



Overview of postpartum hemorrhage

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Literature review current through: **Apr 2025**.

This topic last updated: **Apr 09, 2025**.

INTRODUCTION

Postpartum hemorrhage (PPH) is an obstetric emergency. It is one of the top five causes of maternal mortality in both resource-abundant and resource-limited countries, although the absolute risk of death from PPH is much lower in the former. Timely recognition, availability of appropriate resources, and appropriate response are critical for preventing death and severe maternal morbidity. Provider and institutional planning and preparation are essential to ensure an appropriate response. In addition to the traditional proven methods of hemorrhage control, several novel and improvised methods have been described that are promising and may provide clinicians with last-resort options in desperate situations where conventional management has failed [1].

This topic will present an overview of major issues relating to PPH. Clinical use of specific medical and minimally invasive interventions, and surgical interventions at laparotomy, for the management of PPH are discussed separately. (See "[Postpartum hemorrhage: Medical and minimally invasive management](#)" and "[Postpartum hemorrhage: Management approaches requiring laparotomy](#)".)

TERMINOLOGY

- **Primary or early PPH** – Occurs in the first 24 hours after giving birth (the subject of this topic).
- **Secondary, late, or delayed PPH** – Occurs from 24 hours to 12 weeks after birth (discussed separately). (See "[Secondary \(late\) postpartum hemorrhage](#)".)

CRITERIA FOR DIAGNOSIS

Our approach — We make the diagnosis of PPH in postpartum patients with bleeding that is greater than expected **and** results in signs and/or symptoms of hypovolemia ([table 1](#)). Some patients will have signs and/or symptoms of hypovolemia before excessive blood loss is seen because the bleeding is intraabdominal, retroperitoneal, or in the pelvic floor (eg, vaginal hematoma).

Excessive blood loss can be inferred from large studies evaluating uterotonic drugs for prevention of PPH. In such studies, at vaginal birth, less than 10 percent of patients receiving routine prophylaxis against PPH had blood loss ≥ 500 mL and less than 2 percent had blood loss ≥ 1000 mL [2]. At cesarean birth, 63 percent had blood loss > 500 mL and 30 percent had blood loss > 1000 mL [3].

Other criteria — Various criteria for diagnosis of PPH are used worldwide, as shown in the table ([table 2](#)). Although some guidelines utilize the classic definition of PPH for diagnosis (ie, estimated blood loss [EBL] ≥ 500 mL after vaginal birth or ≥ 1000 mL after cesarean birth), this is problematic because bleeding may not be visible externally, blood in collection devices may be mixed with amniotic fluid, and postpartum morbidity is relatively infrequent among patients with blood loss of 500 to 999 mL [4]. Because of these limitations, the American College of Obstetricians and Gynecologists (ACOG) revised its definition of PPH in 2017 to the following [5]:

- Cumulative blood loss ≥ 1000 mL, **or**
- Bleeding associated with signs/symptoms of hypovolemia within 24 hours of the birth process

The criteria apply to both vaginal and cesarean birth. However, ACOG emphasized that despite this updated definition, a blood loss >500 mL in a vaginal birth should be considered abnormal, particularly if heavy bleeding persists, and should prompt evaluation and close monitoring by the health care provider.

Classification of severity — PPH may be classified as severe, although criteria vary. In a systematic review of over 300 PPH studies from 1960 to 2024, those that included a severe PPH classification generally defined it as blood loss ≥ 1000 mL in the first 24 hours after birth as opposed to ≥ 500 mL [6]. The best classification systems are more detailed, considering factors such as signs and symptoms, mode of birth, ongoing bleeding, and tiered blood loss thresholds.

California Maternal Quality Care Collaborative staging system — The California Maternal Quality Care Collaborative (CMQCC) obstetric hemorrhage toolkit describes the following stages of PPH and level of intervention for each stage [7]:

- Stage 0 – Every patient in labor/giving birth
 - Intervention: Prophylactic [oxytocin](#), quantitate cumulative blood loss, closely monitor
- Stage 1 – Cumulative blood loss ≥ 500 mL at vaginal birth or ≥ 1000 mL at cesarean birth with continued bleeding or signs of concealed hemorrhage, such as vital signs that are abnormal or trending in that direction (heart rate ≥ 110 beats per minute [bpm], blood pressure $\leq 85/45$ mmHg, O₂ saturation <95%, confusion, or shock index [SI] >0.9 where SI = heart rate/systolic blood pressure).
 - Intervention: Activate institutional PPH protocol
- Stage 2 – Continued bleeding or vital sign instability, and cumulative blood loss <1500 mL
 - Intervention: Begin next level of interventions in the institutional PPH protocol, mobilize PPH rapid response team and blood bank support
- Stage 3 – Continued bleeding, with cumulative blood loss >1500 mL or transfusion of >2 units of red blood cells or abnormal vital signs or suspicion of disseminated intravascular coagulation

- Intervention: Move on to next level of interventions in the institutional PPH protocol, initiate massive transfusion protocol and surgical approaches for control of hemorrhage

Advanced trauma life support classification — The Advanced Trauma Life Support manual describes four classes of hemorrhage to emphasize the progressive signs and symptoms leading to shock [8]. The following classes were derived from nonpregnant populations and thus may be slightly different in postpartum patients, but remain useful:

- Class I hemorrhage involves a blood volume loss of up to 15 percent. The heart rate is minimally elevated or normal, and there is no change in blood pressure, pulse pressure, or respiratory rate.
- Class II hemorrhage involves a blood volume loss of 15 to 30 percent. It manifests clinically as tachycardia (heart rate of 100 to 120 bpm), tachypnea (respiratory rate of 20 to 24 breaths per minute), and decreased pulse pressure, although systolic blood pressure changes minimally, if at all. The skin may be cool and clammy, and capillary refill may be delayed. An increasing maternal heart rate and tachypnea with stable systolic blood pressure should be regarded as evidence of compensated shock and should prompt investigation and institution of a PPH protocol, even if only light vaginal bleeding is observed.
- Class III hemorrhage involves a blood volume loss of 30 to 40 percent, resulting in a significant drop in blood pressure and changes in mental status. Any hypotension (systolic blood pressure less than 90 mmHg) or drop in blood pressure greater than 20 to 30 percent of the measurement at presentation is cause for concern. While diminished anxiety or pain may contribute to such a drop, the clinician must assume it is due to hemorrhage until proven otherwise. Heart rate (≥ 120 bpm and "thready") and respiratory rate are markedly elevated, while urine output is diminished. Capillary refill is delayed.
- Class IV hemorrhage involves a blood volume loss of >40 percent, leading to significant depression in blood pressure and mental status. Most patients are hypotensive (systolic blood pressure less than 90 mmHg). Pulse pressure is narrowed (≤ 25 mmHg), and tachycardia is marked (>120 bpm). Urine output is minimal or absent. The skin is cold and pale, and capillary refill is delayed.

EPIDEMIOLOGY

PPH is generally reported to occur in 1 to 3 percent of births [9-11]. In an analysis of population-based data from the United States National Inpatient Sample, the PPH rate increased from 2.7 percent in 2009 to 4.3 percent in 2019 [11]. However, many reports are based on subjective estimates of blood loss; when blood loss is measured quantitatively, prospective studies have reported postpartum blood loss ≥ 500 mL in as many as 10 percent of births [12]. Variations in criteria for PPH (eg, >500 versus >1000 mL, presence/absence of symptoms) also contribute to variations in reported incidence.

Population-based surveillance data show an increasing frequency of severe PPH in the US and elsewhere [13,14]. This is likely related, at least in part, to increased rates of cesarean birth and the increased risk of placenta previa and placenta accreta spectrum (PAS) in patients with a prior cesarean birth.

Higher rates of PPH have been reported in some races/ethnicities [15], but race/ethnicity is not a biologic construct and does not make an individual physiologically at higher risk for hemorrhage [7]. The higher rate in some races/ethnicities has been attributed to disparities in quality of care.

PHYSIOLOGIC MECHANISMS THAT LIMIT POSTPARTUM BLOOD LOSS

Normally, hemostasis occurs upon placental separation because uterine bleeding is controlled by a combination of two mechanisms:

- Mechanical hemostasis, whereby contraction of the myometrium compresses the blood vessels supplying the placental bed, resulting in severely reduced blood flow.
- Local thrombosis, whereby the presence or release of local decidual hemostatic factors (tissue factor [16,17] and type-1 plasminogen activator inhibitor, respectively [18,19]) and systemic coagulation factors (eg, platelets, circulating clotting factors) lead to thrombosis of damaged blood vessels supplying the placental bed, resulting in severely reduced blood flow.

Abnormalities in these normal physiologic mechanisms have a high potential for massive hemorrhage because uterine artery blood flow in late pregnancy is 500 to 700 mL/min (versus 60 mL/min in the nonpregnant state) and accounts for approximately 15 percent of cardiac output.

RISK FACTORS FOR PPH

Many risk factors for PPH have been reported and are often interdependent [6]. Identification of most of these risk factors is of limited usefulness because of low positive and negative predictive values for PPH. (See '[PPH risk assessment tools and risk-based preparation](#)' below.)

In a systematic review (327 studies, >800 million patients) [6]:

- Risk factors with a strong association (OR >2) with PPH included: anemia, previous postpartum hemorrhage, cesarean birth, female genital mutilation, sepsis, no antenatal care, multiple pregnancy, placenta praevia, assisted reproductive technology use, macrosomia with a birth weight >4500 g, and shoulder dystocia.
- Risk factors with moderate association (OR 1.5 to 1.9) with PPH included: BMI ≥ 30 kg/m², COVID-19 infection, gestational diabetes, polyhydramnios, preeclampsia, and antepartum hemorrhage.
- Risk factors with weak association (OR >1 and <1.5) with PPH included: Black and Asian race/ethnicity, BMI 25 to 29.9 kg/m², asthma, thrombocytopenia, uterine fibroids, antidepressant use, induction of labor, vacuum- or forceps-assisted vaginal birth, and prelabor rupture of membranes.

CAUSES OF POSTPARTUM HEMORRHAGE

The most common causes of PPH can be considered using the Four Ts mnemonic [20]:

- Tone: uterine atony
- Trauma: laceration, rupture
- Tissue: retained tissue, blood clots, or placenta accreta spectrum (PAS)
- Thrombin: coagulopathy

Approximately 8 percent of patients with PPH have more than one cause [6].

Focal or diffuse atony — Uterine atony (ie, lack of effective uterine contraction after birth) prevents mechanical hemostasis from occurring. In a systematic review, it was responsible for 70.6 percent of all PPH cases and 41.4 percent of cases that were severe (defined as ≥ 1000 mL) or refractory (requiring second-line treatments [eg, additional uterotonics, bimanual uterine compression]) [6]. Atony-related PPH complicates approximately 1 in 40 births in the United States [21]. Although diffuse uterine atony is the most common cause of PPH, it is often responsive to the administration of uterotonic medications; thus, it is not the most common reason for massive intrapartum or postpartum transfusion [22]. Nevertheless, atony-related PPH is the indication for 27 percent of peripartum hysterectomies [23].

- **Diagnosis** – Atony is diagnosed when the uterus does not become firm to palpation after expulsion of the placenta. Administration of prophylactic uterotonic medications after birth is standard practice worldwide to prevent atony. (See ["Prophylactic pharmacotherapy to reduce the risk of postpartum hemorrhage"](#).)

With diffuse atony, the flaccid, dilated uterus may contain a significant amount of blood so blood loss can be much greater than observed. With focal localized atony, the fundal region may be well contracted while the lower uterine segment is dilated (ballooned) and atonic, which is difficult to appreciate on abdominal examination, but may be detected on vaginal examination or at cesarean birth.

- **Risk factors for atony** – Prior PPH and prolonged labor are the most well-established risk factors for atony-related PPH [24]. Other risk factors include chorioamnionitis, therapeutic use of [magnesium sulfate](#), labor induction or augmentation, fibroids, or uterine overdistention from multiple gestation, macrosomia, or polyhydramnios. Uterine inversion can be associated with fundal atony, even though there is constriction of the lower uterine segment/cervix.

Individuals with a PPH due to atony and requiring transfusion in their first pregnancy are at high risk for recurrence. In one study, over 10 percent of these patients had recurrent atony requiring transfusion in the next pregnancy [25]. A genome-wide association meta-analysis identified five loci associated with PPH [26]. These loci appeared to be involved with progesterone-binding and contraction of the myometrium.

Trauma — Trauma-related bleeding can be due to lacerations (including complete or partial myometrial rupture [27]) or surgical incisions. Cervical and vaginal lacerations may occur from natural processes during birth or as a result of provider interventions. Genital tract trauma accounted for 16.9 percent of cases of PPH and 12.8 percent of severe and refractory cases

of PPH in a systematic review [6]. In a series of 349 cases of massive PPH (ie, >2500 mL and/or ≥ 5 units red blood cell transfusion), trauma was the most common cause and accounted for 55 percent of cases compared with 23 percent for atony alone [28].

- **Diagnosis** – Tissue trauma after a vaginal birth is diagnosed on physical examination but may not be noted until excessive postpartum vaginal bleeding prompts careful examination of the lower genital tract beyond the perineum, including examination for vaginal and vulvar hematomas and cervical lacerations.

At cesarean birth, hemorrhage from the uterine incision is generally caused by lateral extension of the incision, which can result from spontaneous tearing of an edematous lower uterine segment after prolonged labor, a lower uterine segment incision that was too low or not sufficiently curved, or delivery of the fetus through an incision that is too small. Bleeding from a lateral extension of the uterine incision is readily ascertained by inspecting the incision, lateral pelvic sidewalls, and broad ligament. Retroperitoneal enlargement and bulging of the broad ligament at cesarean birth can be signs of retroperitoneal hemorrhage.

