Antepartum and Postpartum Hemorrhage

CHAPTER 18

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KEY ABBREVIATIONS	
Bilevel positive airway pressure	BiPAP
Continuous positive airway pressure	CPAP
Fresh frozen plasma	FFP
Packed red blood cells	pRBCs

Obstetric hemorrhage is one of the leading causes of maternal morbidity and mortality throughout the world. Hemorrhage following delivery is the leading reason for an obstetric admission to the intensive care unit (ICU), and it is responsible for one third of all pregnancy-related deaths in both high- and low-income countries.[1] Therefore it is critical for the obstetrician to have a thorough understanding of the hemodynamic changes that accompany pregnancy, the maternal adaptations that occur with excessive blood loss, and the management principles for obstetric hemorrhage.

PREGNANCY-RELATED HEMODYNAMIC CHANGES

Pregnancy is associated with five significant hemodynamic changes (see Chapter 3). The first of these changes is **plasma volume expansion.** The average singleton pregnancy is accompanied by a 40% to 50% increase in plasma volume by the thirtieth week of gestation. This increase in plasma volume occurs along with the second change, an **increase in red blood cell (RBC) mass**. With appropriate substrate availability, RBC mass can be expected to increase 20% to 30% by the end of pregnancy. Third, **maternal cardiac output rises** with normal pregnancy owing to both increased stroke volume and increased heart rate. According to consensus, the average rise in cardiac output is 30% to 50% above nonpregnant levels, and the peak occurs in the early third trimester. Fourth, **systemic vascular resistance falls** in parallel with this rise in cardiac output and blood volume expansion. Fifth, **fibrinogen and the majority of procoagulant blood factors (II, VII, VIII, IX, and X) increase** during pregnancy. These five changes are protective of maternal hemodynamic status and thus allow for certain physiologic adaptations that accompany obstetric hemorrhage.

PHYSIOLOGIC ADAPTATION TO HEMORRHAGE

During pregnancy and the puerperium, a **defined sequence of physiologic adaptations occurs with hemorrhage** (Fig. 18-1). When 10% of the circulatory blood volume is lost, vasoconstriction occurs in both the arterial and venous compartments in order to maintain blood pressure and to preserve blood flow to essential organs. As blood loss reaches 20% or more of the total blood volume, increases in systemic vascular resistance can no longer compensate for the lost intravascular volume, and blood pressure decreases with a commensurate rise in heart rate. Cardiac output falls in parallel because of a loss in preload that results in poor end-organ perfusion. If the intravascular volume is not appropriately replaced, shock will ensue.

In severe preeclampsia (PE), these physiologic adaptations are altered. Unlike in most pregnant women, the protective mechanism of blood volume expansion is diminished with severe PE. It is estimated that plasma volume expansion is 9% lower in the setting of PE. In addition, because of the significant vasoconstriction that accompanies PE, blood loss in these patients may be underestimated because blood pressure is often maintained in the normotensive range. Finally, oliguria may not be as reliable an indicator of poor end-organ perfusion secondary to hemorrhage because reduced urine output is often a manifestation of the severity of PE.

CLASSIFICATION OF HEMORRHAGE

A standard classification for acute blood loss is illustrated in Table 18-1. **Understanding the physiologic responses that accompany varying degrees of volume deficit can assist the clinician when caring for hemorrhaging patients.**

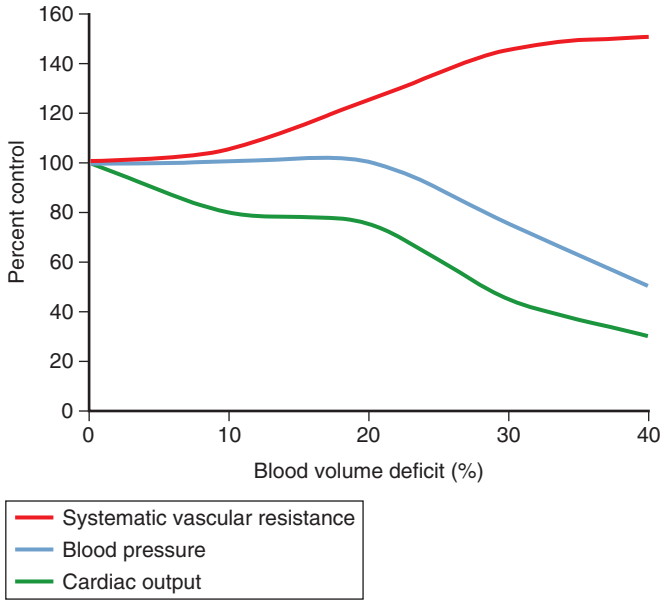

FIG 18-1 Relationships among systemic vascular resistance, blood pressure, and cardiac output in the face of progressive blood volume deficit.

TABLE 18-1 HEMORRHAGE CLASSIFICATION AND PHYSIOLOGIC RESPONSE

CLASS	ACUTE BLOOD LOSS (mL)	% LOST	PHYSIOLOGIC RESPONSE
1	1000	15	Dizziness, palpitations, minimal blood pressure change
2	1500	20-25	Tachycardia, tachypnea, sweating, weakness, narrowed pulse pressure
3	2000	30-35	Significant tachycardia and tachypnea, restlessness, pallor, cool extremities
4	≥2500	40	Shock, air hunger, oliguria or anuria

Modified from Baker RJ. Evaluation and management of critically ill patients. *Obstet Gynecol Annu.* 1977;6:295; and Bonnar J. Massive obstetric haemorrhage. *Baillieres Best Pract Res Clin Obstet Gynaecol.* 2000;14:1.

Determination of the hemorrhage class reflects the volume deficit, which may not be the same as the volume loss. **The average 70-kg pregnant woman maintains a blood volume of 6000 mL by 30 weeks of gestation (85 mL/kg).**

***Class 1 hemorrhage* corresponds to approximately 1000 mL of blood loss.** This blood loss correlates to a **15% volume deficit.** Women with this amount of volume deficit exhibit mild physiologic changes such as dizziness and palpitations owing to the hemodynamic adaptations that accompany normal pregnancy.

***Class 2 hemorrhage* is characterized by 1500 mL of blood loss, or a 20% to 25% volume deficit.** Early physical changes that occur during a hemorrhage of this class include **tachycardia and tachypnea**. Although tachycardia is usually recognized as a compensatory mechanism to increase cardiac output, the significance of tachypnea is unclear and is often unappreciated clinically. Tachypnea can represent a sign of impending clinical decompensation. **Narrowing of the pulse pressure** is another sign of a class 2 hemorrhage. The pulse pressure represents the difference between the systolic and diastolic blood pressures. Systolic blood pressure is a good representation of stroke volume and β_1 stimulation. Diastolic blood pressure is a reflection of systemic vasoconstriction; therefore the pulse pressure represents the interrelationship between these entities. With a class 2 volume deficit, the sympathoadrenal system is activated, which results in a diversion of blood away from nonvital organs (skin, muscle, and kidney) and a redistribution of the circulation to vital body organs, including the brain and heart. The end result is increased vasoconstriction, increased diastolic blood pressure, maintenance of systolic blood pressure, and a narrowing of the pulse pressure. With greater narrowing of the pulse pressure, more compensatory vasoconstriction occurs to accommodate for a loss in stroke volume. A final physiologic response of class 2 hemorrhage is **orthostatic hypotension.** Although blood pressure comparisons can be made in the supine, sitting, and standing positions to document this response, a practical approach is to assess the time needed to refill a blanched hypothenar area on the patient's hand. Typically, a patient with normal volume status can reperfuse this area within 1 to 2 seconds after pressure is applied. A patient with a class 2 hemorrhage and orthostatic hypotension will have significant reperfusion delay.

***Class 3 hemorrhage* is defined as a blood loss of 2000 mL and corresponds to a volume deficit of 30% to 35%.** Within this hemorrhage class, the physiologic responses noted in class 2 hemorrhage are exaggerated. Patients demonstrate significant **tachycardia** (120 to 160 beats/min), **tachypnea** (30 to 50 breaths/min), **overt hypotension, restlessness, pallor, and cool extremities.**

***Class 4 hemorrhage* is characterized by more than 2500 mL of blood loss.** This amount of blood loss exceeds **40% of the patient's total blood volume.** The clinical manifestations of this volume deficit include **absent distal pulses, shock, air hunger, and oliguria or anuria.** When significant hemorrhage occurs, renal blood flow is reduced and is redirected from the outer renal cortex to the juxtamedullary region. In this region, increased water and sodium absorption occur and result in decreased urine volume, lower urinary sodium concentration, and increased urine osmolarity. A urine sodium concentration less than 10 to 20 mEq/L or a urine/serum osmolar ratio greater than 2 indicates significantly reduced renal perfusion in the face of hemorrhage.

ANTEPARTUM HEMORRHAGE

Placental Abruption

Definition and Pathogenesis

***Placental abruption,* or *abruptio placentae,* refers to the premature separation of a normally implanted placenta from the uterus prior to delivery of the fetus.** The diagnosis is typically reserved for pregnancies at greater than 20 weeks of gestation. Abruption is characterized by defective maternal vessels in the decidua basalis, which rupture and cause the separation. On rare occasions, the separation may be caused by a disruption of the fetal-placental vessels. These damaged vessels cause bleeding, which results in a decidual hematoma that may promote placental separation, destruction of placental tissue, and a loss of maternal-fetal surface area for nutrient and gas exchange.

Whereas some placental abruptions may occur acutely after a sudden mechanical event (e.g., blunt trauma, sudden uterine decompression, or motor vehicle accident), most

cases result from more chronic processes.[2] Abnormal development of the spiral arteries can lead to decidual necrosis, inflammation, infarction, and bleeding due to vascular disruption.[3-6] Thrombin, which is released in response to decidual hemorrhage or hypoxia, appears to play an active role in the pathogenesis of placental abruption. Thrombin acts as a direct uterotonic, enhances the action of matrix metalloproteinases, upregulates apoptosis genes, increases the expression of inflammatory cytokines, triggers the coagulation cascade, and initiates functional progesterone withdrawal.[7-9] These thrombin-mediated events initiate a cyclic pathway of vascular disruption, hemorrhage, inflammation, contractions, and rupture of membranes.[5]

Incidence

The overall incidence of placental abruption is approximately 1 in 100 births; however, a range of 1 in 80 to 1 in 250 deliveries has been reported.[10,11] The range in incidence likely reflects variable criteria for diagnosis as well as an increased recognition in recent years of milder forms of abruption. **About one third of all antepartum bleeding can be attributed to placental abruption, which peaks in the third trimester; 40% to 60% of abruptions occur prior to 37 weeks of gestation.**[10]

Clinical Manifestations

Several factors determine the clinical manifestations of placental abruption. These factors include (1) the **temporal nature of the abruption** (acute vs. chronic), (2) **clinical presentation** (overt vs. concealed), and (3) **severity.** An acute, overt abruption typically presents with vaginal bleeding, abdominal pain, and uterine contractions. As the placental separation worsens, uterine tenderness, tachysystole, fetal heart rate (FHR) patterns consistent with hypoxia, and fetal death may occur. **The amount of vaginal bleeding correlates poorly with the extent of placental separation and its potential for fetal compromise. In fact, concealed abruption occurs in 10% to 20% of cases.**[12] With severe abruptions, more than 50% of the placental surface area separates. With extensive abruption, a significant risk for fetal death exists, and maternal compromise in the form of consumptive coagulopathy may result from the triggering of the clotting cascade by hemorrhage and extensive thrombin deposition.

Chronic abruption may be insidious in its presentation and is often associated with ischemic placental disease.[13] Typically, these cases present with intermittent, light vaginal bleeding and evidence of chronic placental inflammation and dysfunction, such as oligohydramnios, fetal growth restriction, preterm labor, premature preterm rupture of membranes (PPROM), and PE.

Risk Factors

Although the exact etiology of placental abruption is unclear, a variety of risk factors have been identified (Box 18-1).

INCREASING PARITY AND MATERNAL AGE

Several studies have noted a **higher incidence of placental abruption with increasing parity**. Among primigravid women, the frequency of placental abruption is less than 1%; however, 2.5% of grand multiparas experience placental abruption. Theories suggest that damaged endometrium, impaired decidualization, and aberrant vasculature may have causal roles with increasing parity or age.

Maternal age is often cited as an associated risk factor for placental abruption. Although a 15-year population-based study in Norway was able to demonstrate a strong relationship between maternal age and placental abruption for all levels of parity, others studies suggest that there is no increased risk for placental abruption among older women when parity and hypertensive disease are excluded.

BOX 18-1 RISK FACTORS FOR PLACENTAL ABRUPTION

- Increasing parity and maternal age
- Maternal substance use
  - Cigarette smoking
  - Cocaine abuse
- Trauma
- Maternal diseases
  - Hypertension
  - Hypothyroidism
  - Asthma
- Preterm premature rupture of membranes
- Rapid uterine decompression associated with multiple gestation and polyhydramnios
- Uterine and placental factors
  - Anomalies
  - Synechiae
  - Fibroids
  - Cesarean scar
  - Abnormal placental formation
  - Chronic ischemia
- Prior abruption
- Hyperhomocysteinemia

MATERNAL SUBSTANCE ABUSE

Cigarette smoking is associated with a significantly increased incidence of placental abruption and fetal death. There appears to be a dose-response relationship with the number of cigarettes smoked and the risks for placental abruption and fetal loss. Compared with nonsmokers, smokers have a 40% increased risk for fetal death from placental abruption with each pack of cigarettes smoked. In addition, smoking and hypertensive disease appear to have an additive effect on the likelihood of placental abruption. Proposed etiologies include placental hypoperfusion with resulting decidual ischemia and necrosis.

Cocaine abuse in the third trimester has been associated with as high as a 10% placental abruption rate. The pathogenesis appears to be related to cocaine-induced vasospasm with subsequent decidual ischemia, reflex vasodilation, and vascular disruption within the placental bed.

TRAUMA

Blunt or penetrating trauma to the gravid abdomen has been associated with placental abruption. After a **minor trauma,** the risk for placental abruption is **between 7% and 9%,** whereas the risk may be **as high as 13%** after severe injury.[14] The two most common causes of maternal trauma are motor vehicle crashes and domestic abuse. With motor vehicle crashes, uterine stretch, direct penetration, and placental shearing from acceleration-deceleration forces are the primary etiologies of trauma-related placental abruption (see Chapter 26).

MATERNAL DISEASES

Maternal hypertension has been the most consistently identified risk factor for placental abruption.[13] This relationship has been observed with both chronic and pregnancy-related hypertensive disease. Compared with normotensive women, hypertensive women have a fivefold increased risk for placental

abruption. Unfortunately, antihypertensive therapy has not been shown to reduce the risk for placental abruption in women with chronic hypertension.

Maternal subclinical hypothyroidism and asthma have also been associated with placental abruption in some studies.[15,16]

PRETERM PREMATURE RUPTURE OF MEMBRANES

Placental abruption occurs in 2% to 5% of pregnancies with PPROM. Intrauterine infection and oligohydramnios significantly increase the risk for placental abruption, and nonreassuring FHR patterns occur in nearly half of these pregnancies.

It is unclear whether placental abruption is the cause or consequence of PPROM. Hemorrhage and associated thrombin generation may stimulate cytokine and protease production, which results in membrane rupture. Alternatively, the cytokine-protease cascade that follows ruptured membranes may cause damage to the decidual vasculature, which predisposes the placenta to separation.

RAPID UTERINE DECOMPRESSION ASSOCIATED WITH MULTIPLE GESTATIONS AND POLYHYDRAMNIOS

Rapid decompression of an overdistended uterus can precipitate an acute placental abruption. This may occur in the setting of multiple gestations or with polyhydramnios. Compared with singletons, twins have been reported to have nearly a threefold increased risk for placental abruption. Although the exact timing of placental abruption in multiple gestations is difficult to ascertain, it has been attributed to rapid decompression of the uterus after the delivery of the first twin. Likewise, rapid loss of amniotic fluid in pregnancies complicated by polyhydramnios has been implicated in placental abruption. This can occur with spontaneous rupture of membranes or may follow therapeutic amniocentesis. For this reason, controlled artificial rupture of membranes with induction of labor may be advisable if significant polyhydramnios complicates pregnancy.

UTERINE AND PLACENTAL FACTORS

Suboptimal placental implantation in patients with uterine anomalies, synechiae, fibroids, and cesarean scars is associated with abruption.[17] In addition, abnormal placental formation (e.g., circumvallate placenta) or chronic ischemia associated with PE and fetal growth restriction have been implicated in placental abruption.[18]

PRIOR ABRUPTION

Women who have had a previous abruption are at significant risk for recurrent abruption. **After one abruption, the recurrence risk is 5% to 15%, whereas the risk increases to 20% to 25% after two abruptions.**[19] **The risk of recurrence is greater after a severe abruption. When an abruption is associated with fetal demise, there is a 7% incidence of the same outcome in a future gestation.**

THROMBOPHILIA

Inconsistent data exist regarding an association among thrombophilias and placental abruptions.[20,21] Hyperhomocysteinemia (a fasting homocysteine level >15 μmol/L) may be associated with recurrent abruption.

Diagnosis

Placental abruption is primarily a clinical diagnosis that is supported by radiographic, laboratory, and pathologic studies. Any findings of vaginal bleeding, uterine contractions, abdominal and/or back pain, or trauma should prompt an investigation for potential placental abruption. Vaginal bleeding may range from mild to severe. Unfortunately, bleeding may be underestimated because it can be retained behind the placenta. The typical abruption contraction pattern is high frequency and low amplitude; however, it may simulate labor in some circumstances.

RADIOLOGY

Although early studies that evaluated the use of **ultrasound** for the diagnosis of placental abruption identified less than 2% of cases, recent advances in imaging and its interpretation have improved detection rates. Early hemorrhage is typically hyperechoic or isoechoic, whereas resolving hematomas are hypoechoic within 1 week and sonolucent within 2 weeks of the abruption. Acute hemorrhage may be misinterpreted as a homogeneous thickened placenta or fibroid.

Ultrasound can identify **three predominant locations for placental abruption.** These are **subchorionic** (between the placenta and the membranes), **retroplacental** (between the placenta and the myometrium), and **preplacental** (between the placenta and the amniotic fluid). Figure 18-2 illustrates the classification of hematomas in relation to the placenta. Figure 18-3 demonstrates a sonographic representation of a subchorionic abruption.

The location and extent of the placental abruption identified on ultrasound examination is of clinical significance. **Retroplacental hematomas are associated with a worse prognosis for fetal survival than subchorionic hemorrhage.** The size of the hemorrhage is also predictive of fetal survival. Large retroplacental hemorrhages (>60 mL) have been associated with a 50% or greater fetal mortality, whereas similarly sized subchorionic hemorrhages are associated with a 10% mortality risk.[22]

Magnetic resonance imaging (MRI) has been used occasionally for the diagnosis of placental abruption when sonography is equivocal.[23,24]

LABORATORY FINDINGS

Few laboratory studies assist in the diagnosis of placental abruption. **Hypofibrinogenemia and evidence of consumptive coagulopathy may accompany severe abruption**; however, clinical correlation is necessary. Moreover, most abruptions are not accompanied by maternal coagulopathy.

