Postpartum hemorrhage: Medical and minimally invasive management

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All topics are updated as new evidence becomes available and our peer review process is complete.
Literature review current through: Apr 2018. | **This topic last updated:** Mar 12, 2018.

INTRODUCTION — Postpartum hemorrhage (PPH) is an obstetric emergency with many potentially effective medical and surgical interventions for management (table 1). The key to management is to recognize excessive bleeding before it becomes life threatening, identify the cause, and initiate appropriate intervention based on the clinical setting (eg, cause and severity of bleeding, whether the abdomen is open or not).

This topic will discuss medical and minimally invasive management of patients with PPH. An overview of issues related to PPH (terminology, incidence, pathogenesis, causes, risk factors, diagnosis, general principles of planning and management, morbidity and mortality, recurrence) is available separately (see "Overview of postpartum hemorrhage"). Treatment approaches to PPH that are performed at laparotomy are also reviewed separately. (See "Postpartum hemorrhage: Management approaches requiring laparotomy".)

INITIAL PATIENT ASSESSMENT

Postvaginal delivery — Patients with persistent excessive vaginal bleeding after vaginal delivery should be assessed immediately by a provider who can initiate necessary medical and surgical emergency care. Assessment includes:

- **Vital signs** – Evaluate blood pressure, heart rate, respiratory rate, peripheral oxygen saturation, and urine output. Tachypnea, tachycardia, hypotension, low oxygen saturation, and air hunger are signs of hypovolemia, which may be due to both inadequate hemoglobin level and circulatory volume (table 2).

  Trends in vital signs over time after delivery should also be evaluated to identify and address patterns suspicious for ongoing bleeding or inadequately replaced blood loss. Assume progressively increasing heart rate and decreasing blood pressure are due to blood loss/hypovolemia until these causes are positively excluded. Deterioration of maternal vital signs out of proportion to vaginal bleeding suggests intraperitoneal or retroperitoneal bleeding.

- **Estimated blood loss** – Quantify the amount of blood loss by collecting blood in graduated volumetric containers, using visual aids that correlate the size and appearance of blood on specific surfaces (eg, maternity pad, emesis basin, bed sheet, lap sponge) with the volume of blood absorbed by that surface (picture 1), and measuring the difference in the total weight of bloody materials and 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).

- **Coagulation –** Suspicion of coagulopathy should prompt blood and blood product replacement. (see 'Transfuse red blood cells, platelets, plasma' below). Thromboelastography is quick and accurate, where available. (See 'Thromboelastography and rotational thromboelastometry' below.)

  In the absence of on-unit thromboelastography, check for non-clotting blood or very anemic-appearing vaginal bleeding while waiting for results from the first set of laboratory studies. One method is to draw 5 mL of blood from an arm vein, place it in clean dry red top glass tube, and note the clotting time (ie, time until the blood no longer flows when the tube is inverted). The clotting time is 5 to 8 minutes when the patient likely has adequate fibrinogen stores: If the blood in the tube does not clot within 8 minutes or the initial clot dissolves, then it is likely she is markedly deficient in key clotting factors. Over a dozen methods for testing whole blood clotting time have been described and vary in number of syringes used to draw the blood, whether some of the blood is discarded, the degree and frequency of tilting the tube(s), the diameter of the tube(s), the number of tubes, the volume of blood, whether tubes are pre-rinsed with saline, the temperature at which the test is performed (room temperature versus 37°C), etc [1,2]. All of these variables affect the time to clotting, which make this an insensitive test.

- **Review drugs** that the patient has received as some drugs can have unanticipated hemodynamic side effects that may confound the situation. For example, beta-blockade may prevent a normal heart rate response in a bleeding patient; histamine release due to an analgesic (morphine) may lead to peripheral vasodilation and destabilized compensated shock with resultant sudden hemodynamic collapse.

Postcesarean delivery — At cesarean delivery, the presence and cause of excessive bleeding is usually readily apparent (atony, uterine laceration, retained placental fragments). However, in patients with vital signs that are normal or near normal and have no oozing from wounds, it may not be recognized when blood is retroperitoneal, confined to the uterine cavity after closure of the hysterotomy, or hidden under surgical drapes. When compensated shock is present (normal blood pressure with increasing heart rate) at cesarean delivery, these sites should be actively evaluated.

After a cesarean delivery has been completed (ie, abdomen has been closed), persistent excessive vaginal bleeding is evaluated as described above for vaginal delivery. Signs of compensated or uncompensated shock without vaginal bleeding should prompt consideration of internal bleeding related to delivery. A modified focused assessment with sonography for trauma (FAST) examination in the recovery room may show fluid in the upper abdomen suggestive of intraabdominal bleeding, but sensitivity is low [3] and an equivocal scan or logistic issues have the potential to delay emergency and life-saving interventions. In all cases, clinical signs suggestive of ongoing bleeding (tachycardia, falling blood pressure, expanding abdomen, change in level of consciousness) should overrule a negative ultrasound scan in determining further management.

BLOOD LOSS >500 ML AT VAGINAL DELIVERY OR >1000 ML AT CESAREAN DELIVERY BUT <1500 ML WITH ONGOING EXCESSIVE BLEEDING — These patients are generally hemodynamically stable, but may have mild tachycardia (heart rate ≥110 beats/min), mild hypotension (systolic blood pressure 80 to 85 mmHg), fall in oxygen saturation (O_2 sat <95 percent), and/or lightheadedness, before initiation of therapy.

Basic interventions

- Obtain assistance, if not already assembled. Team may include obstetricians, nurses, anesthesiologists, hematologists/blood bank personnel, laboratory medicine, surgical subspecialists (eg, vascular, urology), and interventional vascular specialists.
- Continue to monitor vital signs and quantify blood loss.
- If not already in an operating room, move potentially unstable and unstable patients to an operating room as soon as practicable, since this is the safest place to initiate and maintain definitive treatment. In those facilities where the labor and delivery operating room may not be equipped and/or staffed for emergency major abdominal surgery, the patient should be stabilized and moved to the main operating room suite (or other appropriately equipped unit, eg, hybrid operating room on labor unit) for further management.

In addition, apply the following interventions.

Establish adequate intravenous access — Adequate intravenous access should be provided with two lines, at least one of which should be a large bore catheter (14 or 16 gauge), for administration of fluids and blood (see 'Transfuse red blood cells, platelets, plasma' below) and medications. (See 'Increase oxytocin infusion' below and 'Administer tranexamic acid' below.)

Resuscitate with crystalloid — Isotonic crystalloid should be infused to prevent hypotension (target systolic pressure 90 mmHg) and maintain urine output at >30 mL/hour [4]. Rapid infusion of large volumes of crystalloid (eg, >3 to 4 L) may promote dilutional coagulopathy, electrolyte imbalances, and hypothermia, so appropriately monitoring of hematocrit, coagulation status, core temperature, and electrolytes is essential. Women with preeclampsia may have a contracted intravascular volume and hemoconcentration, and are prone to tissue hypoperfusion from blood loss. The approach to fluid therapy is similar to that in trauma patients. (See "Intraoperative management of shock in adults" and "Initial management of moderate to severe hemorrhage in the adult trauma patient", section on 'Intravenous fluid resuscitation'.)

