Hemorrhage is commonly encountered in obstetric practice and is at times deadly. Historically, hemorrhage, along with hypertension and infection, have been the largest contributors to maternal mortality, which as recently as 1930 was 1% in the United States and Britain.\(^1\,^2\) Although the overall risk of death in childbirth is now greatly diminished, hemorrhage remains a major causative factor. Clark and colleagues\(^3\) reported on the causes of maternal death among 1.5 million deliveries from 2000 to 2006 and 12% were related to obstetric hemorrhage. Hemorrhage was the direct cause of death in 17% of the more than 4000 pregnancy-related deaths in the United States as reported by the Pregnancy Mortality Surveillance System of the Centers for Disease Control and Prevention.\(^4\,^5\) In the world, hemorrhage has been reported as the single most common cause of death and accounts for more than half of all deaths worldwide.\(^6\,^7\)

Although maternal mortality is low in the United States, there may have been a recent increase. The Joint Commission on Accreditation of Healthcare Organizations issued a sentinel event alert in 2010 in response to an increase in maternal mortality from 6 per 100,000 to 13 to 15 per 100,000.\(^8\) Although the rates are still quite low by historical standards, any increase is concerning when one recognizes the great improvements seen over the past 80 or so years.\(^1\) The recent increase in maternal mortality is likely multifactorial and related to increased maternal age at delivery and the associated comorbidities, improved management of chronic disease, and
increased elective interventions that result in an increased cesarean delivery rate. The latter greatly increases the risk of placenta accreta and may explain in part increased deaths due to obstetric hemorrhage. This is addressed in detail in the article by Wortman elsewhere in this issue.

Obstetric hemorrhage can be classified as antepartum, intrapartum, and postpartum. Antepartum hemorrhage is seen in placenta previa and sometimes abruption. Intrapartum hemorrhage is the focus of this article and has several causes and predisposing factors, as shown in Box 1. Many of these causes, such as uterine atony, are also associated with postpartum hemorrhage, which is usually immediate, although sometimes can be delayed. Increased risk of intrapartum hemorrhage from placenta accreta has been recently seen and is related to the unprecedented rise in cesarean deliveries. Although placenta accreta can be seen in an unscarred uterus, a cesarean delivery greatly increases the risk in subsequent pregnancies.

**INTRAPARTUM HEMORRHAGE**

The consequences of intrapartum hemorrhage are related to the degree of blood loss and the timeliness of resuscitative measures. The uterus at full gestation receives 600 mL of blood per minute, placing the woman at risk for massive amounts of blood loss in a short amount of time, a true obstetric emergency. The physiologic response to hemorrhage in the pregnant woman is no different from other patients experiencing acute blood loss. Initially, cardiac output, stroke volume, and mean arterial blood pressure decrease, tissue oxygen extraction increases, and pulmonary capillary wedge pressure falls. Redistribution of blood flow occurs through selective, arteriolar-mediated vasoconstriction and results in diminished perfusion to the kidneys, skin, and uterus while maintaining flow to the heart and brain. Cardiac output is maintained.

<table>
<thead>
<tr>
<th>Box 1</th>
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<tbody>
<tr>
<td><strong>Some causes and predisposing factors of obstetric hemorrhage</strong></td>
</tr>
<tr>
<td><strong>Abnormal placentation</strong></td>
</tr>
<tr>
<td>• Previa</td>
</tr>
<tr>
<td>• Abruption</td>
</tr>
<tr>
<td>• Accreta</td>
</tr>
<tr>
<td><strong>Injury during labor and delivery</strong></td>
</tr>
<tr>
<td>• Vaginal or cervical laceration</td>
</tr>
<tr>
<td>• Uterine rupture</td>
</tr>
<tr>
<td>• Uterine laceration during cesarean delivery</td>
</tr>
<tr>
<td><strong>Uterine atony</strong></td>
</tr>
<tr>
<td>• Overdistended uterus</td>
</tr>
<tr>
<td>o Macrosomia</td>
</tr>
<tr>
<td>o Multiple gestation</td>
</tr>
<tr>
<td>o Polyhydramnios</td>
</tr>
<tr>
<td>• Labor induction</td>
</tr>
<tr>
<td>• Prolonged labor</td>
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<tr>
<td>• Rapid labor</td>
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</tbody>
</table>
through increases in the heart rate and contractility. Systemic vascular resistance increases as well. Adequate resuscitative measures during this initial response will quickly reverse the developing shock and full recovery will typically be seen if the source of the hemorrhage is identified and controlled.