Abnormal serum markers early in pregnancy, such as an unexplained elevated maternal serum α-fetoprotein (MSAFP) or human chorionic gonadotropin (hCG) and decreased pregnancy-associated plasma protein A (PAPP-A) or estriol, have been associated with an increased risk for subsequent placental abruption.[18,25]

PATHOLOGIC STUDIES

Macroscopic inspection of the placenta may demonstrate adherent clot and depression of the placental surface. Fresh or acute placental abruptions may not have any identifiable evidence on gross pathologic examination, but histologic analysis may show preservation of the villous stroma, eosinophilic degeneration of the syncytiotrophoblast, and scattered neutrophils with villous agglutination.[5] Chronic abruptions may demonstrate histologic signs of chronic deciduitis, maternal floor decidual necrosis, villitis, decidual vasculopathy, infarction,

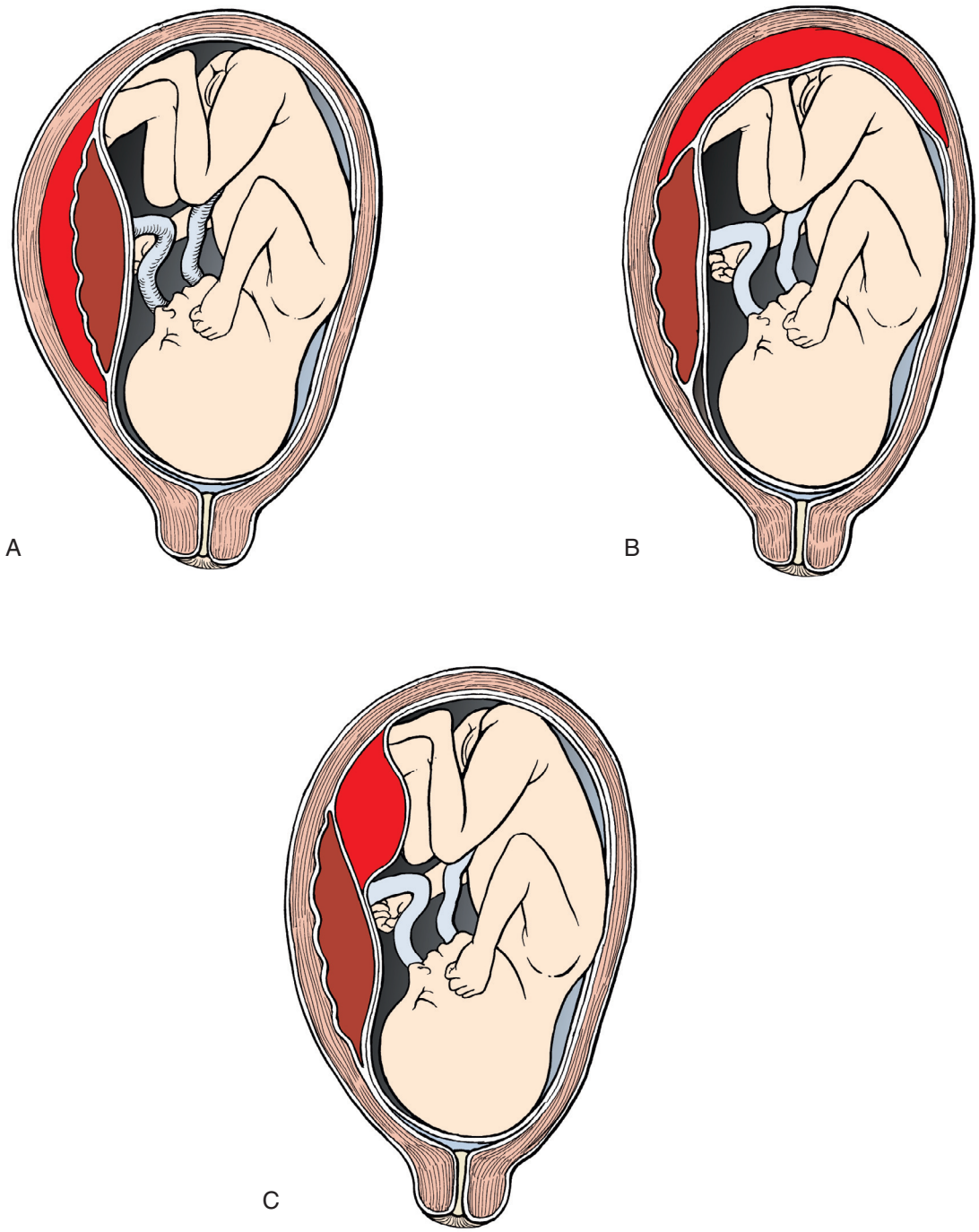

FIG 18-2 The classification system of placental abruption. **A,** Retroplacental abruption. The bright red area represents a blood collection behind the placenta (*dark red*). **B,** Subchorionic abruption. The bright red area represents subchorionic bleeding, which is observed to dissect along the chorion. **C,** Preplacental abruption. The bright red area represents a blood collection anterior to the placenta within the amnion and chorion (subamniotic). (From Trop I, Levine D. Hemorrhage during pregnancy: sonography and MR imaging. *AJR Am J Roentgenol.* 2001;176:607.)

intervillous thrombosis, villous maldevelopment, and hemosiderin deposition.[9]

Management

Both maternal and fetal complications may occur with placental abruption. Maternal complications include blood loss, consumptive coagulopathy, need for transfusion, end-organ damage, cesarean delivery, and death. Fetal complications include intrauterine growth restriction (IUGR), oligohydramnios, prematurity, hypoxemia, and stillbirth. Although maternal complications are related to the severity of the abruption, fetal complications are related to both the severity and timing of the hemorrhage.[12]

Despite its relative frequency, no randomized trials and few studies have examined management approaches for placental abruption.[26] **Typically, management of placental abruption depends on the severity, gestational age, and maternal-fetal status.** Once the diagnosis of placental abruption has been

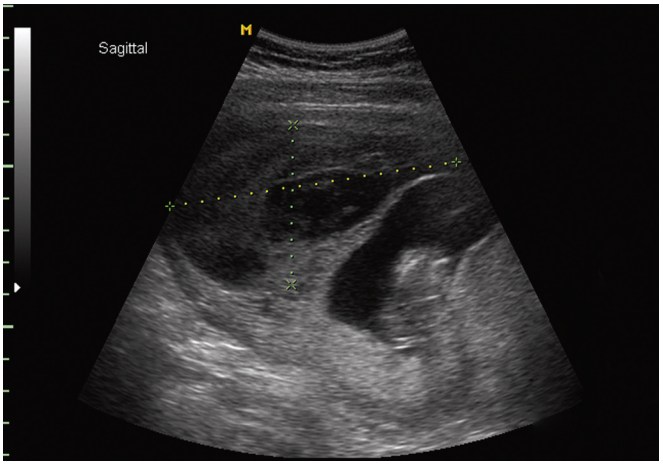

FIG 18-3 Ultrasonic image of a subchorionic abruption. (Courtesy K. Francois.)

made, precautions should be taken to anticipate the possible life-threatening consequences for both mother and fetus. These precautions include baseline **laboratory assessment** (hemoglobin, hematocrit, platelet count, type and screen, fibrinogen, and coagulation studies), **appropriate intravenous (IV) access** (large-bore catheter), **availability of blood products, continuous FHR and contraction monitoring, and communication with operating room (OR) and neonatal personnel.**

Small placental abruptions remote from term (<34 weeks) may be managed expectantly. With cases of chronic abruption, clinical circumstances that include gestational age and the extent of the abruption influence the need for prolonged hospitalization until delivery. In many cases, a cyclic event of bleeding, thrombin generation, contractions, and further placental separation occurs. Tocolysis may help prevent contractions and thus may break the abruption cycle. If the maternal-fetal status is stable, a trial of tocolysis for documented preterm labor and administration of antenatal corticosteroid therapy can be considered. Whereas the choice of tocolysis should be individualized, magnesium sulfate administration may confer an added benefit of fetal neuroprotection. Reported series of expectant management in preterm gestations with placental abruption have shown a significant prolongation of the pregnancy (>1 week) in more than 50% of patients without adverse maternal or fetal outcomes. In a large series of preterm patients who presented with placental abruption and received tocolysis, about one third delivered within 48 hours of admission, one third delivered within 7 days, and one third delivered more than 1 week from initial presentation. No cases of intrauterine demise were reported in women who presented with a live fetus. Although these results are encouraging, the clinician must always keep in mind that placental abruption can result in both maternal and fetal morbidity. Any attempt to arrest preterm labor in a known or suspected placental abruption must be weighed against the likelihood of neonatal survival and morbidity, the severity of the abruption, and the safety of the mother.

Women who present at or near term with a placental abruption should undergo delivery. Induction or augmentation of labor is not contraindicated in the setting of an abruption; however, close surveillance for any evidence of maternal or fetal compromise is advised. Continuous FHR monitoring is recommended because 60% of fetuses may exhibit intrapartum heart rate patterns consistent with hypoxia. Intrauterine pressure catheter (IUPC) placement and internal FHR monitoring can assist the clinician during the intrapartum course. IUPC monitoring may demonstrate elevated uterine resting tone, which can be associated with fetal hypoxia. Maternal hemodynamic and clotting parameters must be followed closely to detect signs of evolving coagulopathy. **Although vaginal delivery is generally preferable, operative delivery is often necessary owing to fetal or maternal decompensation.** When cesarean delivery is required, a rapid decision-to-delivery time is optimal because an interval of less than 20 minutes from the onset of fetal bradycardia is associated with improved outcomes. A *Couvelaire uterus,* also known as *uteroplacental apoplexy,* is characterized by extravasation of blood into the myometrium; it may be noted in some cases and is often associated with significant uterine atony. Administration of uterotonic therapy usually improves the condition. Hysterectomy should be reserved for cases of atony and hemorrhage unresponsive to conventional uterotonic therapies and replacement of blood products.

The management of women with consumptive coagulopathy and fetal demise requires a thorough knowledge of the natural history of severe placental abruption. Nearly five decades ago, Pritchard and Brekken noted several clinically important observations: (1) about 40% of patients with placental abruption and fetal demise will demonstrate signs of consumptive coagulopathy; (2) within 8 hours of initial symptoms, hypofibrinogenemia will be present; (3) severe hypofibrinogenemia will not recover without blood product replacement; and (4) the time course for recovery from hypofibrinogenemia is roughly 10 mg/dL per hour after delivery of the fetus and placenta.

When managing women with severe placental abruptions and fetal demise, maintenance of maternal volume status and replacement of blood products is essential. Although operative delivery may appear to lead to the most rapid resolution of the problem, it may pose significant risks to the patient. Unless the consumptive coagulopathy is corrected, surgery can result in uncontrollable bleeding and an increased need for hysterectomy. The uterus does not need to be evacuated before coagulation status can be restored. Blood product replacement and delayed delivery until hematologic parameters have improved are generally associated with good maternal outcomes.

Neonatal Outcome

Placental abruption is associated with increased perinatal morbidity and mortality. When compared with normal pregnancies, pregnancies complicated by abruption have a **tenfold increased risk for perinatal death.** A case-control study has also shown a **greater risk for adverse long-term neurobehavioral outcomes** in infants delivered after placental abruption. Neonates at risk for abnormal outcomes had higher incidences of abnormal FHR tracings (45%) and emergency cesarean deliveries (53%) compared with controls (10% and 10%, respectively). **Finally, hypoxia-associated periventricular leukomalacia and sudden infant death syndrome (SIDS) are more common in newborns delivered after placental abruptions.**

Placenta Previa

Definition and Pathogenesis

***Placenta previa* is defined as the presence of placental tissue over or adjacent to the cervical os.** Traditionally, four variations of placenta previa were recognized: 1) complete, 2) partial,

3) marginal, and 4) low lying.[27] Although *complete placenta previa* has been the term used to refer to the total coverage of the internal cervical os by placental tissue, the differences among the terms *partial* (placental edge partially covering the internal cervical os), *marginal* (placental edge at the margin of the internal cervical os), and *low lying* (placental edge within 2 cm of the interval cervical os) were often subtle and varied by the timing and method of diagnosis. Improved ultrasound technology and precision have allowed for more accurate assessments of the placental location in relation to the cervical os. **Recent revised classification of placenta previa consists of two variations: true *placenta previa,* in which the internal cervical os is covered by placental tissue, and *low-lying placenta,* in which the placenta lies within 2 cm of the cervical os but does not cover it.**[27] Although not a true placenta previa, low-lying placentas are associated with increased risks for bleeding and other adverse pregnancy events.[28]

Incidence

The overall reported incidence of placenta previa at delivery is 1 in 200 births. In the second trimester, placenta previa may occur in up to 6% of pregnancies.[29] **The term *placental migration* has been used to explain this "resolution" of placenta previa that is noted near term. Three theories have been suggested to account for this phenomenon.** The first hypothesis proposes that as the pregnancy advances, the stationary lower placental edge relocates away from the cervical os with the development of the lower uterine segment. Indeed, the lower uterine segment has been noted to increase from 0.5 cm at 20 weeks to more than 5 cm at term. Secondly, the placenta-free uterine wall has been proposed to grow at a faster rate than the uterine wall covered by the placenta. A final hypothesis suggests that *trophotropism,* the growth of trophoblastic tissue away from the cervical os toward the fundus, results in resolution of the placenta previa.[30]

Clinical Manifestations

Placenta previa typically presents as painless vaginal bleeding in the second or third trimester. The bleeding is believed to occur from disruption of placental blood vessels in association with the development and thinning out of the lower uterine segment. **Between 70% and 80% of patients with placenta previa will have at least one bleeding episode.** About 10% to 20% of patients present with uterine contractions before bleeding, and fewer than 10% remain asymptomatic until term. Of those with bleeding, **one third of women will present before 30 weeks of gestation, one third between 30 and 36 weeks, and one third after 36 weeks.** Early-onset bleeding (<30 weeks) carries with it the greatest risk for blood transfusion and associated perinatal morbidity and mortality.

Risk Factors

Several risk factors for placenta previa have been noted (Box 18-2). Additionally, some reports have documented a higher association of fetal malpresentation, preterm labor, PPROM, IUGR, congenital anomalies, and amniotic fluid embolism with placenta previa.[31]

INTRINSIC MATERNAL FACTORS

Studies have reported **more cases of placenta previa with increasing parity**. Grand multiparas have been reported to have a 5% risk for placenta previa compared with 0.2% among nulliparous women. **Maternal age also seems to influence the occurrence of placenta previa.** Women older than 35 years of age have more than a fourfold increased risk for placenta previa, and women older than 40 years of age have a ninefold greater risk. Finally, **maternal race has been associated with placenta previa.** In a large population-based cohort,[32] the rate of placenta previa among white, black, and other races was 3.3, 3, and 4.5 per 1000 births, respectively. Asian women appear to have the highest rates of placenta previa.

BOX 18-2 RISK FACTORS FOR PLACENTA PREVIA

Intrinsic maternal factors
- Increasing parity
- Advanced maternal age
- Maternal race

Extrinsic maternal factors
- Cigarette smoking
- Cocaine use
- Residence at higher elevation
- Infertility treatments

Fetal factors
- Multiple gestations
- Male fetus

Prior placenta previa

Prior uterine surgery and cesarean delivery

EXTRINSIC MATERNAL FACTORS

Cigarette smoking has been associated with as high as a threefold increased risk for previa formation. Likewise, a case-control study has demonstrated that **maternal cocaine use increases the risk of placenta previa fourfold.** Residence at higher elevations may also contribute to previa development. The need for increased placental surface area secondary to decreased uteroplacental oxygenation may play a role in this association. Finally, prior **infertility treatment is statistically associated with higher rates of placenta previa.**[33]

FETAL FACTORS

Controversy exists regarding an increased risk for placenta previa with multiple gestations. Although some studies have shown a higher incidence of placenta previa among twins, others have not documented a significantly increased risk.[34] A consistently **higher proportion of offspring in women with placenta previa are male.** This association is unexplained; however, two theories suggest larger placental sizes among male fetuses and delayed implantation of the male blastocyst in the lower uterine segment.

PRIOR PLACENTA PREVIA

Having had a prior placenta previa increases the risk for the development of another previa in a subsequent pregnancy. This association has been reported to be as high as an eightfold relative risk. The exact etiology for this increased risk is unclear.

PRIOR UTERINE SURGERY AND PRIOR CESAREAN DELIVERY

Prior uterine surgery has been associated with placenta previa formation. Although a history of curettage and/or myomectomy attends a slightly elevated previa risk, prior cesarean delivery has been the most consistent risk factor. **In the pregnancy following a cesarean delivery, the risk for placenta previa has been reported to range from 1% to 4%.**[35,36] A **linear increase is seen in placenta previa risk with the number**

of prior cesarean deliveries. Placenta previa occurs in 0.9% of women with one prior cesarean delivery, in 1.7% of women with two prior cesarean deliveries, and in 3% of those with three or more cesarean deliveries.[37] In patients with four or more cesarean deliveries, the risk for placenta previa has been reported to be as high as 10%.[35] Endometrial scarring is thought to be the etiologic factor for this increased risk.

Diagnosis

The timing of the diagnosis of placenta previa has undergone significant change in the past four decades. Painless third-trimester bleeding was a common presentation for placenta previa in the past, whereas **most cases of placenta previa are now detected antenatally with ultrasound** prior to the onset of significant bleeding.

RADIOLOGY

Transabdominal and transvaginal ultrasound provide the best means for diagnosing placenta previa. Although transabdominal ultrasound can detect at least 95% of placenta previa cases, transvaginal ultrasound has a reported diagnostic accuracy that approaches 100%. Typically, a combined approach can be used in which transabdominal ultrasound is the initial diagnostic modality, followed by transvaginal ultrasound for uncertain cases. Transvaginal ultrasound is safe and is not contraindicated in these circumstances. Of note, quality images can be obtained using transvaginal ultrasound without the probe contacting the cervix (Fig. 18-4).

If a placenta previa or low-lying placenta is diagnosed in the second trimester, repeat sonography should be obtained in the early third trimester at 32 weeks.[27] More than 90% of the cases of placenta previa diagnosed in the second trimester resolve by term. The potential for placenta previa resolution is dependent on the timing of the diagnosis, extension over the cervical os, and placental location. For example, one study of 714 women with an ultrasound diagnosis of placenta previa noted that the earlier the diagnosis, the more likely the previa would resolve by term (Table 18-2). In addition, complete placenta previa diagnosed in the second trimester will persist into the third trimester in 26% of cases, whereas a low-lying placenta will persist in only 2.5% of cases. Finally, anterior placenta previa is less likely to migrate away from the cervical os than posterior placement.

Occasionally, MRI may be used to diagnose placenta previa. MRI is particularly helpful with posterior placenta previa identification and assessment of invasive placentation (see below).

Management

General management principles for patients with placenta previa in the third trimester include serial ultrasounds to assess placental location and fetal growth, avoidance of cervical examinations and intercourse, activity restrictions, counseling regarding labor symptoms and vaginal bleeding, dietary and nutrient supplementation to avoid maternal anemia, and early medical attention if any vaginal bleeding occurs.

