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Wolters Kluwer

Overview of postpartum hemorrhage

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INTRODUCTION

Postpartum hemorrhage (PPH) is an obstetric emergency. It is one of the top five causes of maternal mortality in both high and low per capita income countries, although the absolute risk of death from PPH is much lower in high-income countries. Timely diagnosis, appropriate resources, and appropriate management are critical for preventing death.

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 management of PPH are discussed separately (See "Postpartum hemorrhage: Medical and minimally invasive management" and "Postpartum hemorrhage: Management approaches requiring laparotomy".)

TERMINOLOGY

PPH occurring in the first 24 hours after delivery may be called primary or early PPH, and is the subject of this topic. PPH occurring from 24 hours to 12 weeks after delivery is usually called secondary, late, or delayed PPH, and is discussed separately. (See "Secondary (late) postpartum hemorrhage".)

DEFINITION/DIAGNOSIS

We make the diagnosis of PPH in postpartum women with bleeding that is greater than expected and results in signs and/or symptoms of hypovolemia (table 1). Diagnosis may be delayed in symptomatic

women when bleeding is not observed, such as intra-abdominal bleeding after a vaginal delivery or after closure of the abdomen in a cesarean delivery.

Multiple other criteria for diagnosis of PPH are in use worldwide (table 2). Although PPH is classically defined by the volume of blood loss (ie, estimated blood loss ≥500 mL after vaginal birth or ≥1000 mL after cesarean delivery), this diagnosis is problematic because the mean blood loss reported after vaginal and cesarean deliveries is approximately 400 to 600 mL and 1000 mL, respectively [1-3], bleeding may not be visible externally, or blood in collection devices may be mixed with amniotic fluid.

In 2017, the American College of Obstetricians and Gynecologists revised their definition of PPH from the classic one (≥500 mL after vaginal birth or ≥1000 mL after cesarean delivery) to (1) cumulative blood loss ≥1000 mL **or** (2) bleeding associated with signs/symptoms of hypovolemia within 24 hours of the birth process regardless of delivery route in order to reduce the number of women inappropriately labeled with this diagnosis [4].

INCIDENCE

The incidence of PPH varies widely, depending upon the criteria used to diagnose the disorder. A reasonable estimate is 1 to 5 percent of deliveries [5,6]. In an analysis of population-based data from the United States National Inpatient Sample, the incidence was between 2 and 3 percent during the years 1994 to 2006 [7] and 3 percent in 2012 to 2013 [8].

PHYSIOLOGIC MECHANISMS THAT LIMIT POSTPARTUM BLOOD LOSS

The potential for massive hemorrhage after delivery is high because, in late pregnancy, uterine artery blood flow is 500 to 700 mL/min and accounts for approximately 15 percent of cardiac output. Normally, hemostasis occurs upon placental separation because uterine bleeding is controlled by a combination of two mechanisms:

- Contraction of the myometrium, which compresses the blood vessels supplying the placental bed and causes mechanical hemostasis.
- Local decidual hemostatic factors (tissue factor [9,10], type-1 plasminogen activator inhibitor [11,12], systemic coagulation factors [eg, platelets, circulating clotting factors]), which cause clotting.

The pathogenesis of most cases of PPH is a disturbance in one or both of these mechanisms. The pathogenesis for most of the remaining PPH cases is loss of intact vasculature (ie, trauma).

PATHOGENESIS

Focal or diffuse atony — The most common cause of PPH is uterine atony (ie, lack of effective contraction of the uterus after delivery), which complicates 1 in 40 births in the United States and is responsible for at least 75 percent of cases of PPH [8]. The diagnosis of atony is generally made when the uterus does not become firm after routine management of the third stage of labor (ie, uterine massage and oxytocin). Atony may or may not be associated with retained tissue. Placental disorders (eg, morbidly adherent placenta, placenta previa, abruptio placentae), retained products of conception, and uterine inversion result in PPH because they inhibit effective uterine contraction, either focally or diffusely.