Provide adequate anesthesia — In the nonanesthetized patient, local anesthesia rarely provides sufficient pain relief for thorough examination and treatment. Gentle digital exploration of the lower segment of the uterus may be performed without anesthesia; however, if thorough manual examination is needed, this should be performed in an operating room with immediate recourse to anesthesia, surgical therapy, and laparotomy, if needed. The choice of a regional or general anesthetic depends upon the planned interventions and the patient's hemodynamic status. (See "Anesthesia for the patient with peripartum hemorrhage".)

Examine the lower genital tract and uterus to determine the cause of bleeding

- **Perform thorough vaginal, abdominal, and rectal examinations** – It is not sufficient to look only for obvious vaginal or incisional bleeding when determining the source of PPH because significant hemorrhage can occur into the retroperitoneum or into a vaginal/vulval hematoma without visible blood loss.

  After a completed delivery, we examine the genital tract with the woman in stirrups (dorsal lithotomy position) in a room with facilities for general anesthesia and both vaginal and abdominal surgery. Lower genital sources of bleeding not previously detected can usually be readily identified by assessing the birth canal with adequate assistance, exposure, lighting, instruments, and anesthesia, which allow performance of a thorough examination.

  The entire vagina from perineum to cervix should be inspected for significant lacerations. The uterine cavity should be palpated for defects indicating uterine rupture or dehiscence. This examination should be performed in all patients with PPH who delivered vaginally, as well as those who attained significant cervical dilatation and descent of the presenting fetal part before a cesarean delivery. Even if inspection for lacerations had already been performed at delivery (or afterwards without

adequate analgesia), a thorough examination should be repeated, as it is possible that a bleeding site was missed. Risk factors for significant cervical lacerations (ie, associated with excessive bleeding or requiring repair) include precipitous labor, operative vaginal delivery, and cerclage [5]. However, absence of such risk factors should not preclude re-examination of the birth canal.

Intense pain in the anus may be a warning sign of an enlarging vaginal or vulvar hematoma and should prompt examination and repeat examination as necessary to exclude this life-threatening blood loss. (See "Management of hematomas incurred as a result of obstetrical delivery".)

- **Rapidly assess uterine tone**, but be aware that there can still be significant bleeding from a poorly contracted and dilated lower segment despite adequate upper segment contraction.
- **Examine the uterine cavity for retained products of conception**. This is more common after vaginal than cesarean delivery, unless the cesarean was performed for a morbidly adherent placenta.
- **Assess for uterine inversion**, which appears as a smooth round mass protruding from the cervix or vagina on vaginal examination and/or lack of a normally positioned fundus on abdominal examination. (See "Puerperal uterine inversion".)
- **Consider the possibility of uterine rupture** – Although uterine rupture is infrequent, examination for this condition is important in patients with signs of PPH who have undergone a trial of labor after a previous cesarean delivery. Rupture of the unscarred uterus is rare, but can occur if labor was induced or augmented and, even more rarely, after instrumental delivery.

  After delivery, uterine rupture is often characterized by pain and persistent vaginal bleeding despite use of uterotonic agents. Even mild hemodynamic instability in any postpartum patient, whether she has observed bleeding or not, should prompt consideration of uterine rupture and intra-abdominal bleeding. Hematuria may occur if the rupture extends into the bladder. Maternal symptoms of hypovolemia that appear to be out of proportion to the observed blood loss and abdominal distention should also prompt consideration of intra-abdominal hemorrhage. Palpation of the uterine cavity may reveal the opening, which can be anterior, fundal, posterior, or lateral. Ultrasound examination may reveal blood in the abdomen and/or a broad ligament hematoma. (See 'Postcesarean delivery' above.)

  Posterior rupture is more common in the unscarred uterus than the scarred uterus. When laparotomy is performed, a posterior rupture is not readily observed upon entering the abdomen so the entire uterus needs to be inspected carefully. (See "Uterine rupture after previous cesarean delivery" and "Uterine rupture: Unscarred uterus".)

Administer tranexamic acid — Tranexamic acid is an anti-fibrinolytic drug that has been useful for prevention and treatment of bleeding in various clinical settings, such as surgery and trauma. The World Maternal Antifibrinolytic Trial (WOMAN) found that tranexamic acid reduced death due to bleeding in women with postpartum hemorrhage by 20 to 30 percent, and was not associated with an increase in adverse effects [6]. This pragmatic, randomized, double-blind, placebo-controlled trial involved 193 hospitals in 21 countries and evaluated the effect of early administration of tranexamic acid (1 g by intravenous injection) on mortality, hysterectomy, and other morbidities in over 20,000 women with clinically diagnosed PPH. Women were eligible for randomization if blood loss was >500 mL at vaginal delivery, >1000 mL at cesarean, or associated with hemodynamic instability and the provider was uncertain whether to use the drug. All other aspects of management of PPH were per usual standards and determined by the provider. Approximately 70 percent of the deliveries were vaginal and 30 percent cesarean. Compared with placebo, tranexamic acid:

- Reduced death due to bleeding by 19 percent overall (1.5 versus 1.9 percent; relative risk [RR] 0.81, 95% CI 0.65-1.00).
  - The reduction in death due to bleeding was observed after both vaginal and cesarean deliveries. Death due to bleeding was reduced by 31 percent when treatment was initiated within three hours of delivery (1.2 versus 1.7 percent; RR 0.69, 95% CI 0.52-0.91) and by 26 percent for bleeding due to atony (1.2 versus 1.6 percent; RR 0.74, 95% CI 0.55-0.99). In contrast, the reduction was not significant when the time from delivery was greater than three hours and in patients with other or unknown causes of bleeding.
- Reduced the incidence of laparotomy to control bleeding by 36 percent (0.8 versus 1.3 percent; RR 0.64, 95% CI 0.49-0.85).
- Did not reduce hysterectomy; however, the decision to perform hysterectomy was sometimes made at the same time as randomization, so some hysterectomies were performed before or concurrently with administration of tranexamic acid. (For this reason, the trial was extended and the sample size was increased).
- Did not reduce all-cause mortality, which included death from sepsis, organ failure, eclampsia, pulmonary embolism, etc and accounted for over 25 percent of the deaths. There was no significant increase or decrease in any specific cause of death, other than death from bleeding.
- Did not increase the risk of thromboembolic events, which had been a concern based on data in case reports.

We agree with the authors of this trial and recommend administering tranexamic acid soon after diagnosis of PPH, ideally within three hours of delivery, and alongside oxytocin and other uterotonics. Delay in treatment, even if short, reduces the benefit of

tranexamic acid administration.

Meta-analyses of randomized trial have concluded that tranexamic acid reduces mortality due to bleeding in women with primary PPH, irrespective of mode of birth [7,8]. As an example, in a meta-analysis using individual level data from patients with acute severe bleeding (traumatic and post-partum hemorrhage), tranexamic acid increased overall survival from bleeding (OR 1.20, 95% CI 1.08-1.33), and immediate treatment improved survival by more than 70 percent (OR 1.72, 95% CI 1.42-2.10). The survival benefit decreased by 10 percent for every 15 minutes of treatment delay until 3 hours, after which there was no benefit [7].

Tranexamic acid is given concomitantly with other drugs and procedures for control of bleeding, and should not be regarded as an alternative therapy. One gram (10 mL of a 100mg/mL solution) is infused over 10 to 20 minutes, as infusion >1 mL/minute can cause hypotension. If bleeding persists after 30 minutes, a second 1 g dose may be administered. The half-life is two hours and the antifibrinolytic effect lasts up to 7 to 8 hours in serum.