ASYMPTOMATIC PLACENTA PREVIA

A recent working group has given specific recommendations for management of asymptomatic placenta previa at varying gestational ages.[27] For pregnancies at greater than 16 weeks of gestation with a low-lying placenta (placental edge within 2 cm from the internal cervical os) or a placenta previa, repeat ultrasound to assess placental location is recommended at 32 weeks. If a low-lying placenta or placenta previa persists at 32 weeks, repeat sonography is again obtained at 36 weeks.

Asymptomatic women with placenta previa may be managed expectantly as outpatients. Although some sonographic features have been associated with a higher likelihood of bleeding—such as complete placental coverage of the cervical os, a thickened placental edge, echo-free placental space over the cervical os, and cervical length less than 3 cm—it is not possible to predict all cases of bleeding that result from placenta previa.[30] With this in mind, asymptomatic patients should be instructed

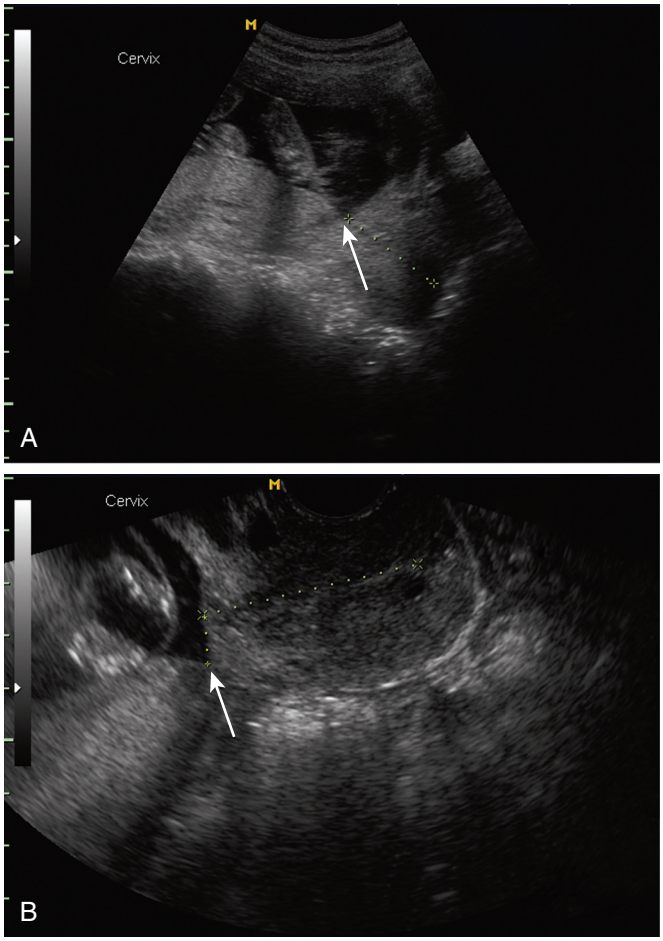

FIG 18-4 Transabdominal and transvaginal ultrasounds of low-lying placenta. Arrows identify the placental edge. (Courtesy K. Francois.)

TABLE 18-2 POTENTIAL FOR PLACENTA PREVIA AT TERM BY GESTATIONAL AGE AT DIAGNOSIS

GESTATIONAL AGE AT DIAGNOSIS (WK)	PREVIA AT TERM (%)
15-19	12
20-23	34
24-27	49
28-31	62
32-35	73

From Dashe JS, McIntire DD, Ramus RM, et al. Persistence of placenta previa according to gestational age at ultrasound detection. *Obstet Gynecol.* 2002;99:692.

to avoid activities that may stimulate uterine contractions and/or cervical irritation, such as strenuous exercise, intercourse, and digital cervical examinations. Several studies have documented the safety, efficacy, and cost savings of outpatient management for asymptomatic placenta previa. **Candidates for outpatient management must (1) be compliant, (2) live within a short commute from the hospital, (3) have 24-hour emergency transportation to the hospital, and (4) verbalize a thorough understanding of the risks associated with placenta previa.**

BLEEDING PLACENTA PREVIA

Women with placenta previa who present with acute vaginal bleeding require hospitalization and immediate evaluation to assess maternal-fetal stability. They should initially be managed in a labor and delivery unit with hemodynamic surveillance of the mother and continuous FHR monitoring. Large-bore IV access and baseline laboratory studies (hemoglobin, hematocrit, platelet count, blood type and screen, and coagulation studies) should be obtained. **If the pregnancy is less than 34 weeks of gestation, administration of antenatal corticosteroids should be undertaken** as well as an assessment of the facility's emergency resources for both the mother and the neonate. In some cases, maternal transport and consultation with a maternal-fetal medicine specialist and a neonatologist may be warranted. **Finally, tocolysis may be used if the vaginal bleeding is preceded by or associated with uterine contractions.** Whereas various agents have been used, magnesium sulfate is often preferred as a first-line agent because of its limited potential for hemodynamic-related maternal side effects and its added benefit for fetal neuroprotection.[30]

Once stabilized, most women with symptomatic placenta previa can be maintained on hospitalized bed rest and expectantly managed. In several observational studies, 50% of women with bleeding placenta previa were undelivered in 4 weeks, including those with initial bleeding episodes of more than 500 mL. Minimizing maternal anemia by using blood conservation techniques is recommended. Although some patients may require transfusion, many patients can be supplemented with iron replacement (oral or IV), vitamin C to enhance oral iron absorption, and B vitamins. Erythropoietin may be used in selected cases to hasten red cell formation. Lastly, autologous donation may be considered in patients with hemoglobin concentrations greater than 11 g/dL.[38,39]

Although maternal hemorrhage is of the utmost concern, **fetal blood can also be lost during the process of placental separation with a bleeding placenta previa. Rh0(D) immune globulin should be given to all Rh-negative unsensitized women with third-trimester bleeding from placenta previa.** A Kleihauer-Betke preparation of maternal blood should be considered. Occasionally, a fetomaternal hemorrhage of greater than 30 mL occurs that necessitates additional doses of Rh0(D) immune globulin. One study noted that 35% of infants whose mothers received an antepartum transfusion were also anemic and required a transfusion following delivery.

DELIVERY

Cesarean delivery is indicated for all women with sonographic evidence of placenta previa and most women with low-lying placenta. When the placental distance is between 1 and 20 mm from the internal cervical os, the rate of cesarean delivery ranges from 40% to 90%.[40] If a vaginal trial of labor is attempted for a low-lying placenta, precautions should be taken for the possibility of an emergent cesarean delivery and need for blood transfusion.

A consensus panel has given delivery-timing guidelines for uncomplicated placenta previa,[22] which includes cases with normal fetal growth and no other pregnancy-associated complications. **Cesarean delivery of asymptomatic placenta previa should occur between $36^{0/7}$ and $37^{0/7}$ weeks of gestation. In cases of complicated placenta previa, delivery should occur immediately regardless of gestational age. Complicated placenta previa includes bleeding associated with a nonreassuring fetal heart pattern despite resuscitative measures, life-threatening maternal hemorrhage, and/or refractory labor.**[30]

When performing a cesarean delivery for placenta previa, the surgeon should be aware of the potential for rapid blood loss during the delivery process. Blood products that are crossmatched should be readily available for delivery. In addition, before incising the lower uterine segment, the surgeon should assess the vascularity of this region. Although a low transverse incision is not contraindicated in patients with placenta previa, performing a vertical uterine incision may be preferable in some cases. This is particularly true with an anterior placenta previa. Ideally, the placenta should not be disrupted when entering the uterus. If disruption occurs, expedited delivery is essential. Given the potential for invasive placentation, the physician should allow the placenta to spontaneously deliver. If it does not separate easily, precautions should be taken for placenta accreta management (see below). Once the placenta separates, bleeding is controlled by the contraction of uterine myometrial fibers around the spiral arterioles. Because the lower uterine segment often contracts poorly, significant bleeding may occur from the placental implantation site. Aggressive uterotonic therapy, surgical intervention, and/or tamponade techniques should be undertaken to rapidly control bleeding. Finally, some studies have shown reduced bleeding at the placental site with the injection of subendometrial vasopressin after delivery of the fetus.[41]

Associated Conditions

Placenta Accreta

DEFINITION AND PATHOGENESIS

Placenta accreta represents the abnormal attachment of the placenta to the uterine lining due to an absence of the decidua basalis and an incomplete development of the fibrinoid layer. Variations of placenta accreta include placenta increta and placenta percreta, in which the placenta extends to or through the uterine myometrium, respectively (Fig. 18-5).

INCIDENCE AND RISK FACTORS

The overall incidence of placenta accreta or one of its variations is 3 per 1000 deliveries. Based on histologic diagnosis, placenta accreta is the most common form of invasive placentation (79%) followed by placenta increta (14%) and placenta percreta (7%), respectively. **The two most significant risk factors for placenta accreta are placenta previa and prior cesarean delivery.** The risk for placenta accreta in patients with placenta previa and an unscarred uterus is approximately 3%.[35] This risk dramatically increases with one or more cesarean deliveries (Table 18-3). Even without a coexisting placenta previa, placenta accreta is more common in women with a prior cesarean delivery.[37]

Other reported risk factors include increasing parity and maternal age, submucosal uterine fibroids, prior uterine surgery, cesarean scar, and endometrial defects.[42,43] Unlike

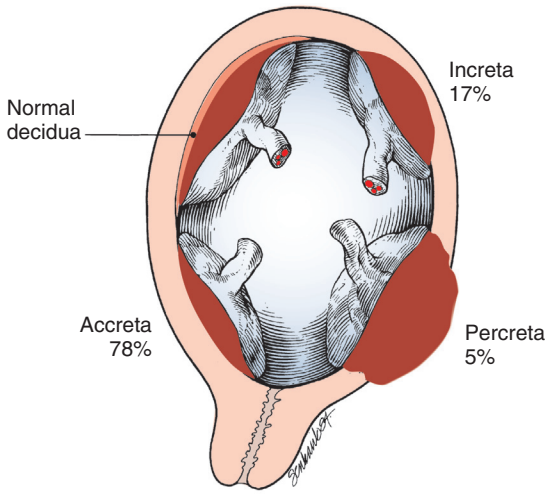

FIG 18-5 Uteroplacental relationships found with invasive placentation.

TABLE 18-3 RISK OF PLACENTA ACCRETA WITH PLACENTA PREVIA AND PRIOR CESAREAN DELIVERY

NO. OF PRIOR CESAREAN DELIVERIES	PLACENTA ACCRETA RISK (%)
0	3
1	11
2	40
3	61
≥4	67

From Silver RM, Landon MB, Rouse DJ, et al. Maternal morbidity associated with multiple repeat cesarean deliveries. *Obstet Gynecol.* 2006;107:1226.

placenta previa, a **female fetus is more common with invasive placentation.**

CLINICAL MANIFESTATIONS

The clinical manifestations of placenta accreta are often similar to those of placenta previa; however, **profuse bleeding usually follows attempted manual placental separation. Hematuria can be a feature of placenta percreta with bladder invasion.**

DIAGNOSIS

Most cases of placenta accreta are now diagnosed antenatally by advanced radiographic techniques. Prenatal diagnosis has been shown to improve maternal outcomes, resulting in less blood loss and decreased transfusion requirements.[44]

RADIOGRAPHIC TECHNIQUES

Ultrasound is the preferred radiographic modality for the diagnosis of placenta accreta. Findings suggestive of placenta accreta include a loss of the normal hypoechoic retroplacental-myometrial zone, thinning and disruption of the uterine serosa–bladder wall interface, focal exophytic masses within the placenta, and numerous intraplacental vascular lacunae (Fig. 18-6).[45] A recent systemic review and meta-analysis of 23 studies that used prenatal sonographic identification of placenta accreta demonstrated a sensitivity of 90%, a specificity of 97%, a positive likelihood ratio of 11, and a negative likelihood ratio of 0.16.[46]

Color Doppler ultrasound is also useful as an adjunctive tool in diagnosing placenta accreta. Specific color Doppler findings that differentiate placenta accreta from normal placentation include diffuse and focal intraparenchymal placental lacunar blood flow, hypervascularity of the bladder and uterine serosa, prominent subplacental venous complexes, and loss of subplacental Doppler vascular signals.[47] Some color-flow mapping studies suggest that a myometrial thickness less than 1 mm with large intraplacental venous lakes is highly predictive of invasive placentation (sensitivity, 100%; specificity, 72%; positive predictive value, 72%; and negative predictive value, 100%).

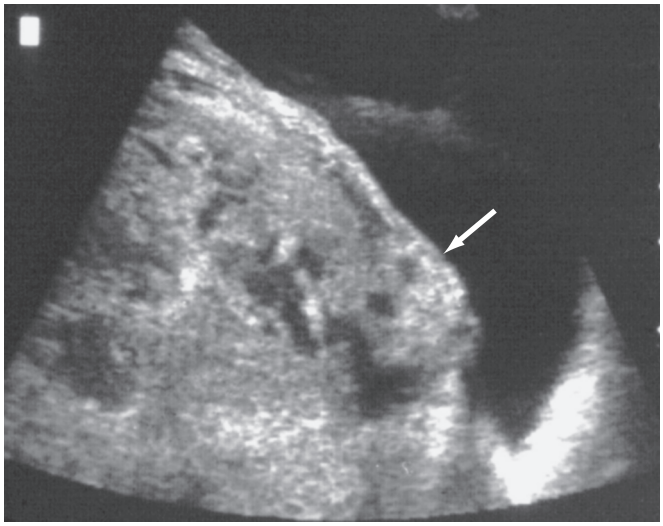

FIG 18-6 Ultrasonic image of focal placenta accreta (*arrow*). An area at the uterine-placental interface demonstrates loss of the normal hypoechoic zone, thinning and disruption of the uterine serosa–bladder interface, and a focal exophytic mass within the placenta.

In addition to the above ultrasound modalities, three-dimensional ultrasound has been used successfully to identify invasive placentation.[48,49] Diagnostic criteria include irregular intraplacental vascularization and hypervascularity of the uterine serosa–bladder wall interface.

Finally, **MRI can be used in conjunction with sonography to assess abnormal placental invasion.** MRI is particularly helpful when ultrasound findings are equivocal, the placenta is in a posterior location, and for determination of the extent of placental invasion within surrounding tissue, such as the parametrium and bladder. In a review and meta-analysis[50] of 1010 pregnancies at risk for placenta accreta, MRI had a diagnostic sensitivity of 94% and a specificity of 84%.

LABORATORY FINDINGS

Placenta accreta has been associated with unexplained elevations in maternal serum α-fetoprotein (MSAFP).

PATHOLOGIC STUDIES

Placenta accreta is confirmed by the pathologic examination of a hysterectomy specimen. Histologic evaluation demonstrates placental villi within the uterine myometrium and absence of a decidual plate. In focal accreta cases in which the uterus is not removed, curettage specimens may show myometrial cells adherent to the placenta.[51]

MANAGEMENT

Because of its associated risk for massive postpartum hemorrhage, **placenta accreta accounts for a large percentage of peripartum hysterectomies.**[52] **A multidisciplinary team approach is the ideal way to manage these cases.** Preoperative

assessments by **maternal-fetal medicine specialists, neonatologists, blood conservation teams, anesthesiologists, advanced pelvic surgeons, and urologists**—especially for a suspected placenta percreta—are recommended. Timing of delivery depends upon clinical circumstances; however, **most authorities favor delivery at $34^{0/7}$ to $35^{6/7}$ weeks** with or without antenatal corticosteroid administration.[38] Ideally, the delivery should be scheduled at a time with optimal personnel availability at a facility prepared to manage significant obstetric hemorrhage. **Adequate IV access with two large-bore catheters and ample blood product availability are mandatory.** Cell-saver technology, donor-directed or autologous blood donation, and recombinant VIIa should be considered. Placement of ureteral stents preoperatively or intraoperatively can assist in maintaining urinary tract integrity. When performing the surgery, **it is recommended that the uterus be incised above the placental attachment site and that the placenta be left in situ after clamping the cord because disruption of the implantation site may result in rapid blood loss.** Finally, adjuvant use of aortic and/or internal iliac artery balloon occlusion catheters with postsurgical embolization has been shown to reduce blood loss, transfusion requirements, and duration of surgery in some studies.[53]

In specific circumstances, uterine conservation may be attempted. These situations include focal accreta, desired future fertility, and fundal or posterior placenta accreta. Uterine conservation techniques typically include leaving the placenta in situ at the delivery with subsequent expectant management, delayed manual placental removal, wedge resection or oversewing of the placental implantation site, tamponade of the lower uterine segment, curettage, uterine artery embolization, hemostatic sutures, arterial ligation, and/or administration of methotrexate.[54] Although each of these techniques has reported success, each is also associated with potential complications, including delayed hemorrhage, infection, fistula formation, subsequent surgery and/or hysterectomy, uterine necrosis, and even death.[55] Data are limited regarding long-term reproductive outcomes in women treated conservatively for invasive placentation. Although most women are able to conceive after conservative management, they remain at risk for spontaneous abortion, uterine synechiae and rupture, preterm delivery, recurrent placenta accreta, and peripartum hysterectomy.[54,56-58]

Vasa Previa

DEFINITION AND PATHOGENESIS

***Vasa previa* is defined as the presence of fetal vessels over the cervical os.** Typically, these fetal vessels lack protection from Wharton jelly (velamentous cord insertion) and are prone to rupture and compression. When the vessels rupture, the fetus is at high risk for exsanguination. **Velamentous cord insertion may occur without vasa previa and can occasionally exist as fetal vessels that run between a bilobed or succenturiate-lobed placenta.**

INCIDENCE AND RISK FACTORS

The overall incidence of vasa previa is 1 in 2500 deliveries; however, data have shown a range from 1 in 2000 to 1 in 5000 deliveries.[59] **Reported risk factors for vasa previa include bilobed and succenturiate-lobed placentas; pregnancies that result from assisted reproductive technology (ART); multiple gestations; and history of second-trimester placenta previa or low-lying placenta.**[59-61]

CLINICAL MANIFESTATIONS

In the past, most cases of vasa previa presented after rupture of membranes with acute onset of vaginal bleeding from a lacerated fetal vessel. If immediate intervention was not provided, fetal bradycardia and subsequent fetal death occurred. Today, many cases of vasa previa are diagnosed antenatally by ultrasound. In rare cases, **pulsating fetal vessels may be palpable in the membranes that overlie the cervical os.**

DIAGNOSIS

Vasa previa is often diagnosed antenatally by **ultrasound with color and pulsed Doppler mapping.** Transabdominal and transvaginal approaches are most often used. **The diagnosis is confirmed by documenting umbilical vessels over the cervical os using color and pulsed Doppler imaging** (Fig. 18-7).

