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# The Normal and Pathologic Postpartum Uterus

*P. Kelehan and E. E. Mooney*

## BACKGROUND AND AIMS

Significant postpartum hemorrhage (PPH) may occur immediately after delivery or may be delayed by weeks or months. In either circumstance, a hysterectomy may be life saving. The uterus normally will be sent for pathologic examination. To facilitate preparation of a useful surgical pathology report, however, the pathologist must be given details of the antepartum course and delivery. Considering how uncommon these specimens are, direct communication between pathologist and clinician is recommended so that the many important details of the case may be fully appreciated. The aim of this chapter is to provide a structured approach to the analysis of the specimen, in order to permit a clinically relevant and pathologically sound diagnosis.

## CLINICAL CORRELATION

The patient's parity and gestation should be provided. Any abnormality of the clinical course, in particular pre-eclampsia or polyhydramnios, may be of relevance. Magnetic resonance imaging (MRI) may have been performed for fibroid, placenta creta or congenital abnormality and these images should be available for review. A history of the use of instruments such as forceps is important. The initial clinical appearance of the uterus at the time of operation may provide valuable information on atony. Any therapeutic measures undertaken such as uterine massage or placement of a compression suture(s) should be noted, along with transfusion and fluid replacement. A description of the surgery (ideally a full copy of the operative report) will help the pathologist to interpret tears and sutures that normally characterize these specimens. The patient's postoperative condition will help to guide sampling in the event that amniotic fluid embolism is also a consideration. Finally, the placenta must also be available for examination.

## GROSS EXAMINATION

Photography is essential at each step of the dissection, with notes as to what each picture is intended to show. Without a clinical input, however, much effort may be wasted on documenting features of little relevance at the expense of missing more important ones. A detailed macroscopic description of sutures, tears, etc. is important and may be relevant medicolegally. Our approach is to examine the specimen in its fresh state, using photography, and then to open the specimen, avoiding tears and sutures, to permit fixation and further examination. The uterus may be opened laterally, but more information can be gained by complete longitudinal anteroposterior section. This approach should be modified to suit the circumstances as predicted from the clinical information obtained along with the specimen. A useful technique that allows good exposure and photographic demonstration is the placing of two parallel complete longitudinal anteroposterior sections about 2–3 cm apart on either side of the midline. How well the uterine cavity has compressed is immediately apparent, contraction band (if present) formation can be demonstrated, and blood clot and placental tissue fragments can be assessed in the lumen (Figure 1).

In the immediate postpartum period, the uterus is characteristically large. It will weigh 700–900 g on average and will have substantially reduced in size and volume from its antepartum state. Clamp marks on the broad and round ligaments should be inspected for residual hematoma, remembering that the pathology may be outside the clamp. In the fresh specimen with intact vessels, it may be possible to perfuse the vasculature for contrast angiography or vascular casting[1]. In this manner the subplacental vasculature can be demonstrated and the location of any arteriovenous malformations identified (Figures 1 and 2).

## UTEROPLACENTAL VASCULATURE

Schapps and Tsatsaris *et al.*[1] have proposed a model of human uteroplacental vascularization. They replace the old concept of maternal blood spurting into the intervillous space from uterine arteries with the blood flowing past fetal chorionic villi in a low-resistance high-flow circuit and behaving as an arteriovenous shunt in the placenta as it flows back into uterine veins. They propose and show evidence for a uterine vascular anastomotic network in the subplacental

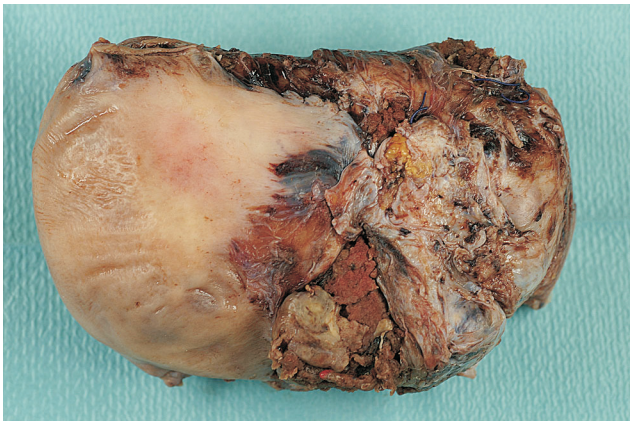

**Figure 1** Fixed uterus showing a large anterior and right-sided diverticulum originating in a cesarean section scar. The specimen was sutured at operation, but placental villous tissue can be seen adjacent to the suture