Tranexamic acid should not be mixed with blood or given through a line with blood, or mixed with solutions containing penicillin. It should not be given to patients with subarachnoid hemorrhage or active intravascular clotting (disseminated intravascular coagulation). The dose should be reduced in patients with renal insufficiency, venous or arterial thrombosis, or ureteral bleeding.

About 90 percent of the maternal drug is eliminated within 24 hours after intravenous administration. The concentration in breast milk is about one hundredth of the serum peak concentration, so it is unlikely to have antifibrinolytic effects in the infant.

We are not administering tranexamic acid prophylactically, except in a research protocol. It is generally not administered before delivery since it freely crosses the placenta, but limited evidence has not shown fetal harm.

Begin treatment of the cause of bleeding

- Manage atony. (See 'Manage atony' below.)
- Repair bleeding lacerations. (See 'Repair genital tract lacerations' below.)
- Remove any retained placental fragments or fetal membranes manually, if possible, or with ring forceps. Ultrasound examination can be helpful for diagnosis of retained tissue and to guide removal [9]. Curettage with a 16 mm suction catheter or (preferably) a large blunt curette (banjo curette) is performed if manual removal is unsuccessful in controlling hemorrhage. (See "Retained placenta after vaginal birth".)

  Management of the morbidly adherent placenta is reviewed separately. (See "Management of the placenta accreta spectrum (placenta accreta, increta, and percreta)".)
- Manually replace an inverted uterus, if present. (See "Puerperal uterine inversion".)
- If uterine rupture is diagnosed, definitive surgical management involves hysterectomy, but uterine repair may be possible, depending on patient plans for future pregnancies, extent of uterine damage, hemodynamic stability, and the surgeon's skills. (See "Uterine rupture after previous cesarean delivery" and "Uterine rupture: Unscarred uterus".)

Manage atony — Uterine massage and compression and administration of uterotonic drugs are the key interventions for treatment of atony.

Perform uterine massage and compression — Fundal massage stimulates the atonic uterus to contract. Bimanual uterine massage, which manually compresses the corpus between the clinician's two hands, is another effective technique: One hand is made into a fist and placed vaginally in the anterior fornix, while the other massages the fundus abdominally while firmly compressing it against the vaginal hand.

Massage should be maintained while other interventions are being initiated, and continued until the uterus remains firm and bleeding has abated. If the fundus is well contracted but bleeding continues unabated, then further massage is not likely to be effective and progression to other methods of hemorrhage control should occur promptly.

Increase oxytocin infusion — Oxytocin is routinely initiated just before or after placental separation to reduce postpartum bleeding and risk of hemorrhage. The rate can usually be increased if bleeding is greater than normal.

We administer oxytocin 40 units in 1 L of normal saline intravenously at a rate sufficient to control uterine atony or 10 units intramuscularly (including directly into the myometrium). While higher doses of oxytocin have been used intravenously for a short duration to manage atony (eg, up to 80 units in 500 mL over 30 minutes) [10], this is not advisable since lower doses appear to be just as effective. Moreover, rapid infusion of high-dose oxytocin, as may occur in an emergency situation, can cause significant hypotension and cardiovascular collapse. Therefore, if a high-dose oxytocin regimen is used, it is advisable to prepare smaller volumes (ie, 15 units in 250 mL) to limit the total dose infused over a short period of time. These issues and dosing are discussed in more detail separately. (See "Management of the third stage of labor: Drug therapy to minimize hemorrhage", section on 'Oxytocin'.)

Administer additional uterotonic drugs — Since uterine atony is the most common cause of PPH, uterotonic drugs are administered for presumed atony until a therapeutic effect is observed or until it is obvious that these drugs are ineffective. The important point is not the sequence of drugs, but the prompt initiation of uterotonic therapy and the prompt assessment of its effect. It should be possible to determine within 30 minutes whether pharmacologic treatment is reversing uterine atony. If it does not, prompt invasive intervention (interventional endovascular procedure, laparotomy) is usually warranted.

If bleeding persists after administering oxytocin, we promptly administer carboprost tromethamine (contraindication: asthma) and/or methylergonovine (contraindications: hypertension, coronary or cerebral artery disease, Raynaud's syndrome). Up to eight doses of carboprost tromethamine may be given intramuscularly at least 15 minutes apart, but if bleeding continues unabated after two doses are given, further benefit of repeated doses is unclear. Methylergonovine may be repeated every two to four hours as needed.

If these pharmacologic interventions are ineffective or only partially effective, or if there is any delay in getting these uterotonic drugs, we expeditiously move on to placing a tamponade balloon (see 'Intrauterine balloon catheter' below) to decrease bleeding and plan for interventional radiology or surgical options. Uterotonic drugs are continued until bleeding is controlled.

Preferred uterotonic drugs and doses:

- If no asthma, carboprost tromethamine (15 methyl-PGF2alpha, Hemabate) 250 mcg intramuscularly every 15 to 90 minutes, as needed, to a total cumulative dose of 2 mg (eight doses). About 75 percent of patients respond to a single dose; move on to a different uterotonic agent if no response after one or two doses. Carboprost tromethamine may be injected directly into the myometrium either transabdominally (with or without ultrasound guidance) or vaginally. The author prefers to use a dilute solution of 250 mcg in 20 mL normal saline for injection given via a six-inch spinal needle. Prior to the blind injection of this solution into the myometrium, aspiration should be performed to prevent intravenous administration.
- If no hypertension or other significant arterial disease, methylergonovine 0.2 mg intramuscularly or directly into the myometrium (never intravenously). May repeat at two- to four-hour intervals, as needed. If there has not been a good response to the first dose, quickly move on to a different uterotonic agent.

Other uterotonic drugs:

- Misoprostol (PGE1) is most useful for reducing blood loss in settings where injectable uterotonics are unavailable or contraindicated (eg, hypertension, asthma). There is no strong evidence that misoprostol is more effective than other uterotonics either for primary therapy of PPH or as an adjunctive treatment to oxytocin infusion [11,12]. In addition, the side effect of hyperthermia is a significant disadvantage of this drug because it is uncomfortable, triggers a work up for sepsis, and may lead to unnecessary empiric antibiotic therapy.

  The optimum dose and route of misoprostol administration are unclear [13-19]. If used, we suggest 400 mcg sublingually. Sublingual misoprostol is rapidly absorbed, achieving a peak concentration within 30 minutes. The peak concentration is higher and sustained longer (about three hours) than with oral administration due to avoidance of first-pass hepatic metabolism; thus, sublingual administration is probably the optimal route of administration for PPH. We use 400 mcg because of the increasing potential for hyperthermia with larger doses [20-22]. A systematic review concluded that a dose of 400 mcg sublingually appeared to be as effective as 600 mcg sublingually and had fewer side effects, but available data on optimal dose were limited [23]. Based on data from randomized trials, other reasonable approaches for misoprostol administration include a combination of 200 mcg orally plus 400 mcg sublingually or 400, 600, or 800 mcg sublingually [13,16,23-25]. The World Health Organization suggests a single dose of 800 mcg sublingually [26,27].

  Oral misoprostol is also rapidly and almost completely absorbed, reaching a peak concentration within 30 minutes, but the level is lower than with sublingual administration and declines rapidly over two hours due to hepatic metabolism.