Uterine rupture, which may be located anteriorly, laterally, or posteriorly, also causes bleeding. (See "[Uterine rupture of the unscarred uterus: Risk factors, clinical manifestations, management, and outcome](#)" and "[Uterine rupture after previous cesarean birth: Prediction, clinical manifestations, diagnosis, management, and outcome](#)".)

- **Risk factors for trauma** – Risk factors for severe perineal trauma during vaginal birth include instrument-assisted vaginal birth, midline episiotomy, and persistent occiput posterior position [29]. In cesarean births, delivery after full dilation is a risk factor for unintended uterine incision extension, particularly in the setting of past cesarean birth or a failed vacuum attempt [30].

Placental disorders — Placental disorders, such as PAS, placenta previa, abruption, low-lying placenta, and retained placenta, cause PPH because effective uterine contraction and hemostasis of decidual vessels are inhibited, either focally or diffusely. In addition, abruption can trigger disseminated intravascular coagulation. Retained placenta accounted for 16.9 percent of cases and abnormal placentation accounted for 3.9 percent of cases of PPH in a systematic review [6]. Retained placenta and abnormal placentation accounted for 13.8 and 8.8 percent of cases, respectively, of severe and refractory PPH in the same review.

- **Diagnosis** – Placenta previa and PAS are typically diagnosed prenatally by ultrasound. The diagnosis of abruption is primarily based on history and physical examination; laboratory studies showing coagulopathy support the diagnosis. Retained placenta is diagnosed when the placenta has not been expelled within 30 minutes of birth.
- **Risk factors** – There are multiple risk factors for occurrence of a placental disorder. Prior cesarean birth is a risk factor for placenta previa and PAS. Hypertension is a risk factor for abruption. Uterine anomalies are a risk factor for retained placenta.
 - (See ["Placenta accreta spectrum: Clinical features, diagnosis, and potential consequences"](#).)
 - (See ["Placenta previa: Epidemiology, clinical features, diagnosis, morbidity and mortality"](#).)
 - (See ["Retained placenta after vaginal birth"](#).)
 - (See ["Acute placental abruption: Pathophysiology, clinical features, diagnosis, and consequences"](#).)

Coagulopathy or other bleeding diathesis — Coagulopathy (reduced hemostasis) was responsible for 2.7 percent of cases of PPH and 1.1 percent of severe and refractory cases of PPH in a systematic review [6]. It complicates approximately 1 in 500 births in the United States [21].

In patients with an inherited or acquired bleeding diathesis, coagulopathy or platelet dysfunction can cause PPH. In patients without an inherited or acquired bleeding diathesis, consumption and hemodilution of clotting factors during PPH can severely impair clotting and exacerbate bleeding. However, coagulopathy is a late finding in these cases. In a study including >18,000 patients with ≥ 1500 mL blood loss at birth, the median prothrombin time (PT) was 12.3 seconds, the longest activated partial thromboplastin time (aPTT) was 30.4 seconds, and the lowest fibrinogen was 360 mg/dL; all of these values are within the normal range for pregnancy.

- **Diagnosis** – In acute PPH, coagulopathy should be suspected in patients with one or more of the following: low fibrinogen level (<300 mg/dL), thrombocytopenia (<100,000/microL), prolonged PT (international normalized ratio [INR] >1.5), and/or prolonged aPTT (varies by laboratory).

Thromboelastography (TEG)/Rotational Thromboelastometry (ROTEM), where available, provides a global assessment of hemostasis in whole blood that includes contributions of platelets, fibrinogen, fibrinolysis, and coagulation factors. It can be

particularly useful for diagnosing dilutional coagulopathy. (See ["Postpartum hemorrhage: Medical and minimally invasive management"](#), section on 'Viscoelastic testing'.)

- **Risk factors for coagulopathy** – Acutely acquired coagulopathies can be caused by amniotic fluid embolism, placental abruption, preeclampsia with severe features, HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets), sepsis, or fetal demise. Patients with von Willebrand disease (VWD) are at increased risk for PPH because VWD factor levels, which typically increase during pregnancy, decline rapidly after birth. After an acute PPH event, an evaluation for VWD factor and platelet function may be warranted. (See ["Approach to the adult with a suspected bleeding disorder"](#).)
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INSTITUTIONAL PLANNING AND PREPARATION

Institutional planning and preparation for PPH has several components and should be proactive rather than reactive [7].

Patient education — All pregnant patients should receive verbal and printed educational material about PPH since most PPHs occur in low-risk patients and PPH may occur after hospital discharge [31]. (See ["Postpartum care"](#) below and ["Information for patients"](#) below.)

PPH risk assessment tools and risk-based preparation

- **Risk assessment** – In the United States, The Joint Commission requires use of an evidence-based tool for determining maternal hemorrhage risk both on admission to labor and delivery and on admission to a postpartum unit [31], but does not provide specific tools for risk assessment and management. The California Maternal Quality Care Collaborative (CMQCC) recommends intrapartum obstetric hemorrhage risk assessment on admission to the labor unit, at the start of the second stage of labor, at transfer to postpartum care, and any time the patient's condition changes [7].

Knowledge of risk factors for PPH and risk assessment tools have limited utility because many patients without risk factors experience severe PPH (eg, 40 percent of patients with PPH in one study had no risk factors [22]) [32,33] and most high-risk patients do not experience significant hemorrhage (risk of severe hemorrhage ranges from 2 to 7 percent [33-35]).

A systematic review of 14 prognostic models for PPH found that none were sufficiently validated in the general obstetric population, and three were potentially useful in high-risk populations (patients undergoing cesarean birth, patients with placenta previa or placenta accreta spectrum [PAS]) [36]. A retrospective study comparing the predictive value of the CMQCC, American College of Obstetricians and Gynecologists Safe Motherhood Initiative (ACOG SMI), and Association of Women's Health, Obstetric and Neonatal Nurses (AWOHNN), PPH risk stratification tools in over 11,000 patients admitted to the labor and delivery unit found that none performed well for predicting significant PPH within 48 hours after birth (defined as transfusion of at least one unit of red blood cells) [35]. Although high-risk patients had a higher incidence of significant PPH than low-risk patients, less than 5 percent of high-risk patients developed significant PPH (3.9-4.6 percent versus 1.5-1.8 percent in low-risk patients).

Despite the low predictive value of current risk assessment tools, use of a tool is likely better than no risk assessment as the risk stratification process may raise consciousness and preparation.

- **Risk assessment tools** – Several tools for risk assessment and risk-based management have been developed. Disadvantages of some prognostic models is that they apply to narrow patient populations, are overly complex for routine use, or include variables that are not routinely available or are only identifiable after PPH has occurred [37]. The advantage of the CMQCC tool and its derivatives are that they apply to the general obstetric population, can be administered by a labor and delivery nurse, include ongoing risk assessment, and can be embedded in the electronic health record:
- CMQCC [toolkit](#). The CMQCC risk classification scheme is used initially for patients admitted to the labor unit [7]. Although it classifies patients as low, medium, or high risk for PPH, in a validation study of an earlier version (version 1.0 in 2010), the incidence of severe PPH (ie, necessitating transfusion) in the three groups was 0.8, 2.0, and 7.3 percent, respectively, and only 22 percent of severe PPH cases occurred in the high-risk group [33].

The CMQCC tool also provides for ongoing risk assessment by including intrapartum and postpartum risk factors in the checklist (eg, retained placenta, cesarean or operative vaginal birth, 3rd or 4th degree laceration or uterine rupture, soaking >1 pad/hour, soaking 1 pad/hour for two consecutive hours, or passing a ≥ 6 cm clot).

The AWOHNN risk assessment tool is used by many facilities in the United States and is based upon the CMQCC tool.

An [online](#) tool created by the ACOG Safe Motherhood Initiative is similar.

The tool used at the author's institution ([table 3](#)) is also similar, but a disadvantage is that it does not include new risk factors developing over the course of labor and postpartum.

- Other tools are available on the UpToDate society guidelines links page for obstetric hemorrhage (see "[Society guideline links: Obstetric hemorrhage](#)").

Planning and intervention for selected groups of high-risk patients — Several UpToDate topics provide detailed information for planning and intervention for specific groups of high-risk patients:

- Patients who decline to accept blood transfusion (see "[Approach to the patient who declines blood transfusion](#)")
- Patients with PAS (see "[Placenta accreta spectrum: Clinical features, diagnosis, and potential consequences](#)" and "[Placenta accreta spectrum: Management](#)")
- Patients with placenta previa (see "[Placenta previa: Epidemiology, clinical features, diagnosis, morbidity and mortality](#)" and "[Placenta previa: Management](#)")
- Patients with bleeding disorders (Von Willebrand disease [VWD], hemophilia, congenital factor XIII deficiency, unclassified bleeding disorder or bleeding disorder of unknown cause) (see "[Perioperative blood management: Strategies to minimize transfusions](#)")
- Should all patients with PPH be screened for an inherited bleeding diathesis? – A prior PPH alone is not a strong indication for screening for inherited bleeding diatheses, given that undiagnosed bleeding disorders are a rare cause of PPH [38]. However, unexplained PPH that does not respond to general measures should alert clinicians to the possibility of a bleeding disorder as a causative factor [39], especially in patients with a history of heavy menstrual bleeding, excessive bleeding after minor trauma, or a family history of a bleeding disorder. (See "[Clinical manifestations and diagnosis of hemophilia A and B](#)", section on 'Obstetric considerations' and "[Anticoagulation during pregnancy and postpartum: Agent selection and dosing](#)", section on 'Labor and delivery' and "[Thrombocytopenia in pregnancy](#)", section on 'Management decisions' and "[von Willebrand disease \(VWD\): Gynecologic and obstetric considerations](#)", section on 'Obstetric considerations'.)

- Patients with thrombocytopenia (see ["Thrombocytopenia in pregnancy"](#))
- Patients with anemia (see ["Anemia in pregnancy", section on 'Management'](#))

Choice of birth facility for patients identified as high-risk prenatally — Patients identified prenatally as high risk for PPH should plan to give birth at a facility that has an appropriate level of care for their needs. For example, both PAS and twin pregnancy are risk factors for PPH. A patient with PAS should plan for delivery at a facility where multidisciplinary expertise is available (maternal-fetal medicine, anesthesiology, interventional radiology, blood bank, surgery [general, vascular, urology], neonatology), whereas a patient with a twin pregnancy is less likely to need this level of care.

PPH management protocols — Labor and delivery units should have a PPH protocol and provide ongoing training to their staff regarding its use [31,40-42]. The protocol should provide a standardized approach to evaluating and monitoring the patient with PPH, notifying a multidisciplinary team, and treatment. Development and consistent application of a comprehensive protocol for managing PPH appears to result in improved outcomes for these patients [43-45]. In an observational study, initiating a PPH protocol was associated with resolution of maternal bleeding at an earlier stage, a 26 percent reduction in use of blood products, a 15 percent reduction in peripartum hysterectomy, and a 64 percent reduction in disseminated intravascular coagulation [44,46].

In the United States, The Joint Commission requires obstetric units to have written evidence-based procedures (developed by a multidisciplinary team) for stage-based management of pregnant and postpartum patients who experience hemorrhage, including [31]:

- Use of an evidence-based tool that includes an algorithm for identification and treatment of hemorrhage
- Use of an evidence-based set of emergency response medication(s) that are immediately available on the obstetric unit
- A response team with a description of required team members and their roles in the event of severe hemorrhage
- A description of how the response team and procedures are activated
- Blood bank planning that includes emergency release of blood products and initiating massive transfusion
- Guidance on when to consult additional experts and consider transfer to a higher level of care
- Guidance on how to communicate with patients and families during and after the event
- Criteria for when a team debrief is required immediately after a case of severe hemorrhage

The author's institution uses a checklist system that addresses some of these requirements ([figure 1](#)).

Resources for developing a PPH protocol include:

- The [California Maternal Quality Care Collaborative](#) (CMQCC), which provides comprehensive information in several formats for management of PPH [7].
- Medical society and government-sponsored guidelines, which can be found on the UpToDate society guidelines links page for obstetric hemorrhage (see "[Society guideline links: Obstetric hemorrhage](#)"). There is large variation in obstetric and hematologic management of severe PPH across resource-abundant countries worldwide [47].

Massive transfusion protocol and algorithm — Massive transfusion is required to support patients with massive hemorrhage and facilitated by use of protocols specific to the hospital. Massive transfusion has been defined as transfusion of ≥ 10 units of whole blood or red blood cells in 24 hours, ≥ 3 units of red blood cells in one hour, or ≥ 4 blood components in 30 minutes, recognizing that blood loss is a continuum and these are arbitrary cutoffs. (See "[Massive blood transfusion](#)".)

All providers should be very familiar with application of their institution's protocol. Regular simulation of activation of PPH and massive transfusion protocols can improve compliance and facilitate team performance of low-frequency/high-complexity/high-risk events [48,49]. (See '[Training and simulation](#)' below.)

The protocol should describe activation criteria, process of activation (phone or electronic), how blood products are provided, type and frequency of laboratory testing, when to transfuse, and criteria for termination of the protocol [50]. It should include specific recommendations for empiric calcium replacement, potassium monitoring (hyperkalemia), and core body temperature management. Hypothermia must be prevented by warming fluids and blood products to be transfused, keeping the room warm, and covering the patient with warm blankets to the extent possible. Detailed information is available separately. (See "[Postpartum hemorrhage: Medical and minimally invasive management](#)", section on '[Transfuse blood products](#)' and "[Perioperative temperature management](#)".)

Equipment

PPH carts/kits — Planning for PPH involves ensuring the availability of resources that might be needed, including personnel, uterotonic and other medications, equipment to control bleeding, adequate intravenous access, topical hemostatic agents ([table 4](#)), and blood products. One way to achieve this is to assemble carts/kits that contain medications, devices, and instruments that may be needed to manage PPH so that these resources ([table 5](#)) are readily available when needed (similar to a "code cart") [7,49]. In the United States, The Joint Commission requires obstetric units to have a standardized, secured, dedicated hemorrhage supply kit stocked per the organization's defined process and, at a minimum, emergency hemorrhage supplies as determined by the organization and the organization's approved procedures for severe hemorrhage response [31].