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

Although diffuse uterine atony is the most common cause of PPH, it is often responsive to administration of additional uterotonic drugs; thus, it is not the most common reason for massive transfusion at delivery [13].

Trauma — Trauma-related bleeding can be due to lacerations (including uterine rupture) or surgical incisions.

Cervical and vaginal lacerations may develop as a result of the natural processes of delivery or may be related to provider interventions. They may not be noted until excessive postpartum vaginal bleeding prompts lower genital tract examination, including examination for vaginal and vulvar hematomas.

Corpus lacerations may be complete transmyometrial ruptures or incomplete lacerations of the inner myometrium [14]. (See "Uterine rupture: Unscarred uterus" and "Uterine rupture: After previous cesarean delivery".)

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

Coagulopathy — Coagulopathy is a cause of PPH in women with an inherited or acquired bleeding diathesis, and a result of PPH when there is a severe reduction of clotting factors due to persistent heavy bleeding and hemodilution of the remaining clotting factors. Acute coagulopathies can be caused by amniotic fluid embolism, placental abruption, preeclampsia with severe features, or HELLP syndrome.

RISK FACTORS AND SPECIFIC ETIOLOGIES

Many risk factors for PPH have been reported and are often interdependent. The types and frequencies are illustrated by the following large series:

- In a study including over 154,000 deliveries that compared 666 cases of PPH to controls without hemorrhage, factors significantly associated with hemorrhage were, in decreasing order of frequency [15]:
    - Retained placenta/membranes (odds ratio [OR] 3.5, 95% CI 2.1-5.8)
    - Failure to progress during the second stage of labor (OR 3.4, 95% CI 2.4-4.7)
    - Morbidly adherent placenta (OR 3.3, 95% CI 1.7-6.4)
    - Lacerations (OR 2.4, 95% CI 2.0-2.8)
    - Instrumental delivery (OR 2.3, 95% CI 1.6-3.4)
    - Large for gestational age newborn (eg, >4000 g) (OR 1.9, 95% CI 1.6-2.4)
    - Hypertensive disorders (preeclampsia, eclampsia, HELLP [Hemolysis, Elevated Liver enzymes, Low Platelets]) (OR 1.7, 95% CI 1.2-2.1)
    - Induction of labor (OR 1.4, 95% CI 1.1-1.7)
    - Prolonged first or second stage of labor (OR 1.4, 95% CI 1.2-1.7)
- In a study including over 690,000 deliveries, the four risk factors associated with the highest odds for predicting the need for massive transfusion (n = 406) during hospitalization for delivery were [16]:
    - Abnormal placentation (placenta accreta or previa) (1.6/10,000 deliveries, adjusted OR [aOR] 18.5, 95% CI 14.7-23.3)
    - Placental abruption (1.0/10,000 deliveries, aOR 14.6, 95% CI 11.2-19.0)
    - Severe preeclampsia (0.8/10,000 deliveries, aOR 10.4, 95% CI 7.7-14.2)
    - Intrauterine fetal demise (0.7/10,000 deliveries, aOR 5.5, 95% CI 3.9-7.8)

Other purported risk factors include: personal or family history of previous PPH (see 'Recurrence' below), obesity, high parity, Asian or Hispanic race, precipitous labor, uterine overdistention (eg, multiple gestation, polyhydramnios, macrosomia), chorioamnionitis, uterine inversion, leiomyoma,

Couvelaire uterus, inherited bleeding diathesis, acquired bleeding diathesis (eg, amniotic fluid embolism, abruptio placentae, sepsis, fetal demise), assisted reproductive technology, and use of some drugs (uterine relaxants, antithrombotic drugs, possibly antidepressants) [13,17-27].

Although there are many known risk factors for PPH, knowledge of these risk factors is not always clinically useful for prevention of hemorrhage.

