vaginal arteries have no relation to the territory of the uterine arteries, and there is consequently a strong possibility of bleeding from these vessels, especially in the lower uterine area and in the posterior wall of the bladder. However, distal hemostasis through the uterine arteries can be possible in abnormal placentation. During the development of abnormal placentation, a number of growth and vascular factors are produced by the placenta in order to increase blood flow. As a consequence, a microscopic network of anastomoses among the bladder, uterus, and vagina becomes visible as NFV. Therefore, embolic material driven with pressure through the uter-

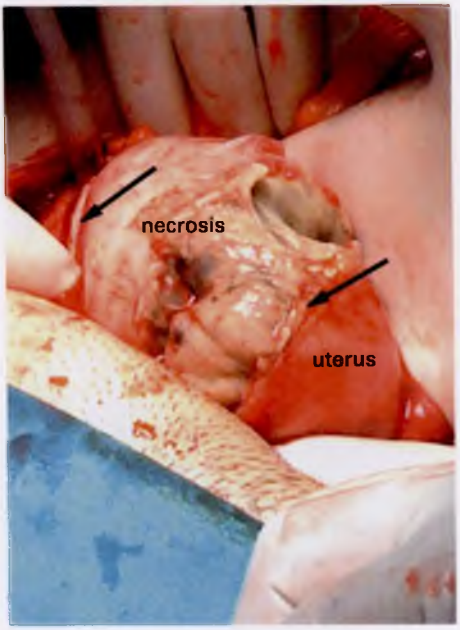

Fig. 6.44: Late uterine necrosis after insertion of compression sutures and embolization. Because the endovascular procedure was unable to arrest bleeding, a B-Lynch procedure was done. Twenty-two days later, the patient developed endometritis with leukocytosis. An MRI study demonstrated signs of uterine necrosis, which were evident during laparotomy. Black arrows show placement of B-Lynch sutures.

ine arteries can occlude these vessels by countercurrent action. However, it is essential that this procedure be performed with caution and expertise, because it carries the possibility of causing a nontargeted embolization and unwanted necrosis (Cottier et al., 202; Porcu et al., 2005; La Folie et al., 2007). To avoid this, the interventional radiologist should use large particles of gelfoam rather than small particles. It is essential to know that gelfoam is a reabsorbable material but capable of producing necrosis, because endovascular elimination of this material is slow, and it has been found inside vessels at 30 days after embolization (Cylwik et al., 1985). If small particles of resorbable or nonresorbable material block the distal anastomotic network, tissue necrosis can occur even with gelfoam, owing to the differing size distribution of gelatin sponge particles according to the method used to make them. More uniform particle sizes can be achieved by cutting than by pumping (Katsumori and Kasahara, 2006). A recent report described uterine necrosis occurring 10 days after uterine artery embolization even with gelfoam calibrated in 1 to 2 mm-particles, and similar cases are described in the literature (Tseng et al., 2011). Recently, a possible effect of deformation of 1 to 2 mm calibrated gelatin particles was associated with areas of uterine necrosis in animal studies (Sone et al., 2010). The deformation of plastic embolic particles may allow their entry into small vessels and blockade of the capillary collateral network to cause unwanted damage. However, this can also happen, in theory, from the high-pressure injection of moldable particles. Although gelfoam is widely known as a rapidly resorbable agent, particles remain inside vessels for weeks (Vlahos et al., 1980; Sniderman et al., 1981; Ohta et al., 2007), a fact that may explain ischemia or necrosis when the final organ collateral network is occluded. of unwanted uterine necrosis (Fig. 6.44).

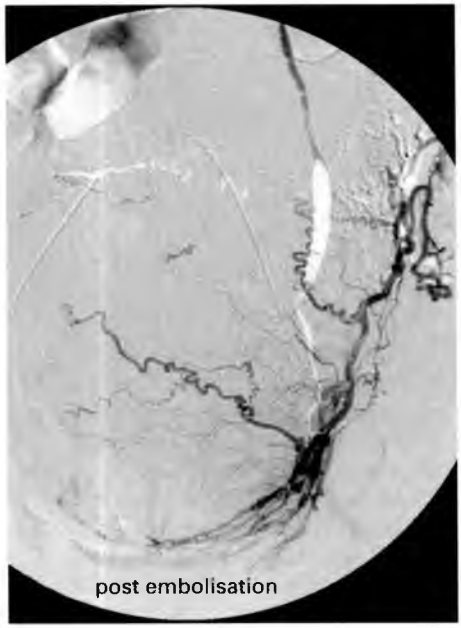

Fig. 6.45: Pelvic arteriography: Embolization view of the right uterine artery with placenta left in situ. The uterine artery was occluded with gelatin particles.

For this reason, the use of compression sutures is recommended before embolization because of the impossibility of knowing the degree of inadvertent anastomotic obliteration caused by embolic particles (Palacios-Jaraquemada, 2011).