Cell salvage — Use of a cell saver (blood salvage) is an option for patients at high risk of PPH undergoing cesarean birth. Routine use in all cesarean births is not cost-effective [51]. (See "[Postpartum hemorrhage: Management approaches requiring laparotomy](#)", section on 'Role of intraoperative cell salvage'.)

Cell salvage has been used successfully in patients with PPH after a vaginal birth, but data are sparse and the procedure should be considered investigational [52]. The potential presence of blood contaminants, including stool, urine, cleansing agents (eg, betadine, [chlorhexidine](#)), and vasoactive medications (eg, [misoprostol](#)) is a contraindication, thus severely limiting use.

Personnel

Multidisciplinary team — In the author's opinion, clinical training programs that encourage a team approach for early recognition of PPH can improve outcomes by engaging the spectrum of necessary providers before development of hypovolemia and uncompensated shock.

Obstetricians, maternal-fetal medicine specialists, midwives, nurses, anesthesiologists, hematologists, blood bank personnel, laboratory medicine, general surgeons and surgical subspecialists (eg, vascular, urology, gynecologic oncology), and interventional radiologists may be involved in managing PPH. These individuals are often summoned and required to work together under conditions of great stress and time pressures. Coordination is essential and can be facilitated by protocols and flow diagrams that anticipate how the team will communicate and function together [7].

Training and simulation — In the United States, The Joint Commission recommends that obstetric staff [31]:

- Undergo team training to teach staff to work together and communicate more effectively when PPH occurs
- Conduct clinical drills to help staff prepare for PPH
- Conduct debriefings after PPH to evaluate team performance and identify areas for improvement

Simulation team training can help to identify areas that need practice, and regular unannounced simulated PPH scenarios in a real-life setting, such as the labor and delivery unit or post-anesthesia care unit, may also increase comfort with the protocols and teamwork required in such emergencies [53]. (See "[Reducing adverse obstetric outcomes through safety sciences](#)", [section on 'Postpartum hemorrhage'](#).)

Post-event review — PPH cases that meet severity criteria established by the hospital should be reviewed to evaluate the effectiveness of the care, treatment, and services provided during the event [31].

EARLY RECOGNITION AND ASSESSMENT

Ongoing risk assessment/close observation enables early recognition of excessive bleeding and prompt mobilization of the resources needed for an appropriate response ([algorithm 1](#)). This principle applies globally to high-, middle-, and low-resource countries.

- A nationwide confidential enquiry of maternal deaths related to PPH in the Netherlands concluded that the key factors in preventing maternal death were [54]:
 - Early recognition of persistent bleeding
 - Prompt involvement of a senior clinician
 - Early determination of the cause of bleeding
 - Early assessment of severity of blood loss and presence of coagulopathy
 - Timely intervention to control bleeding (including timely recourse to surgical interventions, including hysterectomy, when other management options fail to stop bleeding)

- A randomized trial compared a multicomponent clinical intervention for PPH versus usual care in over 210,000 patients having a vaginal birth in 80 secondary-level hospitals across Kenya, Nigeria, South Africa, and Tanzania [55]. The components included all of the following:
 - Early detection and trigger criteria (eg, clinical monitoring and a calibrated blood-collection drape to quantify blood loss with triggers at 300 mLs if one abnormal clinical sign or at 500 mLs)
 - A bundle of first-line treatments (uterine massage, uterotonic medications, [tranexamic acid](#), intravenous fluids)
 - Examination (genital tract, placenta)
 - Escalation of response when necessary
 - An implementation strategy

The trial had a baseline observational phase (110,473 patients) followed by an implementation phase (99,659 patients).

In the implementation phase, the intervention resulted in a 60 percent reduction in the primary composite outcome (blood loss ≥ 1000 mL, laparotomy for bleeding, or maternal death from bleeding: 1.6 versus 4.3 percent with usual care; risk ratio 0.40, 95% CI 0.32-0.50), which likely resulted from much better detection of PPH (93.1 versus 51.1 percent; rate ratio 1.58, 95% CI 1.41-1.76) coupled with high adherence to an evidence-based treatment bundle (91.2 versus 19.4 percent; rate ratio 4.94, 95% CI 3.88-6.28).

Although these findings represent outcomes in a low-resource environment where nursing availability, expert consultant level clinicians, blood products, and access to surgical services are more limited than in high-resource countries, they validate the key principle that close observation (using a calibrated drape) and implementation of a bundle of actions in an incremental way is effective.

Quantify blood loss — The term "quantitative blood loss" (QBL) describes the systematic use of volumetric containers and weighing scales, or computerized image recognition, to quantify blood loss. The term "estimated blood loss" (EBL) describes a qualitative approach to blood loss. EBL is determined visually by looking at the extent to which bedsheets and pads are soaked with blood. It is less sensitive and specific than QBL [56].

We recommend QBL for all births because delay in the recognition of excessive blood loss delays the timely initiation of life-saving interventions and is a common finding in cases of maternal morbidity and mortality from hemorrhage [57-59]. Standardizing as many procedures as possible improves quality and safety; thus, if QBL is reserved only for cases of significant bleeding, staff may be unfamiliar with the process/procedures and less likely to obtain accurate data. With practice and routine adoption, QBL takes only minutes in most births [7]. The Association of Women's Health, Obstetric and Neonatal Nurses (AWOHNN) recommends QBL and many anesthesiologists prefer QBL; however, the American College of Obstetricians and Gynecologists (ACOG) has not recommended QBL over EBL.

QBL options include:

- **Volumetry** – Collect blood in graduated measurement containers, such as V-drapes with calibrated pockets and calibrated suction canisters.
- **Gravimetry** – Measure the total weight of bloody materials and subtract the known weight of the same materials when dry. The difference in weight between wet and dry in grams approximates the volume of blood in milliliters.
- **Colorimetry with artificial intelligence** – Use a smartphone application to calculate blood loss. The app analyzes photographs of used surgical gauze and canisters taken by the phone and then filters out the effects of nonblood components mixed into each sponge and canister. The hemoglobin mass present in the gauze or canister is then subtracted from the preoperative hemoglobin level. A meta-analysis found that this method correlated well with a validated reference, but more data are needed before it can be recommended for clinical use [60].
- **Visual aids** – Use visual aids (eg, posters) that correlate the size and appearance of blood on specific surfaces (eg, maternity pad, bed sheet, lap sponge) with the volume of blood absorbed by that surface ([picture 1](#)). Regularly scheduling standardized training in the use of these charts can be helpful. This approach is different from the traditional visual impression of EBL.

For each of the above methods other than colorimetry, the clinician should attempt to account for fluids other than blood (eg, amniotic fluid, irrigation fluid, urine) that are collected or absorbed.

A systematic review found that using a calibrated drape to measure the volume of blood loss, along with clinical observations (heart rate, blood pressure, uterine tone), had good sensitivity and specificity: 93 out of 100 patients with PPH would be identified and seven would be missed (false negatives) [56]. Five out of 100 patients without PPH would be wrongly diagnosed (false positive). Although objective methods of QBL are more accurate (visual estimation is more likely to underestimate the actual blood loss when volumes are excessive and overestimate when volumes are normal), use of objective methods or a specific objective method of QBL has not been proven to improve maternal and neonatal clinical outcomes [61]. Additional appropriately powered, randomized trials that correlate method of assessing blood loss with relevant clinical outcomes are needed

Calculated blood loss (CBL) is sometimes used in retrospective and research studies. It is the peripartum hematocrit change (estimated blood volume \times [(antepartum hematocrit–postpartum hematocrit)/antepartum hematocrit], where estimated blood volume [mL] = booking weight [kg] \times 85). In a study that compared QBL and CBL in over 8000 patients, QBL and CBL were moderately correlated after vaginal birth, but QBL was less than CBL and the difference increased with increasing blood loss [62].

Recognize alarm findings and intervene early — Timely recognition of PPH followed by rapid determination of the cause and initiation of appropriate treatment before the patient becomes moribund is critical to prevent death, as almost 90 percent of deaths due to PPH occur within four hours of giving birth [63,64]. Early intervention by a multidisciplinary team led by an experienced clinician(s) may prevent shock and the development of the potentially lethal triad of hypothermia, acidosis, and coagulopathy. The types and choices of intervention depend, in part, on whether the birth was vaginal or cesarean (with the abdomen still open), and are described in detail separately. (See "[Postpartum hemorrhage: Medical and minimally invasive management](#)" and "[Postpartum hemorrhage: Management approaches requiring laparotomy](#)".)

Alarm findings include:

- **Early warning vital sign criteria** – Standardized maternal early warning systems (MEWS) that target specific vital sign criteria ([table 6](#)) and mandate an immediate response at these thresholds can reduce maternal morbidity [65,66]. However, the immediate response does not always occur: Several maternal mortality review committees have found that delayed response to abnormal vital signs is a common factor in preventable mortality [67,68]. In some cases, the delay may be related to alarm fatigue because the trigger values used in the system have low specificity.

From a pragmatic perspective, it is wise to always assume, and rule out, PPH as the cause of symptoms of hypovolemia before assigning a less concerning diagnosis. It is important to recognize worrisome trends in vital signs because they may appear before early warning system criteria are met. A large reduction in blood pressure is a late sign of severe PPH as it generally does not manifest until substantial bleeding has occurred; up to 25 percent of a patient's blood volume (≥ 1500 mL in pregnancy) can be lost before systolic pressure falls to <90 mmHg, heart rate rises above 120 beats/minute, and respiratory rate rises above 30 breaths/minute [69]. This is the reason that blood loss ≥ 500 mL with continued bleeding for a vaginal birth and >1000 mL with continued bleeding for a cesarean birth are considered alarm triggers [7].

A lack of excessive vaginal bleeding does not exclude the diagnosis since the possibility of concealed hemorrhage must always be considered. Pain unrelieved by standard pharmacotherapy can be associated with tachycardia and can be a sign of concealed hemorrhage. Although vasodilatation due to neuraxial anesthesia and vasovagal reactions may result in lightheadedness/syncope, tachycardia, and hypotension, these entities are less likely postpartum than PPH, and they are readily reversible and generally not dangerous. Lightheadedness, tachycardia, or hypotension is unlikely to be due to neuraxial anesthesia if the patient was hemodynamically stable prior to birth, the level of the block did not become significantly higher immediately following birth, and symptoms did not abruptly follow systemic administration of a drug known to cause hypotension. Although fever from infection is associated with tachycardia, the combination tachycardia and fever does not exclude the possibility of coexisting infection and concealed hemorrhage. (See ["Adverse effects of neuraxial analgesia and anesthesia for obstetrics", section on 'Hypotension'.](#))

- **High shock index (SI)** – SI is calculated by dividing the heart rate by the systolic blood pressure (ie, HR/SBP). The upper limit of normal in obstetric patients appears to be higher than in nonpregnant patients, and has been reported to be 0.9 to 1.1 [70-74]. In a retrospective case-control study performed in a county safety net hospital, SI and delta-SI (ie, peak SI minus baseline SI) appeared to be superior to heart rate and systolic blood pressure in predicting PPH and the need for intervention (transfusion, surgery); SI >1.14 and SI >1.41 were strong "initial" and "critical" thresholds whereas SI ≤ 1.1 could be normal in peripartum individuals [75]. SI remained sensitive and specific when adjusted for potential confounders, including maternal age, maternal weight, gestational age at delivery, prior parity, laboring on presentation, preeclampsia, chorioamnionitis, and mode of delivery. Delta-SI was the strongest classifier overall. (See ["Postpartum hemorrhage: Medical and minimally invasive management", section on 'Initial patient assessment'.](#))

- **Low fibrinogen** – A fibrinogen level less than 200 mg/dL is an excellent predictor of severe PPH, defined as need for transfusion of multiple units of blood and blood products, need for angiographic embolization or surgical management of hemorrhage, or maternal death. It was the most frequently observed coagulation deficit in a cohort of patients with massive PPH, occurring in 17 percent of cases [28]. For this reason, measuring the fibrinogen level as soon as PPH is suspected and keeping the level above 200 mg/dL in patients at high risk for, or experiencing, PPH is important. The author of this topic aims for a level >300 mg/dL in patients with active bleeding where large amounts of blood products and crystalloid are often being transfused, given the higher normal baseline fibrinogen level in pregnancy and the desire to maintain the fibrinogen level well above the danger zone in these patients, although the benefit of increasing fibrinogen levels above 200 to 250 mg/dL in this setting has not been established.
- **Unexpected or increasing vasopressor requirements after placental delivery** – Because hypotension is a frequent side effect of neuraxial anesthesia for cesarean birth, best practice now includes routine, and preferably prophylactic, use of vasopressors to maintain blood pressure and placental perfusion [76]. The vasopressor requirements gradually decrease after the initial neuraxial-associated sympathectomy and the infusion is often weaned after fetal extraction. By the completion of surgery, the patient should be able to maintain their blood pressure and heart rate without the need for these medications. If the vasopressor cannot be weaned after delivery or the dose needs to be increased to maintain blood pressure, hemorrhage should be suspected.

Phenylephrine is a pure alpha-1 adrenergic receptor agonist with a longer duration of action than norepinephrine. It is the most commonly used vasopressor in obstetrics and is often administered as a continuous infusion to improve hemodynamic stability and minimize side effects (eg, nausea). It increases systemic vascular resistance and afterload, which can lead to baroreceptor-mediated decreases in heart rate. Venoconstriction exceeds arterial vasoconstriction, thus venous return increases [77]. The combination of the alpha-1 receptor arterial vasopressor effect (afterload increase), venoconstriction (increased venous return) and baroreceptor-mediated bradycardia may maintain what appears to be normal blood pressure and heart rate despite hemodynamically significant hemorrhage from ongoing bleeding and/or insufficient resuscitation. The anesthesiologist should inform the obstetrician about unexpected or increasing vasopressor requirements after placental delivery during and/or after cesarean birth to avoid a delay in evaluation for PPH.