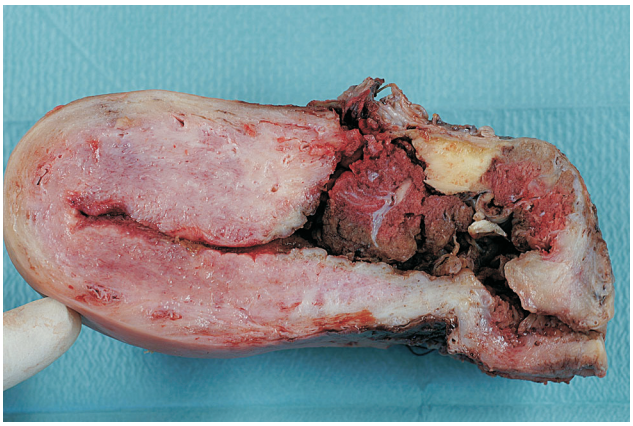

**Figure 2** Anteroposterior section of uterus from Figure 1 showing anterior placenta creta


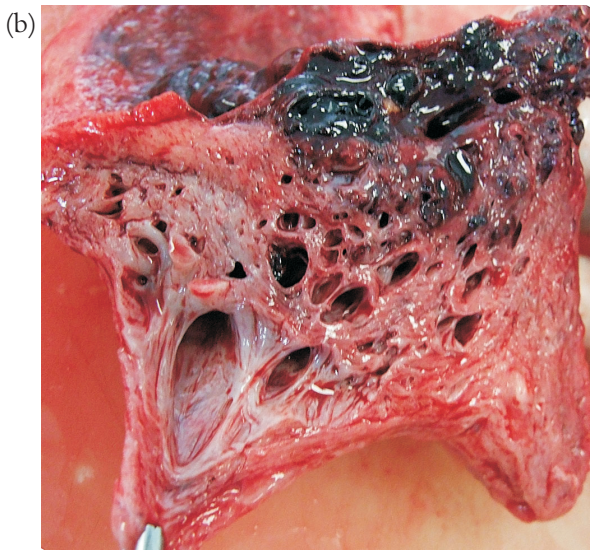


**Figure 3** Demonstration of the subplacental uterine vascular anastomotic network described by Schapps and Tsatsaris *et al.*[1]. (a) Near term undelivered indirect maternal death, flacid myometrium, postmortem allows the dilated vessels beneath the placenta to be easily seen. (b) Postpartum indirect maternal death day 4, vessels remain dilated. There is in addition adherent blood clots associated with subinvolution of the placental site

myometrium, which controls blood flow to the decidual 'spiral arterioles'. Maternofetal exchange occurs within a system in which the uterine vascular network plays an important role, supplying blood to the intervillous space, which is positioned in parallel to the main circuit. In this model, the intervillous space is not a high flow shunt and the fetal circulation is protected from acute maternal hypertensive events. Placental detachment after delivery does not open up high flow vessels, the spiral arterioles are more easily compressible and uterine arterial blood flow is diverted through its anastomoses to the venous system[1]. This concept is of interest in the light of evolving theories of placental mechanical and oxidative stress resulting from pulsatile flow into the intervillous space consequent on inadequate transformation of the spiral arteries[2] (Figure 3). *[Editor's note: Interested readers will find more information on this topic in Chapter 24. L.G.K.]*

## CERVIX

Tears are among the most important pathologies found in the cervix. Small shallow endocervical tears are almost invariably present in the postpartum state, and may be seen even in those cases where cesarean section has prevailed. In contrast, significant and deep tears tend to be lateral in location, may penetrate through to the serosa (with or without hematoma formation), and may extend up into the lower uterine segment or down into the vagina. If extension is upward, involvement of large uterine arteries is possible and should be sought. As it is common to find meconium staining of the endocervical mucus in the presence of fetal distress, meconium may contaminate the tear. It is axiomatic that cervical tears may have severe consequences; for example, an endocervical tear may cause severe and life-threatening blood loss despite a fully contracted uterus. Tears can be associated with amniotic fluid embolus or with amniotic infusion and local defibrination. Upwardly extending tears may be associated with bleeding into the broad ligament with hematoma formation. Suturing of the cervical tear *per se* without considering the patient's

general condition may not prevent a deep hematoma from forming; secondary rupture can result in shock, despite cessation of external vaginal hemorrhage.

In the dilated postpartum cervix, edema, hemorrhage and disarray of the normal architecture of the muscle fibers may make it difficult to identify tears on histologic examination. Torn and contracted muscle fibers and torn arteries with fibrin plugs and tense hematomas provide corroboratory evidence of a tear. Histologic sampling should include blocks from above the apex and from below the tear to identify deep extension and for identification of large torn vessels.

Following amniotic fluid embolism, histologic examination of the uterus will show no evidence of intravascular disease in many cases. Occasionally, however, there may be fibrin clots adherent to vascular endothelium, and squames admixed with fibrin have been found in vessels in the body of the uterus. In some cases of PPH, when there have been no clinical features of acute amniotic infusion but bleeding and unexpected severe onset of consumptive coagulopathy, histological sections of the endocervix will reveal localized areas where amniotic debris fills and expands venules and capillaries. This dramatic appearance is present not only adjacent to the endocervical surface and tears, but also its presence deeper in the stroma distinguishes it from contamination of the surface mucosa by meconium and amniotic fluid at delivery (Figures 4–6).

A subgroup of patients have a lesion of local amniotic infusion associated with disseminated intravascular coagulopathy (DIC) and PPH without systemic collapse. Squamous cells may be present in only one or two sections taken from around the circumference of the cervix. It is usually on one side. Extensive sampling of the cervix may be required to demonstrate amniotic debris in cases of suspected amniotic fluid embolism[3]. It is possible that ongoing blood loss from a tear in this site may occur before the onset of systemic DIC, because the local thromboplastin effect alone of the amniotic debris in the wound may inhibit hemostasis.

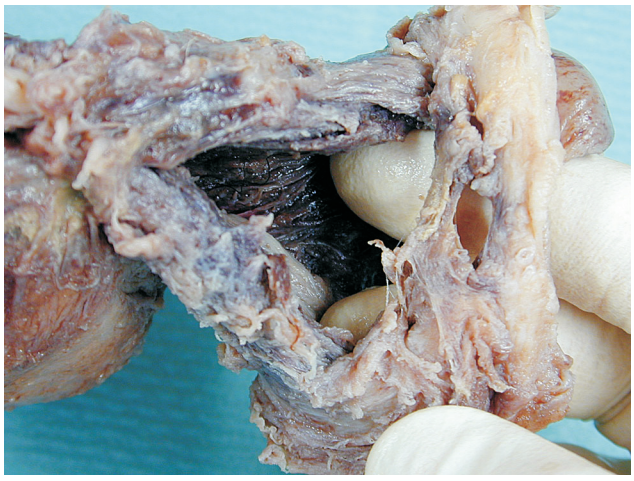

**Figure 4** Right lateral endocervical tear at hysterectomy for PPH

## LOWER UTERINE SEGMENT

Important pathologies at this location involve placental implantation on a previous cesarean section scar, with abnormal adherence or formation of a diverticulum. A prior cesarean section results in chronic changes in the lower uterine segment, which may include distortion and widening, inflammation, giant cell reaction, implantation endometriosis and adenomyosis[4]. In some instances, a distinctive V-shaped defect of the anterior uterine wall ('tenting') may be present.