In some countries, leaving the placenta in situ is a common treatment in cases of abnormal placentation. The main advantage of this approach is that it reduces the possibility of postoperatory bleeding. Postoperative follow-up can have complications such as infection and secondary bleeding; for this reason, some authors decide to use uterine artery embolization after closing the uterus with the placenta inside (Sentilhes et al., 2010). They believe that this procedure protects the patient from a secondary bleeding. But this opinion is not shared by other doctors who also leave the placenta in situ, who prefer to use embolization in a real case of secondary, postpartum hemorrhage. In theory, prophylactic bilateral uterine arterial embolization in cases of placenta in situ may have an additional risk. It is well known that leaving placental tissue inside the uterus increases the possibility of infection, and it is also known that devascularized tissue has a similar risk; thus, leaving a large portion of retained tissue with reduced blood supply may increase the possibility of infection or sepsis without evident advantages.

However, uterine artery embolization or endovascular treatment can be effective for making a preoperative map and for stopping bleeding in abnormal placentation (Figs. 6.45–6.48), or as a hemostatic method in cases of bleeding after hysterectomy. The accuracy of endovascular treatment in detecting and treating specific points of bleeding is established. However, extensive training and management of pelvic-subperitoneal pedicles (such as the pudendal artery and its branches) is

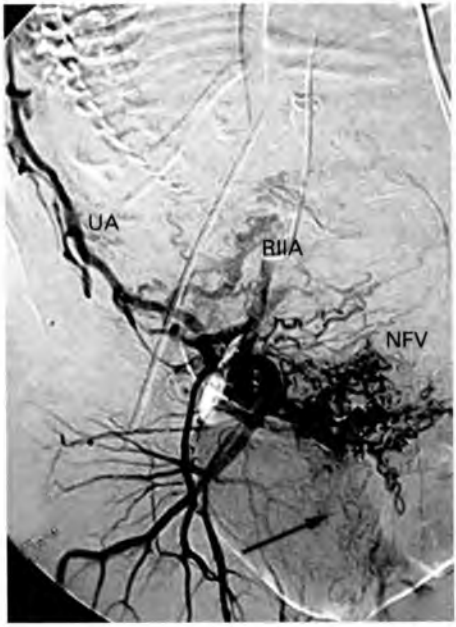

Fig. 6.46: Pelvic arteriography (anterior placenta percreta): An arterial catheter has been placed in the right internal iliac artery (RIIA). UT: Uterine artery, NFV: Newly-formed vessels. Black arrow shows collateral anastomosis with vaginal pedicle.

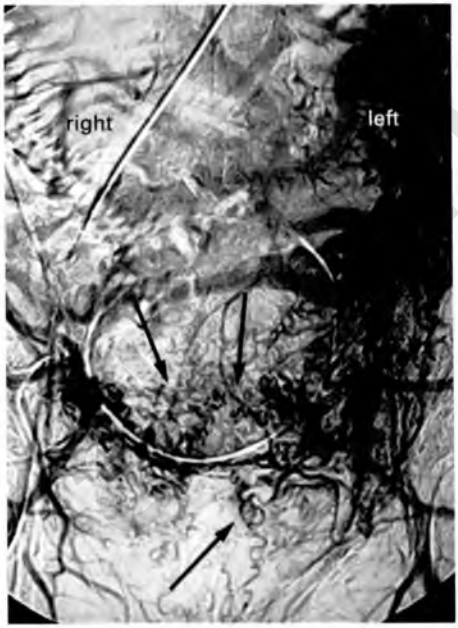

Fig. 6.47: Pelvic arteriography (anterior and lateral placenta percreta): An enlarged collateral blood supply is seen on the left. Upper arrows show an interuterine anastomosis behind the bladder. Low arrow shows a connection with components of the vaginal blood supply.

needed for the accurate accomplishment of this in the treatment of abnormal placentation. The S2 region in abnormal placentation is a network of both known and not very well known anastomotic vessels; to identify and treat them requires a

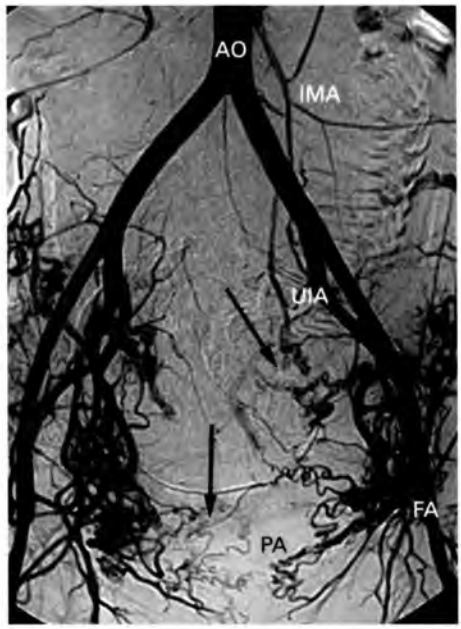

Fig. 6.48: Pelvic arteriography (anterior placenta accreta): Black arrows show anastomotic components among uterine arteries and internal iliac artery. IMA: Inferior mesenteric artery, AO: Aorta, PI: Pudendal internal artery.

high level of training and resources. One proof of this is that nearly 50 % of failures of embolization in abnormal placentation occur in the S2 region. In order to enhance uterine hemostasis in anterior abnormal placentation, many interventional radiologists use bilateral embolization of the uterine arteries and also of the anterior branch of the internal iliac artery. This procedure occludes the most important sector of arterial NFV, which includes the vesical artery and anastomotic branches of the obturator artery. However, the bilateral embolization procedure does not reach the vaginal anastomotic component of the arterial NFV because its main collateral vessels arise from the internal pudendal artery (posterior trunk of the internal iliac artery). For the same reason, embolization of the uterine and anterior branches of the internal iliac artery is usually followed by hysterectomy, because placental detachment can cause hemorrhage from the lower anastomotic component that is out of the reach of control by embolization.