MANAGEMENT

When diagnosed antenatally, vasa previa should be managed similarly to placenta previa. Some authorities have recommended twice-weekly nonstress testing at 28 to 30 weeks of gestation to assess for cord compression; others have favored

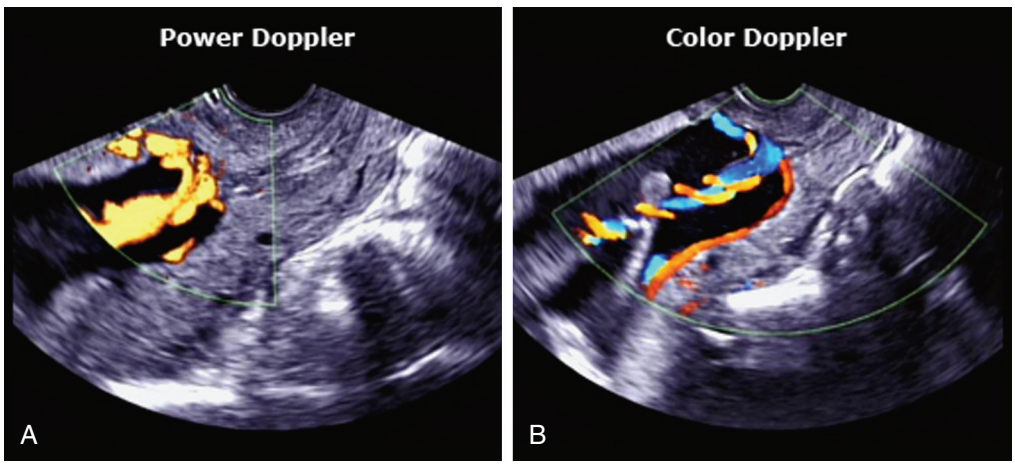

FIG 18-7 Transvaginal ultrasound images showing vasa previa and velamentous cord. The placenta is posterior with an anterior succenturiate lobe. (From Lockwood CJ, Russo-Steiglitz K. *Velamentous umbilical cord insertion and vasa previa,* www.uptodate.com, July 17, 2015.)

hospitalization in the third trimester with administration of antenatal corticosteroids, serial antepartum testing, and **cesarean delivery between $34^{0/7}$ to $36^{0/7}$ weeks of gestation.**[62,63] If an intrapartum diagnosis of vasa previa is made, expeditious delivery is needed. Immediate neonatal blood transfusion is often required in these circumstances.

POSTPARTUM HEMORRHAGE

Postpartum hemorrhage is an obstetric emergency that complicates between 1 in 20 and 1 in 100 deliveries. In the past decade, reported postpartum hemorrhage has increased 26% within the United States.[64] Because it is a major cause of maternal morbidity and mortality, obstetricians need to have a clear understanding of normal delivery-related blood loss so that postpartum hemorrhage can be efficiently recognized and managed.

Normal Blood Loss and Postpartum Hemorrhage

Normal delivery-related blood loss depends on the type of delivery. Based on objective data, the mean blood losses for a vaginal delivery, cesarean delivery, and cesarean hysterectomy are 500, 1000, and 1500 mL, respectively.[65] These values are often underestimated and unappreciated clinically owing to the significant blood volume expansion that accompanies normal pregnancy.

Postpartum hemorrhage has been variably defined in the literature. Definitions have included subjective assessments greater than the standard norms, a 10% decline in hemoglobin concentration, and the need for blood transfusion. A more practical definition is excessive delivery-related blood loss that causes the patient to be hemodynamically symptomatic and/or hypovolemic.

Postpartum Hemorrhage Etiologies

The etiologies of postpartum hemorrhage can be categorized as primary (early) or secondary (late). ***Primary postpartum hemorrhage*** **refers to excessive bleeding that occurs within 24 hours of delivery, whereas** ***secondary postpartum hemorrhage*** **refers to bleeding that occurs from 24 hours until 12 weeks after delivery.** Box 18-3 lists the most common causes of primary and secondary postpartum hemorrhage. Because primary postpartum hemorrhage is more common than the secondary kind, the remainder of this discussion focuses on its etiology and management (Fig. 18-8).

BOX 18-3 ETIOLOGIES OF POSTPARTUM HEMORRHAGE

Early

Uterine atony
Lower genital tract lacerations (perineal, vaginal, cervical, periclitoral, periurethral, rectum)
Upper genital tract lacerations (broad ligament)
Lower urinary tract lacerations (bladder, urethra)
Retained products of conception (placenta, membranes)
Invasive placentation (placenta accreta, placenta increta, placenta percreta)
Uterine rupture
Uterine inversion
Coagulopathy

Late

Infection
Retained products of conception
Placental site subinvolution
Coagulopathy

Uterine Atony

DEFINITION AND PATHOGENESIS

Uterine atony, or the inability of the uterine myometrium to contract effectively, is the most common cause of primary postpartum hemorrhage. At term, blood flow through the placental site averages 500 to 700 mL per minute. After placental delivery, the uterus controls bleeding by contracting its myometrial fibers in a tourniquet fashion around the spiral arterioles. If inadequate uterine contraction occurs, rapid blood loss can ensue.

INCIDENCE AND RISK FACTORS

Uterine atony complicates 1 in 20 deliveries and is responsible for 80% of postpartum hemorrhage cases. Risk factors for uterine atony include **uterine overdistention** (multiple gestation, polyhydramnios, fetal macrosomia), **labor induction, rapid or prolonged labor, grand multiparity, uterine infection, uterine inversion, retained products of conception, abnormal placentation, and use of uterine-relaxing agents** (tocolytic therapy, halogenated anesthetics, nitroglycerin).

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Uterine atony is diagnosed clinically by rapid uterine bleeding associated with a lack of myometrial tone and an absence of other etiologies for postpartum hemorrhage. Typically, bimanual palpation of the uterus confirms the diagnosis.

PREVENTION AND MANAGEMENT

By recognizing risk factors for uterine atony and quickly initiating a treatment cascade, the clinician can minimize blood loss. Three preventive methods for atonic postpartum hemorrhage are 1) active management of the third stage of labor, 2) spontaneous placental separation during cesarean delivery, and 3) prolonged postpartum oxytocin infusion. Active management of the third stage of labor includes early cord clamping, controlled cord traction, uterine massage, and administration of uterotonic therapy before placental separation. A systematic review[66] of seven studies that compared active to expectant management of the third stage of labor showed significant reductions in maternal blood loss, postpartum hemorrhage, prolonged third stage of labor, and the need for additional uterotonic therapies. Whereas controversy exists regarding the timing of uterotonic administration, a recent systematic review[67] suggested that giving uterotonic therapy before delivery of the placenta results in less blood loss and fewer postpartum transfusions.

A second strategy to minimize uterine atony is to allow spontaneous placental separation during cesarean delivery. In one controlled study, spontaneous placental separation reduced blood loss by 30% and reduced postpartum endometritis sevenfold compared with manual removal.

A final preventive approach for atonic postpartum hemorrhage is prolonged postpartum oxytocin infusion. A recent clinical trial[68] assessed two postpartum oxytocin regimens following delivery: a bolus of oxytocin after delivery versus a bolus of oxytocin followed by a 4-hour IV infusion of oxytocin. A significant reduction was seen in uterine atony and need for additional uterotonic therapy in the women who received the prolonged oxytocin infusion. Additional evidence-based review

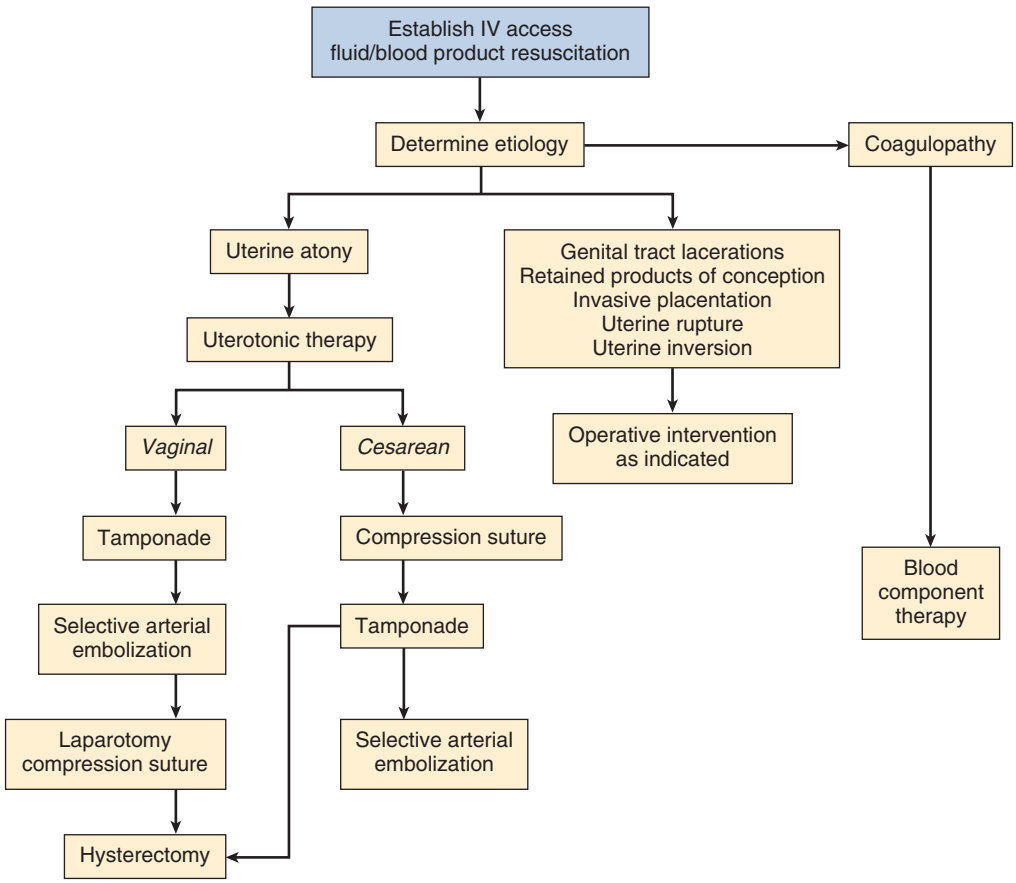

FIG 18-8 Management of postpartum hemorrhage. IV, intravenous.

data support prolonged (4 to 8 hours) oxytocin administration after delivery.[69]

If preventive measures are unsuccessful, medical management for uterine atony should be initiated. This treatment includes bimanual uterine massage and uterotonic therapy.

BIMANUAL UTERINE MASSAGE

To provide effective bimanual uterine massage, **the uterus should be compressed between the external, fundally placed hand and the internal, intravaginal hand** (Fig. 18-9). **Care must be taken to avoid aggressive massage that can injure the large vessels of the broad ligament.**

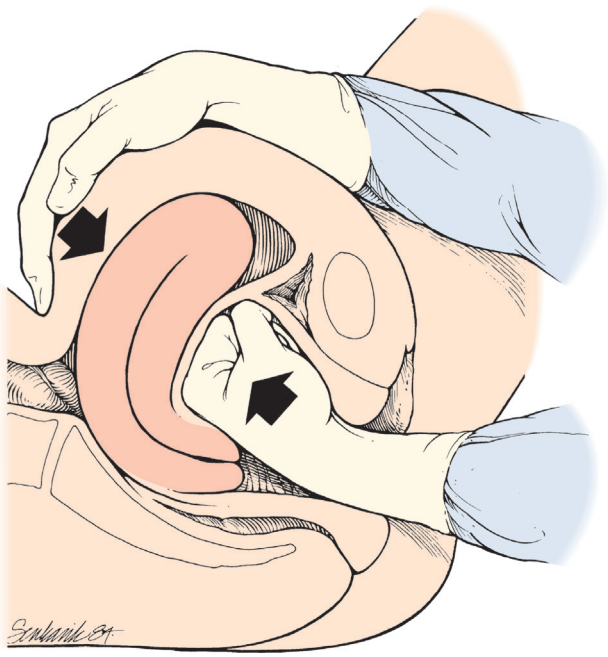

FIG 18-9 Bimanual uterine massage.

UTEROTONIC THERAPY

Uterotonic medications represent the mainstay of drug therapy for postpartum hemorrhage secondary to uterine atony. Table 18-4 lists available uterotonic agents with their dosages, side effects, and contraindications. **Oxytocin** is usually given as a first-line agent. IV therapy is the preferred route of administration, but intramuscular and intrauterine dosing is possible. Initial treatment starts with 10 to 30 units of oxytocin in 500 to 1000 mL of crystalloid solution. Higher doses (80 units in 500 to 1000 mL) have proved safe and efficacious, with a 20% reduction in the need for additional uterotonic therapy and reduced composite hemorrhage treatment (uterotonic drugs, transfusion, tamponade, embolization, surgery).[70]

When oxytocin fails to produce adequate uterine tone, second-line therapy must be initiated. Currently, a variety of additional uterotonic agents are available. The choice of a second-line agent depends on its side-effect profile as well as its

TABLE 18-4 UTEROTONIC THERAPIES

AGENT	DOSE	ROUTE	DOSING INTERVAL	SIDE EFFECTS	CONTRAINDICATIONS
Oxytocin (Pitocin)	10 to 80 U in 500-1000 mL crystalloid solution	First line: IV Second line: IM or IU	Continuous	Nausea, emesis, water intoxication	None
Misoprostol (Cytotec)	600-1000 µg	First line: PR Second line: PO or SL	Single dose	Nausea, emesis, diarrhea, fever, chills	None
Methylergonovine (Methergine)	0.2 mg	First line: IM Second line: IU or PO	Every 2-4 hr	Hypertension, hypotension, nausea, emesis	Hypertension, migraines, scleroderma, Raynaud syndrome
Prostaglandin $F_{2\alpha}$ (Hemabate)	0.25 mg	First line: IM Second line: IU	Every 15-90 min (maximum of 8 doses)	Nausea, emesis, diarrhea, flushing, chills	Active cardiac, pulmonary, renal, or hepatic disease
Prostaglandin E_2 (Dinoprostone)	20 mg	PR	Every 2 hr	Nausea, emesis, diarrhea, fever, chills, headache	Hypotension

IM, intramuscular; *IU,* intrauterine; *IV,* intravenous; *PO,* per os; *PR,* per rectum; *SL,* sublingual.

contraindications. **Misoprostol, a synthetic prostaglandin E_1 analogue,** is a safe, inexpensive, and efficacious uterotonic medication that does not require refrigeration. It has been used for both the prevention and treatment of postpartum hemorrhage.[71-73] Misoprostol is attractive as a second-line agent in that it has multiple administration routes that can be combined. Although higher doses (600 to 1000 µg) have traditionally been used rectally, the sublingual route allows for lower dosing (400 µg) with higher bioavailability.[71] Although helpful in some settings, **methylergonovine** has limited usefulness for acute postpartum hemorrhage because of its relatively long half-life and potential for worsening hypertension in patients with preexisting disease.

Prostaglandins are highly effective uterotonic agents. Both natural and synthetic prostaglandin formulations are available. Intramuscular and intrauterine administration of **prostaglandin $F_{2\alpha}$** can be used for control of atony. Recurrent doses (0.25 mg) may be administered as often as every 15 minutes to a maximum of eight doses (2 mg total dose). It is important to note that asthma is a *strong contraindication* to the use of prostaglandin $F_{2\alpha}$ because of its bronchoconstrictive properties. **Prostaglandin E_2 (dinoprostone)** is a naturally occurring oxytocic that can dramatically improve uterine tone; however, it has an unfavorable side-effect profile often precludes its use (fever, chills, nausea, emesis, diarrhea, and headaches). Lastly, oxytocin analogues and combined ergometrine-oxytocin preparations are available outside of the United States for control of uterine atony.

When atony is due to tocolytic drugs that have impaired calcium entry into the cell (magnesium sulfate, nifedipine), calcium gluconate should be considered as an adjuvant therapy. Given as an IV push, one ampule (1 g in 10 mL) of calcium gluconate can effectively improve uterine tone and resolve bleeding due to atony.

If pharmacologic methods fail to control atony-related hemorrhage, alternative measures must be undertaken. **The genital tract should be carefully inspected for lacerations before proceeding with these measures, which include uterine tamponade, selective arterial embolization, and surgical intervention.**

UTERINE TAMPONADE

Uterine packing is a safe, simple, and effective way to control postpartum hemorrhage by providing tamponade to the bleeding uterine surface. Although packing techniques vary, a few basic principles should be followed. The pack should be made of long, continuous gauze (e.g., Kerlix) rather than multiple small sponges. Some authorities have had success with the use of thrombin-impregnated or chitosan-covered gauze.[74] When packing the uterus, placement should begin at the fundus and progress downward in a side-to-side fashion to avoid dead space for blood accumulation. Transurethral Foley catheter placement and prophylactic antibiotic use should be considered to prevent urinary retention and infection, respectively. Finally, prolonged packing should be avoided (not more than 12 to 24 hours), and close attention should be paid to the patient's vital signs and blood indices while the pack is in place in order to minimize unrecognized ongoing bleeding.

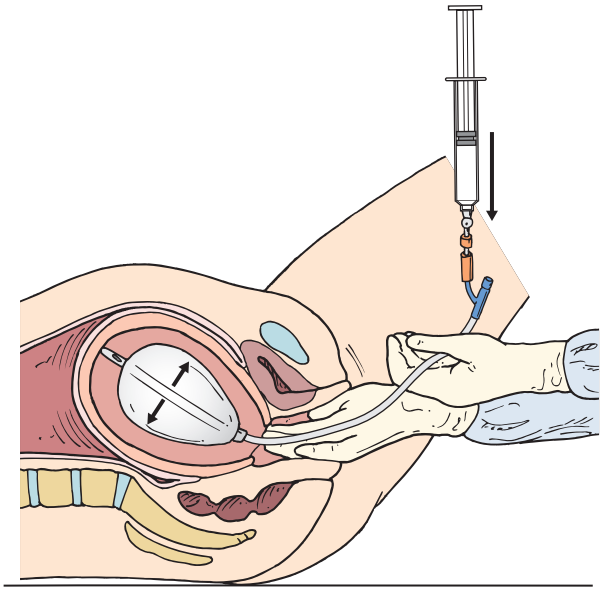

FIG 18-10 The Bakri tamponade balloon. (From Bakri YN, Arulkumaran S. *Intrauterine balloon tamponade for control of postpartum hemorrhage,* www.uptodate.com, July 7, 2015.)

In recent years, **intrauterine tamponade balloons** have largely replaced traditional uterine packing. Multiple balloon types have been used and include the Bakri tamponade balloon, the BT-Cath, the Belfort-Dildy Obstetrical Tamponade System, the Sengstaken-Blakemore tube, and the #24 Foley catheter with 30 mL balloon. The **Bakri tamponade balloon**, the **BT-Cath,** and the **Belfort-Dildy Obstetrical Tamponade System** were all developed specifically for postpartum hemorrhage management. The Bakri tamponade balloon (Cook Women's Health; Fig. 18-10) consists of a silicone balloon attached to a catheter. The catheter is inserted into the uterus either manually or under

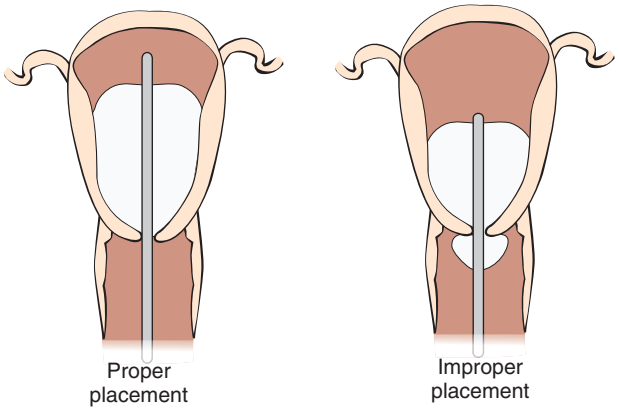

FIG 18-11 Proper placement of the Bakri tamponade balloon. (Courtesy Cook Women's Health.)