If the blood volume deficit exceeds 25 volume percent, cardiac output and blood pressure will begin to drop and there will be rapid clinical deterioration.\textsuperscript{11–14} The initial benefits of redistribution of blood flow will be lost as cellular hypoxia and death begin to occur in the organs that blood flow is being shunted away from. The resultant tissue and organ damage lead to an inflammatory response and endothelial activation. This endothelial involvement leads to loss of the integrity of the vascular wall, increased cell membrane permeability, and capillary leak.

Pregnancy has a couple of advantages when compared with other clinical scenarios, both related to maternal age and pregnancy-related maternal adaptation, which includes volume expansion. The average maternal age at delivery is 24 years and occurs at a time in life when women are at their peak physiologic condition.\textsuperscript{4} Women at this age are typically healthy and do not have significant comorbidities. Unlike other patient populations, women who are pregnant handle moderate amounts of hemorrhage well and usually recover unscathed. More importantly than this, the adaptation of blood volume to pregnancy confers a protective advantage against excessive blood loss that is unique. This volume expansion begins at the end of the first trimester and results in a 50% increase in the blood volume, which leads to an additional 1000 to 1500 mL of circulating blood volume at the time of delivery. Considering that the average blood loss at vaginal delivery is 500 mL and at cesarean delivery is 1000 mL, most women have ample reserve at term for routine blood loss and moderate amounts of hemorrhage.\textsuperscript{15}

There are some conditions in which maternal volume expansion does not occur. Women with preeclampsia typically have decreased plasma volumes as compared with those who do not have preeclampsia.\textsuperscript{16} The degree of contraction appears to be related to the severity of disease and has been reported to be a 50% decrease from normal in nulliparous women with eclampsia. There continues to be debate about whether this occurs after clinical onset of the disease and is really a volume constriction or if the changes occur before the onset of disease. Chronic renal disease has also been associated with abnormal volume expansion. The consequence of this is profound, as these women will not tolerate hemorrhage and even the normal blood loss seen at delivery may result in decompensation and hemorrhagic shock.

IDENTIFICATION OF EXCESSIVE BLOOD LOSS

The first key to management of intrapartum hemorrhage is to recognize its occurrence in a timely fashion. Bleeding often begins at the time of placental separation, and although it can be brisk and obvious, it is sometimes more subtle and can be steady and relentless. In the face of other bodily fluids associated with delivery, including amnionic and urine that are also present, the amount of blood loss is often underestimated. Pritchard and colleagues\textsuperscript{17} reported that the estimated blood loss is commonly only half the actual loss. Although techniques such as calibrated drape markings can improve accuracy, underestimation is still common.\textsuperscript{18,19} The steady bleeding seen initially appears moderate, but will persist until serious hypovolemia has occurred. Further complicating this is the failure of the pulse and blood pressure to change significantly until large amounts of blood have been lost. In fact, as peripheral vasoconstriction occurs and shunting of blood to the core organs begins, the woman may initially become hypertensive.\textsuperscript{20} This may be particularly pronounced in
preeclampsia where the blood pressure is already high. One of the most accurate measures of the degree of blood loss is urine output. The kidneys are particularly sensitive to acute blood loss and subsequent hypovolemia and will decrease urine production relatively quickly. Ultimately, experience may be the most significant factor in identifying hemorrhage quickly. Because of the subtleties involved in the initial diagnosis of excessive blood loss, it is often the appreciation that it is “more than normal” that prompts recognition of the blood loss and the emergency at hand.

Once identified, resuscitative measures should be quickly taken including obtaining intravenous access with 2 large-bore intravenous lines for rapid infusion of crystalloid and blood, placement of a Foley catheter to monitor urine output, an effort made to identify the cause (ie, uterine atony, genital laceration, or other diagnoses) and mobilization of surgical and anesthesia teams as needed.