Emergency cesarean section in particular may be associated with circumstances that cause the incision repair to be suboptimal. In examining hysterectomy specimens for menorrhagia considerable variation in the morphology of the lower uterine segment repair is seen, sometimes with persistent inflammatory foci many years after the last cesarean section. Approximately one-third of lower segment scars examined

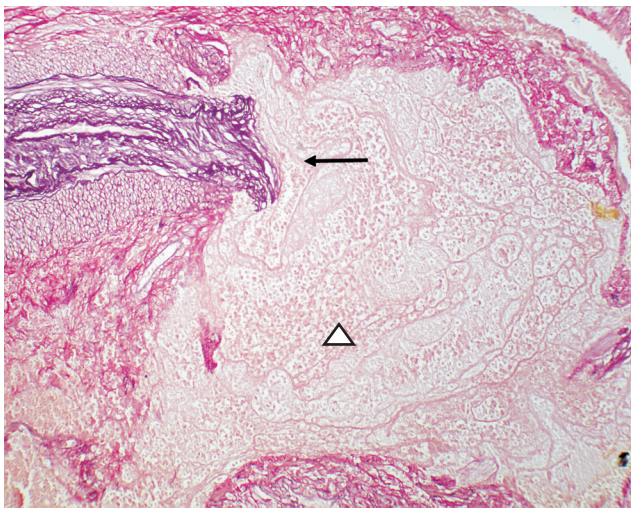

**Figure 5** Elastin Van Geisson stain showing torn artery at apex of tear. Arrow, torn elastic artery; arrowhead, thin fibrin blood clot

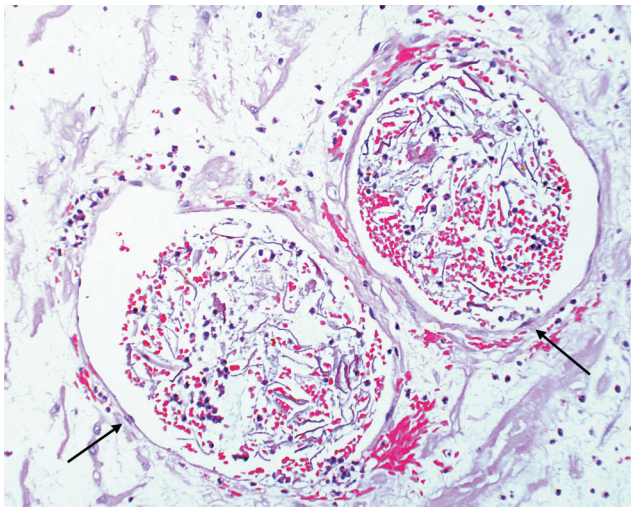

**Figure 6** Amniotic debris in venules (arrows) of cervical stroma following a small endocervical tear in labor. PPH and disseminated intravascular coagulopathy necessitated hysterectomy

histologically will show very deficient interweaving or meshing of myometrial fiber bundles. Occasionally they will show only a thin collagen scar or scar and foci of implantation endometriosis which may extend from lumen to serosa.

An important cause of the weakening of any cesarean section scar is infection. Postoperative wound infection (overt and covert) is not uncommon following cesarean section, particularly the emergency variety. Prophylactic antibiotics can modify the extent and rate of infection, as can the quality of closure, the amount of local tissue trauma, the technique used (one- or two-layer), the presence of swelling and/or hematoma and the nature of the organisms infecting the wound. There may be extensive disruption and inflammation in the uterine wound despite a normal healing appearance of the skin wound. Conservative treatment of the skin wound is normal, and surgical debridement the exception. Accordingly, the consequences of infection at the scar site may be only appreciated in a subsequent pregnancy. If the patient does present before this, hemorrhage and/or vaginal discharge may prompt internal examination. A defect may be identified on palpation. Curettage may be undertaken and may retrieve inflammatory exudate, degenerating decidua, polypoid endometrium or fragments of necrotic myometrium that have prolapsed into the endocervical lumen from the internal edge of the cesarean section scar. Sometimes, quite large pieces of myometrial tissue with edema and coagulative necrosis are obtained. This site related myonecrosis or incisional necrosis is caused by local ischemia[5] (Figures 7–9). Remodeling of blood vessels at the site of the former scar may influence subsequent placental implantation. This is important, because implantation on either a normally healed or a diseased scar will not have the protective effect of the presence of decidua vera (see below), and accordingly postpartum separation at the time of a subsequent pregnancy is less likely to occur. A cesarean section at first birth is associated with increased risks of placenta previa and abruption in second pregnancies[6].