  Rectal administration takes longer to reach peak concentration compared with oral or sublingual administration (up to an hour versus within 30 minutes), which is disadvantageous in the hemorrhaging patient [24,25]. The most commonly used rectal doses are 800 and 1000 mcg [14,15,26,27]. Rectally administered misoprostol has a longer duration of action than oral/sublingual routes (four hours versus two to three hours), which is advantageous in PPH and may be necessary in semi-conscious or unconscious patients.

  Vaginal administration is not recommended because the drug will be washed away by heavy bleeding, thus impairing absorption.

  Maternal temperature should be monitored closely, as pyrexia ≥40 degrees Celsius (104 degrees Fahrenehit) can occur at these doses and should be treated (eg, acetaminophen). The frequency of pyrexia increases with increasing misoprostol dose. High fever may be accompanied by accompanied by adverse autonomic and central nervous system effects.

  In one randomized trial of women with PPH, those who received misoprostol 600 mcg sublingually plus standard uterotonics (oxytocin in 98 percent) had a threefold higher rate of temperature ≥38°C than those who received standard uterotonics alone (58 versus 19 percent); for temperature ≥40°C, the rates were 7 and <1 percent, respectively [28].

- Dinoprostone (PGE2) 20 mg vaginal or rectal suppository is an alternative prostaglandin to misoprostol (PGE1). It can be repeated at two-hour intervals.

- Carbetocin, a long-acting analog of oxytocin, is in use in many countries (but not the United States) for prevention of uterine atony and hemorrhage. In this capacity, it appears to be as effective as oxytocin [29]. Carbetocin 100 mcg is given by a single slow intravenous injection (ie, over one minute), although lower doses may be effective [30]. The toxicity spectrum is similar to that of oxytocin. It seems reasonable to use this drug as an alternative to oxytocin in countries where it is available, as it is easy to administer and has a long duration of action, but its efficacy in treating rather than preventing uterine atony is not well documented.

Repair genital tract lacerations — We repair heavily bleeding vaginal and cervical lacerations with a running locked #0 absorbable suture. Exposure is facilitated by using a Gelpi retractor (figure 1) to spread the distal vaginal sidewalls and Heaney (figure 2) or Breisky (figure 3) retractors to access the upper vagina. If available, use of several assistants with Deaver retractors placed laterally is also effective. Adequate lighting and exposure are crucial in such repairs, often necessitating that repairs are performed in the operating room with appropriate anesthesia, patient positioning, retraction, and suction apparatus.

It is often difficult to begin a suture line at the apex of the laceration because of problems with exposure and visualization. In such cases, one can begin the suture line at the distal end of the laceration and sew toward the apex, while using the suture to pull the lacerated tissue toward the surgeon. Alternatively, these patients are good candidates for angiographic embolization, if stable. (See 'Consider uterine or hypogastric artery embolization' below.)

Three pitfalls to avoid:

- Sutures should not be placed cephalad to the fornix, as this can result in ureteral ligation. When an extension exists high in the vagina, possibly extending into the cardinal ligament, we perform a laparotomy. The patient's thighs are abducted in stirrups to allow surgery to proceed simultaneously via the abdominal and vaginal routes, as needed for optimal exposure and access. This facilitates identification of the bladder and ureters, minimizing the chance of inadvertent damage to these structures. If appropriately skilled surgeons and equipment are available, the abdominal portion may be possible with a laparoscopic approach.

- Vaginal hematomas should not be drained unless expanding. Attempts at operative drainage can result in significant additional blood loss because it is often difficult to identify and ligate bleeding vessels in a fresh vaginal sulcus hematoma. A stable hematoma may be drained later if it becomes infected or pain is not relieved adequately with analgesics. Continuous expansion of a hematoma leading to hypovolemia may necessitate drainage and packing. Alternatively, embolization may be the best approach. Management of vaginal hematomas is discussed in more detail separately. (See "Evaluation and management of female lower genital tract trauma".)

- Arterial or heavy active vaginal bleeding should not be treated with packing, as this has the potential to divert blood into the retroperitoneum. A vaginal balloon or packing can be useful to tamponade venous oozing, before or after repair of the vaginal laceration. A balloon with a drainage channel can compress lacerated sidewalls while the drainage channel allows monitoring of any ongoing significant bleeding. Such patients should be closely monitored for ongoing concealed bleeding, which would necessitate active intervention.

A laparotomy (or angiographic embolization) may be needed when a vaginal laceration has extended above the fornix and appears to be expanding (either on imaging or because of persistent hemodynamic instability). The patient's thighs are abducted in stirrups (modified lithotomy position) to allow surgery to proceed simultaneously via the abdominal and vaginal routes, as needed, for optimal exposure and access. This facilitates identification of the bladder and ureters, minimizing the chance of inadvertently damaging these structures.

Uterine artery laceration will require interventional radiology if the patient is hemodynamically stable, or surgical exploration and ligation if she is not. (See 'Consider uterine or hypogastric artery embolization' below and "Postpartum hemorrhage: Management approaches requiring laparotomy".)

BLOOD LOSS >1500 ML WITH ONGOING EXCESSIVE BLEEDING — These patients may be hemodynamically unstable.

Basic interventions

- Do all of the above. (See 'Initial patient assessment' above and 'Blood loss >500 mL at vaginal delivery or >1000 mL at cesarean delivery but <1500 mL with ongoing excessive bleeding' above.)

- Any unstable patient who is not already in an operating room should be moved to an operating room as soon as practically possible, since this is the safest place to initiate and maintain definitive treatment.

- In addition to large bore IVs for fluid resuscitation, central venous access should be considered early (while the patient is still in compensated shock) as it is often very difficult to gain such access in a shocked and hemodynamically unstable patient. In addition, it may take time to assemble appropriate personnel (anesthesia team, vascular access team) to place the line. A central venous pressure line enables rapid volume infusion and provides supplemental data regarding intravascular volume

status, but these parameters are inaccurate surrogates to determine cardiac preload, are poor predictors of fluid responsiveness, and do not detect or predict impending pulmonary edema indicative of hypervolemia. (See "Overview of central venous access" and "Intraoperative management of shock in adults".)

- A bladder catheter with urometer should be inserted to monitor urine output.

In addition to these basic interventions, apply the following interventions. Although the interventions described below are often successful, in the setting of cardiovascular instability, it is important to avoid prolonged, futile attempts at conservative therapy before proceeding to laparotomy (or re-laparotomy if a cesarean delivery was performed) and, if necessary, hysterectomy. (See 'Consider laparotomy' below.)

Laboratory evaluation

Routine — Routine laboratory evaluation should include [31]:

- **Complete blood count**, including platelet count – For every 500 mL of blood loss, hemoglobin levels will fall by about one gram/dL; however, the initial hemoglobin/hematocrit value does not accurately reflect the amount of blood loss acutely.
- **Type and crossmatch** for multiple units of packed red cells, if not already done.
- **Coagulation studies –** Fibrinogen concentration, prothrombin time, activated partial thromboplastin time. The coagulation panel should be repeated every 30 to 60 minutes to observe trends until PPH is controlled. Coagulation studies are usually normal in the early stages of hemorrhage, but may be abnormal when comorbidities are present, such as abruptio placentae, liver disease, intrauterine fetal demise, sepsis, or amniotic fluid embolism. Eventually, significant hemorrhage without replacement of coagulation factors will result in coagulation abnormalities.