CAUSES OF HEMORRHAGE

The cause of intrapartum hemorrhage can include placenta previa, accreta abruption, uterine atony that occurs immediately after delivery, uterine injury and laceration, vaginal or cervical laceration, and vaginal hematoma. Although labor can initiate the bleeding that occurs with a placenta previa, this more often occurs spontaneously and antepartum, so is not considered here. Accreta is also not commented on here, as it is covered in the article by Wortman elsewhere in this issue. Abruption is most often categorized as an antepartum event; however, because it is so often associated with labor, and the coagulopathy that ensues greatly complicates intrapartum management, it is covered in this article. Management of intrapartum hemorrhage is medical or surgical depending on the cause; medical management is covered with postpartum atony and surgical management is discussed after uterine, cervical, and vaginal injury and laceration.

Abruption

Abruption is the diagnosis given when the placenta separates from the uterus before the onset of labor. The bleeding that occurs is a result of this blood tracking between the membranes and uterus until it passes through the cervix. Often, a large amount of the blood lost is retained between the detached placenta and uterus, where it is concealed. Large amounts of blood can build up here and the extent of hemorrhage is usually not fully appreciated until delivery.21 The morbidity of an abruption is not only related to the volume of blood loss, but is also related to the coagulopathy that develops.

In one-third of abruptions severe enough to kill the fetus, significant hypofibrinogenemia occurs (fibrinogen levels of less than 150 mg/dL). This is because of tissue thromboplastin released into the maternal venous bloodstream.22,23 Abnormal activation of the extrinsic intravascular coagulation cascade and massive fibrinolysis then ensues. Fibrinogen-fibrin degradation products and d-dimers are seen and thrombocytopenia sometimes occurs. Delivery of the fetus stops the process and the abnormal coagulation status will correct itself relatively quickly, with appropriate resuscitation and transfusion of blood products. A vaginal delivery can be ideal as the uterus does not require an intact coagulation system for hemostasis; however, the fetus will not always tolerate labor in an abruption, and a cesarean will be required for fetal indication. When a cesarean delivery is required, significant bleeding will occur from the surgery if appropriate replacement of clotting factors does not occur. In abruption cases associated with a stillbirth, vaginal delivery is always preferred because of the coagulopathy so often present.22,24
Placental abruption occurs in 1 of every 200 deliveries. The National Center for Health Statistics reports an incidence of 1 in 160 in singleton gestations, and US birth certificate data from 2003 showed an incidence of 1 in 190 deliveries.\(^4,25\) Parkland hospital has reported a decrease in abruptions severe enough to kill a fetus from 1 in 420 between 1956 and 1967 to 1 in 1600 in the years between 1996 and 2003.\(^21\) Abruptions are most commonly associated with hypertension and abdominal trauma, but all of the following risk factors have been reported\(^{26-31}\):

- Maternal age older than 40
- African American or Caucasian race
- Premature rupture of the membranes
- Smoking
- Cocaine
- Leiomyomas
- Previous abruption
- Polyhydramnios

The management of abruption once identified is to effect delivery. If the fetus is alive, then a cesarean delivery will often be necessary, unless vaginal delivery is imminent or the abruption affects a small part of the placenta. Two very important caveats must be kept in mind:

- The blood loss may be much greater than is evident on vaginal/cervical examination
- A coagulopathy may be present even though clinical evidence may be lacking

With adequate resuscitative measures using crystalloid blood products, including red blood cells, plasma, and even platelets, the maternal outcome can be excellent even in cases in which the fetus is lost.

Although effecting delivery is necessary to resolve the abruption and save the fetus, delaying the delivery if the fetus is premature is sometimes beneficial.\(^{32,33}\) Bond and associates\(^32\) demonstrated a benefit to expectant management at less than 35 weeks with a reported 12-day delay in delivery from the time of diagnosis. No stillbirths were reported and most deliveries were by cesarean. This management should be approached with trepidation, and decompensation of the fetus may be sudden, resulting in stillbirth. Women undergoing expectant management of a preterm abruption should be hospitalized with immediate access to the delivery suite in case of acute decompensation.