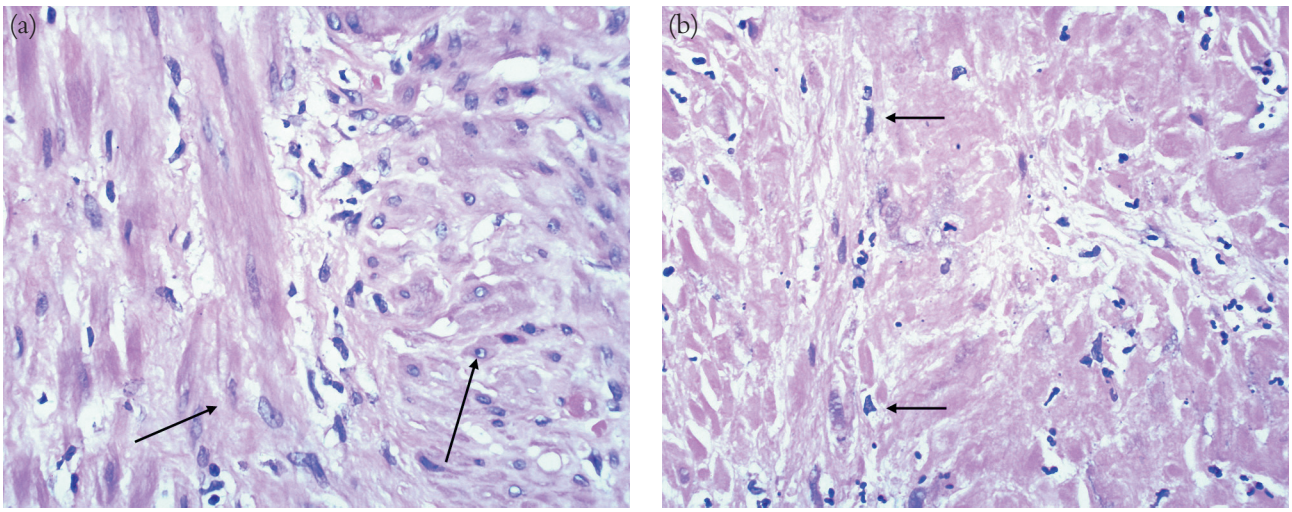


**Figure 7** H/E comparison of (a) normal myometrial fibers and (b) myonecrosis in lower uterine segment in hysterectomy specimen for postpartum hemorrhage following cesarean section. Long arrows, normal viable cell nuclei; short arrows, non-viable necrotic cells

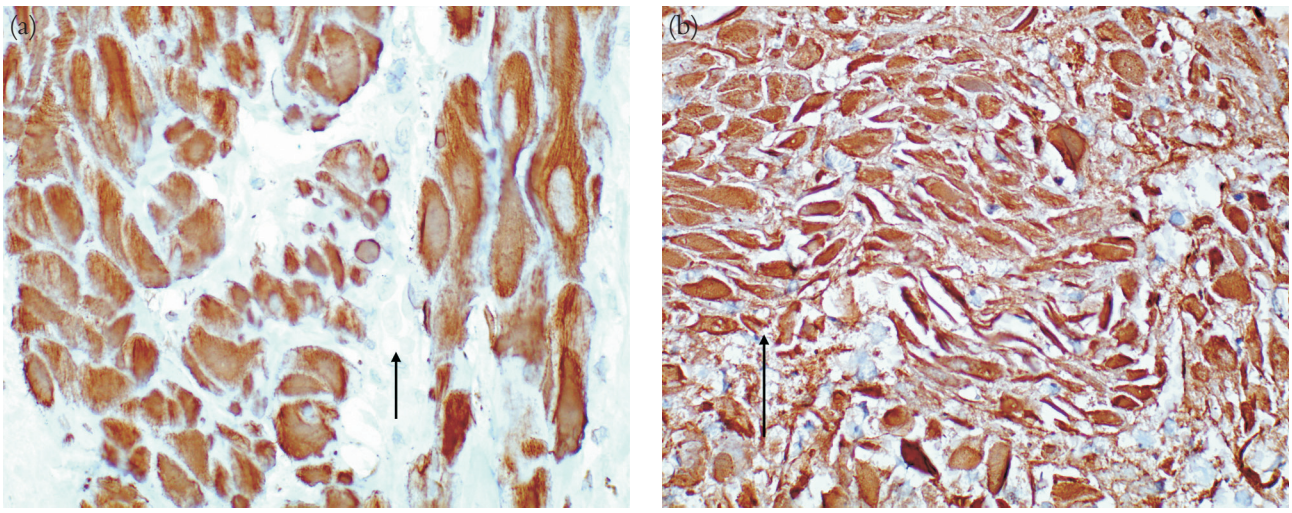


**Figure 8** Desmin comparison of same myometrial fibers accentuates the necrosis. (a) Normal; (b) myonecrosis. Long arrow, normal myometrial cells with intercellular edema; short arrow, dense, compacted necrotic myometrial cells at same magnification

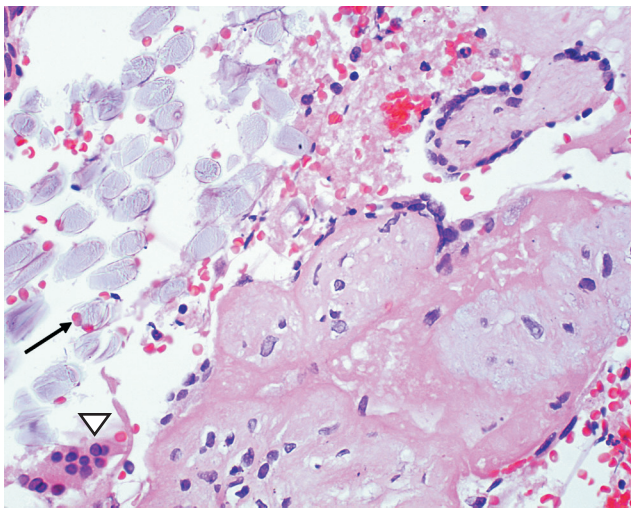

**Figure 9** H/E section showing stitch material in uterine curettings following cesarean section. Arrow, absorbable suture; arrowhead, giant cell reaction to suture material

Implantation in the lower segment (adjacent to the defect) can cause expansion of the defect, dehiscence of the wall and the formation of a pulsion diverticulum which will further enlarge and progress with growth of the placenta. If the implantation is fundal, on the other hand, a fortuitous elective section may reveal a thin, almost transparent anterior lower segment wall. This should be more easily resected at the time of closure, as the scar will not be excessively vascular. If implantation is in the lower segment or in the scar, then the potential for catastrophic hemorrhage on attempt at delivery of the placenta is real.