  Fibrinogen falls to critically low levels earlier than other coagulation factors during PPH, thus the fibrinogen level is a more sensitive indicator of ongoing major blood loss than the prothrombin time, activated partial thromboplastin time, or platelet count [32,33]. The fall is likely related to loss of fibrinogen through bleeding, increased fibrinolytic activity, and hemodilution from fluids given to support blood pressure, and the contribution of each of these factors may be affected by the cause of PPH [34].

  The fibrinogen level at the time of diagnosis of PPH has been called the ‘canary in the coal mine’ for coagulopathy because fibrinogen depletion is an early predictor of hemorrhage severity and can be used to guide the aggressiveness of management [35-38]. The normal fibrinogen level in a term pregnancy is 350 to 650 mg/dL, which is nearly double that of nonpregnant adults (200 to 400 mg/dL) [39]. In multiple studies of women with PPH, a low fibrinogen level (less than 200 mg/dL) was 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 [35-37,40]. The positive predictive value for progression to severe PPH has been reported to be 100 percent at this level, with a 79 percent negative predictive value for progression at fibrinogen values >400 mg/dL [35].

  In one study, compared with patients with fibrinogen >300 mg/dL, the odds of severe PPH (hemoglobin decrease ≥4 g/dL, red cell transfusion, arterial embolization or emergency surgery, admission to intensive care, or death) for patients with fibrinogen between 200 and 300 mg/dL were almost doubled (odds ratio [OR] 1.90, 95% CI 1.16-3.09) and increased 12-fold for fibrinogen less than 200 mg/dL (OR 11.99, 95% CI 2.56-56.06) [36].

Thromboelastography and rotational thromboelastometry — Thromboelastography (TEG) and rotational thromboelastometry (ROTEM), where available, can be useful for guiding plasma and coagulation product therapy. These tests provide a global assessment of whole blood hemostasis (clot development, stabilization and dissolution) and can be performed at the bedside, so results are available within minutes [41]. In nonpregnant patients, TEG and ROTEM results can be useful for choosing specific blood components for transfusion and assessing the efficacy of interventions [42], and increasing information on use of the tests in pregnancy is becoming available [43-48]. In pregnancy, mean clot firmness and alpha angle (TEG) are larger, and clot time (ROTEM) and reaction time (TEG) are shorter [49]. (See "Platelet function testing", section on 'Thromboelastography (TEG)'.)

ROTEM provides evidence of slowly developing hypofibrinogenemia in patients with slow, but steady, bleeding. The author has found that use of ROTEM in his hospital has led to fibrinogen replacement well before replacement would have occurred based on standard coagulation testing, and believes this early and aggressive fibrinogen replacement has prevented severe coagulopathy and has been a key factor in reducing maternal morbidity and mortality.

Using the ROTEM machine, fibrin-based clot strength can be measured in blood after platelet inhibition; this measurement has been termed Fibtem. The amplitude of the Fibtem after five minutes (Fibtem A5) is related to the maximum clot firmness (MCF), but is available sooner than MCF, and has been tested as an alternate biomarker for this reason. Fibtem A5 <10 mm has been suggested as a way to detect the progression of bleeding from mild hemorrhage to severe PPH and has been associated with more prolonged bleeding and longer stay in a high-dependency unit [37,50].

A randomized trial among women with 1 to 1.5 L PPH found that fibrinogen replacement was not beneficial if the Fibtem A5 was >12 mm or Clauss fibrinogen was >200 mg/dL, but the authors could not exclude a benefit below these levels [51]. Blood losses of greater than 2.0 L were not studied, so the performance of this biomarker in severe PPH cannot be inferred from this study. In another study by the same group, if Fibtem A5 remained >15 mm or bleeding stopped, FFP could be withheld without resulting in clinically significant hemostatic impairment [52]. These studies show that use of point-of care-testing can help direct early fibrinogen and FFP replacement to women who can benefit from these blood products while reducing the number of unnecessary transfusions.

Perform uterine tamponade in patients with atony or lower segment bleeding — Uterine tamponade is effective in many patients with atony or lower segment bleeding. Either an intrauterine balloon catheter or an intrauterine pack can be used for tamponade, but a balloon device designed for uterine tamponade is preferable because it can be placed quickly, allows some assessment of ongoing hemorrhage, and is probably more effective [53,54].

Regardless of the method employed, continued blood loss, hemoglobin, and urine output should be closely monitored. This is especially important when a gauze pack is used because a large amount of blood can collect behind the pack and conceal ongoing blood loss. If successful, the balloon or pack is removed after 24 hours.

Blood product replacement should be aggressively pursued as well to stabilize the patient as much as possible in case emergency surgery is needed. Continued excessive bleeding indicates that tamponade is not effective and surgery or embolization should be performed. (See 'Consider uterine or hypogastric artery embolization' below and 'Consider laparotomy' below.)

Intrauterine balloon catheter — The early use of balloon tamponade is a way of limiting ongoing blood loss while attempting to reverse uterine atony [55]. It is important to remember that even a slow trickle of blood can add up over time to significant hemorrhage. Similarly, repeated uterine expression of clots and unclotted blood between periods of normal lochia can result in significant blood loss over time.

Commercially available uterine balloon tamponade devices and devices improvised for balloon tamponade (#24 Foley catheter with a 30 mL balloon, Sengstaken-Blakemore tube) have been used successfully to tamponade the uterine cavity. For each device, the balloon is filled until bleeding is controlled or the manufacturer's suggested fill volume is reached. The exact mechanism of action of these devices is unclear, but is likely related to a reduction in uterine artery perfusion pressure [56]. Whether this is the result of direct compression of the uterine artery in the lower segment or due to wall conformational changes has not been determined [56]. A description of these devices, their placement, and data on efficacy are described in detail separately. (See "Intrauterine balloon tamponade for control of postpartum hemorrhage".)

Intrauterine pack — Uterine packs have also been used to control PPH with variable success; proper technique requires firmly packing the entire uterine cavity with gauze, such as Kerlix, to achieve tamponade [57-59]. The gauze can be impregnated with 5000 units of thrombin in 5 mL sterile saline to enhance clotting. A small series including 19 cases of PPH at vaginal or cesarean delivery also reported success using chitosan-covered gauze (CELOX Gauze, each gauze is three meters in length) [60].

A regimen of intravenous broad-spectrum antibiotics, such as gentamicin, 1.5 mg/kg every eight hours, and either metronidazole, 500 mg every eight hours, or clindamycin, 300 mg every six hours, are administered while the pack is in place (typically 24 hours). If packing does not control hemorrhage, repacking is not advised [61].

Other — A single-use, vacuum-induced tamponade device has been developed and is undergoing testing [62]. A distal loop with pores is positioned inside the uterine cavity and a proximal occlusion balloon is positioned at the external cervical os to seal the outlet. When the uterine cavity is subjected to symmetrically distributed low level vacuum, the differential pressure between the inside and outside of the uterus collapses the space, creating self-tamponade. Residual blood drains out a vaginal tube connected to the distal loop and the uterus regains normal tone.

Transfuse red blood cells, platelets, plasma

Initial approach — Replacement of blood components is more important than crystalloid infusion if massive hemorrhage has occurred or is likely [63]. In a postsurgical patient who repeatedly drops her blood pressure and/or urine output despite reasonable volume replacement, the clinician should assume ongoing hemorrhage. In such patients volume replacement should be with blood products and fibrinogen as necessary, rather than crystalloid, which may result in a dilutional coagulopathy and worsen bleeding.