Reinitiation of vasopressors after they have been discontinued also should prompt clinical concern for PPH. Vasopressor use should become a part of standardized intraoperative and immediate postoperative communication to facilitate a shared mental model about the patient's hemodynamic status.

Hemoglobin and hematocrit values are poor indicators of acute blood loss since they may not decline immediately after an acute bleed. It can take four hours for changes in laboratory values to be seen, and the nadir may not be seen for 48 to 72 hours [7]. (See ["Postpartum hemorrhage: Medical and minimally invasive management"](#), section on 'Laboratory evaluation'.)

Identify the cause of bleeding — Physical examination to determine the cause of hemorrhage includes vaginal examination to look for vaginal and cervical lacerations and vaginal hematomas, abdominal examination to evaluate uterine tone and look for signs of intraabdominal hemorrhage, and possibly bimanual examination of the uterus.

Ultrasound can be useful if the clinician suspects retained placental fragments or membranes or concealed hemorrhage (eg, lower uterine genital tract hematoma with extension, uterine rupture, broad ligament laceration, or another source of internal bleeding).

Monitor bleeding, vital signs, and laboratory results and perform an examination — Close maternal monitoring is critical to assess the best approach to and aggressiveness of intervention, and requires bedside evaluation by the provider.

Laboratory evaluation may include, depending on the clinical scenario, (see ["Postpartum hemorrhage: Medical and minimally invasive management"](#), section on 'Routine'):

- Complete blood count
- Coagulation studies (fibrinogen level, prothrombin time [PT], activated partial thromboplastin time [aPTT])
- Type and screen or crossmatch
- Potassium and ionized calcium levels
- Maternal blood gas analysis and serum lactate

In patients with major bleeding and coagulopathy, point-of-care viscoelastic testing provides nearly real-time data for guiding the transfusion of blood products. Its use leads to better hemorrhage control, in part because of rapid detection and targeted treatment of hypofibrinogenemia and thrombocytopenia. This is a highly complex laboratory test, requiring a skilled technician,

specialized equipment, and specialized reagents. It is often used in trauma, cardiac, and thoracic surgery during daytime hours when many resources are available. While data in nonpregnant patients suggest that viscoelastic tests may reduce mortality [78], a reduction in maternal mortality has not yet been demonstrated despite data showing that these tests reduce the need for transfusion of blood products and the rate of circulatory overload [79,80]. Detailed information on point-of-care viscoelastic testing is available separately. (See ["Postpartum hemorrhage: Medical and minimally invasive management", section on 'Viscoelastic testing'.](#))

OVERVIEW OF TREATMENT

Many potential interventions for treatment of PPH are available and listed in the table ([table 7](#)). Treatment goals and our approach to achieving these goals are described in the following sections.

Goals

- Restore or maintain adequate circulatory volume to prevent hypoperfusion of vital organs
- Restore or maintain adequate tissue oxygenation
- Reverse or prevent coagulopathy
- Eliminate the obstetric cause of PPH

Initial approach — The initial treatment approach is based on a combination of factors, including the cause and severity of bleeding and whether the abdomen is already open because of cesarean birth. The obstetric provider should initiate a sequence of nonoperative and operative interventions to control bleeding based on the cause and promptly assess the success of each measure. If an intervention fails, the next treatment in the sequence should be swiftly instituted. Indecisiveness delays therapy and results in excessive hemorrhage, which eventually causes dilutional coagulopathy and severe hypovolemia, tissue hypoxia, hypothermia, and acidosis. This will make control of hemorrhage much more difficult and will increase the likelihood of hysterectomy, major morbidity from hemorrhagic shock, and death.

The following is a synopsis of the treatment approach for the four causes of PPH. Regardless of the cause, all patients should receive initial circulatory support with crystalloid. In those with severe bleeding, switching to blood transfusion when blood is

available and early administration of [tranexamic acid](#) (an antifibrinolytic drug) can reduce the risk of death due to bleeding. These interventions are discussed in detail separately. (See "[Postpartum hemorrhage: Medical and minimally invasive management](#)", section on 'Resuscitate with crystalloid and blood' and "[Postpartum hemorrhage: Medical and minimally invasive management](#)", section on 'Transfuse blood products' and "[Postpartum hemorrhage: Medical and minimally invasive management](#)", section on 'Administer tranexamic acid'.)

- **Atony** – Treatment of atony, the most common cause of PPH, is influenced by both the route of birth and severity of bleeding. After a vaginal birth, treatment begins with uterotonic drugs and minimally invasive procedures (eg, intrauterine devices such as a balloon for tamponade or low-level vacuum to facilitate uterine compressive forces) and progresses to more invasive procedures (eg, uterine artery embolization or surgical intervention) until hemorrhage is controlled. Treatment of atony after vaginal birth (eg, choice of drugs, dosing) is discussed in detail separately. (See "[Postpartum hemorrhage: Medical and minimally invasive management](#)", section on 'Manage atony'.)

Uterotonic medications are also used to treat atony at cesarean birth, but since the abdomen is already open, surgical procedures to control bleeding (eg, uterine artery and utero-ovarian artery ligation, uterine compression sutures) are employed much sooner than after a vaginal birth, and uterine artery embolization may be considered if these procedures fail. Treatment of atony at laparotomy is discussed in detail separately. (See "[Postpartum hemorrhage: Management approaches requiring laparotomy](#)".)

Hysterectomy is the definitive therapy when bleeding cannot be controlled by other measures within a timeframe appropriate for the clinical scenario.

- **Trauma** – Traumatic, hemorrhaging lacerations are controlled surgically, either via a transvaginal or transabdominal approach, as appropriate for the site of bleeding. (See "[Postpartum hemorrhage: Medical and minimally invasive management](#)", section on 'Repair genital tract lacerations' and "[Postpartum hemorrhage: Management approaches requiring laparotomy](#)", section on 'Myometrial lacerations' and "[Postpartum hemorrhage: Management approaches requiring laparotomy](#)", section on 'Laceration of the uterine artery or utero-ovarian artery branches'.)

Topical hemostatic agents and tissue adhesives are useful adjuncts to manage bleeding from surgical surfaces. They are particularly useful for diffuse nonanatomic bleeding, bleeding associated with sensitive structures, and bleeding in patients

with hemostatic abnormalities ([table 4](#)). (See "[Overview of topical hemostatic agents and tissue adhesives](#)".)

- **Retained placental tissue** – Retained placental tissue can be identified visually and by palpation at a cesarean birth or by palpation after a vaginal birth. It can be removed manually or with the use of instruments (eg, hemostat, Kelly clamp, or curette). In cases of delayed (secondary) hemorrhage, retained placental tissue is usually detected by ultrasound and removed by curettage. (See "[Retained placenta after vaginal birth](#)".)

Placenta accreta spectrum (PAS) generally requires a hysterectomy, but uterine conservation with placental resection may be successful without excessive risk in selected cases of focal accreta or a posterior or fundal accreta. Diagnosis, preoperative planning, and management are reviewed separately. (See "[Placenta accreta spectrum: Clinical features, diagnosis, and potential consequences](#)" and "[Placenta accreta spectrum: Management](#)".)

- **Coagulopathy** – Coagulopathy is treated medically with transfusion of blood products and/or clotting factors to correct the clotting factor deficiencies. Treatment of coagulopathy is reviewed in detail separately. (See "[Postpartum hemorrhage: Medical and minimally invasive management](#)", section on 'Clotting factors'.)

Additional considerations for hemodynamically unstable patients — In addition to the initial approach to treatment described above, the following considerations apply to patients who are hemodynamically unstable.

- **Move the patient to an appropriate area** – Unstable patients in a coagulopathic state with active bleeding should be managed in the most appropriate area for resuscitation and emergency surgery. Under most circumstances, this is a warm operating room with a full multidisciplinary team in attendance.
- **Transfuse as soon as possible** – When hemorrhage is the cause of hemodynamic instability, initial (and expedited) aggressive volume resuscitation with whole blood or red cells and other appropriate blood products (eg, platelets, fresh frozen plasma, cryoprecipitate) is required (as opposed to large volume crystalloid infusion). Transfusion should keep up with blood loss and may require massive transfusion. In such cases, early activation of standardized institutional approach to massive transfusion can improve outcome. (See '[Massive transfusion protocol and algorithm](#)' above.)

Our approach to transfusion is described in detail separately. (See "[Postpartum hemorrhage: Medical and minimally invasive management](#)", section on 'Transfuse blood products'.).

- **Correct severe hypofibrinogenemia with high-concentration fibrinogen products** – If the patient is severely coagulopathic with an extremely low fibrinogen level (50 to 100 mg/dL), cryoprecipitate and/or other high-concentration fibrinogen products (eg, [fibrinogen concentrate](#)) are required since whole blood and fresh frozen plasma will not increase the fibrinogen level to the normal range (ideally greater than 200 mg/dL) without requiring excessive volume infusion. (See ["Postpartum hemorrhage: Medical and minimally invasive management"](#), section on 'Clotting factors' and ["Plasma derivatives and recombinant DNA-produced coagulation factors"](#) and ["Postpartum hemorrhage: Management approaches requiring laparotomy"](#), section on 'Evaluation of the abdomen at laparotomy'.)
- **Begin temporizing measures** – Temporizing measures allow resuscitation to a point where general anesthesia and surgery, if necessary, are better tolerated. These interventions include bimanual uterine compression, intrauterine tamponade or vacuum device, aortic compression, and resuscitative endovascular balloon occlusion of the aorta (REBOA). (See ["Postpartum hemorrhage: Medical and minimally invasive management"](#), section on 'Perform uterine massage and compression' and ["Postpartum hemorrhage: Medical and minimally invasive management"](#), section on 'Consider external aortic compression' and ["Postpartum hemorrhage: Medical and minimally invasive management"](#), section on 'Consider resuscitative endovascular balloon occlusion of the aorta'.)

Intrauterine tamponade can be performed with an intrauterine balloon ([table 8](#) and [table 9](#)), packing ([table 10](#)), or a low-level vacuum device that induces physiologic uterine contraction. The choice is largely driven by local availability, provider preference, and cost (see ["Postpartum hemorrhage: Use of an intrauterine hemorrhage-control device"](#)). The author believes early recourse to intrauterine tamponade can be useful to decrease ongoing uterine blood loss following vaginal birth or after the abdomen is closed following cesarean birth, and that this measure will allow additional time for assessment and evaluation, stabilization, and institution of resuscitative procedures. In some cases, intrauterine tamponade may avoid the need for surgical management of PPH [81]. (See ["Postpartum hemorrhage: Medical and minimally invasive management"](#), section on 'Use an intrauterine postpartum hemorrhage control device in patients with atony or lower segment bleeding'.)

In patients who continue to bleed at cesarean and still have an open abdominal incision, temporizing measures, compression sutures and devascularization are more easily accomplished than intrauterine tamponade. (See ["Postpartum hemorrhage: Management approaches requiring laparotomy"](#), section on 'Temporary measures for stabilizing hemodynamically unstable patients' and ["Postpartum hemorrhage: Management approaches requiring laparotomy"](#), section on 'Uterine compression

sutures' and ["Postpartum hemorrhage: Management approaches requiring laparotomy"](#), section on 'Patients not at imminent risk of exsanguination'.)

- **Role of hysterectomy** – Early resort to hysterectomy is appropriate in patients with severe bleeding due to diffuse PAS or a large uterine rupture, which require aggressive surgical management. In contrast, hysterectomy is generally not the first intervention in patients with atony, as these patients can often be managed successfully with medical therapy and less aggressive surgical interventions. (See ["Postpartum hemorrhage: Management approaches requiring laparotomy"](#), section on 'Role of hysterectomy'.)

In coagulopathic patients who require an emergency hysterectomy to control bleeding, concomitant blood product resuscitation during surgery is required. (See ["Disseminated intravascular coagulation \(DIC\) during pregnancy: Management and prognosis"](#), section on 'Role of hysterectomy'.)

- **Keep unstable patients with persistent bleeding in the operating room** – If the abdomen was opened for management of PPH but bleeding was not completely controlled, temporarily closing the abdomen with towel clips allows the surgical team rapid direct access to the pelvis to repack or readdress ongoing bleeding (eg, digital pressure or a temporary clamp applied to the aorta).

The author is aware of situations where unstable, actively bleeding patients in a coagulopathic state have had their abdomen packed and then were transported to an intensive care unit (ICU), where they expired. In the author's opinion, in these desperate situations, keeping the patient under anesthesia on a surgical table in a warm environment gives the team the most options for gaining control of the situation (eg, acid-base resuscitation; replacement of volume, electrolytes, and blood products), even if logistically difficult. ICU consultants can be summoned to the operating room for assistance. Even in the direst situations, as long as transfusion of appropriate blood products can be continued and the volume of such products exceeds the volume of the ongoing loss, then blood pressure can be maintained and efforts to reverse the coagulopathy, acidosis, and hypothermia should be continued and ultimately may be successful.

Hemodynamically stable patients with persistent bleeding after initial therapy — Arterial embolization is an effective treatment for hemodynamically stable patients with persistent bleeding in whom the capacity for blood replacement exceeds that of the ongoing hemorrhage. In a systematic review of 20 observational studies (1739 patients), a single procedure

completely arrested bleeding in 89 percent of cases, re-embolization was necessary in 4 percent, and hysterectomy was required in 7 percent, primarily after embolization failure [82]. Sixty-two percent of the patients in these studies were post-cesarean birth.