Because the chief advantage of leaving the placenta in situ is that it avoids hemorrhage, and the second is fertility, any kind of endovascular devascularization must be considered for preserving reproductive function. The literature is very extensive and controversial on this point, because when fertility is analyzed after uterine artery embolization, some reports indicate damage and others do not. Some details are very important during uterine embolization, especially for avoiding unwanted secondary damage (Fig. 6.49). The size and type of the particles used for embolization, the pressure at which they are injected, and anastomotic pathways leading from the region of their injection are variables that can modify good hemostatic results, because the vesical blood supply is widely connected with the

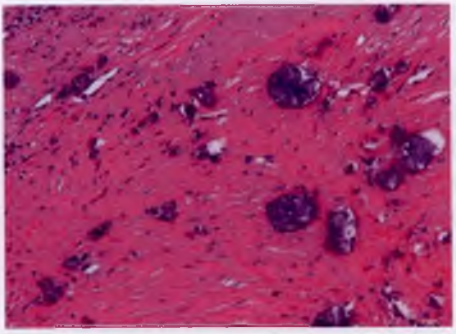

Fig. 6.49: Histologic view of embolized uterine tissue: Embolization was realized with Bead Block particles (500–900 µ) and completed with Gelfoam particles. Magnification is 200X, staining is with hematoxylin–eosine, and the zone of fibrosis shows multiple calcifications; in the middle and the upper edges are seen some pale oval areas, which are completely involuted zones of implantation.

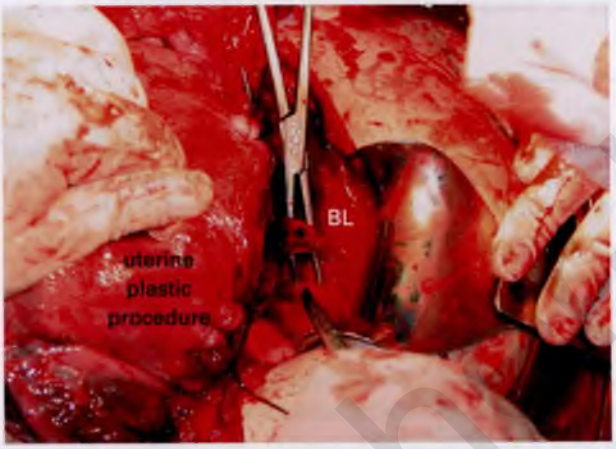

Fig. 6.50: Intrasurgical view: Asterisk shows a thick vessel that directly links the uterine artery with the bladder. Unwanted embolization of this vessel could result in non-targeted embolization of the bladder.

uterine arteries in abnormal placentation (Fig. 6.50). Blocking the distal anastomotic system can produce ovarian failure and also a late reduction of the endometrial blood supply (Amato and Roberts, 2001; Tropeano et al., 2003; Chitrit et al., 2006). However, a lack of ovarian damage and the safety of this procedure in the hands of skilled operators has been reported (Lei et al., 2007). A recent review (Berkane and Moutafoff-Borie, 2010) indicates that the use of small particles, especially during embolization of a myoma, can induce a real risk of occlusion of endometrial and ovarian vessels. For this reason, except in prospective trials with a fully informed patient, their use is inappropriate for women who wish to have a chance at childbirth. Because of the small size of published series, further studies on future fertility after uterine artery embolization, and also on fertility after surgery, are needed to refine these conclusions. For this reason, it is strongly recommended that uterine devascularization or embolization be performed by an

interventional radiologist especially trained in obstetrics, because not all techniques or embolic agents are equivalent.

In abnormal placentation, and especially in cases of placenta percreta, the rate of success of selective arterial embolization observed in a large population was lower than previously reported, and one of the reasons for this is revascularization by pedicles of the internal pudendal artery (Chou, 2004). It is most likely to succeed for uterine atony but is not recommended in cases of hemodynamic shock or after cesarean section. In cases of abnormal placentation, selective arterial embolization must be discussed with a multidisciplinary treatment team (Touboul et al., 2008). In some countries there is the idea that uterine hemostasis is equivalent to uterine artery embolization, and also as a simple procedure that can be done by operators without a high level of training, but perhaps this idea should be abandoned.

Video 6.6: Normal embolization.

Uterine arterial embolization occludes the blood flow of the uterine arteries and their first divisions, and leaves the distal anastomotic vascular network free, in order to avoid ischemia or necrosis. The procedure is performed as a controlled devascularization.

http://dx.doi.org/10.1515/9783110282382_v6.6

Video 6.7: Small particle embolization.