There are no universally accepted guidelines for replacement of blood components in patients with PPH [64,65]. Recommendations are usually based upon expert opinion since there is no strong evidence from randomized trials, and these opinions are often extrapolated from data from studies in trauma patients.

Before laboratory studies are available, we suggest transfusing 2 units of packed red blood cells (pRBCs) if hemodynamics do not improve after the administration of 2 to 3 liters of normal saline, estimated blood loss is under 1500 mL, and continued bleeding is likely. In addition, aggressive use of plasma replacement is important to reverse dilutional coagulopathy [66]. One guideline suggests 4 units pRBCs followed by 4 units fresh frozen plasma (FFP) if no laboratory results are available, bleeding is ongoing, and bleeding is due to atony; the 1:1 pRBC:FFP ratio is maintained until tests of hemostasis are available [49]. FFP is begun

sooner in patients with abruption, amniotic fluid embolism, or prolonged hemorrhage as impaired hemostasis is more likely in these settings. (See 'Transfusion targets' below.)

Whenever transfusing large volumes of blood products, particularly stored pRBCs, it is imperative to consider the electrolyte effects of such massive transfusion, and to take steps to prevent or ameliorate changes in calcium (hypocalcemia) and potassium (hyperkalemia) levels. The blood bank should have compatible blood available for massive transfusion in obstetric emergencies, and eliminate barriers to rapid access of O-negative and O-positive uncrossmatched blood when needed [67,68]. (See "Massive blood transfusion".)

Institutions should adopt an obstetric hemorrhage massive transfusion protocol for obstetric patients with massive hemorrhage and continued bleeding; several such protocols exist, including:

- Texas Children's Hospital Pavilion for Women (algorithm 1).
- Stanford University Medical Center: An initial package consisting of 6 units RBCs, 4 units FFP, and 1 apheresis platelet unit [67].
- Brigham and Women's Hospital: immediate availability of 2 units RBCs and 2 units of FFP followed by 4 units each of RBCs and FFP and thawing of one pool (6 bags) cryoprecipitate.
- California Maternal Quality Care Collaborative OB Hemorrhage Protocol: For patients with unstable vital signs, suspicion of disseminated intravascular coagulation, or blood loss >1500 mL, transfuse pRBCs, FFP, and platelets in a ratio of 6:4:1 or 4:4:1. If coagulopathy persists after 8 to 10 units pRBCs and coagulation factor replacement, recombinant activated factor VIIa is a reasonable option. (See 'Recombinant factor VIIa' below.)

Monitoring — Blood loss should be estimated every 15 to 30 minutes and laboratory studies drawn every 30 to 60 minutes to guide blood product replacement. In the massively transfused patient, assumptions about possible dilutional coagulopathy secondary to crystalloid infusion or RBC transfusion should be confirmed by measurement of the prothrombin time, activated partial thromboplastin time, and platelet count or results of thromboelastography after the administration of every five to seven units of red cells. Subsequent to the initial set of components transfused, further replacement therapy should be based on these parameters rather than on any formula. (See "Massive blood transfusion".)

In any massive transfusion situation where multiple units of blood are rapidly transfused, electrolytes should be monitored, with prompt treatment of abnormalities. The most common electrolyte abnormalities are hyperkalemia and low ionized calcium levels. Both electrolyte disturbances, if severe, can lead to cardiac arrest or significantly depressed cardiac function that precludes optimal resuscitation.

- **Ionized calcium** – Ionized calcium should be measured at baseline and then every 15 to 30 minutes during a massive transfusion, and then hourly for the next few hours after transfusions have been stopped because of potential rebound hypercalcemia and hypokalemia.

  An ionized calcium level <1 mmol/L (normal 1.1 to 1.3 mmol/L) impairs coagulation and places the patient at risk of cardiac arrest. Emergency replacement may be accomplished by infusing 1 gram of calcium chloride over two to five minutes via a central line. Alternatively, 1 to 2 grams of calcium gluconate can be infused intravenously over two to three minutes empirically for every four units of pRBCs transfused [69]. Hypocalcaemia has a linear, concentration-dependent relationship more important in predicting hospital mortality than the lowest fibrinogen concentration, the development of acidosis, or the lowest platelet count [70].

- **Potassium** – Hyperkalemia may result from the rapid transfusion of multiple units of pRBCs, especially if they are older units. The potassium (K+) concentration in the supernatant increases from 2 to approximately 45 mEq/L as a unit of blood ages from 2 to 42 days. In an older unit of pRBCs (300 mL), there may be as much as 5 mEq of K+. When a massive transfusion protocol is instituted and large numbers of pRBCs are given at a high rate of infusion (eg, >500 mL/minute using a rapid transfusion device), dangerously high (>6 mEq/dL) K+ levels may result.

  To some extent, hyperkalemia may be prevented by using washed units of blood, and an in-line K+ filter; however, in a massive transfusion situation, this is usually impractical. Patients undergoing massive transfusion should have electrolyte levels evaluated serially to detect hyperkalemia. When urgent reduction of K+ is needed, one commonly used regimen for administering insulin and glucose is 10 to 20 units of regular insulin in 500 mL of 10 percent dextrose, given intravenously over 60 minutes. (See "Treatment and prevention of hyperkalemia in adults", section on 'Insulin with glucose'.)

Transfusion targets — We continue to transfuse RBCs, platelets, cryoprecipitate, and FFP in women with ongoing bleeding to achieve the following targets:

- Hemoglobin greater than 7.5 g/dL
- Platelet count greater than 50,000/mm^3

- Fibrinogen greater than 300 mg/dL
- Prothrombin time less than 1.5 times the control value
- Activated partial thromboplastin time less than 1.5 times the control value

As an example, 4 units of FFP are given if the prothrombin time is more than 1.5 times the control value, one apheresis platelet pack is given if the platelet count is less than 50,000/mm^3, and 10 bags of cryoprecipitate (usually supplied in one or two pools) are given if the fibrinogen is less than 100 mg/dL (table 3).

Most providers continue to transfuse patients with hemoglobin values less than 7.5 to 8 g/dL [71]. A hemoglobin level of at least 8.0 g/dL after transfusion has been recommended since values below this level can be associated with impaired hemostasis from lower platelet adhesion and high blood velocity [72], as well as myocardial ischemia [4]. Transfusion is rarely indicated when the hemoglobin is greater than 10 g/dL [73]. In other critical care settings, a restrictive transfusion policy (ie, hemoglobin threshold for initiating transfusion 7.0 g/dL) has been advocated and widely adopted, based on data from randomized trials [74].

It is important to stress that critically low fibrinogen levels cannot be returned to normal using only FFP without the use of cryoprecipitate, and in some cases of established coagulopathy, without fibrinogen concentrate (see 'Fibrinogen concentrate' below). These substances should be given together in patients undergoing massive transfusion who have severe coagulopathy.

Once hemostasis and hemodynamic stability are achieved, it is important to stop aggressive transfusion of blood components (ie, plasma, platelets, cryoprecipitate). When bleeding is controlled and the patient is stable, the infusion of further blood products is likely to only add risk (eg, fluid overload and transfusion complications) without clear benefit.