The points noted above should be borne in mind when examining a postpartum hysterectomy specimen where there is a history of previous cesarean section. The recently sutured section incision may be carefully reopened. Following photography, the edges and margins should be inspected for thinning and scar tissue formation. An enlarged, ragged and open defect of the anterior lower uterine segment, now tightly contracted and rigid with formalin fixation, may be all that is left of a huge, thin-walled, placenta-filled diverticulum, the result of scar dehiscence and rupture. It is easy to destroy this thin structure with hurried dissection. Examination of the lateral margins of the defect may indicate left- or more often right-sided extension of the bulging diverticulum into parametrial soft tissue of the pelvis. A complete section through the anterior lower uterine segment can identify previous cesarean section scars with tenting defects and the shape and edges of a recent section. Most importantly, en-face examination of the lateral sides of the lower segment will show the cavity and lateral extension of a dehiscence diverticulum, fresh tears and/or adherent placenta. The issue of abnormal adherence is addressed below.

## FUNDUS

Important pathologies include retained products, placenta creta and subinvolution. Placenta creta is the name given to abnormally adherent or ingrowing placenta that does not detach with full contraction of the uterus after expulsion of the fetus. This term covers placenta accreta (abnormal attachment to the wall), increta (extension of villi into the myometrium) and percreta (extension of villi through to the serosa). The intimate relationship of villous tissue to myometrium, without intervening decidua, is key to the diagnosis. Descriptions of placenta percreta based on illustrations or descriptions of chorionic villi displaced between torn myometrial fibers should be evaluated critically. If MRI has been obtained, views may show the loss of zonation associated with penetration rather than invasion of chorionic villi.

Full-thickness anteroposterior sections of the fundus make it easier to recognize the position of the contracted placental site. It is surprisingly difficult to identify the exact placental site on inspection of the raw decidual surface that is seen if the uterus is opened laterally.

Detachment of the placenta is dependent on the presence of a normal spongy decidua vera, where shearing of the placenta from the myometrium occurs. This soft compressible area is not seen when the postpartum uterine lining is examined histologically, because its many mucous glands are disrupted to facilitate the normal plane of cleavage. It is seen to its full extent in the tragic cases of maternal death prior to labor. Either Alcian blue stain or diastase-PAS can be used to demonstrate mucopolysaccharides in the swollen gland crypts that help to identify this layer. Deficiency of this layer may be focal or, rarely, complete. When it is absent, the thinned Nitabuch's layer with anchoring villi lies in close proximity to muscle fiber bundles or the interstitial fibrous cesarean section scar. An occasional finding is the presence of abundant intermediate trophoblast infiltrating between muscle fibers beneath a firmly adherent Nitabuch's layer. Histological examination of multiple sections can show anchoring villi penetrating Nitabuch's fibrinoid and ghost villi in dense fibrin adherent to muscle. The often described appearance of chorionic villi infiltrating between muscle fibers is characteristic only of invasive mole; the key to the diagnosis of placenta percreta is the absence of decidua. An increased number of implantation site intermediate trophoblasts has been shown in cases of placenta creta compared with controls[7]. Retained placental fragments reflect some degree of placenta creta and are more common in women with a spectrum of changes in previous pregnancies, such as pre-eclamptic toxemia, growth restriction, spontaneous abortion and retained placental fragments. It has been hypothesized that these latter conditions reflect abnormal maternal–trophoblast interaction[8].

Placenta creta is therefore due to a deficiency of the decidua. The end result of penetration of the placenta through a weakened part of the uterine wall includes rupture and secondary changes, including serosal peritoneal reaction. Curette penetration may cause secondary infection or hematoma formation and

provide the nidus for dehiscence into the adherent bladder wall, if this had been injured at previous surgery.

Placenta creta is only part of the problem of uncontrolled PPH. The thin myometrium, with little muscle, interstitial fibrosis and increased intermediate trophoblast will contain large dilated arteries of pregnancy and often widespread extrauterine extension of these changes into the parametrium, as demonstrated on Doppler ultrasound. The degree of constriction–contraction of the myometrium is insufficient to close off these vessels. Where there is severe thinning of the muscle of the lower segment with diverticulum formation, abnormal adhesion is not necessary to sustain bleeding (Figures 10 and 11). Conversely, on histological examination of the lining of the postpartum uterus, the finding of chorionic villi in clefts in the placental bed may be an artifact rubbed in following clearance of uterine contents and is of no diagnostic consequence. Smearing of DNA due to crush artifact may be helpful in distinguishing this from true extension.