The use of small particles is not recommended during uterine arterial embolization. Although hemostasis can be used, blockade of the distal uterine capillary network may result in ischemia or necrosis. Successful control of bleeding is possible, but may result in late complications such as endometrial adhesions, restriction of intrauterine growth, or abortion. The use of small particles has also been associated with their migration to the ovarian circulation and the possibility of premature ovarian failure in young patients.

http://dx.doi.org/10.1515/9783110282382_v6.7

Video 6.8: High pressure embolization.

Technical details are highly important during uterine arterial embolization. It was thought that the use of particles of 1000 μ or larger could not cause uterine necrosis. However, recent publications have described uterine necrosis with calibrated particles of 1000 μ–2000 μ in size. One possible explanation folr this is the deformation of large reabsorbable particles into small ones as a result of hyperpressure. In this case, the deformation of particles can lead to the blockade of a distal capillary network and stop circulation.

http://dx.doi.org/10.1515/9783110282382_v6.8

6.5 Postoperative care

Abnormal placentation usually requires extensive care in all of its stages, which include presurgical diagnosis, clinical–surgical decisions, and intense postoperative care. In the last stage, payoff postolperative care, attention must first be given controlling any possibility of hemorrhage, hypotension, shock, or coagulopathy. Regardless of the specific procedure (conservative or extirpative) used for treating abnormal placentation and its surgical complexity, intensive control is recommended as a component of initial care. It is obvious that the structure of different hospitals varies, and some of them probably have a special facilities for complicated surgeries, but almost all have an intensive care unit.

6.5.1 Thromboprophylaxis

The complications and recovery from cesarean section are dominated by the patient's medical condition preoperatively. With emergency cesarean section, mortality associated with the procedure can range up to five times that for vaginal delivery. Thromboprophylaxis at the time of cesarean section decreases morbidity in the index pregnancy, but can also be reasonably expected to reduce complications of future pregnancy (Jackson and Paterson-Brown, 2001). Women in the pregnant and postpartum state are approximately four to five times more likely than nonpregnant women to develop venous thrombosis and embolism (Heit et al., 2005). Three elements that contribute to the development of thrombosis are stasis, vascular trauma, and hypercoagulability, all of which are present during pregnancy and the postpartum period (Davis and Branch, 2010).

Use of an intermittent leg compressor is advisable during surgery in patients with a known risk of thromnbosis or in the postoperative stage, until they begin to walk again. Mechanical thromboprophylaxis is also considered to be a cost-effective strategy under a wide range of circumstances (Casele and Grobman, 2006).

Pregnancy increases the concentrations of prothrombotic factors in the body and extensive pelvic surgery adds nonmeasurable risks for postoperative thrombosis. In patients with thrombophilia, the use of an intermittent leg compressor is preferred before starting surgery because of the risk of formation of blood clots in the leg when the patient is in the decubitus position in the operating room. If the decision to use an intermittent leg compressor is made after surgery, a previous Doppler US examination of the legs is recommended to detect possible instances of undetected deep-vein thrombosis (DVT). If the examination is negative, a leg-compression device can be connected without risk. Low-molecular-weight heparin is usually prescribed for 8–12 hours after surgery if the platelet count is above 100,000/mm^3 and if there is no evidence of bleeding. The usual dose is 40 U of enoxaparin every 24 hours until the patient is able to walk without restrictions.

Decision analysis suggests that the benefits of low-molecular-weight heparin after cesarean delivery exceed the risks of its use (Blondon et al., 2010).

6.5.2 Analgesia

Standard analgesia is usually sufficient for peripartum hysterectomy or in cases of placenta in situ, but needs to be modified for one-step conservative surgery. Tissue dissection, bladder mobilization, removal of the placentally invaded area, and uterine tissue repair are causes of additional pain. Two schemes of analgesia have been used, one that includes intrarachideal opioids such as morphine or fentanyl, and the other that uses a morphine infusion by pump plus a nonsteroidal analgesic agent such as ketorolac. Analgesia provided by intrarachideal morphine is excellent, but when adverse symptoms appear, such as nausea or vomiting, it is more problematic to manage. The scheme for infusion of morphine is more flexible and manageable, especially if the patient needs additional doses. Parenteral morphine is used for 1 or 2 days, after which a combination of a synthetic morphine analog such as dextropropoxiphen together with a nonsteroidal analgesic is recommended for the next 4 days. In a recent study, midazolam added to hyperbaric low-dose bupivacaine administered in the subdural space was reported to significantly improve the quality of surgical anaesthesia and prolong the duration of analgesia without any adverse effects (Akhtaruzzaman et al., 2010).

7 Results

Results of the treatment of abnormal placental adhesion are considered in terms of mortality, morbidity, and reproductive outcomes. Early maternal mortality is closely related to the degree of hemorrhage in a given case, while late complications are associated with organ damage after sustained shock. Morbidity is also high in peripartum hysterectomy, with prevalent complications including cardiac arrest, DIC, pulmonary edema, septicemia, bladder injury (Smith and Mousa, 2007), and Sheehaṅs syndrome (Tessnow and Wilson, 2010). Placenta accreta is becoming a leading cause of emergency postpartum hysterectomy. Although hysterectomy is a lifesaving operation, it is associated with a high maternal mortality (Varras et al., 2010).