The following UpToDate topic reviews discuss blood transfusion therapy in detail:

- (See "Indications and hemoglobin thresholds for red blood cell transfusion in the adult".)
- (See "Initial evaluation of shock in the adult trauma patient and management of NON-hemorrhagic shock".)
- (See "Red blood cell transfusion in adults: Storage, specialized modifications, and infusion parameters".)
- (See "Massive blood transfusion".)

Issues relating to patients who are unwilling to accept transfusions (eg, Jehovah's Witnesses) are addressed in a separate topic review. (See "The approach to the patient who refuses blood transfusion".)

Blood salvage and infusion has been used for management of massive hemorrhage at cesarean delivery (see "Surgical blood conservation: Blood salvage"). Its use is under investigation as an option for management of massive hemorrhage at vaginal delivery [75-79].

Transfusion ratios — There is no consensus on the optimal ratio of blood product replacement; recommendations for RBC:FFP ratios vary widely (eg, RBC:FFP: 1:1, 2:1, 3:2, 6:4) [64,67,80]. A pragmatic approach is 1 unit FFP for every 2 to 3 units of RBCs [81,82] or 4 units FFP for every 6 units of RBCs [83]. Clinical experience in Iraq and Afghanistan, as well as domestic and foreign trauma centers and an obstetrical unit at a military hospital, suggest administration of 1 unit of FFP for every 1 to 2 units of RBCs until the clinical situation is stable or absence of coagulopathy is confirmed by laboratory studies (discussed below) [63,84-86]. When a massive transfusion is needed, the recommended initial transfusion ratio for RBCs:FFP:platelets is typically 1:1:1 to mimic replacement of whole blood [87].

> In a multisite randomized trial in patients with severe trauma and major bleeding (> 2 liters), early administration of plasma, platelets, and RBCs in a 1:1:1 ratio compared with a 1:1:2 ratio resulted in a significant increase in the proportion of patients who achieved hemostasis within 24 hours (86 versus 78 percent) and a significant reduction in the proportion of patients who exsanguinated (9.2 versus 14.6 percent), without a significant increase in complications (eg, acute respiratory distress syndrome, multiple organ failure, venous thromboembolism, sepsis, transfusion-related complications) [88]. There was no significant difference in mortality at 24 hours or at 30 days. However, it must be borne in mind that data from male and female patients with severe trauma cannot necessarily be directly extrapolated to pregnant women, and that postpartum hemorrhage has unique aspects that may require a different approach. For example, massive tissue trauma and its attendant issues are rarely present in PPH, and acute pre-existing thrombocytopenia (as seen in severe preeclampsia) is rarely present in trauma victims. Data addressing specific massive transfusion protocols and outcomes as they relate to severe hemorrhage in pregnant and postpartum women are needed to guide therapy in this population.

Correct clotting factory deficiencies — Although FFP contains a small amount of fibrinogen, cryoprecipitate and fibrinogen concentrate are preferable for treatment of hypofibrinogenemia because they have a higher fibrinogen concentration per infused volume. Thromboelastography (TEG) and rotational thromboelastometry (ROTEM), where available, can be useful for guiding plasma and coagulation product therapy. (See 'Thromboelastography and rotational thromboelastometry' above.)

Standard approach

Cryoprecipitate — Cryoprecipitate is primarily used for correcting fibrinogen deficiency, but also contains other clotting factors. The dose depends on the measured and target fibrinogen levels; dosing is described in the table (table 3). If no laboratory results are available and 8 units of pRBCs and 8 units of FFP have been transfused, one guideline advises infusion of two pools of cryoprecipitate [49].

Advantages of cryoprecipitate are that large amounts of fibrinogen can be administered in a low-volume product and it is less costly than the commercial products described below. Disadvantages are that it takes time to thaw and prepare for transfusion, and it carries a risk of transmissible infections since it is a pooled blood product that has not undergone any pathogen inactivation procedures.

The following specific clotting factor therapies can be useful instead of or in addition to cryoprecipitate in cases of intractable hemorrhage and coagulopathy. Further research is required before any of these products is routinely instituted.

Additional options

Fibrinogen concentrate — Fibrinogen concentrate (RiaSTAP, Fibryga [formerly Fibryna]), a heat-treated, lyophilized fibrinogen (Factor I) powder made from pooled human plasma, may be available in some institutions. Each vial contains about 1000 mg fibrinogen. It is usually administered alone but can be used in combination with cryoprecipitate.

Fibrinogen concentrate may be used when fibrinogen levels are critically low (ie, <100 mg/dL), and FFP and cryoprecipitate are not available. It can be administered sooner than cryoprecipitate since thawing is not required and it is effective, but there are few data that it improves outcome compared with cryoprecipitate [89].

When rapid evaluation and treatment are essential, the combination of intraoperative dynamic monitoring of clotting abnormalities using thromboelastography (TEG) or rotational thromboelastometry (ROTEM) (where available) and administration of fibrinogen concentrate may be the optimum approach. As an example, one study reported ROTEM-guided fibrinogen concentrate administration in major obstetric hemorrhage reduced requirements for blood component therapy (trigger for administration: FIBTEM A5 <7 mm or fibrinogen ≤150 mg/dL) [50]. (See 'Thromboelastography and rotational thromboelastometry' above and "Disorders of fibrinogen", section on 'Fibrinogen concentrate: Dosing and monitoring'.)

Recombinant factor VIIa — Recombinant human activated factor VII (rFVIIa) is used for treatment of individuals with bleeding related to hemophilia A and B inhibitors, acquired inhibitors, and congenital factor VII deficiency. (See "Recombinant factor VIIa: Clinical uses, dosing, and adverse effects".)

It has also been used successfully off-label for control of bleeding in other situations, such as intractable bleeding associated with postpartum uterine atony, placenta accreta, or uterine rupture [90-92]. In a trial that randomly assigned women with severe postpartum hemorrhage unresponsive to oxytocin and sulprostone to treatment with rFVIIa (60 mcg/kg) or standard care, use of rFVIIa resulted in a 41 percent reduction in the primary outcome measure (arterial embolization, arterial ligation, or hysterectomy) (22/42 [52 percent] versus 39/42 [93 percent]; RR 0.56, 95% CI 0.42-0.76), independent of the delivery method [92]. The proportion of patients requiring transfusion was lower in the intervention group, although the absolute number of blood products administered was similar for both groups. Eight of the 42 patients in the standard care group received late rFVIIa as a compassionate treatment in an attempt to avoid hysterectomy and peripartum hysterectomy was avoided in two cases. One patient developed postpartum ovarian vein thrombosis and one developed deep vein thrombosis and pulmonary embolus; both had received thromboprophylaxis and rFVIIa after a cesarean delivery. Although this therapy appears promising for patients with hemorrhage refractory to standard therapy, the drug is very expensive, failed in 50 percent of patients, and may have increased the risk of thrombotic events, as reported by others [93]; thus, we suggest reserving its use for women with postpartum hemorrhage and coagulopathy unresponsive to standard therapies.

The optimal dose is unclear. Doses of 16.7 to 120 mcg/kg as a single bolus injection over a few minutes every two hours until hemostasis is achieved have been effective, and usually control bleeding within 10 to 40 minutes of the first dose [90,94]. It is preferable to start with a low dose (40 or 60 mcg/kg) to reduce the risk of thromboembolic events; doses of 40 mcg/kg [95] to 90 mcg/kg [96] have been suggested for obstetric hemorrhage. The dose may be repeated once in 15 to 30 minutes if there is no response. Additional doses are unlikely to be effective.