ASSESSMENT OF SEVERITY OF HEMORRHAGE

Significant drops in blood pressure are generally not manifested until substantial bleeding has occurred, and up to 25 percent of a patient's blood volume (≥1500 mL in pregnancy) can be lost before blood pressure falls and heart rate rises [28]. Hemoglobin and hematocrit values are poor indicators of acute blood loss since they may not decline immediately after an acute bleed. However, a low fibrinogen level (less than 200 mg/dL) is predictive 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. (See "Postpartum hemorrhage: Medical and minimally invasive management", section on 'Laboratory evaluation'.)

California maternal quality care collaborative staging system — The California Maternal Quality Care Collaborative OB Hemorrhage Protocol describes the following stages of PPH [29]:

- Stage 0: Blood loss <500 mL with vaginal delivery or <1000 mL with cesarean delivery. Stable vital signs.
- Stage 1: Blood loss >500 mL vaginal delivery or >1000 mL cesarean delivery **or** change in vital signs (by >15 percent **or** heart rate ≥110 beats/minute, blood pressure ≤85/45 mmHg, O_2 saturation <95%)
- Stage 2: Continued bleeding with total blood loss <1500 mL
- Stage 3: Continued bleeding with total blood loss >1500 mL **or** transfusion of more than 2 units packed red blood cells **or** unstable vital signs **or** suspicion of disseminated intravascular coagulation

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 the shock state [30]. The following classes were derived from nonpregnant populations and may be somewhat different in postpartum women:

- 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 occurs when there is a 15 to 30 percent blood volume loss and is manifested clinically as tachycardia (heart rate of 100 to 120), tachypnea (respiratory rate of 20 to 24), and a 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 30 to 40 percent blood volume loss, 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 and "thready") and respiratory rate are markedly elevated, while urine output is diminished. Capillary refill is delayed.

- Class IV hemorrhage involves more than 40 percent blood volume loss leading to significant depression in blood pressure and mental status. Most patients in class IV shock are hypotensive (systolic blood pressure less than 90 mmHg). Pulse pressure is narrowed (≤25 mmHg), and tachycardia is marked (>120). Urine output is minimal or absent. The skin is cold and pale, and capillary refill is delayed.

Differential diagnosis of mild hemodynamic instability — 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 woman was hemodynamically stable prior to delivery, the level of the block did not become significantly higher immediately following delivery, and symptoms did not abruptly follow systemic administration of a drug known to cause hypotension. (See "Adverse effects of neuraxial analgesia and anesthesia for obstetrics", section on 'Hypotension'.)

PLANNING

Management of risk — Women with risk factors for PPH should be identified and counseled as appropriate for their level of risk and gestational age (see 'Risk factors and specific etiologies' above).

Planning for these patients involves ensuring availability of resources that might be needed, including personnel, medication, equipment, adequate intravenous access, and blood products. For example, the American College of Obstetricians recommends that women identified prenatally as high risk for PPH (eg, placenta accreta spectrum, prepregnancy body mass index >50, clinically significant bleeding disorder, or other surgical/medical high risk factor) should plan to be delivered in a facility that has an appropriate level of care for their needs [31].

Intrapartum, blood should be typed and screened for women with a medium risk factor for PPH (eg, prior uterine surgery, multiple gestation, grand multiparity, prior PPH, large fibroids, macrosomia, body mass index >40, anemia, chorioamnionitis, prolonged second stage, oxytocin >24 hours, magnesium sulfate administration) and typed and crossmatched for those at high risk of PPH (eg, placental previa or accreta, bleeding diathesis, two or more medium risk factors for PPH). Use of a cell saver (blood salvage) should be considered for women at increased risk of PPH, but is not cost-effective as a routine in all cesarean deliveries [32]. (See "Postpartum hemorrhage: Management approaches requiring laparotomy", section on 'Role of intraoperative cell salvage'.)

Routine prophylactic use of uterotonic drugs, such as oxytocin, reduces the risk of PPH by 50 percent in the overall obstetric population [33]. Prophylactic administration of tranexamic acid is under investigation [34]. (See "Management of the third stage of labor: Drug therapy to minimize hemorrhage" and "Postpartum hemorrhage: Medical and minimally invasive management", section on 'Administer tranexamic acid'.)