7.1 General overview

Abnormal placentation increases with an increasing rate of of cesarean section, and only a few countries have been able to maintain low rates of such section. However, in countries with high parity and high rates of cesarean section, the proliferation of cases of abnormal placentation has been almost epidemic. Multiple and variable factors have been presented to explain this, such as an increase in the number of cases of previous cesarean sections, less use of forceps delivery, altered fetal monitoring, breech presentation, the fear of malpractice claims, and shorter waiting times for vaginal than for cesarean delivery. In other words, it seems impossible to break this close association simply by implementing policies for stimulating vaginal birth. However, the solution may be hidden in the uterine architecture. The uterus is formed mainly of muscle, the myometrium, and collagen tissue for support. In the uterine body, the percentage of muscle is high because of its role in the contraction. In the lower part of the uterus, the situation is completely the opposite, with a high percentage of collagen because of its function of keeping the fetus in the uterus. The uterine segment has a high percentage of collagen, which is increased after every cesarean section through scar formation. This renders a previous cesarean scar inextensible, and it breaks in variable ways in subsequent pregnancies. More damage is related to a higher risk of abnormal placentation, with a correspondence between the number of cesarean sections and possibility of abnormal placental adherence. Uterine collagen exposure has been linked to problems of decidualization in mammals (White et al., 2004), and the same siktuation can pertain to humans. The lack of normal endometrial and myometrial structure is related to abnormal placentation. When myometrial tissue cannot provide an adequate placental blood supply, placental tissue produces growth and vascular factors that enlarge microanastomoses (Tantbirojn et al., 2008) to ensure an adequate placental blood supply.

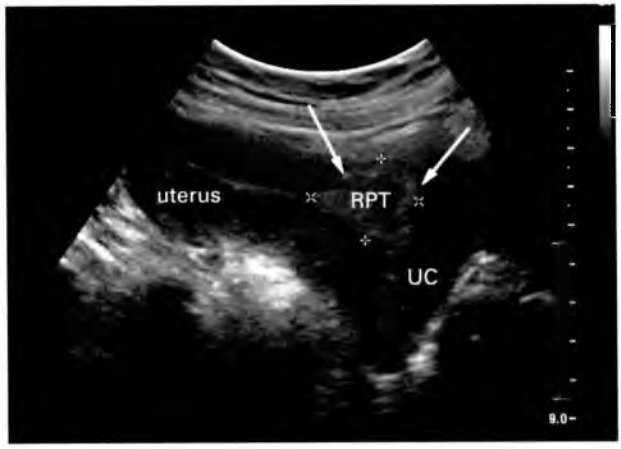

Fig. 7.1: Abdominal US image (placenta in situ) showing retained placental tissue (RPT) at 4 months after delivery. White arrows mark limits of destroyed anterior myometrium. UC: Uterine cervix.

Experience with one-step conservative surgery, in which invaded myometrial tissue and previous cesarean scar are resected, showed almost no cases of recurrence of abnormal placentation in a subsequent pregnancy (1.2%). This rate is extremely low with respect to that with any other conservative treatment (35%–70%) Provensal et al., 2010; Kayem et al. 2007). Except for removal of the placenta, the main difference between the two conservative procedures of one-step conservative surgery and in situ treatment for abnormal placentation is resection of the area of invasion (previous cesarean scar) and repair of the anterior uterine wall. Conservative treatment with the placenta left in situ is an excellent means of avoiding an initial hemorrhage, but requires an intensive clinical follow-up until the placenta is reabsorbed or expelled, in a period of weeks or months. Although its initially showed advantages, this procedure has never resolved the myometrial damage done by placental invasion (Figs. 7.1 and 7.2).

A current binational study (in Argentina and Belgium) is comparing the similarities and differences in uterine scar and placentally invaded tissue. If it is proved that collagen exposure is the initial point of abnormal placentation, the solution can be very simple, accessible, and independent of the number of cesarean sections a patient has had. A prophylactic resection of the entire cesarean scar (uterine segment) may prevent abnormal implantation in a subsequent pregnancy. The new scar created by the resection of invaded tissue and by one-step conservative surgery for uterine reconstruction would heal differently from the previous uterine scar because of the thickness of the myometrium and types of uterine collagen involved in healing.

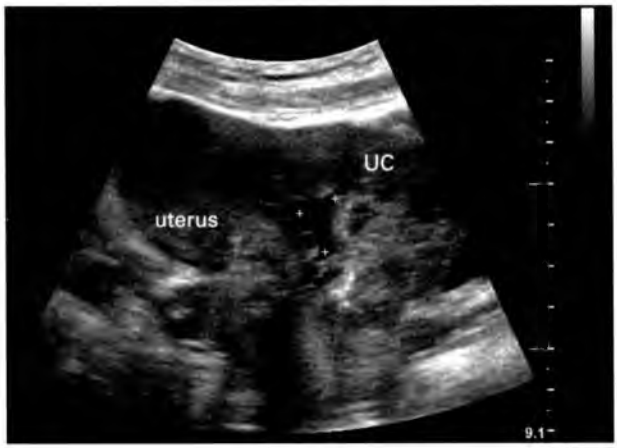

Fig. 7.2: Abdominal US image (placenta in situ): A defect is seen in the anterior uterine wall after conservative treatment. The residual damaged tissue could present a high rate for recurrence in a following pregnancy.