FIG 18-12 The Belfort-Dildy obstetrical tamponade system. (Courtesy Glenveigh Medical.)

ultrasound guidance, and the silicone balloon is subsequently inflated with sterile saline (maximum of 500 mL). Once inflated, the balloon should adapt to the uterine configuration to provide tamponade to the endometrial surface. The intraluminal catheter allows drainage from within the uterus so that ongoing assessment of blood loss can occur. Proper placement of the balloon is essential to provide adequate tamponade (Fig. 18-11). Like the Bakri tamponade balloon, the BT-Cath (Utah Medical Products) is a silicone balloon; however, it is shaped like an inverted pear. This tamponade balloon also has a double-lumen catheter that allows saline filling of the balloon as well as drainage of blood from within the uterus. Finally, the Belfort-Dildy Obstetrical Tamponade System (Glenveigh Medical) incorporates both intrauterine and intravaginal polyurethane balloons that conform to these cavities, respectively (Fig. 18-12).[75] The uterine balloon can be rapidly inflated from a saline bag to a larger volume of 750 mL when the smaller balloons are inadequate (e.g., with multiple gestations). Like the other balloons, this system has a drainage port to assess for ongoing bleeding; however, unlike the others, it also has an infusion port to irrigate the uterus.

SELECTIVE ARTERIAL EMBOLIZATION

Selective arterial embolization is an increasingly common therapeutic option for hemodynamically stable patients with postpartum hemorrhage. The procedure can be performed alone or after failed surgical intervention.[76] Diagnostic pelvic angiography is used to visualize bleeding vessels, and gelatin (e.g., Gelfoam]) pledgets are placed into the vessels for occlusion. **Cumulative success rates of 90% to 97% have been reported.**[76]

Selective arterial embolization has several advantages over surgical intervention. First, it allows for selective occlusion of bleeding vessels. This can be extremely valuable in circumstances of aberrant pelvic vasculature, such as uterine arteriovenous malformations. Second, the uterus and potential future fertility are preserved. Case series[76] have reported successful pregnancies after pelvic embolization. Finally, the procedure has minimal morbidity, enables the physician to forego or delay surgical intervention, and can be performed in coagulopathic patients, which allows more time for blood and clotting factor replacement. Procedure-related complications occur in 3% to 6% of cases.[76] Reported complications include postembolization fever, infection, ischemic pain, vascular perforation, and tissue necrosis. A relative disadvantage of the procedure is its limited availability. Timely coordination of services between the obstetric team and interventional radiology personnel, as well as a hemodynamically stable patient, are necessary to provide this treatment option.

SURGICAL INTERVENTION

When uterine atony is unresponsive to conservative management, surgical intervention by laparotomy is necessary. Possible interventions include arterial ligation, uterine compression sutures, and hysterectomy.

The goal of arterial ligation is to decrease uterine perfusion and subsequent bleeding. Success rates have varied from 40% to 95% in the literature depending on which vessels are ligated. Arterial ligation may be performed on the ascending uterine arteries, the uteroovarian arteries, the infundibulopelvic ligament vessels, and the internal iliac (hypogastric) arteries. Because internal iliac arterial ligation can be technically challenging and time consuming, it is not advised as a first-line technique unless the surgeon is extremely skilled in performing the procedure. Instead, a stepwise progression of uterine vessel ligation is recommended.

Nearly five decades ago, O'Leary described a technique of bilateral uterine artery ligation for control of postpartum hemorrhage. Today, it is still considered the initial ligation technique given its ease in performance and the accessibility of the uterine artery. To perform the procedure, the ascending uterine artery should be located at the border of the upper and lower uterine segment. Absorbable suture is passed through the uterine myometrium at the level of the lower uterine segment and laterally around the uterine vessels through a clear space in the broad ligament. The suture is then tied to compress the vessels against the uterine wall (Fig. 18-13). Because the suture is placed fairly high in the lower uterine segment, ureteral injury is avoided;

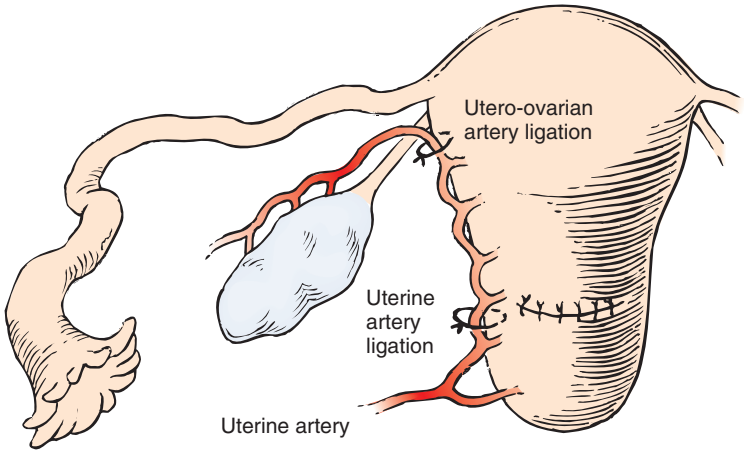

FIG 18-13 Uterine artery ligation.

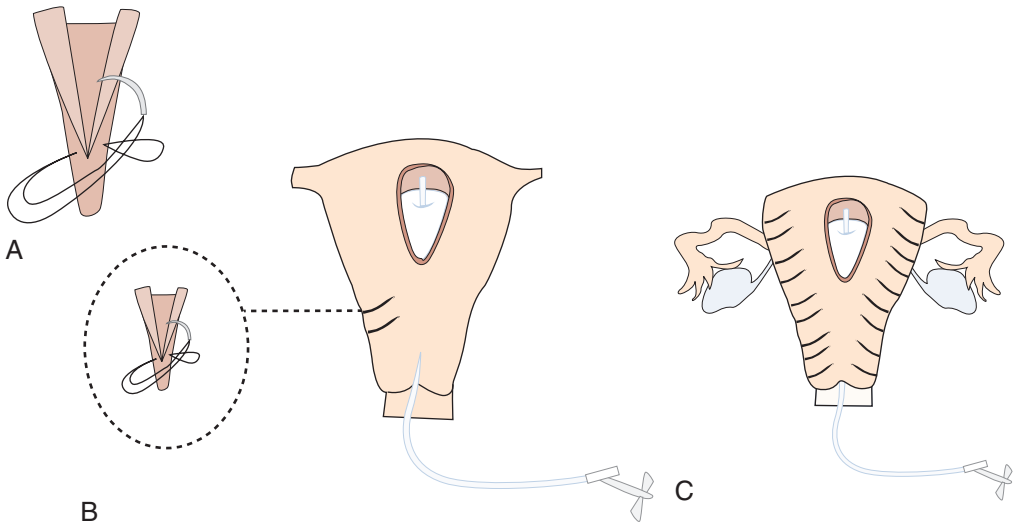

FIG 18-14 Bilateral looped uterine vessel sutures. (From Bakri YN, Arulkumaran S. *Intrauterine balloon tamponade for control of postpartum hemorrhage,* www.uptodate.com, July 7, 2015.)

therefore the bladder does not need to be mobilized. Unilateral artery ligation will control hemorrhage in 10% to 15% of cases, whereas bilateral ligation will control hemorrhage in over 90% of cases.

If bleeding persists, the uteroovarian and infundibulopelvic vessels should be ligated. The uteroovarian arteries can be ligated similarly to what has been described for the ascending uterine vessels. If this measure is unsuccessful, interruption of the infundibulopelvic vessels can be undertaken. Although the ovarian blood supply may be decreased with an infundibulopelvic vessel ligation, successful pregnancy has been reported following this procedure.

Bakri has described a newer technique for bilateral uterine artery ligation in combination with tamponade balloon placement (Fig. 18-14). The procedure, termed ***Bilateral Looped Uterine Vessel Sutures*** (B-LUVS), incorporates absorbable sutures looped bilaterally through the myometrium around the uterine vessels from the lower segment up to the corneal region. Once the sutures are tied, a Bakri tamponade balloon is placed within the uterine cavity to provide internal tamponade. A small series has had high success with this combined ligation-tamponade approach.

In addition to arterial ligation, **uterine compression sutures** have been described for atony control. Several techniques have evolved over the past two decades, including the **B-Lynch suture, Hayman vertical sutures, Pereira transverse and vertical sutures, and multiple square sutures.**[77,78] To place a compression suture, the patient should lie in the dorsal lithotomy position to facilitate assessment of vaginal bleeding. Large absorbable suture is typically anchored within the uterine myometrium both anteriorly and posteriorly. It is passed in a continuous or intermittent fashion around or through the external surface of the uterus and tied firmly so that adequate uterine compression occurs. Figures 18-15 to 18-18 demonstrate proper placements of these sutures. Like arterial ligation, uterine compression sutures can be combined with tamponade balloons for refractory bleeding. The "uterine sandwich" technique refers to placement of a B-Lynch suture followed by a Bakri tamponade balloon within the uterine cavity.[79] Typically, the tamponade balloon is inflated to a lesser volume (median of 100 mL) in these cases. Small series[79] have demonstrated high success rates for this combined approach.

The final surgical intervention for refractory bleeding due to atony is hysterectomy, which provides definitive therapy.

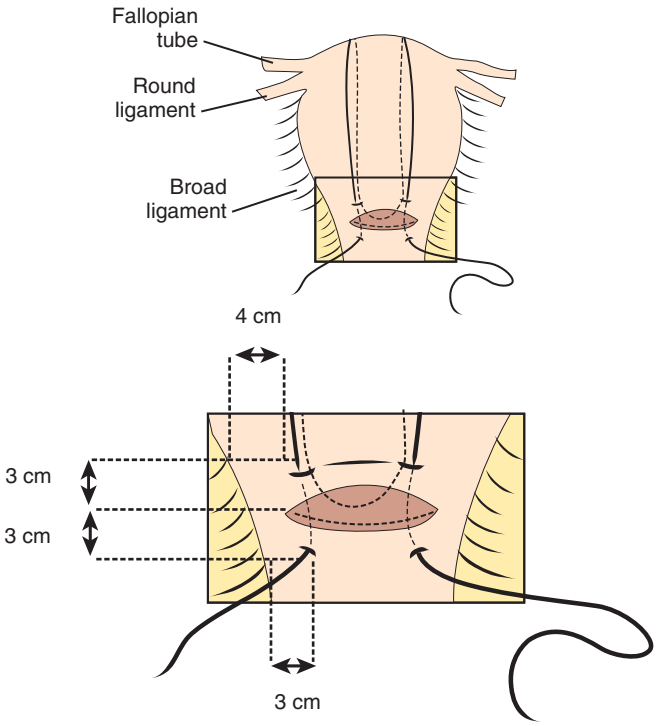

FIG 18-15 B-Lynch compression suture. (From Belfort MA. Management of postpartum hemorrhage at cesarean delivery, www.uptodate.com, June 26, 2015.)

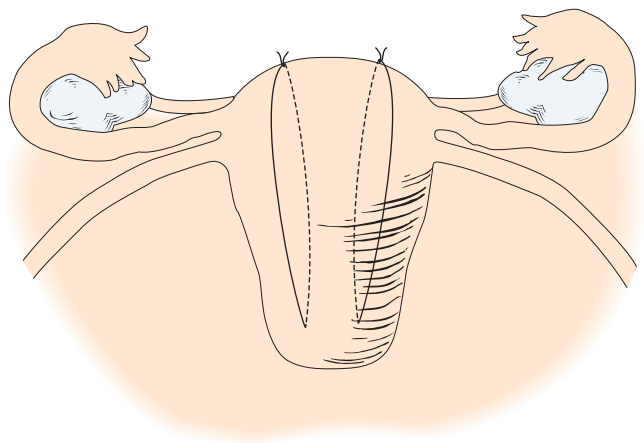

FIG 18-16 Hayman vertical sutures. (From Belfort MA. *Management of postpartum hemorrhage at cesarean delivery,* www.uptodate.com, June 26, 2015.)

Because blood loss may be severe, it is often prudent to modify the surgical approach by using the "clamp-cut-drop" technique (Fig. 18-19), performing a supracervical hysterectomy, or both.[80] These considerations are especially important when the patient is hemodynamically unstable (Video 18-1).

Genital Tract Lacerations

DEFINITION AND PATHOGENESIS

Genital tract lacerations may occur with both vaginal and cesarean deliveries. These lacerations involve the maternal soft tissue structures and can be associated with large hematomas and rapid blood loss if unrecognized. **The most common lower genital tract lacerations are perineal, vulvar, vaginal, and**

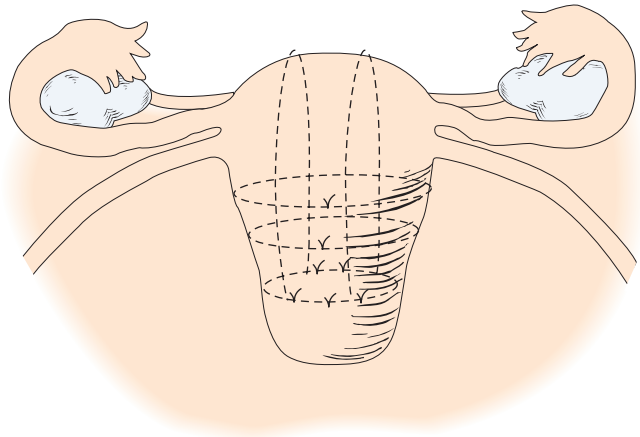

FIG 18-17 Pereira transverse and vertical sutures (From Belfort MA. *Management of postpartum hemorrhage at cesarean delivery,* www.uptodate.com, June 26, 2015.)

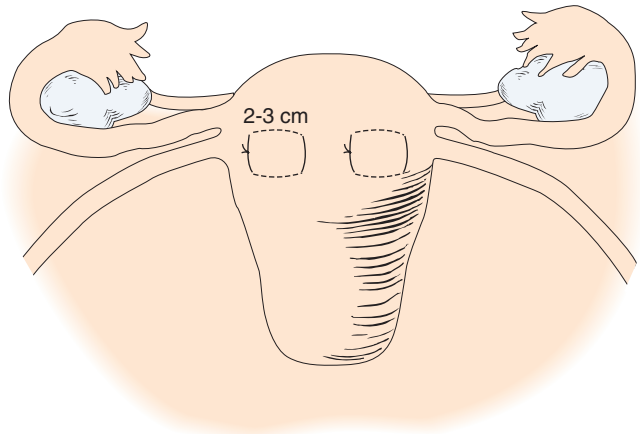

FIG 18-18 Multiple square sutures. (From Belfort MA. *Management of postpartum hemorrhage at cesarean delivery,* www.uptodate.com, June 26, 2015.)

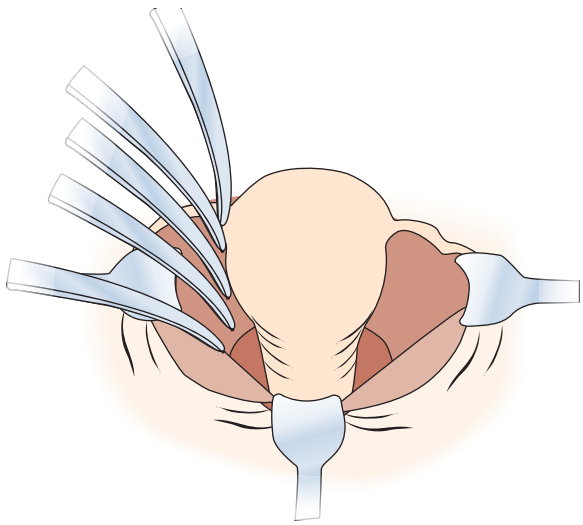

FIG 18-19 Clamp-cut-drop technique for hysterectomy. (From Wright JD, Bonanno C, Shah M, et al. Peripartum hysterectomy. *Obstet Gynecol.* 2010;116:429-434.)

cervical. Upper genital tract lacerations are typically associated with broad ligament and retroperitoneal hematomas.

INCIDENCE AND RISK FACTORS

Although it is difficult to ascertain their exact incidence, **genital tract lacerations are the second leading cause of postpartum hemorrhage. Risk factors include instrumented vaginal delivery, fetal malpresentation or macrosomia, episiotomy, precipitous delivery, prior cerclage placement, Dührssen incisions, and shoulder dystocia.**

CLINICAL MANIFESTATIONS AND DIAGNOSIS

A genitourinary tract laceration should be suspected if bleeding persists after delivery despite adequate uterine tone. Occasionally, the bleeding may be masked because of its location, such as with the broad ligament. In these circumstances, large amounts of blood loss may occur in an unrecognized hematoma. Pain and hemodynamic instability are often the primary presenting symptoms.

For diagnosis, it is best to evaluate the lower genital tract superiorly from the cervix and to progress inferiorly to the vagina, perineum, and vulva. Adequate exposure and retraction are essential for identification of many of these lacerations.

MANAGEMENT

Once a genital tract laceration is identified, management depends on its severity and location. Lacerations of the cervix and vaginal fornices are often difficult to repair owing to their position. In these circumstances, relocation to an OR with anesthesia assistance for better pain relief, pelvic relaxation, and visualization are recommended. For cervical lacerations, it is important to secure the apex of the tear because this is often a major source of bleeding. Unfortunately, exposure of this angle can be difficult. A helpful technique for these scenarios is to start suturing the laceration at its proximal end, thereby using the suture for traction to expose the more distal portion of the cervix until the apex is in view (Fig. 18-20).

Perineal repairs are the most common types of genital tract lacerations. Figures 18-21 to 18-23 illustrate second-, third-, and fourth-degree perineal lacerations and techniques for their repair. If the laceration is adjacent to the urethra, the use of a

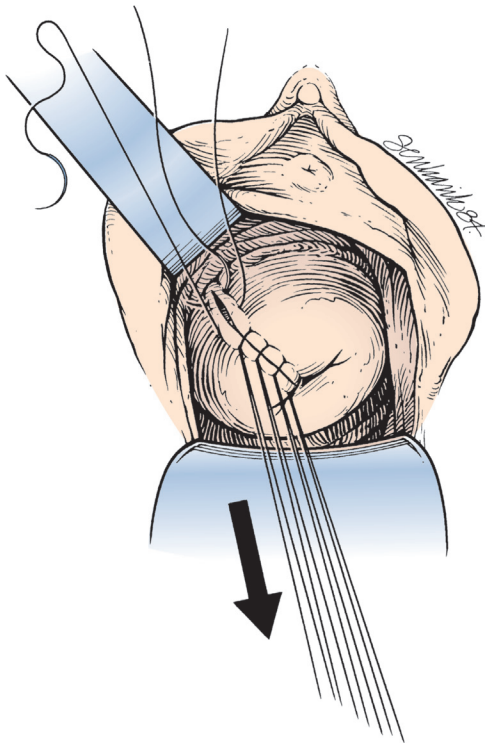

FIG 18-20 Repair of a cervical laceration, beginning at the proximal part of the laceration and using traction on the previous sutures to aid in exposure of the distal defect.