Specific interventions are available for managing risk in women when the following conditions are identified antenatally:

- Abnormal placentation (see "Management of the placenta accreta spectrum (placenta accreta, increta, and percreta)" and "Placenta previa: Management").
- Bleeding diatheses (see "Treatment of von Willebrand disease", section on 'Pregnancy and delivery' and "Clinical manifestations and diagnosis of hemophilia", section on 'Obstetrical issues' and "Use of anticoagulants during pregnancy and postpartum", section on 'Labor and delivery' and "Thrombocytopenia in pregnancy" and "Thrombocytopenia in pregnancy", section on 'Management decisions').

However, for most patients, knowledge of risk factors for PPH is not useful clinically because only a small proportion of at-risk women develop PPH (abnormal placentation is an exception) and many women without risk factors experience PPH [25,35]. As an example, the California quality improvement toolkit classifies patients as low, medium, or high risk for PPH:

- Low risk

    - Singleton pregnancy
    - Fewer than four previous deliveries
    - No previous uterine surgery
    - No history of PPH

- Medium risk

    - Prior uterine surgery
    - More than four previous deliveries
    - Multiple gestation
    - Large fibroids
    - Chorioamnionitis
    - Magnesium sulfate or prolonged oxytocin infusion

- High risk

    - Morbidly adherent placenta
    - Hematocrit <30 percent
    - Bleeding at admission
    - Bleeding diathesis/coagulation defect
    - History of PPH
    - Tachycardia, hypotension

In a validation study, 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 [35].

PPH protocols and algorithms — Ideally, each hospital labor and delivery unit should have a PPH protocol. 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 management of PPH appears to result in improved outcomes for these women [36-38]. In an observational study, the initiation of a PPH protocol was associated with resolution of maternal bleeding at an earlier stage, use of fewer blood products, and a 64 percent reduction in the rate of disseminated intravascular coagulation [39].

Some graphic examples of PPH protocols are provided below:

- Texas Children's Hospital flowchart (algorithm 1A).

- American College of Obstetricians and Gynecologists four-stage system for classifying PPH, and checklist of appropriate interventions at each stage.

- The California Maternal Quality Care Collaborative provides comprehensive information in several formats for management of PPH.

Massive transfusion is often required with severe PPH, and can be facilitated by use of an algorithm (algorithm 1B)

PPH kits — In addition to a protocol, it is useful for labor and delivery units to assemble kits that contain medications and instruments that may be needed to manage PPH so that these resources are readily available when needed (similar to a "code cart") (table 3).

Training and simulation — The Joint Commission recommends that obstetrical staff [40]:

- 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, and
- 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. (See "Reducing adverse obstetrical outcomes through safety sciences", section on 'Postpartum hemorrhage'.)

GENERAL PRINCIPLES OF MANAGEMENT

Objective measurement of blood loss — Once PPH is suspected, or in those cases where there is a high probability that PPH will occur, objective quantitative measurement of blood loss should be initiated. This is an important factor for early recognition of excessive blood loss and timely initiation of life-saving interventions [41-43]:

- Collect blood in graduated measurement containers, including drapes with calibrated pockets.
- 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 for this assessment.
- 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.

A systematic review concluded evidence was insufficient to support the use of one method over another for estimating blood loss after vaginal birth [44]. For all of these methods, the clinician should attempt to account for fluids other than blood (eg, amniotic fluid, irrigation fluid, urine) that are collected or absorbed.

Timely diagnosis and early intervention — Timeliness in recognition of PPH, determining the cause, and initiating treatment is critical, as almost 90 percent of deaths due to PPH occur within four hours of giving birth [45,46]. It is important to not allow the patient to become moribund before initiating life-saving measures. Early intervention may prevent shock (inadequate perfusion and oxygenation of tissues) and the development of the potentially lethal triad of hypothermia, acidosis, and coagulopathy. These interventions are described separately. (See "Postpartum hemorrhage: Medical and minimally invasive management".)