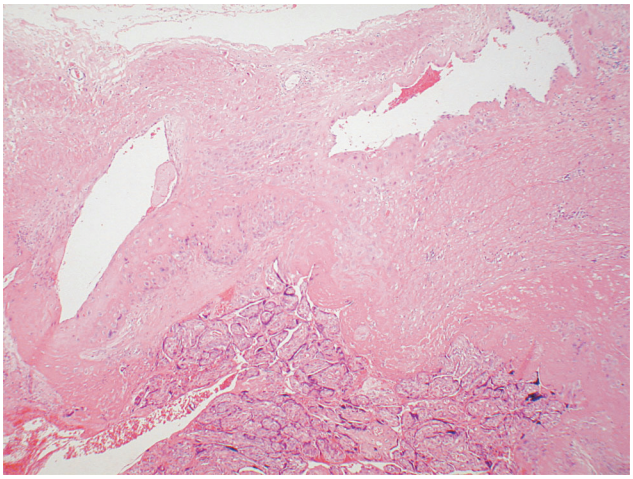

**Figure 10** H/E section of lower uterine segment showing placenta creta and large vessels in thin myometrium

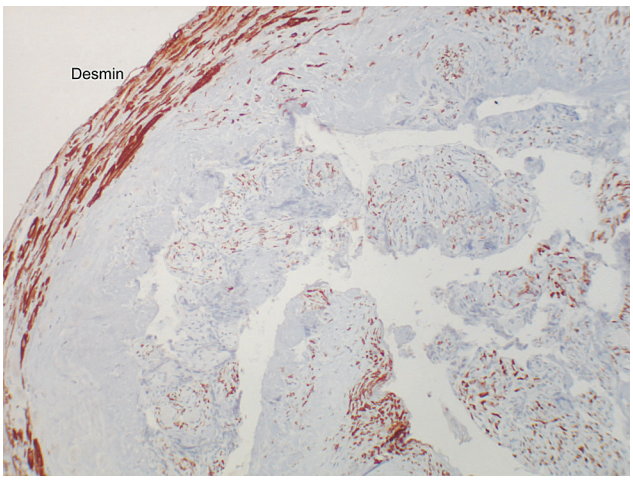


**Figure 11** Immunohistochemical stain for desmin accentuates the thin myometrial fibers in scar

## RETAINED PLACENTA AND RETAINED PRODUCTS OF CONCEPTION

The failure of total expulsion of the placenta may lead to PPH and attempted removal is a major cause of maternal mortality and morbidity worldwide. Herman and colleagues[9] offer perspectives into third stage mechanisms. They have shown by dynamic ultrasound techniques that the normal third stage has three distinct phases: (1) latent phase – in which the whole uterus can be seen to contract except for the area behind the placenta; (2) contraction phase – the retroplacental myometrium contracts; and (3) detachment and expulsion phase. It is further suggested that an as yet unknown placental factor plays a major part in controlling and inhibiting contraction in the subplacental myometrium[10]. Following delivery of the placenta, fragments that remain in the uterus may be attached or detached. A fragment of placenta remaining attached, assumes a polypoid shape ('placental polyp'), and undergoes compression and some devitalization. Viable trophoblast and villous stromal cells may persist due to continuing uteroplacental perfusion.

Plasma cell infiltrate may be present in the adjacent myometrium; however, this is not always diagnostic of (infective) endometritis in this context. Detached placental fragments are devitalized and along with blood clot form a nidus for delayed involution and ascending infection. The frequency of detection of retained products varies from 27 to 88%[8], but much of this literature is decades old and was obtained before the routine use of ultrasonography. Nevertheless, retained placental fragments are more common in women who have experienced pre-eclampsia or growth restriction in previous pregnancies. This observation has been interpreted as indicative of an abnormal maternal–trophoblast relationship[8].

## SUBINVOLUTION

In the absence of retained placental fragments, subinvolution of the blood vessels of the placental bed is an important and distinctive cause of secondary PPH. This important idiopathic and non-iatrogenic condition has been recognized in the pathology literature for more than 50 years, but is rarely mentioned in clinical texts. It is to be distinguished from clinical atony alone, but may also have co-morbidity of retained fragments of placenta or sepsis.

Normal arterial involution involves a decrease in the lumen size, disappearance of trophoblast, thickening of the intima, re-growth of endothelium and regeneration of internal elastic lamina. These changes normally occur within 3 weeks of delivery. With subinvolution, on the other hand, arteries remain distended and contain red cells or fresh thrombus, and trophoblast persists in a perivascular location[11]. In some cases, endovascular trophoblast may be present. Hemorrhage from subinvolution is maximal in the second week postpartum, although it may occur up to

several months later. It is commoner in older, multiparous women and may recur in subsequent deliveries.

Subinvolution is not related to the method of delivery and may be regarded as a specific entity, possibly due to an abnormal immunologic relationship between trophoblast and the uterus[11]. Despite this, subinvolution did not show the association with markers of such an abnormal relationship seen with retained placental fragments in another study[8].

Subinvolutionary changes may be recognized on curettage specimens, whereas hysterectomy specimens are characterized by a uterus that is soft and larger than expected[11]. As normally involuted vessels may be present adjacent to subinvoluted ones, multiple blocks of placental bed should be taken to exclude this process (Figure 12).

## ATONY

Although this is a well-recognized clinical phenomenon, but there is little to report in the way of pathology. The diagnosis is one of exclusion. The uterus is enlarged, edematous and soft, with edema and hemorrhage apparent microscopically. The diagnosis will depend on clinical information, combined with adequate histologic sampling to exclude other causes.

## ARTERIOVENOUS MALFORMATIONS

Uterine arteriovenous malformations (AVMs) are rare and may present with profuse hemorrhage, including hemorrhage in the postpartum period. Congenital AVMs consist of multiple small connections and may enlarge with pregnancy. The more common acquired AVMs are rare in nulliparous women, and are thought to arise following uterine trauma: curettage, myomectomy or even previous uterine rupture (Figure 13). AVMs may co-exist with retained products of conception or trophoblastic proliferation. Pathologically, vessels of arterial and venous caliber are present, along with large vessels of indeterminate nature. It is possible that the special vasculature of the uterus may, in abnormal situations, contribute to the formation of AVMs.

## OTHER CAUSES OF POSTPARTUM HEMORRHAGE

Lacerations of the inner myometrium can cause PPH[14]. Women with leiomyomas are at an increased risk of PPH[15]. Less commonly, endometrial carcinomas and congenital anomalies may also result in reduced decidua formation and subsequent PPH. Trophoblastic disease has also been reported in this context.

## ENDOMYOMETRITIS

Sepsis causing acute endometritis is reported as a cause of PPH, and hemorrhage may be followed by ascending infection. It is relatively uncommon in modern obstetric practice in the developed world and may be due to a variety of organisms. Its incidence is increased following emergency cesarean section. It accounted for less than 5% of cases of delayed PPH in one series[8].