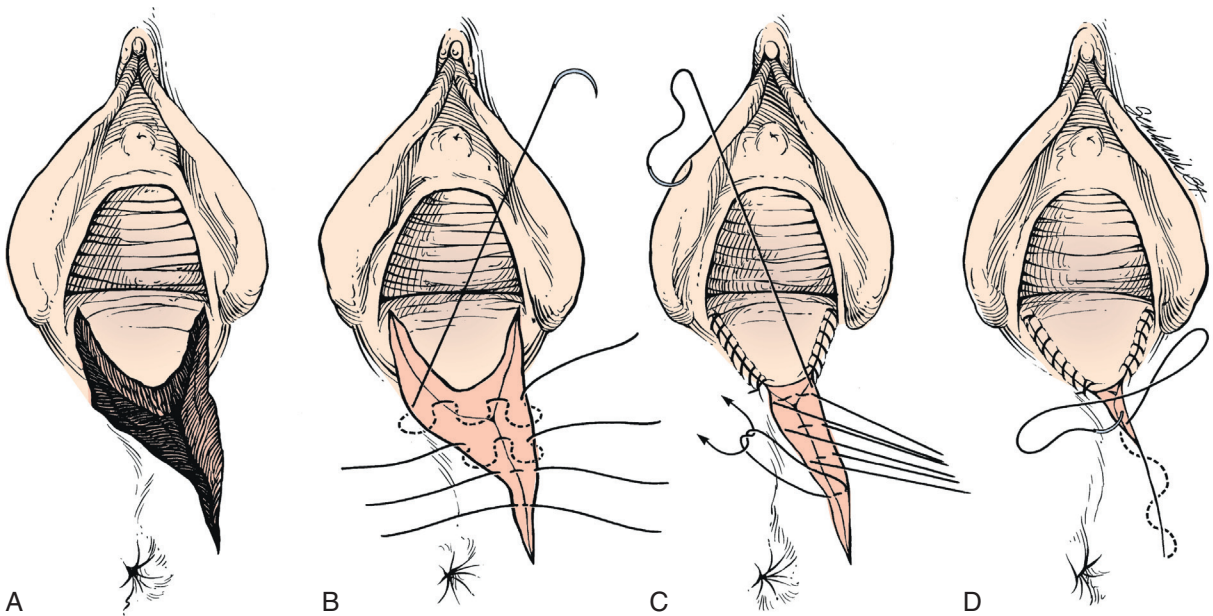

FIG 18-21 Repair of a second-degree laceration. A first-degree laceration involves the fourchette, the perineal skin, and the vaginal mucous membrane. A second-degree laceration also includes the muscles of the perineal body. The rectal sphincter remains intact.

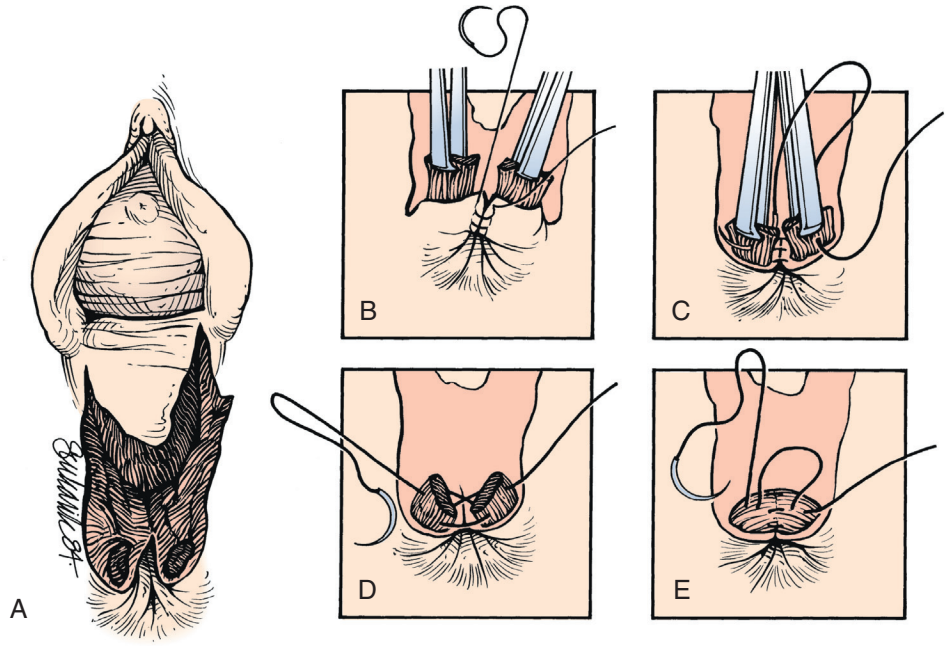

FIG 18-22 Repair of the sphincter after a third-degree laceration. A third-degree laceration extends not only through the skin, mucous membrane, and perineal body but includes the anal sphincter. Interrupted figure-of-eight sutures should be placed in the capsule of the sphincter muscle.

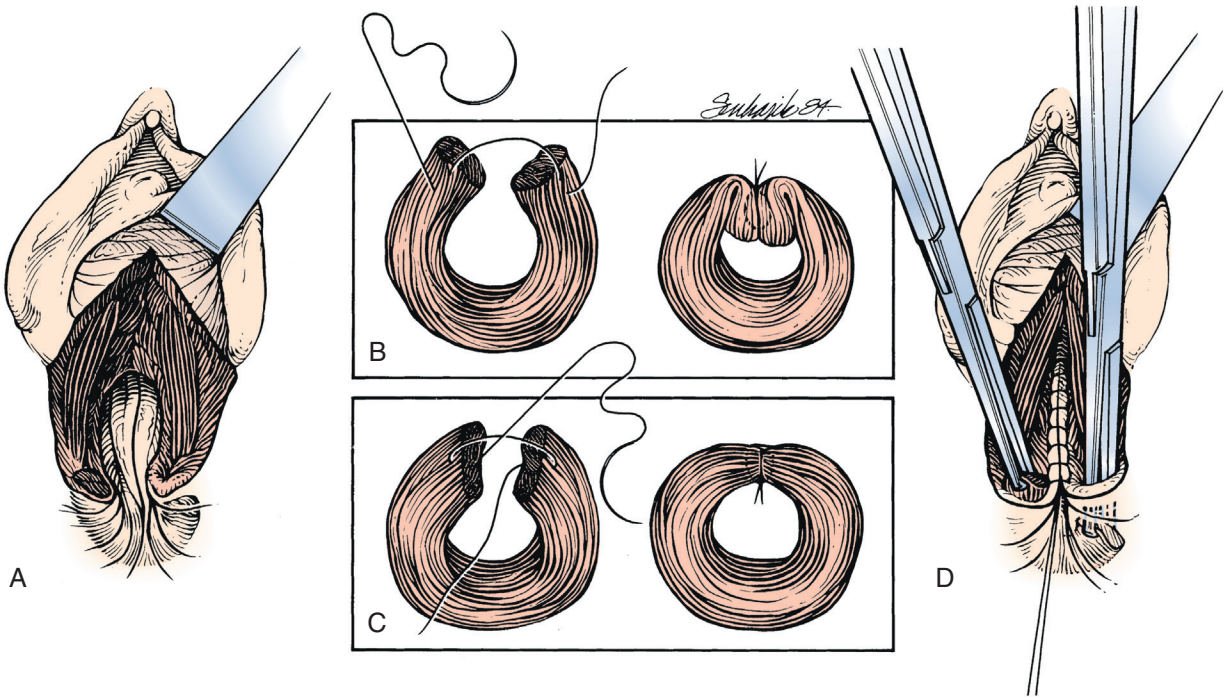

FIG 18-23 Repair of a fourth-degree laceration. This laceration extends through the rectal mucosa. **A,** The extent of this laceration is shown with a segment of the rectum exposed. **B,** Approximation of the rectal submucosa. This is the most commonly recommended method for repair. **C,** Alternative method of approximating the rectal mucosa in which the knots are actually buried inside the rectal lumen. **D,** After closure of the rectal submucosa, an additional layer of running sutures may be placed. The rectal sphincter is then repaired.

transurethral catheter can aid in a more efficient repair and can protect uninjured organs. Digital rectal exam is recommended after repair of third- and fourth-degree lacerations in order to ensure proper integrity of the rectum.

On occasion, a blood vessel laceration may lead to the formation of a pelvic hematoma in the lower or upper genital tract. The three most common locations for a pelvic hematoma are vulvar, vaginal, and retroperitoneal.

VULVAR HEMATOMA

Vulvar hematomas usually result from lacerated vessels in the superficial fascia of the anterior or posterior pelvic triangle.

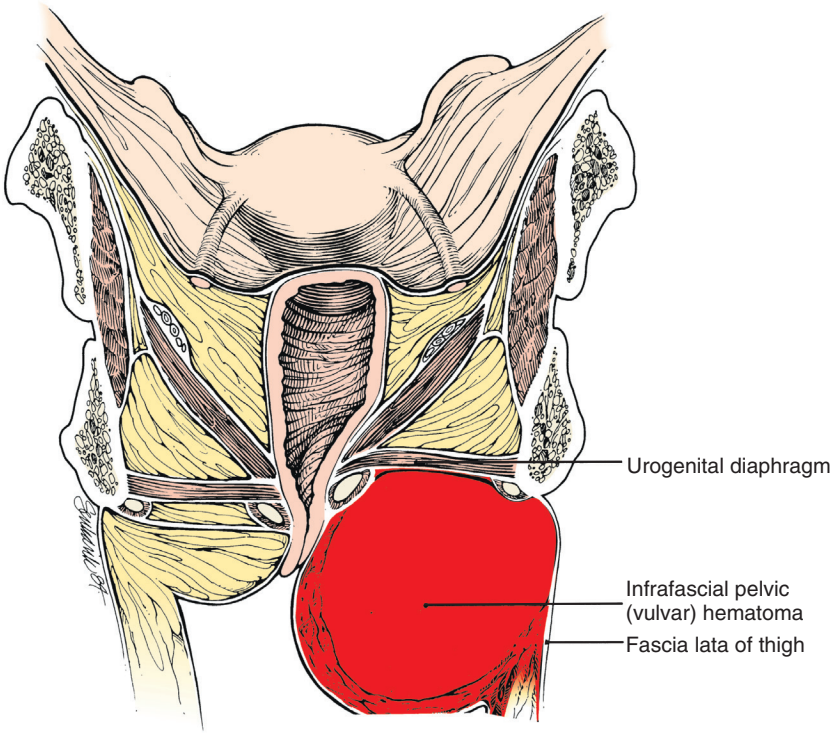

FIG 18-24 Vulvar hematoma fascial boundaries.

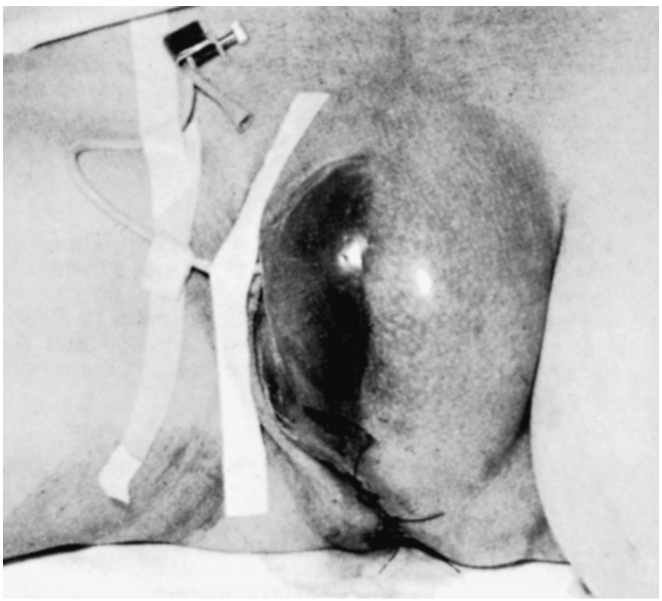

FIG 18-25 Large vulvar hematoma.

Blood loss is tamponaded by Colles fascia, the urogenital diaphragm, and anal fascia (Fig. 18-24). Because of these fascial boundaries, the mass will extend to the skin, and a visible hematoma results (Fig. 18-25).

Surgical drainage is the primary treatment for vulvar hematomas. A wide linear incision through the skin is recommended. Typically, the bleeding is due to multiple small vessels; hence, vessel ligation is not possible. Once the hematoma is evacuated, the dead space should be closed in layers with absorbable suture, and a sterile pressure dressing should be applied. A transurethral catheter should be placed until significant tissue edema subsides.

VAGINAL HEMATOMA

Vaginal hematomas result from delivery-related soft tissue damage. These hematomas accumulate above the pelvic diaphragm (Fig. 18-26), and occasionally, they protrude into the vaginal-rectal area. Like vulvar hematomas, vaginal hematomas are due to multiple small vessel lacerations. Depending on the extent of the hemorrhage, **vaginal hematomas may or may not require surgical drainage.** Small, nonexpanding hematomas are often best managed expectantly. Larger, expanding hematomas require surgical intervention. Unlike vulvar hematomas, the incision of a vaginal hematoma does not require closing, rather a vaginal pack or tamponade device should be placed on the raw edges. If bleeding persists, **selective arterial embolization may be considered.**

RETROPERITONEAL HEMATOMA

Although infrequent, **retroperitoneal hematomas are the most serious and life threatening.** The early symptoms of a retroperitoneal hematoma are often subtle, with the hematoma being unrecognized until the patient is hemodynamically unstable from massive hemorrhage. **These hematomas usually occur after a vessel laceration from the internal iliac (hypogastric) arterial tree** (Fig. 18-27). Such lacerations may result from instrumented vaginal delivery, inadequate hemostasis of the uterine arteries at the time of cesarean delivery, or uterine rupture during a trial of labor after cesarean delivery (TOLAC). **Treatment of a retroperitoneal hematoma typically involves laparotomy, hematoma evacuation, and arterial ligation. In some situations, selective arterial embolization may be used as a primary or adjunctive treatment.**

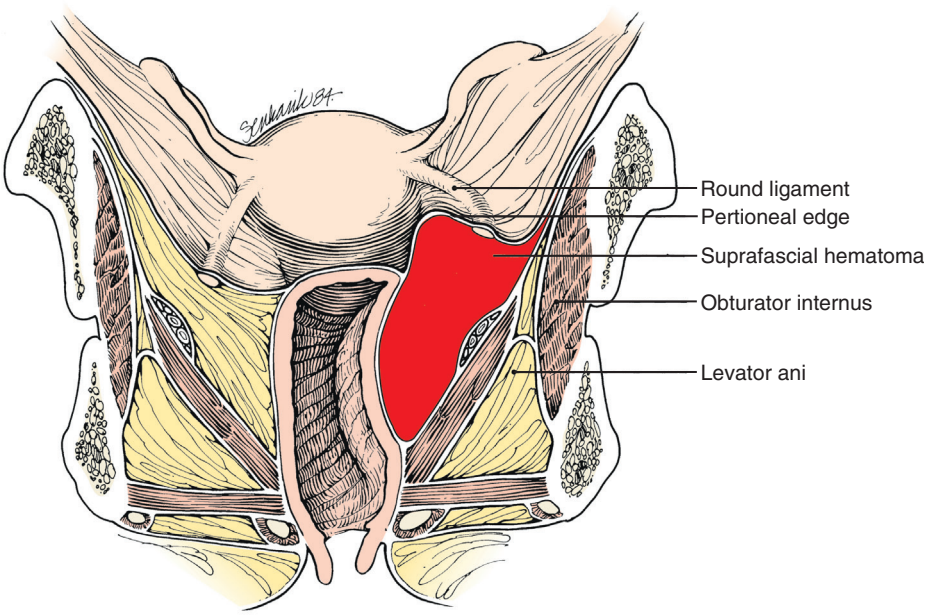

FIG 18-26 Vaginal hematoma.

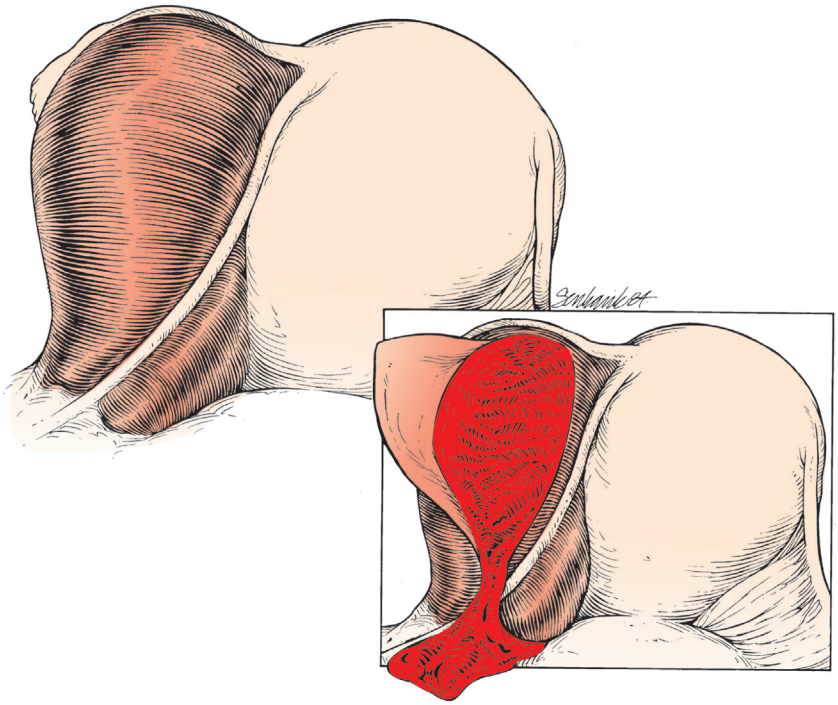

FIG 18-27 Retroperitoneal hematoma.

Retained Products of Conception

DEFINITION AND PATHOGENESIS

Retained products of conception, namely placental tissue and amniotic membranes, can inhibit the uterus from adequate contraction and can result in hemorrhage. The diagnosis is made when spontaneous expulsion of the tissue has not occurred within 30 to 60 minutes of delivery.

INCIDENCE AND RISK FACTORS

Retained products of conception complicate 0.5% to 1% of deliveries. Risk factors include midtrimester delivery, chorioamnionitis, and accessory placental lobes.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Retained products of conception typically present with uterine bleeding and associated atony. To assess the uterus for retained products of conception, the uterine cavity needs to be explored. Manual exploration is not only diagnostic but is also often therapeutic (Fig. 18-28). By wrapping the examination hand with moist gauze, removal of retained placental fragments and amniotic membranes can be facilitated. If manual access to the uterine cavity is difficult or limited owing to maternal body habitus or inadequate pain relief, **transabdominal or transvaginal ultrasound may be used to determine whether retained placental fragments are present.**[81]

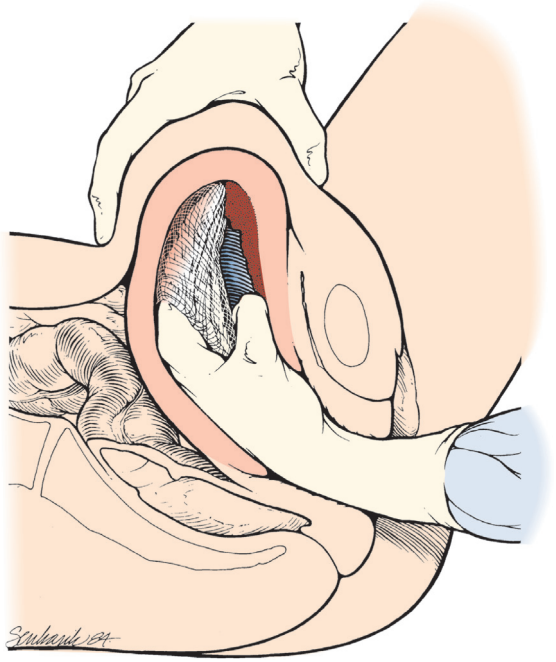

FIG 18-28 Manual uterine exploration.