Teamwork — In the author's opinion, clinical training programs that encourage a team approach for early recognition of PPH can improve outcomes by engaging the necessary providers before hypovolemia and uncompensated shock occur. Obstetricians, midwives, nurses, anesthesiologists, hematologists/blood bank personnel, laboratory medicine, surgical subspecialists (eg, vascular, urology), and interventional radiologists may be involved in managing PPH [47]. 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.

Monitor bleeding, vital signs, and laboratory results — Close maternal monitoring is critical to assess the best approach to and aggressiveness of intervention, and requires bedside evaluation by the provider. Laboratory evaluation includes complete blood count, coagulation studies, potassium and ionized calcium levels. (See "Postpartum hemorrhage: Medical and minimally invasive management", section on 'Laboratory evaluation'.)

Treatment goals — Treatment goals are to:

- 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

Treatment approach — Potential interventions for management of PPH are listed in the table (table 4) and discussed below.

Consider the cause and severity of bleeding, and need for laparotomy — The treatment approach is based on a combination of factors, including the cause and severity of bleeding and

whether the abdomen is already open for cesarean delivery. The four most common causes can be considered using the Four T's mnemonic: Tone: uterine atony; Trauma: laceration, hematoma, inversion, rupture; Tissue: retained tissue or invasive placenta; and Thrombin: coagulopathy [48].

- Treatment of atony, the most common cause of PPH, is influenced by both the route of delivery and severity of bleeding (table 5). After a vaginal birth, treatment of atony begins with uterotonic drugs and minimally invasive procedures (eg, intrauterine balloon tamponade) and progresses to more invasive procedures (eg, uterine artery embolization) until hemorrhage is controlled. It is usually possible and desirable to avoid laparotomy and its associated morbidity. (See "Postpartum hemorrhage: Medical and minimally invasive management", section on 'Manage atony'.)

  Uterotonic drugs are also used to treat atony at cesarean delivery, but since the abdomen is already open, surgical procedures to control bleeding requiring laparotomy (eg, uterine artery and utero-ovarian artery ligation, uterine compression sutures) are employed much sooner than after a vaginal delivery, and uterine artery embolization is considered if these procedure fail. (See "Postpartum hemorrhage: Management approaches requiring laparotomy".)

  The obstetrical provider should initiate a sequence of nonoperative and operative interventions for control of PPH and promptly assess the success or failure of each measure. If an intervention does not succeed, the next treatment in the sequence must 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.

- Traumatic, hemorrhaging lacerations need to be controlled surgically, either transvaginally or transabdominally.

- Retained placental tissue needs to be identified and removed. Placenta accreta spectrum generally requires hysterectomy. (See "Retained placenta after vaginal birth" and "Management of the placenta accreta spectrum (placenta accreta, increta, and percreta)".)

- Coagulopathy is treated medically, with transfusion of blood and blood products.

- Early administration of tranexamic acid, an anti-fibrinolytic drug, can reduce death due to bleeding in women with postpartum hemorrhage related to atony or trauma. (See "Postpartum hemorrhage: Medical and minimally invasive management", section on 'Administer tranexamic acid'.)

Approach to hemodynamically unstable patients — When hemorrhage is suspected as the cause of hemodynamic instability, initial (and expedited) management with blood and blood products is advised (as opposed to large volume crystalloid infusion). Hypovolemic hemorrhagic shock is treated with aggressive volume resuscitation with packed red cells and other appropriate blood products. Transfusion should keep up with blood loss, with early activation of a protocol for large volume transfusion in those patients with heavy bleeding. Development of a standardized institutional approach to massive transfusion improves outcome (algorithm 1B). There are no data from clinical trials of PPH to help guide management of transfusion specifically in PPH [49] (see "Postpartum hemorrhage: Medical and minimally invasive management", section on 'Transfuse red blood cells, platelets, plasma'). Management of patients who refuse to accept blood transfusion is addressed separately. (See "The approach to the patient who declines blood transfusion".)