7.2 Maternal outcomes

When the diagnosis of placenta accreta is made substantially earlier than delivery, the need for hysterectomy should be anticipated and preparations made for delivery in a center with adequate resources, including those for massive transfusion. Intraoperative attention should be paid to blood loss. Early use of blood-products for replacement, with consideration of volume, oxygen-carrying capacity, and coagulation factors, can reduce perioperative complications (Belfort, 2010; Karayalçın et al., 2011; Warschak et al., 2011). Maternal mortality is mainly associated with hemorrhage, coagulopathy, and the consequences of sustained shock. There is a closer relation between maternal mortality and the duration of shock than with the amount of volume loss. This is clinically very important in a patient with heavy bleeding; in all cases the decision should be to stop the bleeding through a method for vascular control, restore volume, and then stop the uterine bleeding by means of hysterectomy or any other method. Unfortunately, proximal vascular control and hemodynamic parameters are often not considered at first, leading to later morbidity. Teamwork is essential if the problem is a patient with massive bleeding and not a uterus with intractable bleeding. Proximal vascular control is not a complex issue, but it is necessary to obtain basic knowledge and use time and practice to acquire the skills for achieving it, because less bleeding is equal to fewer complications.

Conservative treatment, with the placenta left in situ, produces fewer hemorrhagic but more infectious complications, which include infection, septic shock, sepsis,endometritis, wound infection, peritonitis, pyelonephritis, vesicouterine fistula, and uterine necrosis (Sentilhes, 2010), among other medical complications.

Although maternal and reproductive outcomes of one-step conservative surgery have not yet been published, they are encouraging. Resolution of the main problems of abnormal placentation and resection of the entire area of invasion and entire placenta allows avoiding late problems in terms of infection and recurrence. However, intensive training is needed to manage complex dissection and methods of applying proximal vascular control and uterine-specific surgical hemostasis.

7.3 Reproductive outcome

Abnormal placentation increases with the increase in rates of cesarean section in a particular geographic location, and only a few countries have been able to maintain low rates of cesarean section. In countries with high rates of parity and cesarean section, the proliferation of cases of abnormal placentation has been almost epidemic. Multiple and variable factors have been adduced for this, including an increase in the number of previous cesarean sections, a decreased use of forceps-assisted delivery, deficiencies in fetal monitoring, an increased frequency of breech presentations, fear of lawsuits for malpractice, and shorter waiting times for vaginal delivery. In other words, it seems impossible to break the close association of abnormal placentation with the factors mentioned above only by implementing policies that promote vaginal birth. However, the solution to this problem may be hidden in the intrinsic architecture of the uterus. The uterus is formed chiefly of muscle, the myometrium, and collagen tissue for support. The percentage of muscle in the body of the uterus is high because of its specific role in uterine contraction in childbirth. By constrast, the structure of the lower part of the uterus has a high percentage of collagen relative to the myometrium, and this supports the fetus in utero during gestation. The high percentage of collagen in the uterus increases after each cesarean section, and this progressive collagenization renders pre-existing cesarean scars inextensible, leading to their rupture in various ways in subsequent pregnancies. Greater damage in this regard is related to a greater possibility of abnormal placentation, with the number of cesarean sections corresponding to the possibility of abnormal placental adherence. Exposure to uterine collagen has been linked to problems in decidualization (Spiess et al., 2007), and there is preliminary information that the same situation can exist in humans (Pollio et al., 2006).

A study of patients undergoing one-step conservative surgery in which myometrial tissue affected by placental invasion (distorted uterine cesarean scar) were completely resected found almost no cases (1.2%) of recurrent placental adhesion in a subsequent pregnancy. This rate is extremely low with respect to that found with any other conservative treatment for abnormal placentation (35% to 70%). Apart from the complete removal of a temporary organ such as the placenta, the major difference between the procedures in one-step surgery and treatment of the placenta in situ is the resection of the entire area of invasion (previous cesarean scar) and the entire placenta in the one-step procedure.

8 Summary

The treatment of placental adhesive disorders is complex but not impossible when there is accurate preparation. The recognition of risk factors and diagnostic screening of patients with such disorders are essential to planning their subsequent management. A multidisciplinary team is recommended in order to permit discussion of each patient's case and to create suitable management options for each patient. Vascular control is mandatory in cases of resective procedures such as one-step conservative surgery or hysterectomy, and close clinical control is needed in the management of a placenta in situ. Because of the many variables involved in disorders of placental adhesion, training, resources, and knowledge are essential to obtaining optimal results in their management.

8.1 Quick guide

8.1.1 Clinical risk

1) Previous cesarean section and placenta previa
2) Previous iterative cesarean sections
3) Placenta previa and curettage
4) Abrasive abortions
5) Dilatation and curettage (D&C) after cesarean section
6) Previous uterine surgeries
7) Radiation
8) Age over 35 years
9) In vitro fertilization

8.1.2 Ultrasound

1) In patients at risk, disorders of placental adhesion are visible at week 18 of pregnancy.
2) The most reliable sign of a disorder of placental adhesion is the presence of irregular vascular spaces with arterial flow.
3) In the first trimester, low implantation of a sac on a uterine scar strongly suggests placenta accreta.
4) Antenatal identification of placenta accreta is possible with a high degree of sensitivity in patients with placenta previa and a history of cesarean section.
5) Transvaginal ultrasonography (Fig. 8.1) is preferred to abdominal US in cases of lower S2 invasion because it provides better image resolution in this area.