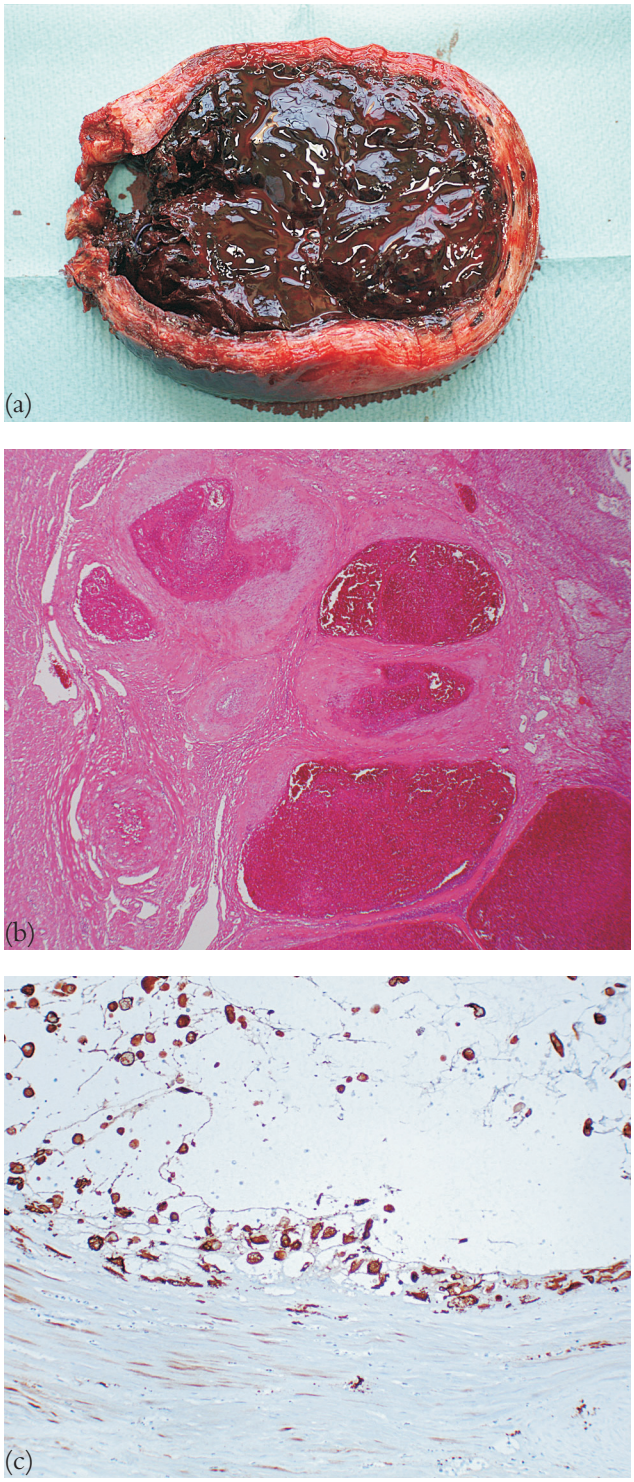


**Figure 12** (a) Anteroposterior section of postpartum uterus 5 days following emergency cesarean section. Secondary PPH with blood filled uterus due to subinvolution of the placental site. Bleeding was from the fundus not the cesarean site. (b) Low power microscopic image of greatly dilated thrombosed subinvoluted blood vessels in the postpartum placental bed. H/E. (c) Cytokeratin staining of endovascular trophoblast

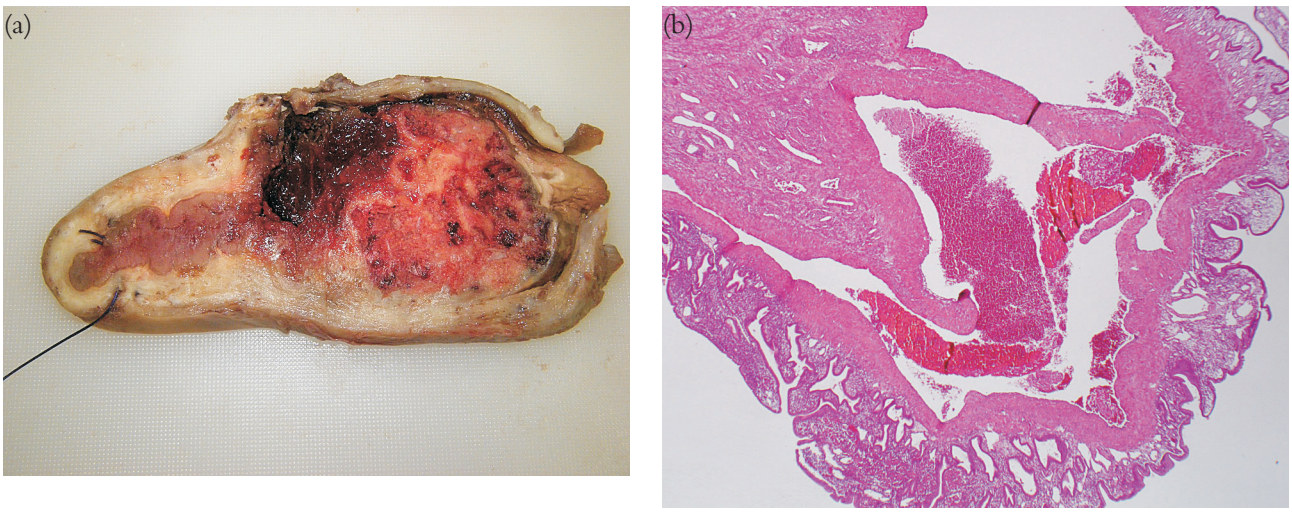


**Figure 13** (a) Anteroposterior section of postpartum uterus 2 months following cesarean section for severe placenta accreta with conservative management including uterine artery embolization. Severe bleeding has recurred in association with arteriovenous malformation. (b) Low power microscopic image of part of large arteriovenous malformation which extends to the endocervical mucosa. H/E

## PLACENTAL PATHOLOGY

The placenta should be examined wherever possible in cases of PPH. Pre-eclampsia may cause retroplacental hemorrhage: recent and old hemorrhages and infarcts may be seen. The characteristic changes of acute atherosis are only present in 50% of cases of pre-eclamptic toxemia. However, examination of the parenchyma will usually show so-called accelerated villous maturation (distal villous hypoplasia) in response to uteroplacental ischemia. Sampling from the center of the placental disc is important to avoid overinterpretation of physiologic changes[16].