In addition, the author believes early recourse to intrauterine balloon tamponade can be useful to decrease ongoing uterine blood loss following vaginal delivery or after the abdomen is closed following cesarean delivery, and that this measure will allow additional time for assessment and evaluation, stabilization, and institution of resuscitative procedures. In those women who continue to bleed at the time of cesarean and the abdomen is still open, compression sutures and devascularization are more easily accomplished than placing a tamponade balloon, and if all else fails hysterectomy remains the definitive treatment. (See "Postpartum hemorrhage: Management approaches requiring laparotomy", section on 'Intrauterine balloon tamponade' and "Postpartum hemorrhage: Medical and minimally invasive management", section on 'Perform uterine tamponade in patients with atony or lower segment bleeding'.)

If the patient is coagulopathic with an extremely low fibrinogen level (50 to 100 mg/dL), cryoprecipitate and other high-concentration fibrinogen products (eg, fibrinogen concentrate) are indicated since fresh frozen plasma alone will not increase the fibrinogen level to the normal range without requiring excessive volume infusion. (See "Postpartum hemorrhage: Medical and minimally invasive management", section on 'Correct clotting factory deficiencies' and "Plasma derivatives and recombinant DNA-produced coagulation factors" and "Postpartum hemorrhage: Management approaches requiring laparotomy", section on 'Evaluation of the abdomen'.)

Under most circumstances, an acutely unstable and/or coagulopathic patient should receive temporizing measures such as bimanual uterine compression, balloon tamponade, aortic compression, transfusion of blood products, and possibly a high coagulation factor concentrate (eg, fibrinogen concentrate, prothrombin complex concentrate) to allow resuscitation to a point where general anesthesia and surgery are better tolerated. Unless absolutely necessary, emergency hysterectomy should be avoided in a coagulopathic patient with inadequate intravenous access for massive transfusion/correction of electrolyte imbalances, as major surgery in this setting may cause further deterioration in maternal status as a result of uncontrolled retroperitoneal hemorrhage and

myocardial depression. (See "Postpartum hemorrhage: Management approaches requiring laparotomy", section on 'Temporary measures for stabilizing hemodynamically unstable patients' and "Postpartum hemorrhage: Management approaches requiring laparotomy".)

Early resort to hysterectomy is appropriate in women with severe bleeding due to diffuse placenta accreta/increta/percreta or a large uterine rupture. In contrast, hysterectomy is generally a last resort in patients with atony, as these patients can often be managed successfully with medical therapy and less aggressive surgical interventions. However, hysterectomy should not be delayed in those who have depleted their clotting factors and require prompt control of uterine hemorrhage to prevent death. (See "Postpartum hemorrhage: Management approaches requiring laparotomy", section on 'Role of hysterectomy'.)

Approach to hemodynamically stable patients — For hemodynamically stable patients in whom the capacity for blood replacement exceeds that of the ongoing hemorrhage, arterial embolization is an effective treatment for persistent bleeding. In a systematic review of 20 observational studies, 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 [50]. Sixty-two percent of the patients in these studies were post cesarean delivery.

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'.)

MORBIDITY AND MORTALITY

Maternal mortality — Maternal mortality after PPH averages approximately 2 percent, with wide variations worldwide depending on both the overall health of pregnant women in the population and the resources for treatment of PPH [51]. Death rates vary from 0.6 percent in the United Kingdom to 20 percent in parts of Africa, and from 1 in 100,000 deliveries in the United Kingdom versus 1 in 1000 deliveries in parts of the developing world. Women who are anemic at delivery due to poor nutrition or malaria are particularly vulnerable to severe sequelae of PPH.