Generally, arterial embolization should not be attempted in unstable patients who have to be transferred to a radiology suite for the procedure and should not be considered an emergency procedure for managing uncontrolled PPH of indeterminate cause. (See ["Postpartum hemorrhage: Management approaches requiring laparotomy"](#) and ["Postpartum hemorrhage: Medical and minimally invasive management"](#), section on 'Consider uterine or hypogastric artery embolization'.)

Role of nonpneumatic anti-shock garments — In ambulances and facilities where definitive treatment of PPH is not possible or will be delayed because of lack of resources, use of a nonpneumatic anti-shock garment (NASG) may reduce bleeding, stabilize patients until they are transferred to an appropriate referral/tertiary facility, and decrease mortality from hypovolemic shock [83-87]. With minimal training, NASG can be applied within two minutes and is reusable.

NASG consists of nine articulated segments that are wrapped tightly and sequentially around the legs, pelvis, and abdomen and then closed with hook-and loop-fastening straps. Application of circumferential counterpressure decreases blood flow to the compressed area (abdominal aorta, pelvis, and lower extremities) and increases blood flow to the heart, lungs, and brain [88]. The addition of a small foam ball centrally provides some degree of aortic as well as uterine compression and blood flow to the pelvis is reduced [88,89]. Intrauterine tamponade can also be employed.

In an analysis of five observational studies, NASG use was associated with a ≥ 50 percent reduction in median blood loss in three of four studies and a smaller reduction in one study [83]. The analysis also found total pooled mortality was reduced 38 percent across the five studies, even though mortality was increased in one study.

Extracorporeal membrane oxygenation — Isolated case reports have described the use of extracorporeal membrane oxygenation (ECMO) in the management of severe PPH and amniotic fluid embolism [90-97]. While this modality is clearly only available in very few hospitals, it may be reasonable to develop an algorithm and process in those units where massive transfusion can be anticipated and where high-risk cases are routinely encountered (eg, referral centers for known placenta percreta, complex maternal comorbidities).

In a systematic review including 358 pregnant patients at the time of cannulation, the three most common indications for ECMO were acute respiratory distress syndrome (49 percent), cardiac failure (19 percent), and cardiac arrest (16 percent) [98]. Approximately 75 percent of the patients survived at 30 days and at one year; fetal survival was 65 percent. The author of this topic has personal experience of successfully instituting venoarterial (VA) ECMO in a patient with cardiac arrest from presumed amniotic fluid embolism and massive hemorrhage who had undergone CPR for 75 minutes without return of spontaneous circulation. Following 30 minutes of VA ECMO, cardiac activity returned with effective and sustained ventricular function. (See ["Extracorporeal life support in adults in the intensive care unit: Overview".](#))

OUTCOME

Mortality — Maternal mortality after PPH has wide variations worldwide depending on both the overall health of the pregnant population and the resources for treatment of PPH [99]. Death rates vary from 0.01 percent of patients with PPH in the United Kingdom to 20 percent of patients with PPH in parts of Africa, and from 1 in 100,000 births in the United Kingdom to 1 in 1000 births in resource-limited regions. Patients with anemia due to poor nutrition or malaria are particularly vulnerable to severe sequelae of PPH.

Severe maternal morbidity — Severe maternal morbidity (ie, health-impacting and life-threatening events that occur during hospitalization for childbirth) has increased substantially in recent years, largely driven by an increase in blood transfusion for PPH ([figure 2](#)). (See ["Severe maternal morbidity"](#).)

Short-term morbidity

- **Anemia** – Postpartum anemia is common and usually defined as a hemoglobin level <11 g/dL at one week postpartum and <12 g/dL at eight weeks postpartum [100]. Patients with PPH often have a 10-point decline in postpartum hematocrit from antepartum levels. The type of treatment depends on severity of anemia.

One or more red blood cell transfusions may be required, depending on the severity of anemia and symptoms attributable to anemia. A common practice is to offer a transfusion to symptomatic patients with a hemoglobin value <7 g/dL [5]. In the WOMAN trial, which included over 20,000 patients worldwide with PPH, 54 percent were transfused. By comparison, in the

United States, 16 percent of births with PPH are transfused [21] (versus 0.4 to 0.7 percent of births in the overall obstetric population [101]). Risks of transfusion include infection, electrolyte abnormalities, allergic reactions, alloimmunization, volume overload, and venous thromboembolism (VTE). (See "[Indications and hemoglobin thresholds for RBC transfusion in adults](#)".)

In most cases of PPH, iron supplementation is required because the amount of iron lost is not fully replaced by any transfused blood. Oral supplements are one option and single-dose parenteral iron therapy is another option. Advantages of parenteral iron are that hemoglobin levels rise faster, symptoms of anemia improve sooner, and less gastric upset occurs compared with oral therapy [102,103]. Nevertheless, most patients with mild to moderate anemia resolve the anemia sufficiently rapidly with oral iron, and it is inexpensive and convenient [104-106]. Assessment and treatment of iron deficiency anemia is discussed in detail separately. (See "[Treatment of iron deficiency anemia in adults](#)".)

Although erythropoietin can increase the rate of recovery to normal hemoglobin levels, it does not have an immediate effect and has not been proven to reduce transfusion requirements after PPH [107]. It is no more effective than iron therapy in this setting [108], and it is expensive. However, for the few patients with severe anemia who do not respond to iron therapy because of blunted erythropoiesis due to infection and/or inflammation, some hematologists consider recombinant human erythropoietin an alternative to transfusion [100].

- **Hysterectomy** – In the WOMAN trial, 3.5 percent of patients with PPH underwent hysterectomy [109]. By comparison, in the United States, 2.1 percent of patients with PPH underwent hysterectomy in 2014, and atony accounted for almost 60 percent of these cases [21].
- **Organ failure related to hemodynamic instability** – In the WOMAN trial, 60 percent of patients with PPH had clinical signs of hemodynamic instability at diagnosis of PPH and nearly 4 percent developed kidney failure, heart failure, respiratory failure, or liver failure [109]. One mechanism is that treatment of hemodynamic instability with fluids and blood can lead to volume overload, resulting in pulmonary edema and dilutional coagulopathy.
- **Thromboembolism** – In the WOMAN trial, 0.3 percent of patients with PPH experienced thromboembolism (deep vein thrombosis, pulmonary embolus, stroke, myocardial infarction) within 42 days of birth [109].

- **Abdominal compartment syndrome** – Abdominal compartment syndrome (organ dysfunction caused by intraabdominal hypertension) is a rare but life-threatening complication of PPH with intraabdominal bleeding. The diagnosis should be considered in patients with a tensely distended abdomen and progressive oliguria who are developing multiorgan failure. Of note, the normal postpartum patient after cesarean birth has been reported to have an intraabdominal pressure that approaches that seen in abdominal compartment syndrome in nonpregnant individuals [110].

Clinical presentation, diagnosis, and management are discussed in detail separately. (See ["Abdominal compartment syndrome in adults"](#).)

Long-term morbidity

- **Sheehan syndrome** – Sheehan syndrome (ie, postpartum hypopituitarism) is a rare but potentially life-threatening complication. The pituitary gland is enlarged in pregnancy and prone to infarction from hypovolemic shock. The resulting pituitary damage ranges from mild to severe, and can reduce secretion of one, several, or all of its hormones. A common presentation is a combination of failure to lactate postpartum and amenorrhea or oligomenorrhea, but any of the manifestations of hypopituitarism (eg, hypotension, hyponatremia, hypothyroidism) can occur at any time from the immediate postpartum period to years after giving birth.

Patients who remain hypotensive after control of PPH and volume replacement should be evaluated and treated for adrenal insufficiency in the immediate postpartum period, whereas evaluation of other hormonal deficiencies can be deferred until four to six weeks postpartum. This evaluation is described in detail separately (see ["Clinical manifestations of hypopituitarism"](#) and ["Diagnostic testing for hypopituitarism"](#)). Treatment is also reviewed separately. (See ["Treatment of hypopituitarism"](#).)

- **Asherman syndrome** – Development of intrauterine adhesions (termed Asherman syndrome) can lead to menstrual abnormalities and infertility. Approximately 90 percent of cases of severe intrauterine adhesive disease are related to uterine curettage for pregnancy complications, such as PPH [111,112]. Uterine compression sutures used to treat PPH have also been associated with the development of intrauterine adhesions [113-116]. Diagnosis and treatment are discussed separately. (See ["Intrauterine adhesions: Clinical manifestation and diagnosis"](#) and ["Intrauterine adhesions: Treatment and prevention"](#).)

POSTPARTUM CARE

- Active management of the third stage of labor, primarily by routine prophylactic use of uterotonic drugs such as [oxytocin](#), substantially reduces the incidence of PPH due to atony. While evidence is lacking regarding the optimal approach specifically in patients who have experienced PPH, it seems reasonable to prolong the duration of postpartum uterotonic administration when the cause was atony. Drug choice, dosing, and efficacy are described separately. (See "[Prophylactic pharmacotherapy to reduce the risk of postpartum hemorrhage](#)".)
- PPH is a traumatic experience for patients that can have both short-term and long-term impacts on their mental health [[117-121](#)]. Helping patients process and understand the events that occurred, discussing their concerns and prognosis, connecting them with a patient resource person, and referring them to emotional support and counseling services can mitigate the impact.
- Before discharge, patients should be given information about normal vaginal bleeding, signs and symptoms associated with excessive bleeding, and when to call the provider, in addition to the usual information about postpartum recovery. (See "[Overview of the postpartum period: Normal physiology and routine maternal care](#)", section on 'Patient education'.)

RECURRENCE

Patients with a prior PPH have as much as an 18 percent risk of recurrence in the subsequent pregnancy and 27 percent after two consecutive pregnancies with PPH [[122-124](#)]. The risk of recurrence likely depends, at least in part, on the underlying cause. In a study of patients with PPH from atony, lacerations, or retained placenta, the risks of atony and retained placenta remained increased in the next two pregnancies with vaginal births, whereas the risk of lacerations decreased [[122](#)]. Although the risk of recurrence was greatest for PPH from the same cause, the risk was also increased for PPH from other causes.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See ["Society guideline links: Obstetric hemorrhage"](#).)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see ["Patient education: Postpartum hemorrhage \(The Basics\)"](#))

SUMMARY AND RECOMMENDATIONS

- **Diagnosis and assessment of severity** – Primary (early) postpartum hemorrhage (PPH) refers to excessive bleeding in the first 24 hours after birth (see ["Terminology"](#) above). We make the diagnosis in postpartum patients with:
 - Bleeding that is greater than expected using a quantitative method to assess blood loss **and**
 - Results in signs and/or symptoms of hypovolemia ([table 1](#))

Other definitions are shown in the table ([table 2](#)). (See ["Criteria for diagnosis"](#) above and ["Quantify blood loss"](#) above.)

Severe PPH, defined as the need for transfusion of multiple units of blood and blood products, the need for angiographic embolization or surgical management of hemorrhage, or maternal death can be predicted by maternal early warning systems (MEWS) vital sign criteria ([table 6](#)) and by a low fibrinogen level (less than 200 mg/dL). (See '[Recognize alarm findings and intervene early](#)' above and '[Monitor bleeding, vital signs, and laboratory results and perform an examination](#)' above.)

- **Causes** – The most common causes are atony, trauma, placental disorders, and coagulopathy/bleeding diatheses. (See '[Causes of postpartum hemorrhage](#)' above.)
- **General approach algorithm** ([algorithm 1](#))
- **Planning**
 - **Risk assessment** – Patients with risk factors for PPH should be identified, when possible, and counseled as appropriate for their level of risk. However, only a small proportion of these patients develop PPH (abnormal placentation is an exception), and many patients without risk factors experience PPH. (See '[Risk factors for PPH](#)' above and '[PPH risk assessment tools and risk-based preparation](#)' above.)
 - PPH alone is not a strong indication for screening for inherited bleeding diatheses, given that undiagnosed bleeding disorders are rarely the cause of PPH. However, unexplained PPH that does not respond to general measures should alert clinicians to the possibility of a bleeding disorder as a causative factor, especially in patients with a history of heavy menstrual bleeding, excessive bleeding after minor trauma, or a family history of a bleeding disorder. (See '[Recurrence](#)' above.)
 - **Preparation** – Planning for PPH involves:
 - Quantifying blood loss in all births
 - Ensuring availability of resources that might be needed (personnel, medications, equipment [eg, intrauterine tamponade, cell salvage], adequate intravenous access [eg, two large-bore cannulas], blood products).

Two useful approaches are PPH carts ([table 5](#)) and protocols to manage PPH (eg, massive transfusion protocol).

Regular training and simulation drills should be instituted to ensure compliance, emergency stage-based response, and

unit preparedness. (See '[Institutional planning and preparation](#)' above.)

- **Treatment**

- **All patients** – The initial treatment approach is based on a combination of factors, including the cause and severity of bleeding. (See '[Goals](#)' above and '[Initial approach](#)' above.)
 - Atony is initially treated with uterotonic drugs. Second-line treatment (eg, manual uterine massage and compression, uterine compression sutures) depends on the route of birth, as the morbidity of open operative interventions is less at cesarean since the abdomen is already open.
 - Lacerations are treated surgically.
 - Coagulopathy is treated with infusion of blood products and/or concentrates.

Regardless of the cause of PPH, initial circulatory support with crystalloid is required in all patients. In those with severe bleeding, switching to blood transfusion when blood is available and early administration of [tranexamic acid](#) reduces the risk of death due to bleeding. (See '[Initial approach](#)' above.)

- **Hemodynamically unstable patients** – Key considerations in patients who are hemodynamically unstable include (see '[Additional considerations for hemodynamically unstable patients](#)' above):
 - Initial management in the most appropriate area for resuscitation and emergency surgery
 - Transfusion as soon as possible
 - Correction of severe hypofibrinogenemia with cryoprecipitate and/or other high-concentration fibrinogen products (eg, [fibrinogen concentrate](#))
 - Use of temporizing measures to allow resuscitation to a point where general anesthesia and surgery, if necessary, are better tolerated
 - Early resort to hysterectomy
 - Ongoing management of unstable patients with persistent bleeding in an operating room rather than an intensive care unit (ICU).