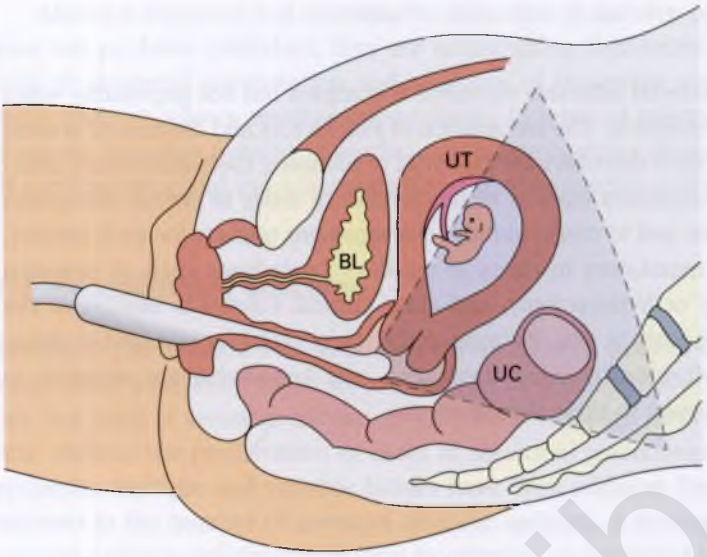

Fig. 8.1: Transvaginal ultrasound scan.

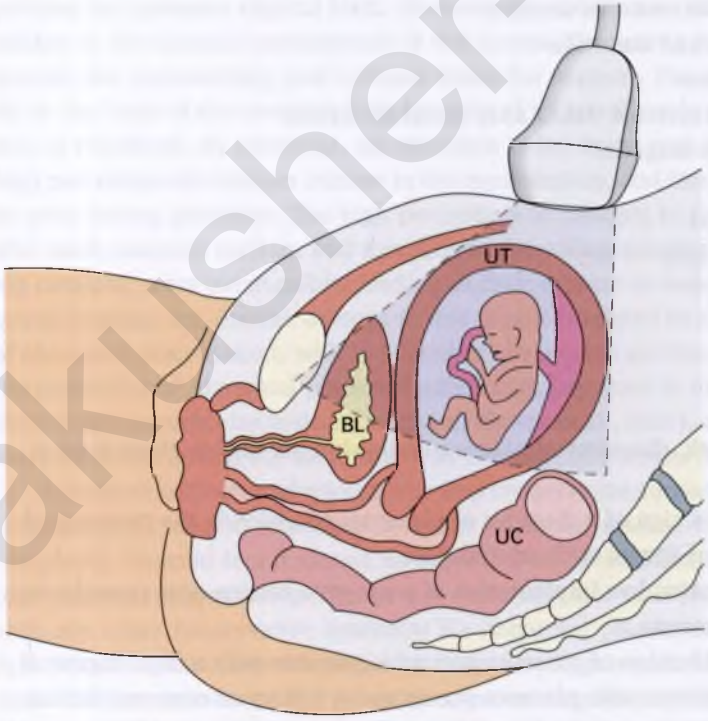

Fig. 8.2: Abdominal ultrasound scan.

8.1.3 Placental magnetic resonance imaging (pMRI)

1) Useful in surgical procedures such as hysterectomy or one-step conservative surgery to delineate the area of invasion, specific features, and the possibility of parametrial invasion (first indication).
2) Useful when US leaves doubt about a diagnosis (second and uncommon indication). Particularly useful in the diagnosis of abnormal placental invasion in patients without recognizable risk factors for this.
3) T2-weighted ultrafast techniques or breath-holding are preferred. Imaging with 5-mm slices in the, sagittal, axial, and coronal planes is recommended.
4) In cases of placental invasion of the anterior uterus, a semifull bladder is necessary for delineating the anatomy of invasion of the posterior bladder.
5) The study must focus on the entire bladder in detail. It is important to keep in mind that this study is different from fetal MRI, which includes the entire uterus.
6) Sagittal and coronal slices of the S2 region provide the most important information; for this reason, it is recommended that this region be examined first. In the few cases in which a patient may not tolerate a full placental MRI study, initial imaging of the most useful sequences guarantees the utility of a study even if it is not completed.

8.1.4 Doppler

1) Doppler US is useful as a diagnostic procedure complementary to US; does not reveal pathognomonic signs.
2) Three-dimensional Doppler US prevents overdiagnosis in cases of placenta previa and is useful for detecting placental invasion of the bladder.

8.1.5 Surgery

1) It is recommended that surgery be scheduled at week 35 for placenta percreta and week 37 for placenta accreta. The purpose of planing the surgery is to avoid expected complications.
2) Surgical alternatives depend on the type of placental invasion, the available resources, and the skills of the treatment team. The best procedure must prevent massive hemorrhage and avoidable complications during fetal delivery.
3) Either conservative treatment or hysterectomy can be better in cases of abnormal placentation, depending on the circumstances in a given case. There are no strict rules in this respect.
4) Knowledge and skill in the proximal vascular control of bleeding are needed to manage all kinds of abnormal placentation safely.