**UTERINE ATONY**

The most common cause of hemorrhage is the failure of the uterus to contract after delivery of the placenta. Risk factors for uterine atony are well known, allowing anticipation of hemorrhage in many cases; however, they are only found in 50% of cases and are not typically predictive.\(^34\) Conditions that overdistend the uterus, including macrosomia, polyhydramnios, and multiple gestations, lead to atony. Grand multiparity is a risk factor with Babinski and colleagues\(^35\) and Fuchs and colleagues\(^36\) both reporting an increased risk of atony with higher parity. Prolonged labor is a long-recognized risk factor and includes both inductions and augmentations. Retained placenta fragments can lead to atony and this is especially true if a succinate lobe is present. Inspection of the placenta after delivery should be routine and the uterine cavity explored if it looks incomplete. **Box 2** summarizes the common risk factors for hemorrhage.
The treatment of uterine atony is usually medical and consists of administration of oxytocic and/or prostaglandin derivatives (Table 1). In addition, bimanual compression is performed, a relatively easy technique that controls most uterine hemorrhage. In this technique, the posterior aspect of the uterine fundus is massaged through the abdomen while the anterior wall of the uterus is massaged through the vagina with the other hand. This should be done while medical treatment is being given. In addition to bimanual compression and medical management, resuscitation of the woman is critical. An effective circulating blood volume is essential for uterine perfusion and for the drugs administered to act on the myometrium. Without appropriate resuscitation, hypovolemic shock will develop and the atony will become persistent and ultimately irreversible. Box 3 shows a typical algorithm for treating uterine atony.

### VAGINAL AND CERVICAL LACERATION

Spontaneous delivery is associated with varying degrees of injury to the vagina and cervix that range from superficial to deep and very serious lacerations. The deeper lacerations of the vagina can involve the anal sphincter; extend into lateral vaginal

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Dose</th>
<th>Route</th>
<th>Contraindication</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxytocin</td>
<td>20–30 mg per 1 L of fluid</td>
<td>IV or IM</td>
<td>None</td>
<td>Should be initiated after delivery of the placenta</td>
</tr>
<tr>
<td>Ergot derivatives</td>
<td>0.2 mg</td>
<td>IM</td>
<td>Hypertension</td>
<td>IV administration can cause hypertensive crisis</td>
</tr>
<tr>
<td>(methylergonovine)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prostaglandin</td>
<td>0.25 mg</td>
<td>IM</td>
<td>Asthma</td>
<td>Side effects include diarrhea, fever, flushing less effective than ergo or carboprost</td>
</tr>
<tr>
<td>analogs F2ₐ – carboprost</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prostaglandin F₂</td>
<td>0.20 mg</td>
<td>Per rectum</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: IM, intramuscular; IV, intravenous.
walls and even the ischiorectal fascia. It is of critical importance to recognize that bleeding after delivery while the uterus is contracted is suggestive of a genital tract laceration and/or retained placental fragments. Efforts to identify the source of the injury should be initiated immediately, as the bleeding may be brisk and can quickly lead to decompensation and shock.

CERVICAL LACERATION

The cervix is lacerated in up to 50% of normal vaginal deliveries. Most of these are short, heal rapidly, and bleed minimally. Occasionally the tear will extend up into the lower uterine segment, uterine artery, and even extend retroperitoneally. These lacerations bleed profusely and repair may be quite difficult, even requiring a transabdominal approach in the most extreme cases. In general, the surgical repair of the cervical lacerations can be effected from the vagina with appropriate assistance, effective anesthesia, and vigorous resuscitation and blood replacement if necessary.

VAGINAL HEMATOMAS

Sometimes there is damage to the vaginal arteries, typically branches of the pudendal, and a hematoma occurs. The incidence of puerperal hematoma varies from 1 in 300 to 1 in 1000 deliveries and the reported risk factors include nulliparity, episiotomy, and forceps delivery. Usually the hematoma is small, does not expand, and will tamponade off. In more significant cases, the bleeding will result in an expanding hematoma that will develop rapidly, cause intense personal pain, and may dissect into the ischiorectal fascia and retroperitoneal spaces. Smaller nonexpanding hematomas may be managed expectantly; however, larger hematomas must be evacuated. Zahn and Yeomans reported that half of women with a hematoma who require surgical intervention will also require a blood transfusion.

When incising and draining a hematoma, one should identify the point of maximal distension, open with a sharp scalpel, and evacuate the clot. If obvious, the bleeding artery should be identified and ligated, but often no obvious bleeding vessel is seen. In these cases, the cavity is closed in layers with hemostatic sutures placed and vaginal packing for 12 to 24 hours.