- **Outcome** – PPH is associated with potentially serious short-term morbidities from hemorrhage and hypotension, and may be lethal. Potential long-term morbidities include Sheehan syndrome (in patients with hypotension) and Asherman syndrome (in patients who were curetted). (See '[Outcome](#)' above.)
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ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges Allan J Jacobs, MD, who contributed to earlier versions of this topic review.

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GRAPHICS

Symptoms related to blood loss with postpartum hemorrhage

Blood loss, % (mL)	Systolic blood pressure, mmHg	Signs and symptoms
10 to 15 (500 to 1000)	normal and ≥90	Palpitations, lightheadedness, no or mild increase in heart rate
15 to 25 (1000 to 1500)	80 to 90	Weakness, sweating, tachycardia (100 to 120 beats/minute), tachypnea (respiratory rate of 20 to 24)
25 to 35 (1500 to 2000)	70 to 80	Restlessness, confusion, pallor, oliguria, tachycardia (120 to 140 beats/minute), cool and clammy skin
35 to 45 (2000 to 3000)	50 to 70	Lethargy, air hunger, anuria, collapse, tachycardia (>140 beats/minute)

Adapted from: Bonnar J. Massive obstetric haemorrhage. Baillieres Best Pract Res Clin Obstet Gynaecol 2000; 14:1.

Examples of criteria for postpartum hemorrhage

Organization	Definition of PPH
World Health Organization ^[1]	<ul style="list-style-type: none"> ▪ Blood loss ≥ 500 mL within 24 hours after birth. ▪ Severe PPH: Blood loss ≥ 1000 mL within the same time frame.
American College of Obstetricians and Gynecologists ^[2]	<ul style="list-style-type: none"> ▪ Cumulative blood loss ≥ 1000 mL or blood loss accompanied by signs or symptoms of hypovolemia within 24 hours after the birth process (includes intrapartum loss) regardless of route of delivery.
Royal College of Obstetricians and Gynaecologists ^[3]	<ul style="list-style-type: none"> ▪ Minor PPH (500 to 1000 mL) and major PPH (>1000 mL). Subdivisions of major PPH include moderate (1001 to 2000 mL) or severe (>2000 mL).
International expert panel ^[4]	<ul style="list-style-type: none"> ▪ Active bleeding >1000 mL within the 24 hours following birth that continues despite the use of initial measures, including first-line uterotonic agents and uterine massage.
Society of Obstetricians and Gynaecologists of Canada ^[5]	<ul style="list-style-type: none"> ▪ Any amount of bleeding that threatens the patient's hemodynamic stability.
California Maternal Quality Care Collaborative ^[6]	<ul style="list-style-type: none"> ▪ Stage 0: Every woman in labor/giving birth. ▪ Stage 1: Blood loss >500 mL after vaginal or >1000 mL after cesarean delivery; or change in vital signs $>15\%$ or heart rate ≥ 110 beats/minute, blood pressure $\leq 85/45$ mmHg, O₂ saturation $<95\%$. ▪ Stage 2: Continued bleeding with total blood loss <1500 mL. ▪ Stage 3: Total blood loss >1500 mL or >2 units packed red cells transfused; or unstable vital signs; or suspicion of disseminated intravascular coagulation.

PPH: postpartum hemorrhage.

References:

1. World Health Organization. WHO recommendations for the prevention and treatment of postpartum haemorrhage. Geneva: World Health Organization; 2012.
2. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin Number 183, October 2017: Postpartum hemorrhage. Obstet Gynecol 2017; 130:e168.
3. Prevention and management of postpartum haemorrhage: Green-top guideline No. 52. BJOG 2017; 124:e106.

4. Abdul-Kadir R, McLintock C, Ducloy AS, et al. Evaluation and management of postpartum hemorrhage: Consensus from an international expert panel. *Transfusion* 2014; 54:1756.
 5. Leduc D, Senikas V, Lalonde AB, et al. Active management of the third stage of labour: Prevention and treatment of postpartum hemorrhage. *J Obstet Gynaecol Can* 2009; 31:980.
 6. CMQCC. www.cmqcc.org/resources-tool-kits/toolkits/ob-hemorrhage-toolkit (Accessed on May 17, 2017).
-

Sample approach to risk stratification and delivery preparation for PPH

Prenatal assessment and planning		
<ul style="list-style-type: none"> Identify and prepare for patients at risk: previa/accreta, bleeding disorder, those who refuse transfusion Screen and treat for anemia (iron panel, hemoglobin electrophoresis, consider oral versus IV iron) 		
Low	Medium	High
≤4 previous SVD	Hgb <8 g/dL	Placenta previa
Singleton	Platelets <100,000/microL	Suspected placenta accreta
<2 prior CD	≥3 prior CD or previous myomectomy	Abruption
No previous PPH	>4 vaginal births	Inherited or acquired coagulopathy
No known bleeding disorder	Chorioamnionitis	
	Magnesium sulfate use	
	Multiple gestation	
	Large uterine fibroids	
	EFW >4250 g	
	Black race	
	History of PPH	
	Severe obesity (BMI >40 kg/m ²)	

Oxytocin preparation: 30 international units in 500 cc solution. 167 cc = 10 international units.

Low:

- Type and screen on admission (if antibody positive, then will need crossmatch)
- Postpartum order: Oxytocin 167 cc bolus, then 42 cc/hour for 4 hours

Medium:

- Type and screen on admission
- Discuss risk of PPH with patient

- Postpartum orders: Oxytocin 167 cc bolus, then 42 cc/hour for 8 hours

Medium "plus" (>1 medium risk factor):

- Crossmatch 2 units
- Discuss risk of PPH with patient
- Postpartum orders: Oxytocin 167 cc bolus, then 42 cc/hour for 8 hours

High:

- Crossmatch 4 (or more) units
- Discuss risk of PPH and transfusion with patient
- Postpartum orders: Oxytocin 167 cc bolus, then 42 cc/hour for 24 hours
- Observation on L&D postoperatively based on QBL or at least 4 hours (whichever is longer)

BMI: body mass index; cc: cubic centimeter; CD: cesarean delivery; EFW: estimated fetal weight; Hgb: hemoglobin; IV: intravenous; L&D: labor and delivery; PPH: postpartum hemorrhage; QBL: quantification of blood loss; SVD: small-vessel disease.

Table updated for this publication. Original sample approach courtesy of Christina Davidson, MD and Catherine Eppes, MD, MPH.

Sample checklist for managing postpartum hemorrhage

Complete all steps in prior stages regardless of stage in which the patient presents

Recognize, call for assistance: ☐ Charge nurse ☐ OB Attending

Designate: ☐ Team lead ☐ Checklist reader/recorder ☐ Second RN

Announce: ☐ Cumulative blood loss ☐ Vital signs

Stage 1: Blood loss >500 mL to 1000 mL

Initial steps:

- ☐ Ensure 16 G or 18 G IV access
- ☐ Empty bladder: straight catheter or place indwelling Foley catheter with urimeter
- ☐ Fundal massage
- ☐ Vital signs every 5 minutes

Medications:

- ☐ Oxytocin infusion at bolus rate for up to maximum cumulative dose of 40 units
- ☐ Administer appropriate medications, consider patient history

Action:

- ☐ PPH kit to bedside
- ☐ QBL assessed, announced and recorded every 15 minutes
- ☐ Determine etiology and treat
- ☐ Consider uterine balloon tamponade

Medications

Oxytocin:

30 units per 500 mL solution; 167 mL = 10 units

Methylergonovine:

0.2 milligrams IM every 2 to 4 hours as needed;
avoid with hypertension

Carboprost:

250 micrograms IM (may repeat every 15 minutes,
maximum 8 doses); avoid with asthma

Misoprostol:

800 micrograms rectal **or**
600 micrograms buccal
(1000 micrograms maximum dose)

Stage 2: Blood loss >1000 mL to 1500 mL

Initial steps:

- ☐ Place second IV (16 to 18 G)
- ☐ Prepare OR if clinically indicated (optimize visualization/examination)

Medications:

- ☐ Continue medications as indicated

Action:

- ☐ Stat labs: CBC, PT/PTT, INR, fibrinogen
- ☐ Type and cross 2 units RBCs
- ☐ Transfuse RBCs per clinical signs/symptoms (do not wait for lab results)

Stage 3: Continued bleeding; blood loss >1500 mL

Initial steps:

- ☐ Activate OB emergency
- ☐ Move to OR; communicate plan (anesthesia/patient position/equipment)
- ☐ Mobilize additional help: Notify back-up provider

Medications:

- ☐ Continue medications as indicated
- ☐ Administer TXA 1 gram IV over 10 minutes; if bleeding persists, administer second dose TXA 1 gram IV
- ☐ Re-dose antibiotics

Action:

- ☐ Initiate Massive Transfusion Protocol: State "Obstetric patient"
- ☐ Stat labs every 30 minutes: CBC, PT/PTT, INR, fibrinogen, blood gas, electrolytes including ionized calcium
- ☐ Monitor TEG **or** ROTEM
- ☐ Warm all transfused fluids
- ☐ Monitor core temperature; direct warming of the patient to maintain euthermia

OB: obstetrician; RN: registered nurse; IV: intravenous; QBL: quantity of blood loss; IM: intramuscular; OR: operating room; CBC: complete blood count; PT: prothrombin time; PTT: partial thromboplastin time; INR: international normalized ratio; RBC: red blood cell; TXA: tranexamic acid; TEG: thromboelastography; ROTEM: rotational thromboelastography.

Courtesy of Christina Davidson, MD and Catherine Eppes, MD, MPH.

Graphic 126212 Version 2.0

Topical hemostatic agents for managing bleeding

Agent	Source	How supplied	Trade names in United States	Absorption time ^[1]	Adverse effect* ^[2]	Relative expense ^{¶[1,3]}
Physical agents						
Wax-based						
Bone wax	Beeswax	Stick form	Not applicable	Indefinite	Impaired bone healing	¢
Ostene (soluble copolymer implant material)	Synthetic alkaline oxide copolymers	Stick form	Ostene	48 hours		¢
Dry matrix						
Absorbable gelatin (gelatin matrix)	Porcine	Sponge, powder	Gelfilm, Gelfoam, Surgifoam, Gelfoam hemostasis kit (also contains human thrombin)	4 to 6 weeks	Infection, abscess or granuloma formation, fibrosis, clot disruption if sponge is removed ^[1]	\$
Oxidized regenerated cellulose	Plant	Mesh	Surgicel (Nu-Knit, Fibrillar, SNoW)	1 to 2 weeks ^Δ	Foreign body reaction, infection, adhesions ^[1]	\$
Microfibrillar collagen	Bovine	Sheets	Actifoam, Avitene (Ultrafoam),	>8 weeks	Granuloma formation, allergenic	\$\$

(collagen hemostat)			Endo Avitene, Instat MCH, Helistat Helitene, Syringe Avitene			
		Powder	Avitene Flour			
Microporous polysaccharide spheres (MPH)	Synthetic	Powder	Arista AH	24 to 48 hours	Use of >50 g can alter glucose load in patients with diabetes	\$
External agents						
Chitosan	Biodegradable complex carbohydrate derived from chitin	Adhesive dressing	HemCon bandage (Chitoflex)	Not applicable	Training needed to ensure proper application	¢
Kaolin-impregnated sponge	Aluminosilicate mineral	Gauze sponge	Quikclot	Not applicable	Possible clot disruption when sponge removed	¢
Hemafiber	Cellulose and silica	Dressing, surgical pad, elastic wrap	NuStat OTC, NuStat Trauma Pad XR, NuStat Flex	Not applicable	Possible clot disruption when pad removed	\$
Biologically active agents						
Topical thrombin	Bovine	Liquid	Thrombin-JMI	Not applicable	Intravascular application leads to thrombosis Antibody formation against bovine thrombin can inhibit	\$\$
	Human		Evithrom			

	Recombinant human (rHuman)		Recothrom		coagulation and prolong prothrombin time/INR	
Fibrin sealant (human fibrinogen and human thrombin) [◇]	Human	Liquid	Artiss, Tisseel (also contains synthetic aprotinin), Evicel, TachoSil, Evarrest, Vistaseal	Immediate	Potential exposure to blood-borne viruses	\$\$\$
	Human autologous		CryoSeal Fibrin Sealant System Vitagel (autologous fibrinogen and bovine thrombin)			Not available
	Human	Powder (spray and direct application; for use with absorbable gelatin sponge)	Raplixa	(Refer to Absorbable gelatin sponge, above)		Not available
Dry Fibrin Sealant Dressing (DFSD) [§]	Freeze-dried fibrinogen and thrombin applied to gauze substrate	Dressing	Not applicable	Not applicable		\$\$\$\$
Bovine albumin-glutaraldehyde tissue adhesive	Bovine	Liquid	Bioglue		Antibody formation against bovine	\$\$\$\$

					thrombin can inhibit coagulation and prolong prothrombin time/INR	
Combination preparations						
Thrombin/gelatin	Bovine or porcine	Liquid	Floseal (bovine), Surgiflo (porcine)	6 to 8 weeks	Antibody formation against bovine thrombin can inhibit coagulation and prolong prothrombin time/INR	\$\$\$
Thrombin/collagen	Bovine	Liquid	Costasis	4 weeks	Antibody formation against bovine thrombin can inhibit coagulation and prolong prothrombin time/INR	\$\$\$

INR: international normalized ratio.

* Although rare, use of protein-containing topical hemostatics and fibrin sealants can be associated with severe hypersensitivity reactions and formation of antibodies; such reactions may be seen especially after repeated application.

¶ The cost of topical hemostatics is highly variable in United States and subject to institutional contracting. Price range (US dollars): ¢: less than 50 dollars; \$: 50 to 100 dollars; \$\$: 101 to 300 dollars; \$\$\$: 301 to 500 dollars; \$\$\$\$: 501 to 750 dollars.