Transfusion — In a trial including over 20,000 women worldwide with PPH (WOMAN), 54 percent received a blood transfusion [52]. By comparison, the rate of transfusion in the overall obstetric population of the United States is 4 to 7 per 1000 deliveries [8,53] and the frequency of transfusion in PPH deliveries was 16 percent in 2012-2013 [8]. Risks of transfusion include infection, electrolyte abnormalities, allergic reactions, alloimmunization, and volume overload. (See "Indications and

hemoglobin thresholds for red blood cell transfusion in the adult", section on 'Risks and complications of transfusion'.)

Hysterectomy — In the WOMAN trial described above, 3.5 percent of women underwent peripartum hysterectomy because of PPH [52]. In the United States, 2.5 percent of women with PPH underwent hysterectomy in 2012-2013 [8]. Hysterectomy was more common in PPH without atony than with atony (6.6 versus 1.0 percent).

Thromboembolism — In the WOMAN trial described above, 0.3 percent of women with PPH had thromboembolic event (deep vein thrombosis, pulmonary embolus, stroke, myocardial infarction) within 42 days of delivery [52].

Thromboembolism prophylaxis — In trauma patients, transfusion is an independent risk factor for development of thromboembolism [54]. For this reason, all women who have been transfused for PPH should receive mechanical thromboprophylaxis (graduated compression stockings or pneumatic compression device) as soon as feasible and continue thromboprophylaxis until discharge [55]. Twelve to 24 hours after bleeding has been controlled, pharmacologic thromboprophylaxis should be added, providing coagulation tests are normal or close to normal. (See "Use of anticoagulants during pregnancy and postpartum".)

Hemodynamic instability and organ failure — In the WOMAN trial described above, 60 percent of women with PPH had clinical signs of hemodynamic instability at diagnosis of PPH and almost 4 percent developed renal failure, heart failure, respiratory failure, or hepatic failure [52]. Treatment of hemodynamic instability with fluids and blood can lead to volume overload, resulting in pulmonary edema and dilutional coagulopathy.

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. Damage to the pituitary can be mild or severe, and can affect the secretion of one, several, or all of its hormones. A common presentation is a combination of failure to lactate postdelivery and amenorrhea or oligomenorrhea, but any of the manifestations of hypopituitarism (eg, hypotension, hyponatremia, hypothyroidism) can occur any time from the immediate postpartum period to years after delivery. If the patient remains hypotensive after control of hemorrhage and volume replacement, she should be evaluated and treated for adrenal insufficiency immediately; 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".)

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 delivery has been reported to have an intraabdominal pressure that approaches that seen in abdominal compartment syndrome in nonpregnant individuals [56].

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

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 [57,58]. Uterine compression sutures used to treat PPH have also been associated with the development of intrauterine adhesions [59-62].

Treatment is discussed separately. (See "Intrauterine adhesions: Clinical manifestation and diagnosis".)

Severe postpartum anemia — Postpartum anemia is common: One classic criteria for PPH was a 10-point decline in postpartum hematocrit concentration from antepartum levels. Postpartum anemia can also be defined as a hemoglobin level of <11 g/dL at 1 week postpartum and <12 g/dL at 8 weeks postpartum [63].

- **Treatment** – Severe anemia due to PPH may require red cell transfusions, depending on the severity of anemia and the degree of symptomatology attributable to anemia. A common practice is to offer a transfusion to symptomatic women with a hemoglobin value <7 g/dL [4]. (See "Indications and hemoglobin thresholds for red blood cell transfusion in the adult".)

  In most cases of PPH, the amount of iron lost is not fully replaced by the transfused blood. Oral iron should lead to a modest reticulocytosis beginning in approximately seven days and a rise in the hemoglobin concentration of approximately 2 g/dL over the ensuing three weeks. Single dose parenteral iron therapy is another option; advantages are that hemoglobin levels rise faster, symptoms of anemia improve sooner, and less gastric upset occurs compared with oral therapy [64]. Nevertheless, most women with mild to moderate anemia resolve the anemia sufficiently rapidly with oral iron and it is cheap and convenient [65-67]. A ferritin level at approximately six weeks postpartum helps to guide iron therapy. 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 [68]. It is no more effective than iron therapy in this setting [69], and it is expensive. However, for the few women 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 [63].