RESUSCITATION

The goal of resuscitation is to restore an effective circulating blood volume as efficiently as possible to avoid significant morbidity. Crystalloid solutions are typically

<table>
<thead>
<tr>
<th>Box 3</th>
<th>Typical steps initiated for control of hemorrhage caused by uterine atony</th>
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</thead>
<tbody>
<tr>
<td>1. Call for assistance</td>
<td></td>
</tr>
<tr>
<td>2. Initiate bimanual uterine compression</td>
<td></td>
</tr>
<tr>
<td>3. Confirm oxytocin is being administered</td>
<td></td>
</tr>
<tr>
<td>4. Start a second large-bore intravenous catheter for blood transfusions</td>
<td></td>
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<tr>
<td>5. Explore uterine cavity for retained placental fragments or lobes</td>
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</tr>
<tr>
<td>6. Inspect the cervix and vagina for lacerations</td>
<td></td>
</tr>
<tr>
<td>7. Insert a Foley catheter to monitor urine output</td>
<td></td>
</tr>
<tr>
<td>8. Begin volume resuscitation and consider a blood transfusion</td>
<td></td>
</tr>
</tbody>
</table>
used for initial volume resuscitation and are effective in cases of mild hemorrhage. The limitation of crystalloid resuscitation is due to reequilibration that results in only a proportion remaining in the intravascular circulation. Shoemaker and Kram\(^4\) reported that only 20% of crystalloid remains in the circulation at 1 hour. Because of this reequilibration, initial crystalloid resuscitation typically occurs at 3 times the estimated blood loss. Depending on the degree of blood loss and crystalloid used, volume overload can be an issue after the initial event as the third spaced fluid returns to the intravascular space.

Colloid is sometimes used for resuscitation and even advocated by many as an alternative to crystalloid, as it is more likely to stay intravascular. A recent Cochrane analysis, however, has demonstrated a significant increase in cost but no benefit in patient outcome in critically ill nonpregnant patients.\(^4\) The SAFE trial of 7000 nonrandomized nonpregnant patients reported similar results.\(^4\)

With continued or massive hemorrhage, red blood cell transfusions will ultimately be required. Although the exact threshold at which replacement should be initiated has never been determined, most agree that a hematocrit (Hct) less than 21% to 25% in the face of ongoing hemorrhage is appropriate.\(^1\) Although Morrison and colleagues\(^14\) demonstrated that an Hct of between 18% and 25% is well tolerated in women who are isovolumic, but anemic, their conclusion drives home the point that interpretation of the Hct value is dependent on the likelihood of continued and ongoing blood loss. That is, an Hct of between 18% and 25% may be tolerated if there is no further bleeding.

Most cases of hemorrhage will require 1 to 2 units of red blood cells (RBCs) and the Hct can be expected to rise 3 to 4 volume percent per unit transfused. In large-volume hemorrhage, RBCs alone will not suffice and plasma and platelets will also be required depending on the amount of hemorrhage and the number of units of RBCs transfused. This need usually occurs in cases that require more than 4 to 8 units of RBCs. The ideal ratio of fresh frozen plasma to RBCs transfused in these cases has not been determined and higher ratios than have been traditionally used were recently recommended, based on data recently published.\(^4\) In the face of this newer data, at least one large obstetric hospital has maintained a policy of transfusion of whole blood when available.\(^4\) Platelets are usually required when levels fall below 50,000/\(\mu\)L and in patients experiencing massive transfusion (defined as 10 units of packed RBCs or more). Ideally, platelets obtained by apheresis from a single donor are transfused. When this is unavailable, which is often, random donor platelets are used and typically 5 to 6 donors are required to make “1” unit of platelets. A “unit” of platelets will usually raise the recipients’ levels by 25,000 to 30,000.

Plasma is separated from whole blood immediately after donation and frozen for later use. When needed, it can be thawed in 30 minutes and is a source of all clotting factors, including fibrinogen. It should be given to correct clotting deficiencies related to large amounts of hemorrhage, as described previously. In addition, hemorrhage associated with a consumption coagulopathy, such as seen with abruption, typically requires plasma transfusion to increase fibrinogen levels if they fall to below 100 mg/dL. Cryoprecipitate is made from plasma and can be given if fibrinogen levels are very low and there is active oozing hemorrhage or oozing. It is particularly useful in cases in which volume overload is a concern. Transfusion-related acute lung injury (TRALI) has been described and is a life-threatening complication characterized by hypoxia, shortness of breath, and noncardiogenic pulmonary edema within 6 hours of transfusion and has been related to plasma infusion.\(^4\) It is said to occur because of injury to the pulmonary capillaries, secondly to lipid products contained in stored blood components and may complicate 1 in 5000 cases. Alexander and colleagues\(^4\)
did not identify any cases in the 1540 women transfused in their study, suggesting that this complication is rare. The diagnosis of TRALI is very difficult to make, especially because its signs and symptoms are very similar to those seen with the volume overload sometimes encountered with those patients. When encountered, treatment is supportive.