5) In cases of undiagnosed but surgically evident abnormal placentation, caution is extremely important until a treatment decision is made. If the fetal or maternal condition can wait until a qualified team is called and resources are requested, the uterus should not be incised. If the maternal or fetal condition make waiting impossible, it is preferable to open the uterus through a safe area to deliver an infant. It should be remembered that once this has ben done, there is time to decide on further measures. To ligate the umbilical cord and close the uterus can be the safest option until the necessary resources and a qualified team are available. If hysterectomy or manual placental removal triggers massive bleeding, there is no option to go back. At this point, accurate vascular control can provide limited additional time in which to request help.

Video 8.1: Bleeding (intraoperative).

Consideration of the removal of an adherent placenta needs to be carefully evaluated beforehand, because if bleeding occurs there is no possibility of going back. Both resources and skills need to be available and basic knowledge is necessary to achieve accurate vascular control. Attention should be given to the amount of blood lost in a period of a few seconds, because if dissection ia not done between the bladder and area of placental invasion, lower vascular control is almost impossible. If this problem arises, it is recommended that the infrarenal aorta be compressed against the pelvic promontory until help is obtained. In such circumstances, rapid hysterectomy is almost impossible or carries a high risk of hypovolemic shock.

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6) Surgical evidence of parametrial invasion presents a major challenge, and the operators treating such invasion should take care to evaluate their skills and resources until starting the surgery for it, whether this is conservative one-step surgery or hysterectomy. Ureteral identification, a narrow anatomic operating space, and invasion by a multitude of NFV are the most important technical problems in the surgical treatment of parametrial invasion.

8.1.6 Interventional radiologist's assistance

1) Embolization of the uterine arteries is not a guarantee of hemostasis in anterior and lower abnormal placentation (the most frequent types of this condition).
2) Occlusion of the internal iliac artery is not a good means of proximal vascular control in lower abnormal placentation.
3) Embolization of the anterior trunk of the iliac internal artery large particles (over 1000 μ) of an embolic agent usually reduces collateral blood flow in the invaded area, permitting hysterectomy with less bleeding.

4) Massive or high-pressure embolization with inappropriate embolic material increases the possibility of nontargeted embolization and unwanted ischemia or necrosis. Small particles can produce accurate hemostasis but also be a cause of late ovarian and endometrial dysfunction.
5) Transfemoral aortic balloon occlusion is one of the best methods for controlling massive pelvic bleeding. The balloon can be put in place in the deflated state until it is necessary to stop the bleeding. After it is inflated, the action of the aortic balloon is immediate, and the resulting blockage can be reversed through deflation of the balloon without hemodynamic effects. It should be remembered that optimal vascular control provides time in which to replenish volume and organize subsequent therap
6) Transferring a patient with active bleeding caused by abnormal placentation to the interventional radiology room is a complex issue. Patient transfer can be dangerous without a specialist's care and high-resource assistance. care should be taken to maintain an optimal blood pressure and safe clotting during transfer; hypotension and coagulopathy are the main causes of failure in such cases. Vascular redistribution and vasospasm can be additional problems in unstable patients.

8.1.7 Hemodynamic and hemostatic status

Having 4 units of blood and good vascular access prevents habitual delay in the management of massive bleeding. The duration of hypotension is closely associated with late morbidity, especially with multiple organ failure. Immediate proximal vascular control and early provision of fluids, blood, and hemoderivatives are needed in trearting massive bleeding. Uncontrolled hemorrhage is a cause of shock, acidosis, coagulopathy and additional disorders, which are sources of serious morbidity.

Massive bleeding due to abnormal placentation produces coagulation disorders. The most common are fibrinogen consumption and platelet malfunction. With the exception of its measurement at a few centers, the assay of fibrinogen levels takes 30 minutes or more. For this reason, and in the face of clinical evidence of coagulopathy, the administration of 1 U of cryoprecipitate per 10 kg body weight is recommended immediately. Dilutional coagulopathy is frequent only after massive blood-loss replacement and fluid replacement, although this can be avoided by using one unit of plasma per unit of packed red blood cells transfused.

8.1.8 Reproductive outcome

Conservative treatments can preserve fertility but are followed by different rates of recurrence of the conditions against which they are used. A complete review of the

resources, expected complications, and follow-up for any particular condition must be discussed before treatment. Although presurgical studies can provide information about the type, degree, and topography of a placental invasion, a complete diagnosis can be made only during surgical exploration.

8.2 Conclusions

The resolution of abnormal placentation is a major challenge for obstetricians. The advances made in diagnostic methods for this disorder makes its identification easy, but its successful surgical management requires skill and a well-organized treatment team. Distortions of anatomy and profuse circulatory perfusion in cases of abnormal placentation make surgical mistakes too costly. However, multiple worldwide investigations have provided useful details for the management of this disorder.

A major change will occur with modification of the etiology of abnormal placentation independently of the number of a patient's cesarean sections, but until that time comes, obstetricians need to coordinate their efforts to fight this challenging disorder.

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