RECURRENCE

Women with a prior PPH have as much as a 15 percent risk of recurrence in a subsequent pregnancy [70,71]. The risk of recurrence depends, in part, on the underlying cause (eg, the risk of recurrent abruption is 5 to 15 percent).

PPH alone is not a strong indication for screening for inherited bleeding diatheses, given that undiagnosed bleeding disorders are rarely the cause of PPH. As an example, one study of 50 women with PPH who underwent postpartum screening identified a bleeding diathesis in only one woman [72]. However, unexplained PPH that does not respond to general measures should alert clinicians to the possibility of a bleeding disorder as a causative factor [73], especially in women with a history of menorrhagia, excessive bleeding after minor trauma, or a family history of a bleeding disorder. (See "Approach to the adult with a suspected bleeding disorder".)

PREVENTION

Active management of the third stage of labor can reduce the incidence of PPH due to atony. (See "Management of the third stage of labor: Drug therapy to minimize hemorrhage".)

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

- Primary PPH occurs in the first 24 hours after delivery (also called early PPH), and secondary PPH occurs 24 hours to 12 weeks after delivery (also called late or delayed PPH). (See 'Terminology' above.)

- The most common causes of PPH are atony (which may be related to placental disorders), trauma, and acquired or congenital coagulation defects.

- We make the diagnosis of PPH in postpartum women with bleeding that is greater than expected and results in signs and/or symptoms of hypovolemia (table 1). Diagnosis may be delayed in symptomatic women when bleeding is not observed, such as intra-abdominal bleeding after a vaginal delivery or after closure of the abdomen in a cesarean delivery. (See 'Definition/diagnosis' above.)

- Significant drops in blood pressure are generally not manifested until substantial bleeding has occurred, and up to 25 percent of a patient's blood volume (≥1500 mL in pregnancy) can be lost before blood pressure falls and heart rate rises. Hemoglobin and hematocrit values are poor indicators of acute blood loss, but a low fibrinogen level (less than 200 mg/dL) is predictive 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. (See 'Assessment of severity of hemorrhage' above.)

- Women with risk factors for PPH should be identified when possible, and counseled as appropriate for their level of risk and gestational age (see 'Risk factors and specific etiologies' above). Planning for these patients involves ensuring availability of resources that might be needed, including personnel, medications, equipment, adequate intravenous access, and blood products. Coordination is essential and can be facilitated by use of graphic protocols (eg,

(algorithm 1A-B)), availability of PPH kits (table 3), and training/simulation. (See 'Planning' above.)

- Routine prophylactic use of uterotonic drugs, such as oxytocin, reduces the risk of PPH by 50 percent in the overall obstetric population. Specific interventions are available for managing risk in women with abnormal placentation or bleeding diatheses. For most patients, knowledge of risk factors for PPH is not useful clinically because only a small proportion of at-risk women develop PPH (abnormal placentation is an exception) and many women without risk factors experience PPH. (See 'Management of risk' above.)

- Many interventions are available for management of PPH (table 4). The approach to management of PPH varies depending on the cause and severity of bleeding (table 5) and whether the patient has had a vaginal birth or cesarean delivery. Traumatic, hemorrhaging lesions are managed surgically and coagulopathy is managed medically, with replacement of blood products. The treatment of atony depends on the route of delivery, as there is less concern about the morbidity of open operative interventions when the patient's abdomen is already open. (See 'General principles of management' above.)

- We treat patients with mild to moderate postpartum anemia with an oral rather than a parenteral iron preparation. Oral treatment is more convenient, cheaper, and not associated with severe side effects. Severe anemia may require transfusion. (See 'Severe postpartum anemia' above.)

- Women with a prior PPH have as much as a 15 percent risk of recurrence in a subsequent pregnancy. 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 women with a history of menorrhagia, excessive bleeding after minor trauma, or a family history of a bleeding disorder. (See 'Recurrence' above.)

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