Δ The low pH of the oxidized regenerated cellulose can inhibit proteases and elastase, which can delay resorption for more than two weeks.

◇ Thrombin and fibrinogen concentrations vary. Fibrinogen concentrations are higher for commercial preparations. As an example, Tisseel, Evicel, and Vistaseal have fibrinogen concentrations of 70, 55 to 85, and 80 mg/mL, respectively, compared with 2.5 to 25 mg/mL for cryoprecipitate (unmanipulated). When rapid clot formation (5 to 10 seconds) is desired, thrombin concentrations of 500 to 1000 NIH units should be used (eg, Tisseel, 500 IU/mL; Evicel, 800 to 1200 IU/mL).^[4,5,6]

§ Investigational.

References:

1. Duenas-Garcia OF, Goldberg JM. Topical hemostatic agents in gynecologic surgery. *Obstet Gynecol Surv* 2008; 63:389.
2. Gabay M, Boucher B. An essential primer for understanding the role of topical hemostats, surgical sealants, and adhesives for maintaining hemostasis. *Pharmacotherapy* 2013; 33:935.
3. Hong YM, Loughlin KR. The use of hemostatic agents and sealants in urology. *J Urol* 2006; 176:2367.
4. TISSEEL VH Kit. Available at:
<https://www.fda.gov/downloads/biologicsbloodvaccines/bloodbloodproducts/approvedproducts/licensedproductsblas/fractionatedplasmaproducts/ucm072968.pdf>
(Accessed on October 27, 2017).
5. EVICEL Fibrin Sealant. Available at:
<https://www.fda.gov/downloads/BiologicsBloodVaccines/BloodBloodProducts/ApprovedProducts/LicensedProductsBLAs/FractionatedPlasmaProducts/UCM270787.pdf>
(Accessed on October 27, 2017).
6. VISTASEAL Fibrin Sealant. Available at: <https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=a8708417-2f74-4dfa-b6f9-ef88f902e0fc> (Accessed on December 15, 2021).

Example of instruments, equipment, and medications to assemble for a postpartum hemorrhage emergency cart

- | |
|---|
| ▪ Equipment/supplies for starting an intravenous line (14-, 16-, 18-, and 20-gauge peripheral venous catheter, 1 L Lactated Ringer's solution for injection, intravenous tubing, four-way stopcock, tape) |
| ▪ Urinary catheter kit, urimeter |
| ▪ Lubricating gel |
| ▪ Assorted sizes of sterile gloves, including elbow-length gloves |
| ▪ Vaginal retractors, including a long right-angle retractor |
| ▪ Sterile speculum, long weighted speculum |
| ▪ Sponge forceps |
| ▪ Vaginal packs, 2 by 2 and 4 by 4 sponge gauze packs, gauze bandage rolls |
| ▪ Balloon catheter kit for intrauterine tamponade |
| ▪ Sterile utility bowl, 20 and 60 mL syringes, irrigation water |
| ▪ Banjo curettes |
| ▪ Long needle holder |
| ▪ Appropriate sutures for cervical and vaginal laceration repair and for uterine compression sutures |
| ▪ Uterine forceps |
| ▪ Freestanding mobile task light, battery-powered headlamp |
| ▪ Illustrations showing how to perform relevant procedures (eg, uterine compression, uterine artery ligation, placement of intrauterine hemorrhage-control devices, replacement of inverted uterus) |

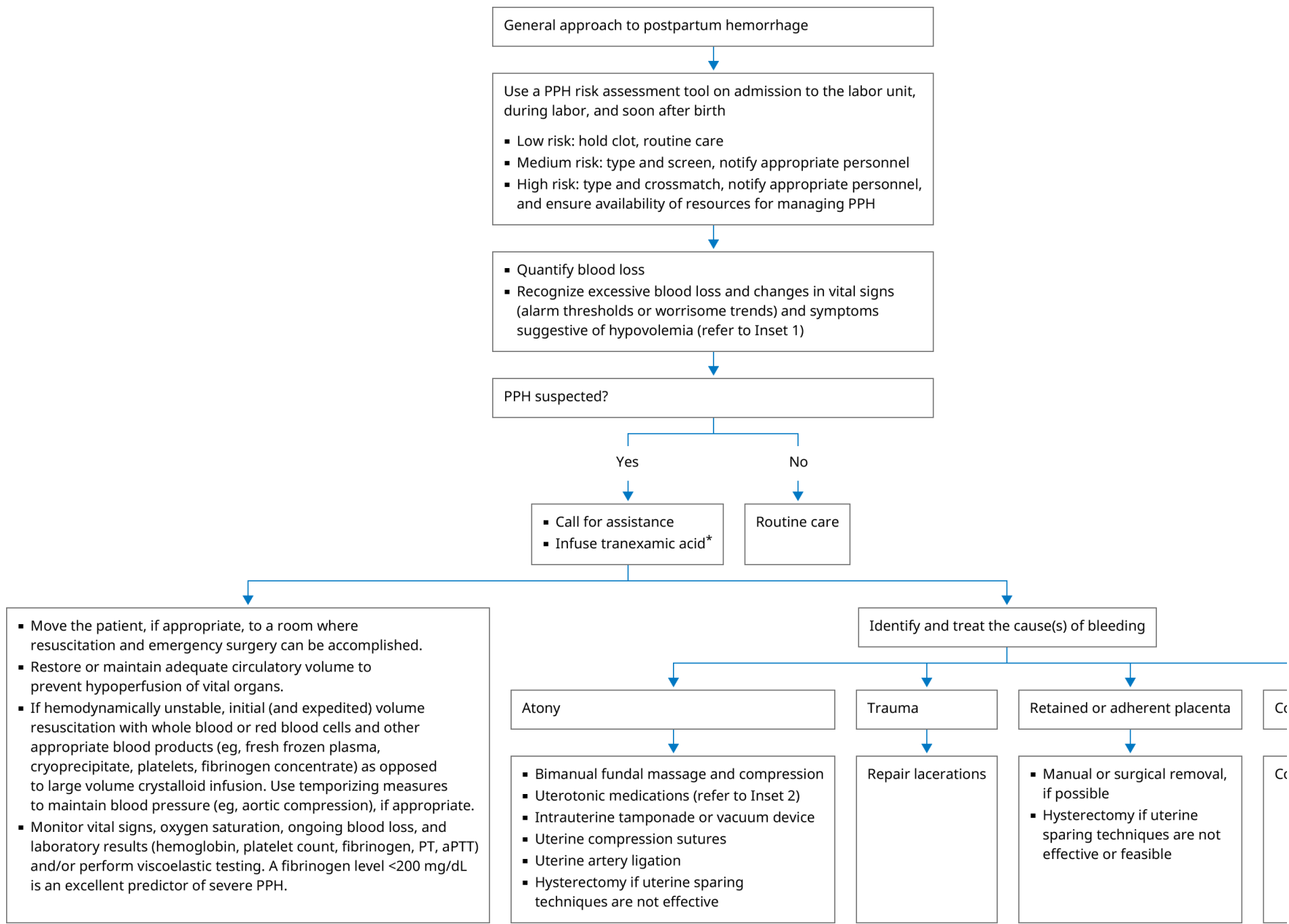
-
- Equipment/supplies for drawing blood (eg, syringes; needles; red, green, blue, and tiger top tubes; alcohol prep pads; tourniquet) for laboratory studies (eg, type and cross, coagulation studies, CBC, platelets, electrolytes, ionized calcium, potassium) with prewritten lab and blood bank requisition orders; instructions on how to order tests and blood and how to activate the massive transfusion protocol
-
- Biohazard bag
-
- Adult oxygen nonrebreather mask
-
- Tubing and filter for blood transfusion
-
- Equipment for warming irrigation and intravenous fluids (including blood)
-
- Pressure infuser bags
-
- Tape, bandages
-
- Medications:
 - Kit for transabdominal intramyometrial injection of carboprost under ultrasound guidance: 20 mL syringe, 20 mL sterile saline for injection, 6-inch 20-gauge and 6-inch 22-gauge amniocentesis needles
 - Misoprostol, five 200 mcg tablets
 - Oxytocin, 10 to 40 units per 500 to 1000 mL NS 2 bags
 - Methylergonovine, 0.2 mg/mL 1 ampule (requires refrigeration)
 - Carboprost, 250 mcg/mL 1 ampule (requires refrigeration)
 - Tranexamic acid 1 gram/10 mL vial, 1 or 2 vials
-

CBC: complete blood count; NS: normal saline.

Adapted from:

1. OB Hemorrhage Toolkit V3.0 - Appendix E: Checklist: Carts, Kits and Trays. California Maternal Quality Care Collaborative. <https://www.cmqcc.org/resource/ob-hemorrhage-toolkit-v30-appendix-e-checklist-carts-kits-and-trays> (Accessed on February 25, 2025).
 2. Postpartum hemorrhage cart inventory list. Texas Children's Pavilion for Women.
-

Overview of the general approach to postpartum hemorrhage



Inset 1

Blood loss, % (mL)	Systolic blood pressure, mmHg	Signs and symptoms
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Inset 2

- Oxytocin (first-line): 10 to 30 units in 500 mL normal saline infused at a rate sufficient to control atony or 10 units IM

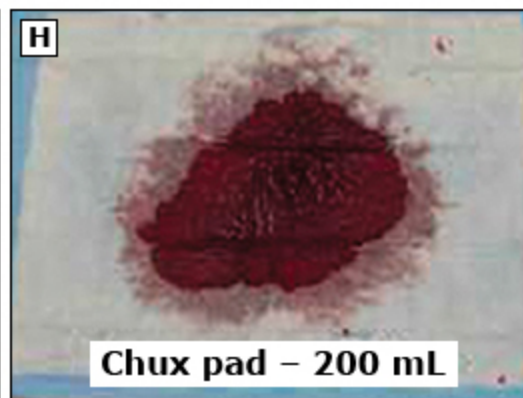
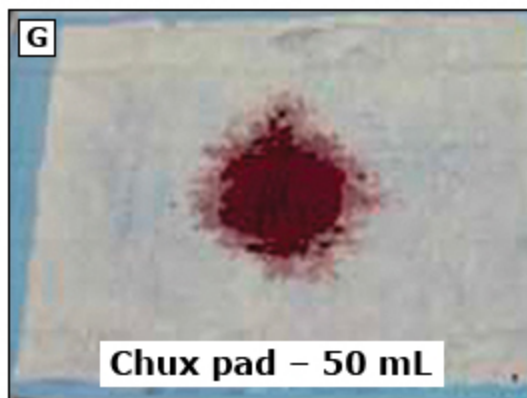
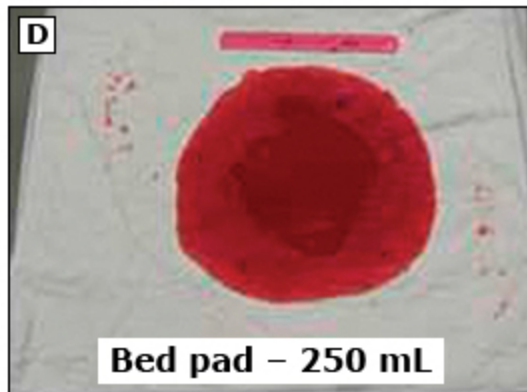
10 to 15 (500 to 1000)	Normal and ≥ 90	Palpitations, lightheadedness, no or mild increase in heart rate
15 to 25 (1000 to 1500)	80 to 90	Weakness, sweating, tachycardia (100 to 120 beats/minute), tachypnea (respiratory rate of 20 to 24)
25 to 35 (1500 to 2000)	70 to 80	Restlessness, confusion, pallor, oliguria, tachycardia (120 to 140 beats/minute), cool and clammy skin
35 to 45 (2000 to 3000)	50 to 70	Lethargy, air hunger, anuria, collapse, tachycardia (>140 beats/minute)

infused at a rate sufficient to control bleeding or 10 units IM.
<ul style="list-style-type: none"> Tranexamic acid (adjunctive agent): 1 g (10 mL of a 100 mg/mL solution) is infused over 10 to 20 minutes; if bleeding persists after 30 minutes, a second 1 g dose is administered.
<ul style="list-style-type: none"> Ergots (second-line): Methylergonovine 0.2 mg IM or ergonovine 0.2 mg IM every 2 to 4 hours.
<ul style="list-style-type: none"> Carboprost (second-line): 0.25 mg IM every 15 to 90 minutes up to 8 doses.
<ul style="list-style-type: none"> Misoprostol: 200 to 600 mcg orally or sublingually once. If oral administration is not possible, 400 to 800 mcg can be administered rectally once.

aPTT: activated partial thromboplastin time; PPH: postpartum hemorrhage; PT: prothrombin time.

* WHO guidelines recommend **not** using tranexamic acid in patients with a known contraindication, including a known thromboembolic event in pregnancy, history of coagulopathy, active intravascular clotting, or known hypersensitivity to the medication.

Visual aid for estimating intrapartum blood loss



Visual aid. Pocket card with images of measured volumes of artificial blood. Panel F is a manikin.

From: Zuckerwise LC, Pettker CM, Illuzzi J, et al. Use of a novel visual aid to improve estimation of obstetric blood loss. Obstet Gynecol 2014; 123:982. DOI: [10.1097/AOG.0000000000000233](https://doi.org/10.1097/AOG.0000000000000233). Copyright © 2014 American College of Obstetricians and Gynecologists. Reproduced with permission from Wolters Kluwer Health. Unauthorized reproduction of this material is prohibited.