MANAGEMENT

Once a diagnosis of retained products of conception is established, removal must be undertaken. Therapeutic options include **manual extraction,** as noted previously, or **uterine curettage.** Nitroglycerin (50 to 200 µg IV) has been used effectively to assist with manual placental extraction. Nitroglycerin provides rapid uterine relaxation to assist with removal of the retained tissue. Uterine curettage may be performed in a delivery room; however, excessive bleeding mandates that an OR be used for the procedure. Either a large blunt (Banjo or Hunter) curette or vacuum suction curette can be used. Transabdominal ultrasound guidance is helpful in determining when tissue evacuation is complete.

Uterine Rupture

DEFINITION AND PATHOGENESIS

Uterine rupture **refers to the complete nonsurgical disruption of all uterine layers**—endometrium, myometrium, and serosa. The severity of hemorrhage and maternal-fetal morbidity depends on the extent of the rupture. A large rupture may be associated with massive hemorrhage and extrusion of the fetus and/or placenta into the maternal abdomen; whereas a small rupture may have minimal bleeding and insignificant maternal-fetal consequences. *Uterine dehiscence* refers to an incomplete or occult uterine scar separation, in which the uterine serosa remains intact. Typically, no adverse obstetric outcomes are associated with a dehiscence.

INCIDENCE AND RISK FACTORS

The overall incidence of uterine rupture (scarred and unscarred uteri) is 1 in 2000 deliveries. Uterine rupture is most common in women with a scarred uterus, including those with prior cesarean delivery and myomectomy. The incidence of uterine rupture in women with prior cesarean delivery varies from 0.3% to 1%.[82] The location of the previous hysterotomy affects the uterine rupture risk. For prior cesarean deliveries, the risk of uterine rupture is illustrated in Table 18-5.

TABLE 18-5 RISK OF UTERINE RUPTURE BASED ON INCISION OF PRIOR CESAREAN DELIVERY

INCISION OF PRIOR CESAREAN DELIVERY	UTERINE RUPTURE RISK (%)
Classical	2-6
T or J shaped	2-6
Low vertical	2
Low transverse	0.5-1

From Landon MB, Hauth JC, Leveno KJ, et al. Maternal and perinatal outcomes associated with a trial of labor after prior cesarean delivery. *N Engl J Med.* 2004;351(25):2581.

Although **multiple risk factors** have been associated with uterine rupture, no single factor or combination of factors can reliably predict all cases.[83] Despite this, consistent data demonstrate strong associations for TOLAC and one or more of the following: **multiple prior cesarean deliveries, no previous vaginal delivery, induced or augmented labor, term gestation, thin uterine scar identified by ultrasound, multiple gestation, fetal macrosomia, postcesarean delivery infection, single-layer closure of hysterotomy incision, and short interpregnancy interval.**[84] Uterine rupture associated with TOLAC is discussed in Chapter 20. Other reported risk factors for uterine rupture include **increasing maternal age, multiparity, fetal malpresentation, uterine manipulation** (e.g., internal podalic version), **mid- to high-operative vaginal delivery, congenital uterine malformations, Ehlers-Danlos syndrome, invasive placentation, and trauma.**[85]

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Uterine rupture is associated with both fetal and maternal clinical manifestations. Fetal bradycardia with or without preceding variable or late decelerations is the most common clinical manifestation of symptomatic uterine rupture and occurs in 33% to 70% of cases.[86] In some circumstances, a **loss of fetal station in labor** may occur. **Maternal clinical manifestations** are variable and may include **acute vaginal bleeding, constant abdominal pain or uterine tenderness, change in uterine shape, cessation of contractions, hematuria** (if extension into the bladder has occurred), and **signs of hemodynamic instability.**

Uterine rupture is suspected clinically but confirmed surgically. Laparotomy will demonstrate complete disruption of the uterine wall with hemoperitoneum and partial or complete extravasation of the fetus into the maternal abdomen.

MANAGEMENT

Once the fetus and placenta are delivered, the **site of rupture should be assessed to determine whether it can be repaired. If feasible, the defect should be repaired in multiple layers with absorbable suture.** Adjacent structures (e.g., bladder and adnexa) should be assessed for damage and repaired accordingly. Hysterectomy is reserved for cases of massive hemorrhage, irreparable uterine defects, and/or maternal hemodynamic instability.

Uterine Inversion

DEFINITION AND PATHOGENESIS

Uterine inversion **refers to the collapse of the fundus into the uterine cavity, and it is classified by degree and timing. With regard to degree, uterine inversion may be first degree (incomplete), second degree (complete), third degree**

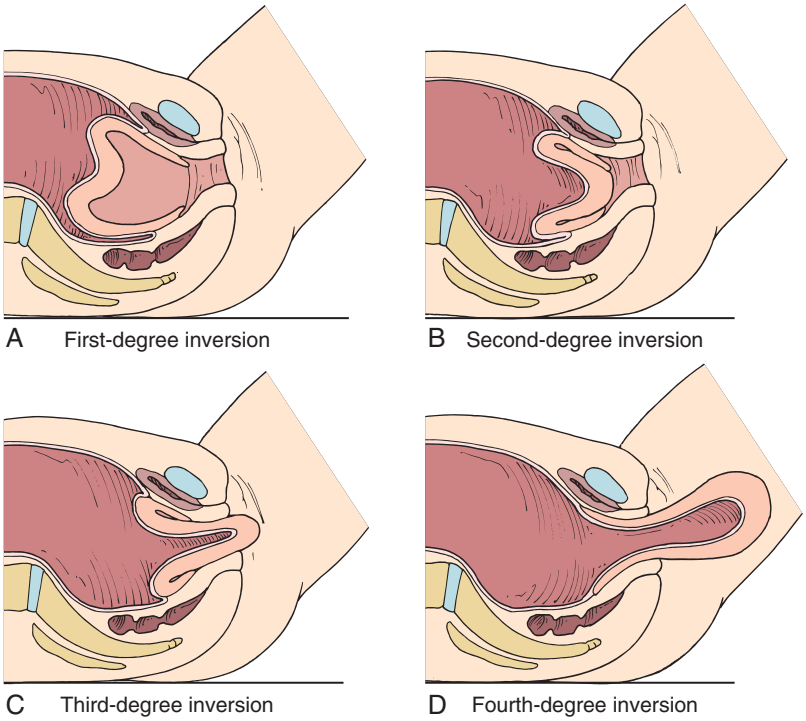

FIG 18-29 Classification of uterine inversion. (From Repke JT. *Puerperal uterine inversion,* www.uptodate.com, Dec 8, 2004.)

(prolapsed), or fourth degree (total; Fig. 18-29). *First-degree uterine inversion* represents a partial extrusion of the fundus into the uterine cavity. In *second-degree uterine inversion,* the internal lining of the fundus crosses through the cervical os, forming a rounded mass in the vagina with no palpable fundus abdominally. *Third-degree uterine inversion* refers to the entire uterus prolapsing through the cervix with the fundus passing out of the vaginal introitus. *Fourth-degree uterine inversion* represents both total uterine and vaginal prolapse through the vaginal introitus. Uterine inversion timing is classified as ***acute*** **(within 24 hours of delivery),** ***subacute*** **(>24 hours postpartum but <4 weeks), or** ***chronic*** **>1 month postpartum).**

The two most commonly proposed etiologies for uterine inversion include excessive umbilical cord traction with a fundally attached placenta and fundal pressure in the setting of a relaxed uterus. However, a causal relationship between active management of the third stage of labor and uterine inversion remains unproven.

INCIDENCE AND RISK FACTORS

Uterine inversion is a rare event that complicates about 1 in 1200 to 1 in 57,000 deliveries.[87] Proposed **risk factors** include **uterine overdistention, fetal macrosomia, rapid labor and delivery, congenital uterine malformations, uterine fibroids, invasive placentation, retained placenta, short umbilical cord, use of uterine-relaxing agents, nulliparity, manual placental extraction, and Ehlers-Danlos syndrome.**[87]

CLINICAL MANIFESTATIONS AND DIAGNOSIS

The clinical presentation of uterine inversion varies by its degree and timing. Whereas incomplete uterine inversion may be subtle in its clinical findings, complete uterine inversion often presents with **brisk vaginal bleeding, inability to palpate the fundus abdominally, and maternal hemodynamic instability.** It may occur before or after placental detachment. The diagnosis is made clinically with bimanual examination, during which the uterine fundus is palpated in the lower uterine segment or within the vagina. Sonography can be used to confirm the diagnosis if the clinical examination is unclear.[88]

MANAGEMENT

Once diagnosed, uterine inversion requires rapid intervention in order to restore maternal hemodynamic stability and control hemorrhage. Maternal fluid resuscitation through a large-bore IV catheter is recommended. The uterus must be replaced to its proper orientation to resolve the hemorrhage, which is best accomplished in an OR with the assistance of an anesthesiologist. The uterus and cervix should initially be relaxed with nitroglycerin (50 to 500 μg), a tocolytic agent (magnesium sulfate or β-mimetic), or an inhaled anesthetic. Once relaxed, gentle manual pressure is applied to the uterine fundus to return it to its proper abdominal location (Fig. 18-30). Uterotonic therapy should then be given to assist with uterine contraction and to prevent recurrence of the inversion.

If manual repositioning is unsuccessful, other options include hydrostatic reduction and surgical correction. With hydrostatic reduction, warmed sterile saline is infused into the vagina. The physician's hand or a Silastic ventouse cup is used as a fluid retainer to generate intravaginal hydrostatic pressure and resultant correction of the inversion (Fig. 18-31). Surgical options include the Huntington and Haultain procedures, laparoscopic-assisted repositioning, and cervical incisions with manual uterine repositioning.[89] The Huntington procedure involves a laparotomy with serial clamping and upward traction of the round ligaments to restore the uterus to its proper position. If this technique fails, the Haultain procedure can be attempted, which includes a vertical incision within the inversion and subsequent repositioning of the fundus. As with manual repositioning, uterotonic

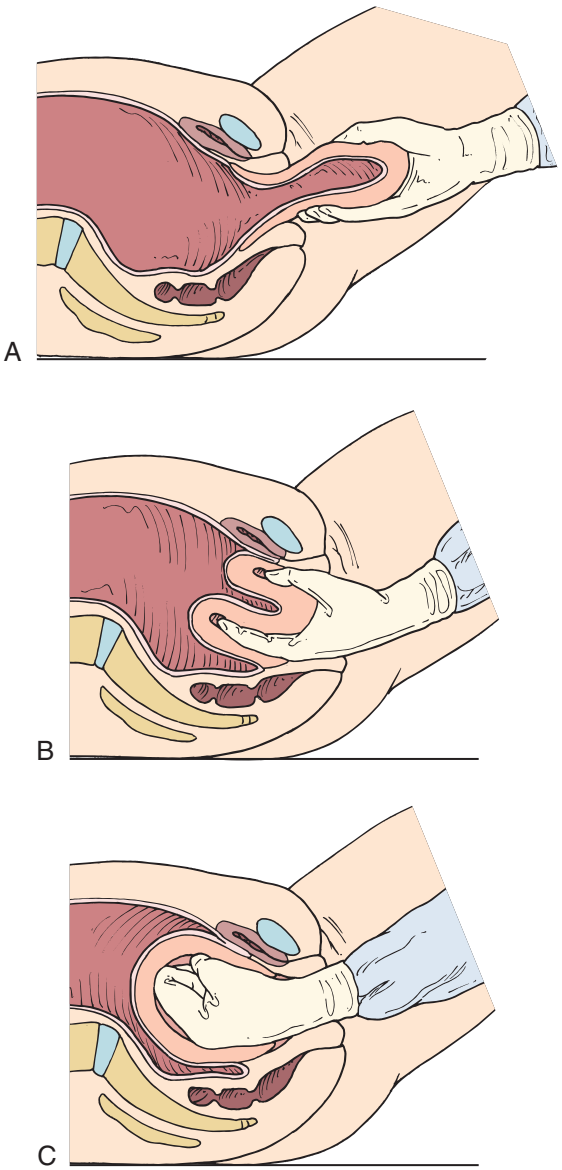

FIG 18-30 Manual replacement of uterine inversion. (From Repke JT. *Puerperal uterine inversion,* www.uptodate.com, Dec 8, 2004.)

therapy should be administered immediately after uterine replacement in order to prevent reinversion.

Coagulopathy

DEFINITION AND PATHOGENESIS

Coagulopathy represents an imbalance between the clotting and fibrinolytic systems. This imbalance may be hereditary or acquired in origin. *Hereditary coagulopathies* are relatively rare and have variable etiologies. Although ***acquired coagulopathy*** can be iatrogenic, such as that associated with anticoagulant administration, it is **usually the result of clotting factor consumption.** Figure 18-32 demonstrates the pathophysiology of consumptive coagulopathy and its association with hemorrhage.

INCIDENCE AND RISK FACTORS

The overall incidence of coagulopathy in the obstetric population has not been reported; however, several **associated clinical conditions** have been documented. These include **massive antepartum or postpartum hemorrhage; sepsis; severe PE; hemolysis, elevated liver enzymes, low platelets (HELLP) syndrome; amniotic fluid embolism; fetal demise; placental abruption; septic abortion; and acute fatty liver of pregnancy.**

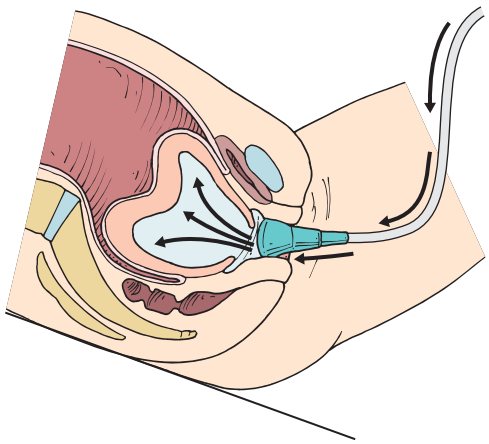

FIG 18-31 Hydrostatic reduction for uterine inversion. (From Repke JT. *Puerperal uterine inversion,* www.uptodate.com, Dec 8, 2004.)

CLINICAL MANIFESTATIONS AND DIAGNOSIS

The primary clinical manifestations of consumptive coagulopathy include bleeding, hypotension out of proportion to blood loss, microangiopathic hemolytic anemia, acute lung injury, acute renal failure, and ischemic end-organ tissue damage.

Consumptive coagulopathy is a clinical diagnosis that is confirmed with laboratory data, such as evidence of thrombocytopenia, hemolytic changes on the peripheral blood smear, decreased fibrinogen, elevated fibrin degradation products, and prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT). When timely laboratory assessment is unavailable, drawing 5 mL of maternal blood into an empty red-topped tube and watching for clot formation will provide the clinician with a rough estimate of the degree of existing coagulopathy. If a clot is not visible within 6 minutes or forms and lyses within 30 minutes, the fibrinogen level is usually less than 150 mg/dL.

MANAGEMENT

The most important factor in the successful treatment of coagulopathy is identifying and correcting the underlying etiology. For most obstetric causes, **delivery of the fetus initiates resolution of the coagulopathy.** In addition, rapid replacement of blood products and clotting factors should occur simultaneously. The patient should **have two large-bore IV catheters for fluid and blood component therapy, and laboratory studies should be drawn serially every 4 hours until resolution** of the coagulopathy is evident. The obstetrician should attempt to achieve a hematocrit greater than 21%, a platelet count greater than 50,000/mm^3, a fibrinogen level greater than 100 mg/dL, and PT and aPTT less than 1.5 times control. It also is important **to maintain adequate oxygenation and normothermia.** Finally, adjuvant therapies should be

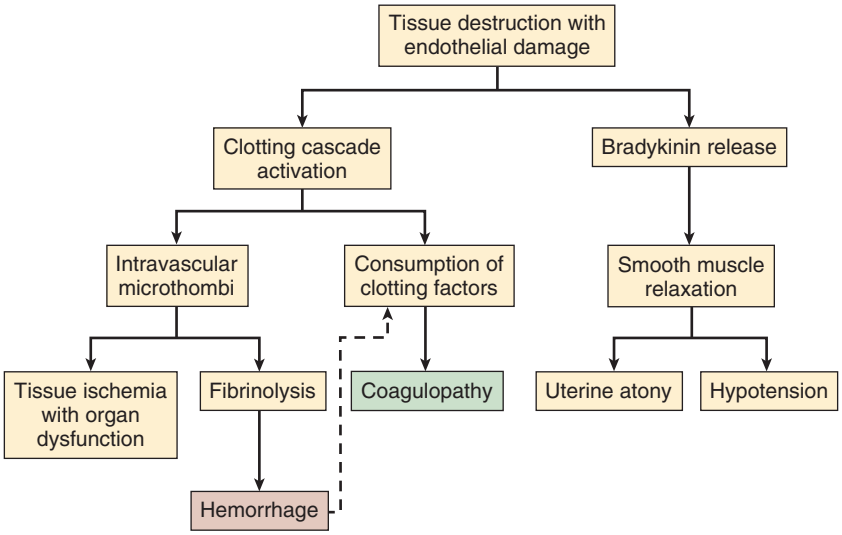

FIG 18-32 Pathophysiology and clinical manifestations of consumptive coagulopathy.

considered such as vitamin K, recombinant activated factor VII, fibrinogen concentrate, prothrombin complex concentrate, tranexamic acid, and hemostatic agents.

VITAMIN K

Factors II, VII, IX, and X are vitamin K–dependent clotting factors. In consumptive coagulopathy, these clotting factors are consumed. Administration of vitamin K (5 to 10 mg) by subcutaneous, intramuscular, or IV routes can assist with endogenous replenishment of these procoagulants.

RECOMBINANT ACTIVATED FACTOR VII

Factor VII is a precursor for the extrinsic clotting cascade. When massive procoagulant factor consumption occurs, replacement is necessary. **Human recombinant factor VIIa has been used successfully in cases of consumptive coagulopathy attributed to postpartum hemorrhage.**[90] The dosage of this IV therapy has ranged from 16.7 to 120 μg/kg.[90] An important advantage of this therapy is its rapid bioavailability (10 to 40 minutes) for reversal of coagulopathy; however, disadvantages include a relatively short half-life (2 hours), cost (approximately $1 per microgram), and thromboembolism risk. Recombinant activated factor VIIa should be considered in cases of refractory coagulopathy or when blood component replacement is delayed.