## THE AUTOPSY AFTER POSTPARTUM HEMORRHAGE

In data drawn from the Confidential Enquiries into Maternal Deaths in the UK for the period 1970–90, approximately 10% of direct maternal deaths are due to hemorrhage[17]. Roughly half were antepartum and half postpartum. Excess blood loss is more common in older women (>35 or 40 years, depending on the study)[18]. This trend and ratio has continued to the 2006–2008 Confidential Enquiries into Maternal Deaths in the UK report[19].

Before beginning an autopsy in a case of maternal death following postpartum or intrapartum hemorrhage, it is critical to plan the procedure and the sequence of the autopsy in the light of the information received and the suspected cause or causes and mode of death. The autopsy must be unhurried and methodical; it is a fundamental mistake to seek to demonstrate immediately the proposed cause of death. Members of the clinical team should be asked to attend, but it is unwise to have everybody there during what will be a long phase of inspection, measurement and initial systematic dissection. When all is ready, the procedure is stopped and members of the clinical team attend. In this manner, the history can be reviewed, pre-existing conditions or disease discussed and demonstrated, e.g. chronic pyelonephritis, and the dissection and demonstration of the focus of main clinical interest can commence.

A fundamental aspect of good autopsy practice is the confident exclusion of specific diseases and conditions in a systematic approach. The understandable desire and pressure to skip to the seat of disease must be resisted. The parametrium, pelvic side-walls and vagina are as important objects of attention as is the uterus.

At the time of external inspection of the body, the pathologist must consider in turn each of the major causes of maternal death. Many require modification of routine techniques, e.g. air embolism, amniotic fluid embolism, ruptured aneurysm, and these modifications are detailed elsewhere[20]. Preparation and sampling of blood and fluids for hematology, hemophilia, toxicology and microbiology may be planned, e.g. sample containers should be pre-labeled and set out in sequence. Cardiopulmonary resuscitation attempts most likely preceded death, and therefore the features and sequence of sustained unsuccessful resuscitation must be identified and the complications and accompanying agonal changes interpreted in this context. It is important from a medicolegal aspect not to allow such artifacts to be construed as a major factor in the cause of death, e.g. liver or mesenteric tear, blood in the abdomen, bone marrow embolus.

The traditional Y-shaped autopsy incision should be extended to an abdominal inverted Y with the incision continued to the inguinal femoral triangle on each side. This allows better examination of the ileofemoral vessels and better exposure of the pelvis. Blood and blood clots are removed from the abdomen and the amounts of each measured. The relative size and position of the abdominopelvic organs are assessed. The peritoneal lining of the pelvis is inspected, noting color, texture and degree of

congestion. Patches of peritoneal decidual reaction of pregnancy can be identified by their gelatinous appearance.

In traditional autopsy practice, the state of pregnancy can be suspected, even when the uterus is still small, by the characteristic dilated and congested appearance of retroperitoneal veins. The degree of dilation and turgidity of the pelvic veins should be noted early at autopsy as they will be dissected and examined in detail later. Retroperitoneal hematoma and broad ligament hematoma should be identified or excluded at this stage as these may be less easily assessed and measured following organ removal. The uterus may be examined and opened *in situ*, but it is better to remove adrenal, renal and pelvic organs as one complete block.

The traditional method of blunt dissection along the pelvic side-wall and pubis with transection at the mid to upper vagina is extended in the investigation of PPH. Following knife separation of the symphysis pubis, the legs are externally rotated and a knife cut is made along the lower edge of the pubic bone. The pubic bones are forcefully separated by 8–10 cm. This, together with the inguinal femoral incisions, gives good exposure of the paracervical and paravaginal soft tissues. Lateral vaginal wall tears and hemorrhage can be inspected and well demonstrated by this modified technique. The ileofemoral vessels are transected and inspected. The complete urogenital block is placed on a dissection board where it can be opened in layers, beginning with the urethra and bladder, then the vagina and cervix. Alternatively, the block can be placed in formalin and later dissected after short fixation.

The aorta is opened posteriorly and incision is extended into the branches of the iliac arteries for a short distance. The inferior vena cava is opened from the anterior side, probed and dissected into the right and left renal veins; the ovarian veins are identified and opened, and dissection is continued into the branches of the pelvic veins out to the limits of the excised specimen. The intima is examined for evidence of tear or abrasion and for adherent thrombus. Pieces of tissue containing venous plexus from the broad ligament and parametrium are selected for formalin fixation and histological examination.

When the patient has died of hemorrhage and where there has been an attempt to stem the bleeding by hysterectomy and under-sewing of bleeding sites and pedicles, it may be very difficult to identify the exact sites of bleeding, and ancillary techniques may be helpful. Prior to pelvic dissection, an infusion of saline through an intravenous infusion set and cannula into the clamped abdominal aorta may identify a bleeding point. With special preparation and ligation of all peripheral vessels, autopsy specimen angiography may be very valuable in selected cases.

The most useful of all techniques is the histological examination of carefully selected blocks of tissue demonstrating vital reaction to injury and the presence or absence of conditions predisposing to disease.

Detailed histological examination is a prerequisite of good autopsy practice in the investigation of maternal death. Amniotic fluid embolus, pregnancy-induced or essential hypertension, pre-eclampsia and rare conditions such as thrombotic thrombocytopenic purpura or cardiac sarcoidosis always need histological examination for diagnosis or confirmation. A minimum of organs to be sampled and sections taken is recommended in the investigation of all maternal deaths[21].

## SUMMARY

In modern obstetrics practice in developed countries, an important cause of life threatening PPH is morbid adhesion of the placenta to a previous cesarean section scar. Recognition of this potential complication is an essential consideration in the investigation and clinical management of the pregnant woman with previous cesarean delivery. Gross and microscopic pathology findings can enhance the interpretation of radiologic images and explain the pathophysiology of this condition. Histological evaluation of poorly appreciated conditions such as subinvolution and arteriovenous malformation are an essential component of the evaluation of PPH.

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