Graphic 103418 Version 5.0

Maternal early warning criteria

Systolic BP (mmHg)	<90 or >160
Diastolic BP (mmHg)	>100
Heart rate (beats per minute)	<50 or >120
Respiratory rate (breaths per minute)	<10 or >30
Oxygen saturation on room air, at sea level, %	<95
Oliguria, mL/hour for ≥ 2 hours	<35
Maternal agitation, confusion, or unresponsiveness; patient with preeclampsia reporting a non-remitting headache or shortness of breath	

These triggers cannot address every possible clinical scenario that could be faced by an obstetric clinician and must not replace clinical judgment. As a core safety principle, bedside nurses should always feel comfortable to escalate their concerns at any point.

BP: blood pressure.

From: Mhyre JM, D'Oria R, Hameed AB, et al. The maternal early warning criteria: A proposal from the national partnership for maternal safety. Obstet Gynecol 2014; 124:782. DOI: [10.1097/AOG.0000000000000480](https://doi.org/10.1097/AOG.0000000000000480). Copyright © 2014 American College of Obstetricians and Gynecologists. Reproduced with permission from Wolters Kluwer Health. Unauthorized reproduction of this material is prohibited.

Potential interventions for treatment of postpartum hemorrhage

Pharmacologic interventions	
Drug	Dosing
Oxytocin (first-line)	10 to 40 units in 500 to 1000 mL normal saline infused at a rate sufficient to control atony or 5 to 10 units IM.
Tranexamic acid (adjunctive agent)	1 g (10 mL of a 100 mg/mL solution) is infused over 10 to 20 minutes; if bleeding persists after 30 minutes, a second 1 g dose is administered.
Ergots (second-line)	Methylergonovine 0.2 mg IM or ergonovine 0.2 mg IM every 2 to 4 hours.
Carboprost (second-line)	0.25 mg IM every 15 to 90 minutes up to 8 doses.
Misoprostol	400 to 800 mcg sublingually as a single dose. Most useful in settings where injectable uterotonics are unavailable or contraindicated (eg, hypertension, asthma)
Recombinant human factor VIIa (adjunctive agent)	50 to 100 mcg/kg. It is preferable to start with a low dose (40 or 60 mcg/kg). The dose may be repeated once in 15 to 30 minutes if there is no response. Additional doses are unlikely to be effective.
Surgical interventions	
<ul style="list-style-type: none">▪ Repair lacerations▪ Curettage▪ Uterine compression suture (eg, B-Lynch suture)▪ Uterine artery ligation▪ Utero-ovarian artery ligation or cross clamp▪ Pelvic packing▪ Uterine tourniquet▪ Focal myometrial excision▪ Use of fibrin glues and patches to cover areas of oozing and promote clotting▪ Placement of figure 8 sutures or other hemostatic sutures directly into the placental bed▪ Resuscitative endovascular balloon occlusion of the aorta (REBOA)▪ Internal iliac artery (hypogastric artery) ligation▪ Aortic/iliac artery compression	

- Hysterectomy, supracervical
- Hysterectomy, total

Interventional endovascular procedures

- Selective arterial embolization
- Intermittent aortic balloon occlusion
- Common iliac artery balloon occlusion

Blood bank

- Packed red blood cells
- Platelets
- Fresh frozen plasma
- Cryoprecipitate

Nonsurgical interventions

- Uterine massage
- Intravenous fluids
- Intrauterine tamponade
 - Intrauterine balloon or alternative device (eg, bladder catheter bulb, Sengstaken-Blakemore tube)
 - Intrauterine vacuum
 - Uterine packing (eg, 4-inch gauge packing)

Consultations

- General surgery
 - Trauma surgery
 - Anesthesia team
 - Interventional radiology
 - Gynecologic oncology
 - Urology
-

IM: intramuscular; IV: intravenous; kg: kilogram; mcg: micrograms.

Data from:

1. *Dahlke JD, Mendoz-Figueroa H, Maggio L, et al. Prevention and management of postpartum hemorrhage: a comparison of 4 national guidelines. Am J Obstet Gynecol 2015; 213.e1.*
 2. *Bienstock JL, Ahizechukwu CE, Hueppchen NA. Postpartum hemorrhage. N Engl J Med 2021; 384:1635.*
-

Graphic 73412 Version 17.0

Types and characteristics of balloon catheters designed for intrauterine use (all are single-use balloon catheters)

Name and manufacturer	Balloon composition	Manufacturer-recommended maximum fill volume*	Actual filling capacity (as demonstrated by in vitro trials) ^{¶ [1]}	Maximum time*	Special features	Limitations
Bakri Cook Medical	Single silicone balloon	500 mL	2850 mL	24 hours	<ul style="list-style-type: none"> ▪ Rapid instillation components available to fill by spiking a bag of fluid ▪ May inflate with a syringe ▪ Drainage port allows efflux ▪ Latex free 	<ul style="list-style-type: none"> ▪ Vaginal packing may be needed ▪ Intrauterine drainage port protrudes pas balloon surface
BT-Cath Utah Medical	Single soft silicone balloon	500 mL	>5000 mL	24 hours	<ul style="list-style-type: none"> ▪ Intrauterine drainage port flush with top of balloon ▪ Allows inflation from syringe or IV bag ▪ Latex free 	<ul style="list-style-type: none"> ▪ Vaginal packing may be required
ebb Complete Tamponade	Double balloon system	300 mL (vaginal balloon)	>5000 mL (intrauterine)	24 hours	<ul style="list-style-type: none"> ▪ Vaginal balloon may 	<ul style="list-style-type: none"> ▪ Two ports required for

System	Intrauterine balloon: Polyurethane	750 mL (intrauterine balloon)	balloon)		reduce expulsion and may be left uninflated ▪ Rapid inflation from IV or syringe	inflation
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IV: intravenous.

* As published by the device manufacturer.

¶ Actual filling capacity (prior to balloon rupture) may be higher than the maximum manufacturer-recommended fill volumes and is important to note if instillation of additional fluid is required to effectively stop bleeding.

Reference:

1. Antony KM, Racusin DA, Belfort MA, Dildy GA 3rd. Under Pressure: Intraluminal Filling Pressures of Postpartum Hemorrhage Tamponade Balloons. *AJP Rep* 2017; 7:e86. Courtesy of Karin A. Fox, MD, MEd.

Types of balloons not designed for intrauterine use that may be effective if an intrauterine balloon is not available (all are single-use and off-label)

Type	Composition	Manufacturer-recommended maximum fill volume	Actual filling capacity (as demonstrated by in vitro studies)* ^[9]	Maximum time	Features/original design	Limitations
Rusch urologic hydrostatic balloon ^[1]	Natural latex	500 to 1500 mL	Not reported	24 hours	<ul style="list-style-type: none"> ▪ Potential for large volume of inflation 	<ul style="list-style-type: none"> ▪ Off-label use
Sengstaken-Blakemore Tube ^[2,3]	Natural latex	250 mL (gastric balloon)	3350 mL	24 hours	<ul style="list-style-type: none"> ▪ Two-balloon catheter designed originally to stop bleeding from esophageal varices 	<ul style="list-style-type: none"> ▪ Off-label use ▪ Long tip on catheter must be trimmed to aid proper placement
Condom catheter ^[2,4-6]	Latex, plastic, lambskin	200 to 500 mL ^[7]	4750 mL	24 hours	<ul style="list-style-type: none"> ▪ Condom affixed to a straight urinary catheter ▪ Kit designed for and tested in resource-poor settings ▪ May assemble out of available local resources ▪ Very low cost 	<ul style="list-style-type: none"> ▪ Requires assembly ▪ Need to clamp catheter to avoid efflux of fluid filling balloon ▪ Single lumen catheter, which may not allow egress of

						blood from uterus
Glove catheter ^[8]	Nonlatex surgical glove affixed by a tie to a catheter	Fill until the balloon starts to bulge at the cervix	Not reported	24 hours	<ul style="list-style-type: none"> ▪ Option for resource-poor settings ▪ Very low cost 	<ul style="list-style-type: none"> ▪ Requires assembly ▪ Need to clamp catheter to avoid efflux of fluid filling balloon ▪ Single lumen catheter, which may not allow egress of blood from uterus

* Actual filling capacity (prior to balloon rupture) may be higher than the maximum manufacturer-recommended fill volumes and is important to note if instillation of additional fluid is required to effectively stop bleeding.

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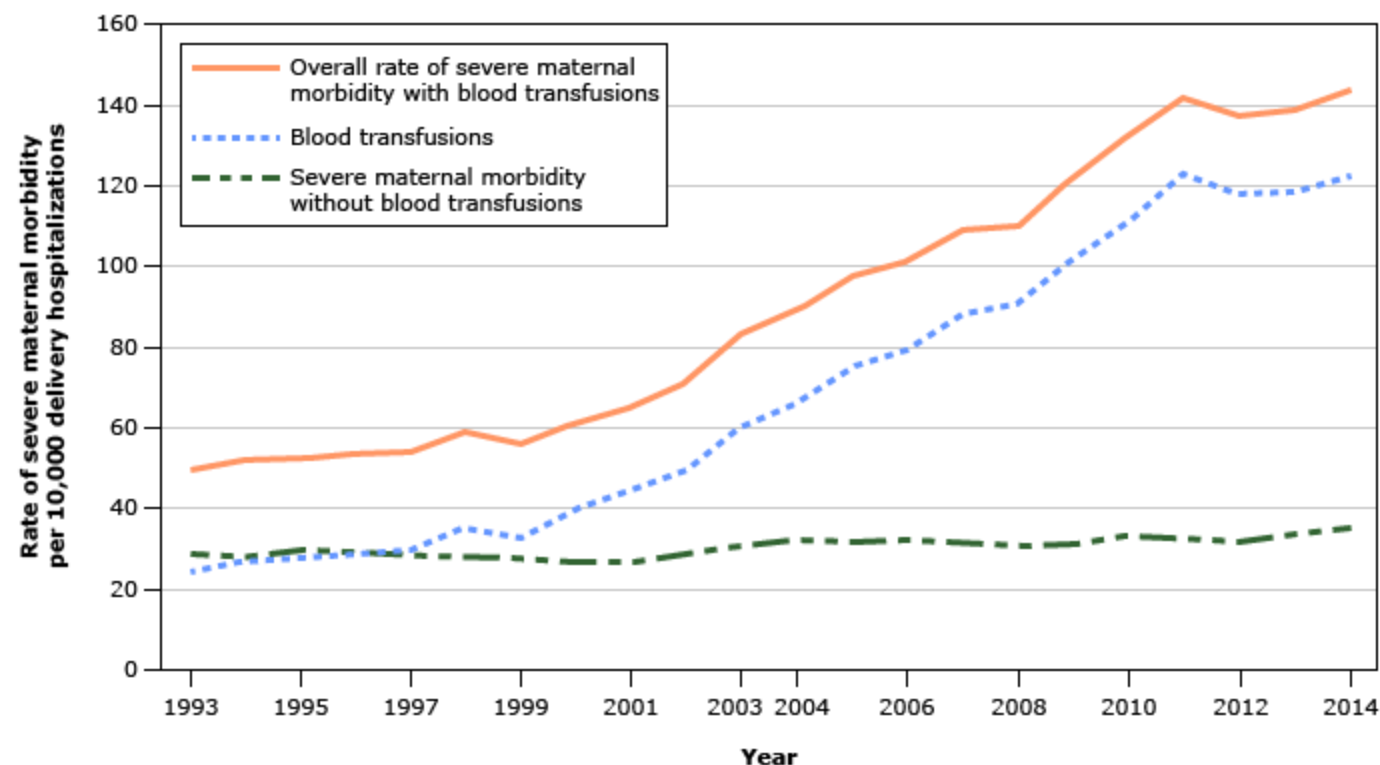
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Types of specialized hemostatic gauze

Type	Composition	Mechanism of action
Celox	Chitosan-derived polysaccharide granules Available impregnated in gauze/ribbon bandages of various sizes	Chitosan polysaccharide granules swell and form a gel-like plug that aids hemostasis. Works in hypothermic conditions and independently of the body's clotting mechanism.
ChitoGauze XR2 Pro	Chitosan-coated gauze dressing with radiographically detectable element	Chitosan polysaccharide granules swell and form a gel-like plug that aids hemostasis. Works in hypothermic conditions and independently of the body's clotting mechanism.
QuikClot	Kaolin-impregnated gauze/dressing of various sizes	Kaolin activates natural clotting cascade upon contact.

Courtesy of Karin A. Fox, MD, MEd.

Rate of severe maternal morbidity per 10,000 delivery hospitalizations



Severe maternal morbidity (SMM) in the United States over time. The overall rate increased almost 200% over the years, from 49.5 in 1993 to 144.0 in 2014. This increase has been mostly driven by blood transfusions, which increased from 24.5 in 1993 to 122.3 in 2014.

Reproduced from: *Severe Maternal Morbidity in the United States*. Centers for Disease Control and Prevention.
<https://www.cdc.gov/reproductivehealth/maternalinfanthealth/severematernalmorbidity.html> (Accessed on March 16, 2020).

Contributor Disclosures

Michael A Belfort, MBBCH, MD, PhD, D.A. (SA), FRCSC, FRCOG, FACOG No relevant financial relationship(s) with ineligible companies to disclose. **Dena Goffman, MD** Grant/Research/Clinical Trial Support: KOKO Medical [Research support to institution – Abnormal postpartum uterine bleeding]; NICHD [Maternal sepsis bundle]; NIH [R01 postpartum hemorrhage simulation; effectiveness of pictographs to prevent wrong-patient errors in the NICU]; Organon [Research support to institution – Abnormal postpartum uterine bleeding]. Consultant/Advisory Boards: Baymatob [Advisory board – AI-guided solutions to improve health outcomes for mothers and their babies]; Cooper Surgical [Obstetric Safety Council – Fertility and women's health]; FetalEase [Clinical review committee – Shoulder dystocia]; Organon [Scientific Advisory Board – Abnormal postpartum uterine bleeding]. All of the relevant financial relationships listed have been mitigated. **Vanessa A Barss, MD, FACOG** No relevant financial relationship(s) with ineligible companies to disclose.

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

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