FIBRINOGEN CONCENTRATE

A human fibrinogen concentrate (RiaStap [CSL Behring]) has been approved by the U.S. Food and Drug Administration (FDA). Each vial of fibrinogen concentrate contains 900 to 1300 mg fibrinogen and 400 to 700 mg human albumin. Fibrinogen concentrate can be used alone or in combination with cryoprecipitate. It has been used successfully in Europe for the treatment of massive obstetric hemorrhage due to consumptive coagulopathy.[91]

PROTHROMBIN COMPLEX CONCENTRATE

Prothrombin complex concentrate (Kcentra [CSL Behring]) contains factors II, VII, IX, and X and proteins C and S. It can be used as an alternative to fresh frozen plasma (FFP). Advantages to its use include no need for thawing or blood group typing and decreased risks of volume overload, transfusion-related acute lung injury (TRALI), and allergic reactions.

TRANEXAMIC ACID

Tranexamic acid is an IV antifibrinolytic drug that may be used for the prevention and treatment of hemorrhage. In a small multicenter trial, tranexamic acid reduced postpartum hemorrhage and transfusion requirements.[92]

HEMOSTATIC AGENTS

A variety of topical hemostatic agents are available for control of coagulopathic surface bleeding. These agents have different clotting factors and mechanisms of action. Examples include oxidized regenerated cellulose (e.g., Surgicel [Ethicon]), fibrin sealants (e.g., Tisseal [Baxter]), microporous polysaccharide spheres (e.g., Arista [Bard Davol]), microfibrillar collagen (e.g. Avitene [Bard Davol]), hemostatic matrices (e.g., Floseal [Baxter]), gelatin matrices (e.g., Gelfoam), and topical thrombin. These agents may be used alone or in combination.

Fluid Resuscitation and Transfusion

All obstetricians will encounter antepartum and postpartum hemorrhage. In most instances, **fluid resuscitation and blood component therapy are life-saving.** Therefore every physician should have a thorough understanding of appropriate volume resuscitation, transfusion therapy, and alternative treatment options and their risks.

Volume Resuscitation

Initial management of a hemorrhaging patient requires appropriate volume resuscitation. Two large-bore IV catheters are recommended. **Warmed crystalloid solution in a 3:1 ratio to the estimated blood loss should be rapidly infused.** Goals of therapy are to maintain an adequate maternal blood pressure (systolic blood pressure >90 mm Hg) and urine output (at least 30 mL/hr). If the hemorrhage is easily controlled, this may be the only therapy needed. The patient should have serial assessments of her vital signs and hematologic profiles to confirm hemodynamic stability.

TABLE 18-6 BLOOD COMPONENT THERAPY

PRODUCT	CONTENTS	VOLUME	ANTICIPATED EFFECT (PER UNIT)
Whole blood	All components	500 mL	Used only in emergencies*
Packed red blood cells	Red blood cells	300 mL	Increase hemoglobin by 1 g/dL Increase hematocrit by 3%
Platelets (single donor pooled)	Platelets	300 mL (6 U)	Increase platelet count by 30,000 to 60,000/mm^3
Fresh frozen plasma	All clotting factors	250 mL	Increase fibrinogen by 5-10 mg/dL
Cryoprecipitate	Fibrinogen, vWF, factors VIII and XIII	10-15 mL	Increase fibrinogen by 5-10 mg/dL

vWF, von Willebrand factor.

COLLOID SOLUTIONS

Colloid solutions contain larger particles, *colloids*, which are less permeable across vascular membranes. These solutions provide a greater increase in colloid oncotic pressure and plasma volume; however, they are more expensive than crystalloids and may be associated with anaphylactoid reactions. Examples of colloid solutions include albumin, hetastarch, and dextran.

Blood Component Therapy

WHOLE BLOOD

Whole blood contains red blood cells (RBCs), clotting factors, and platelets. Whole blood is rarely used in modern obstetrics because of its many disadvantages, including a short storage life (24 hr), large volume (500 mL per unit), and potential for hypercalcemia.

PACKED RED BLOOD CELLS

Packed red blood cells (pRBCs) are the most appropriate therapy for patients who require RBC replacement because of hemorrhage. They are the only blood product to provide oxygen-carrying capacity. **Each pRBC unit contains approximately 300 mL of volume (250 mL RBCs and 50 mL of plasma). In a 70-kg patient, one unit of pRBCs will raise the hemoglobin by 1 g/dL and the hematocrit by 3%.** Transfusion of pRBCs should be considered in any gravida with hemoglobin less than 8 g/dL or with active hemorrhage and associated coagulopathy.

PLATELET CONCENTRATES

Platelets are separated from whole blood and are stored in plasma. Because 1 unit of platelets provides an increase of only approximately 7500/mm^3, platelet concentrates of 6 to 10 units need to be transfused. Platelet concentrates can be derived from multiple donors or single donors. The single-donor concentrates are preferred because they expose the patient to fewer potential antigenic and immunologic risks. Transfusion of a single-donor platelet concentrate will increase the circulating platelet count by 30,000 to 60,000/mm^3. Because sensitization can occur, it is important for platelets to be ABO and Rh specific. **Transfusion of platelets should be considered when the platelet count is less than 20,000/mm^3 after a vaginal delivery or less than 50,000/mm^3 after a cesarean delivery, or when coagulopathy is evident.**

FRESH FROZEN PLASMA

FFP is plasma that is extracted from whole blood. FFP primarily contains fibrinogen, antithrombin, and clotting factors V, XI, and XII. Transfusing FFP not only assists with coagulation but also provides the patient with volume resuscitation because **each unit contains approximately 250 mL.** Typically, fibrinogen levels are used to monitor a patient's response to FFP. Each unit of **FFP should raise the fibrinogen level by 5 to 10 mg/dL,** and FFP does not need to be ABO or Rh compatible. FFP should be considered for hemorrhaging patients with evidence of consumptive coagulopathy, coagulopathic liver disease, and for warfarin reversal.

CRYOPRECIPITATE

Cryoprecipitate is the precipitate that results from thawed FFP. It is rich in fibrinogen, von Willebrand factor, and factors VIII and XIII. Like FFP, **cryoprecipitate can be measured clinically by the fibrinogen response, which should increase by 5 to 10 mg/dL per unit. Unlike FFP, each unit of cryoprecipitate provides minimal volume (10 to 15 mL),** so it is an ineffective agent for volume resuscitation. Cryoprecipitate is indicated for patients with coagulopathy and concerns of volume overload, hypofibrinogenemia, factor VIII deficiency, and von Willebrand disease.

Table 18-6 contains a summary of the available blood component therapies.

MASSIVE TRANSFUSION PROTOCOLS

Although no universally accepted guidelines exist for blood product replacement, expert opinion derived from trauma and military experience suggests that more aggressive replacement of coagulation factors with pRBCs improves outcomes and survival.[93-95] A variety of protocols have been recommended, most of which aim for an equal ratio of pRBCs, FFP, and platelets. **A commonly adopted massive transfusion protocol is 6 units of pRBCs to 4 units of FFP to 1 unit of platelet concentrate (6:4:1).**[93] Some centers have added **6 to 10 units of cryoprecipitate** to this regimen.

TRANSFUSION RISKS AND REACTIONS

METABOLIC ABNORMALITIES AND HYPOTHERMIA

When pRBCs are stored, leakage of potassium and ammonia can occur into the plasma. This may result in hyperkalemia and high ammonia concentrations in patients who require massive transfusion. In addition, because most pRBC units are stored in a citrate solution, hypocalcemia may occur. Serial assessments of metabolic profiles with ionized calcium levels can assist the clinician in managing these changes.

In addition to metabolic abnormalities, hypothermia may complicate the clinical course of massive transfusion and result in cardiac arrhythmias. Hypothermia can be prevented by warming pRBC units before transfusion and by providing alternative heating devices to the patient (e.g., Bair Hugger anesthesia warmer [3M]).

TABLE 18-7 TRANSFUSION-RELATED INFECTION RISKS

INFECTION	TRANSMISSION RISK
HIV-1, HIV-2	1 in 1.4-4.7 million
Hepatitis B	1 in 100,000-400,000
Hepatitis C	1 in 1.6 to 3.1 million
HTLV-I and -II	1 in 500,000 to 3 million
Bacterial contamination:	
Red blood cells	1 in 28,000-143,000
Platelets	1 in 2000-8000

From Hall NR, Martin SR. Transfusion of blood components and derivatives in the obstetric intensive care patient. In: Foley MR, Strong TH Jr, Garite TJ, eds: *Obstetric Intensive Care Manual,* 4th ed. New York: McGraw-Hill; 2014.
HIV, human immunodeficiency virus; *HTLV,* human T-cell lymphotropic virus.

IMMUNOLOGIC REACTIONS

Transfusions introduce interactions between inherited or acquired antibodies and the foreign antigens of the transfused blood products; the most common immunologic reactions are febrile nonhemolytic transfusion reactions. Cytokines are believed to be the primary cause of these reactions. Retrospective cohort studies suggest that transfusion of leukoreduced blood products may decrease the frequency of these reactions; however, randomized controlled trial (RCT) data are limited. Less common immunologic complications include acute or delayed hemolytic transfusion reactions, anaphylaxis, urticarial reactions, posttransfusion purpura, and graft-versus-host disease.

INFECTION RISKS

All blood products have the potential to transmit viral and bacterial infections. Although transmission rates have substantially decreased in the past two decades, they are still a potential risk that must be disclosed when a transfusion is needed. Table 18-7 lists current transfusion-associated infection risks.[96]

TRANSFUSION-ASSOCIATED VOLUME/CIRCULATORY OVERLOAD AND TRANSFUSION-RELATED ACUTE LUNG INJURY

Transfusion-associated volume/circulatory overload (TACO) refers to pulmonary edema that results from excessively large infusions of fluids and blood products. Symptoms include dyspnea, orthopnea, tachycardia, wide pulse pressure, hypertension, and hypoxemia. TACO typically is associated with elevated brain natriuretic peptide (BNP), central venous pressure, and pulmonary artery wedge pressure. TACO is usually treated with diuresis and supplemental oxygen.

Transfusion-related acute lung injury (TRALI) refers to a rare but potentially life-threatening form of acute lung injury that can result from blood product administration. TRALI is thought to result from a "two-hit" mechanism that involves neutrophil sequestration/priming and activation.[97] TRALI is characterized by sudden onset of hypoxemic respiratory insufficiency during or within 6 hours of blood product administration. Additional findings include noncardiogenic pulmonary edema, hypotension, fever, tachypnea, tachycardia, and cyanosis. Treatment is twofold and includes transfusion discontinuation and supportive care with oxygen administration, ventilatory support (continuous positive airway pressure [CPAP], bilevel positive airway pressure [BiPAP], or mechanical ventilation), hemodynamic stabilization (fluid resuscitation and/or vasoactive agents), and possible steroid administration.

Blood Conservation Approaches

Preoperative Autologous Blood Donation and Transfusion

Preoperative autologous blood donation and transfusion refers to the collection of the patient's own RBCs prior to surgery and reinfusion of those cells intraoperatively or postoperatively. Although it is unreasonable to have all pregnant individuals consider autologous blood donation, patients at high risk for transfusion (e.g., those with placenta previa or placenta accreta) may be good candidates. Guidelines for autologous blood donation include a minimum predonation hemoglobin of 11 g/dL, first donation within 6 weeks of anticipated delivery (because of the pRBC storage life of 42 days), a week-long interval between donations, and no donation within 2 weeks of anticipated delivery.[96]

Autologous transfusion should be used selectively. Not only is it significantly more expensive than homologous transfusion, but also the risks for bacterial contamination and subsequent homologous transfusion are not completely eliminated.

Acute Normovolemic Hemodilution

Acute normovolemic hemodilution refers to a blood conservation technique in which blood is removed from the patient preoperatively and is replaced by a crystalloid or colloid solution to maintain normovolemia. During surgery, the blood loss is diluted. The patient is reinfused with her more concentrated blood postoperatively. Acute normovolemic hemodilution can be considered for patients with good initial hemoglobin concentrations who are expected to have a blood loss of at least 1000 mL during surgery (e.g., placenta accreta).

Intraoperative Blood Salvage

Intraoperative blood salvage refers to collecting the patient's blood during surgery, filtering the blood, and then reinfusing the red cells back to the patient. Cell-Saver technology is the most widely used blood-salvage system. In the past, theoretic concerns regarding the risks for infection and amniotic fluid embolism limited the use of blood-salvage technology in obstetrics; however, **several studies have documented its safety and effectivenes.**[98] Intraoperative blood salvage has many advantages over homologous transfusion. It eliminates the risk for infectious disease transmission, alloimmunization, and immunologic transfusion reactions. It is a cost-effective procedure that avoids wastage of blood and thereby reduces the need for homologous transfusion. In addition, it can rapidly provide the patient with RBCs (1 unit of pRBCs per 3-minute interval). Finally, many Jehovah's Witnesses will accept intraoperative blood salvage because the blood remains in a continuous circuit and does not leave the OR.

Alternative Oxygen Carriers

Because some patients refuse to accept blood products (e.g., Jehovah's Witnesses) or are unable to be transfused owing to a lack of blood compatibility, oxygen carriers have been developed as alternatives to transfusion therapy. The two primary products are hemoglobin-based oxygen carriers and perfluorocarbons. Hemoglobin-based oxygen carriers use hemoglobin derived from animals or outdated human blood. The hemoglobin is separated from the red cell stroma and undergoes multiple filtration and polymerization processes before use. Perfluorocarbons are inert compounds that can dissolve gases, including oxygen.

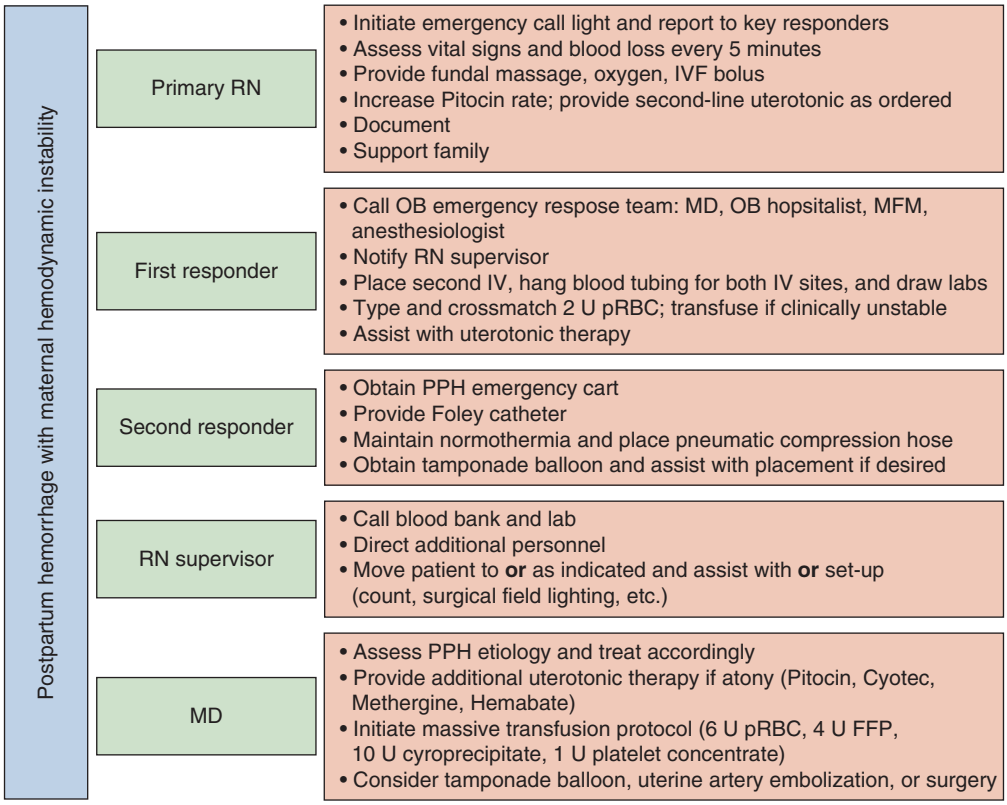

FIG 18-33 Postpartum hemorrhage treatment protocol. FFP, fresh frozen plasma; IV, intravenous; IVF, intravenous fluids; MD, physician; MFM, maternal-fetal medicine; OB, obstetric; PPH, postpartum hemorrhage; pRBC, packed red blood cells; RN, registered nurse. (Courtesy K. Francois.)

Both products have high oxygen-carrying capacities. Unfortunately, cost, supply problems, lack of FDA approval, and poor side-effect profiles have limited the usefulness of these agents.

Hemorrhage Prevention and Protocols

Because obstetric hemorrhage is such a widespread problem, it is important for institutions to develop standardized management protocols and to conduct hemorrhage drills. Multiple organizations have developed guidelines for the diagnosis, management, and prevention of postpartum hemorrhage. The use of obstetric rapid response teams, massive transfusion protocols, and prescriptive checklists is vital to program success. In addition, simulation-based teaching models have been helpful to identify knowledge deficits, improve accuracy of blood loss estimation, and instill provider confidence in the clinical management of postpartum hemorrhage.[99-102] Figure 18-33 provides a modified postpartum hemorrhage treatment protocol from the author's institution.

KEY POINTS

- Hemorrhage is a major cause of obstetric morbidity and mortality throughout the world. It is responsible for one third of all pregnancy-related deaths in both high- and low-income countries.
- Understanding the hemodynamic changes of pregnancy and the physiologic responses that occur with hemorrhage assists in appropriate management. Clinicians should recognize the four classes of hemorrhage to allow for rapid intervention.
- Placental abruption is diagnosed primarily by clinical findings and is confirmed by radiographic, laboratory, and pathologic studies. Management of placental abruption is dependent on the severity, gestational age, and maternal-fetal status.
- Placenta previa is typically diagnosed with sonography. Placenta previa remote from term can be expectantly managed, and outpatient management is possible in selected cases.
- Placenta previa in association with a prior cesarean delivery is a major risk factor for placenta accreta. Additional radiographic surveillance should be attempted in these cases to provide antenatal diagnosis of placenta accreta.
- Placenta accreta is best managed with a multidisciplinary approach that includes maternal-fetal medicine specialists, neonatologists, blood-conservation teams, anesthesiologists, advanced pelvic surgeons, and urologists. Scheduled preterm delivery at 34 to 35 weeks of gestation is recommended.
- Antenatal detection of vasa previa is possible with sonography and significantly improves perinatal outcomes.
- Postpartum hemorrhage complicates 1 in 20 to 1 in 100 deliveries. Every obstetrician and birth attendant needs to have a thorough understanding of normal delivery-related blood loss in order to recognize postpartum hemorrhage.
- Management of uterine atony should follow a rapidly initiated sequenced protocol that may include bimanual

massage, uterotonic therapy, uterine tamponade, selective arterial embolization, or surgical intervention.
- Coagulopathy mandates treatment of the initiating event and rapid replacement of consumed blood products. Transfusion of blood components should not be delayed, and replacement protocols should be followed.
- Blood conservation approaches should be considered if clinically appropriate.
- Standardized obstetric hemorrhage management protocols, checklists, and drills have been shown to improve outcomes.

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