Recombinant Activated Factor VIIa is a synthetic vitamin K–dependent protein that has been approved by the Food and Drug Administration for use in individuals with hemophilia. Recently, its use in surgery, trauma, and obstetrics has increased to control severe hemorrhage. Recombinant Activated Factor VIIa, also known as NovoSeven, acts by binding to exposed tissue factor at the site of the tissue and vascular injury where it generates thrombin, which in turns activates platelets and the coagulation cascade. Thrombosis has been reported with the use of NovoSeven and when it occurs is usually seen with hemorrhage, not hemophilia cases. Reports of thrombosis in obstetrics cases seem to be uncommon at this time.

SURGICAL MANAGEMENT OF HEMORRHAGE

Uterine atony is initially treated with pharmaceutical and resuscitative measures. When these are unsuccessful, or the hemorrhage is the result of a surgical injury, such as a uterine incision extension, surgical management is necessary. Uterine artery ligation, sometimes known as an O’Leary stitch, can be used, especially for uterine artery extensions. This stitch is placed through the lateral uterine wall and encompasses the uterine artery.

Uterine compression sutures were first described by B-Lynch and colleagues in 1997. This technique and others like it compress the anterior and posterior walls of the bleeding uterus together. In a follow-up to the previous report, B-Lynch cited a failure rate of only 7 of 948 cases in which the technique was used. Although successful in many cases, there have been some case reports of ischemic necrosis with peritonitis. At least one report exists of a defect in the uterine wall in a subsequent pregnancy after a B-Lynch and Cho stitch was performed. Nevertheless, these compression sutures are relatively straightforward to perform, reported complications are isolated, and they may prevent a hysterectomy.

Uterine packing was used in the past to stop hemorrhage in women who desired to maintain their fertility, but fell out of favor because of concern over concealed hemorrhage and infection. Newer techniques have been developed that minimize some of these concerns and include use of a Foley catheter and the Bakri balloon. The Foley technique uses a 24F Foley catheter with a 30-mL balloon, which is inserted into the uterine cavity and filled with 60 to 80 mL of saline. This will compress the bleeding vessels, but the open tip also allows for drainage from the uterine cavity and monitoring of ongoing blood loss.

Angiographic embolization is sometimes resorted to when surgical access to bleeding vessels is difficult, such as in an intraperineal bleed. There are several studies reporting effectiveness with intractable hemorrhage and a 90% success rate. Although successful, pelvic embolization in the face of acute hemorrhage is logistically challenging and not always feasible. The patient may be too unstable for transport to the embolization laboratory and the interventional radiologist needed for the procedure may not be immediately available. Recently, prophylactic arterial catheter placement has been advocated for cases in which placenta percreta is likely. The increased accuracy of sonography in predicting these cases allows one to identify the women who are most likely to benefit. The prophylactic placement of the catheter allows for immediate embolization if heavy blood loss is encountered. Balloon-tipped catheters
can be used as well and can be inflated when needed, as opposed to embolization being used. Complications of embolization are low, but include unexpected thrombosis and necrosis of the embolized tissue, leading to infection.

SUMMARY

Intrapartum hemorrhage is commonly encountered in obstetric practice. It can be brisk, resulting in large amounts of blood loss, quickly leading to shock, end-organ damage, and ultimately death. Intrapartum hemorrhage is included in this edition of the Clinics because it constitutes an emergency. Like any obstetric emergency, prompt recognition, identification of the cause, and timely intervention will result in excellent outcomes. Intrapartum hemorrhage is primarily caused by uterine atony, vaginal and cervical lacerations, uterine injury during cesarean delivery, and sometimes abruption. Management may include medical or surgical approaches or both and always requires adequate resuscitation with crystalloid and often transfusion of RBCs and other blood products. Learning to recognize the signs and symptoms of shock and familiarization with techniques for treatment and resuscitation are critical to a successful outcome.

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