The efficacy of rFVIIa depends on the levels of other coagulation factors present. For maximal effectiveness, major sources of bleeding should be controlled and blood products should be administered to correct major deficiencies before administering rFVIIa [97]. In addition, patient temperature, pH, and calcium levels should be adequate. At a minimum, we attempt to achieve:

- Platelet count >50,000/mm^3
- Fibrinogen level >50 to 100 mg/dL
- pH ≥7.2
- Absence of hypothermia
- Absence of hypocalcemia

Prothrombin complex concentrate — Three-factor (II, IX, X) and four-factor (II, VII, IX, X) prothrombin complex concentrates (PCC) are available and have been suggested as an alternative to FFP. The perceived advantages are a reduced risk of volume overload, no need for thawing or blood group typing, and a reduced risk for transfusion-related acute lung injury and allergic reactions. Disadvantages include very high cost and increased risk of thrombosis.

The FDA-approved indication for four-factor PCCs is for urgent reversal of acquired coagulation factor deficiency induced by vitamin K antagonist therapy in adult patients with acute major bleeding or need for an urgent surgery/invasive procedure.

We caution those using PCC off-label in women with postpartum hemorrhage to have evidence (or strong suspicion) of a specific factor deficiency that would be alleviated by PCC because of the risk of thrombosis, the lack of data of efficacy in this population, and the concern that deficiencies in factors II, VII, IX, and X are not common in this setting [49]. The most likely scenario where PCC might be of benefit is in a massive transfusion situation with ongoing disseminated intravascular coagulation (DIC) unresponsive to all of the usual therapies. (See "Plasma derivatives and recombinant DNA-produced coagulation factors".)

Consider uterine or hypogastric artery embolization

Candidates — Where personnel and facilities are readily available, uterine or hypogastric artery embolization by an interventional vascular specialist is an option for appropriate candidates: Women with persistent slow but excessive bleeding, who are hemodynamically and hemostatically stable, and who have failed less invasive therapies.

Consultation with an interventional vascular specialist should be obtained early in the patient's course. This facilitates decision making about the possible need for, and timing of, a procedure. Decision-making and mobilization of personnel and appropriate equipment take time and, in some cases, a significant delay is likely before the uterine vessels can be occluded. Embolization procedures can take one to three hours to complete and fail to control bleeding in 10 percent of cases; furthermore, personnel in a typical interventional suite may not be able to monitor PPH during the procedure. Thus, laparotomy should be performed if the woman is not stable enough to wait for the embolization procedure. However, performing the embolization in an operating room with a full surgical team in attendance is a reasonable option for hemodynamically unstable patients if the facility has a \ an operating room that allows simultaneous surgery and embolization (eg, hybrid operating room, or an appropriately sensitive portable C-arm and carbon fiber table).

If coagulopathy is present, it should be corrected before the procedure, if possible, although some interventional vascular specialists will proceed while a coagulopathy is being treated since the hemorrhage is generally the cause of the coagulopathy. Others consider coagulopathy a relative contraindication to elective interventional procedures; however, under emergency situations, it can be performed as a lifesaving measure even with coagulopathy. In two series, disseminated intravascular coagulation was a risk factor for failure of embolization to control hemorrhage [98,99].

Procedure — The technique of uterine or hypogastric artery embolization is basically the same as with other embolization procedures (see "Interventional radiology in management of gynecological disorders"). Diagnostic angiography is initially performed to identify a bleeding site or abnormal vascular findings, such as extravasation, abnormal arteriovenous communication, pseudoaneurysm, spasm, or truncation (image 1A-B).

Gelfoam, an absorbable gelatin sponge, is the preferred agent for embolization of the uterine or hypogastric arteries since the duration of occlusion is temporary (two to six weeks), but sufficient to reduce hemorrhage. Slow development of collateral arterial flow occurs a few hours after embolization and serves to prevent ischemia [100,101]. DIC is a risk factor for failure of embolization to control hemorrhage

N-butyl cyanoacrylate (NBCA) is a liquid glue that instantly solidifies (polymerizes) when in contact with blood. It has the advantage that it does not depend on maternal clotting factors to plug the bleeding site. In a retrospective cohort study, uterine artery embolization with NBCA was highly effective for hemostasis in patients with PPH and as effective in patients with and without DIC [102]. Disadvantages of NBCA include higher costs compared with Gelfoam, the possibility of systemic embolization, minimal information about subsequent pregnancies, and it is permanent [103,104].

The patient's clinician should monitor her status in the angiography suite at the time of the procedure and be ready to proceed to surgical intervention if the patient becomes hemodynamically unstable. Frequent communication about the patient's status between the interventional vascular specialist and the clinician is important. A prolonged embolization procedure should be avoided if there appears to be little chance of therapeutic success because the patient's condition may deteriorate and increase the risk when surgical intervention is performed.

If the uterine or hypogastric artery procedure is unsuccessful and time permits, angiographic occlusion balloon catheters can be placed to temporarily occlude the hypogastric or common iliac arteries (or even in the aorta) while en route to the operating room or during the surgery for control of hemorrhage (see "Postpartum hemorrhage: Management approaches requiring laparotomy", section on 'Role of embolization'). Prolonged (48 hours) balloon catheter occlusion of the hypogastric arteries alone, without embolization, was reported to successfully control hemorrhage in two hemodynamically unstable patients [105].

Outcome — A literature review of uterine artery embolization for treatment of pelvic hemorrhage unrelated to malignancy reported a 97 percent success rate [106]. Another review described a 95 percent success rate in controlling bleeding related to

PPH; the failure rate was low (5 percent of cases required hysterectomy) [101]. A series of 100 cases performed at a single center mostly by one radiologist reported 89 percent success for control of PPH [107]. Studies of selective arterial embolization by an interventional vascular specialist used a variety of embolization materials in a variety of arteries (but usually the uterine artery), and employed a variety of interventions prior to and concomitantly with embolization, which explains the spectrum of reported success rates [108]. Data are also limited by the small number of published studies and the small number of participants.

Serious complications are unusual, and the procedure-related morbidity of 3 to 6 percent is much less than with laparotomy [106,107,109,110]. Postembolization fever is the most common complication; other less common complications include buttock ischemia, vascular perforation, uterine ischemia and necrosis, leg ischemia, and infection. Ovulation and menses generally resume as long as the uterus and ovaries are intact. However, the author is aware of one patient who died after embolization resulted in peripheral pulmonary vascular occlusion (personal communication).

Menstrual function and fertility generally return to baseline after arterial embolization for PPH [111], and subsequent pregnancies experience no or minimal increase in adverse outcome [112-122]. A case report of uterine artery embolization for treatment of a cervical ectopic pregnancy described regionally decreased blood supply in the mid-posterior wall of the uterine fundus on magnetic resonance imaging on days 5 and 25 postprocedure; this patient had a spontaneous uterine rupture at the mid-posterior wall of the uterus at 32 weeks during a subsequent pregnancy four years later [123]. Some authors have reported placenta accreta in 12 to 39 percent of subsequent pregnancies, but there was a small number of subsequent pregnancies and events in these series [124].

This generally favorable experience appears to contradict the reports describing increased pregnancy loss after uterine artery embolization for treatment of leiomyomas. Possible reasons for this discordance include the typically younger age of pregnant patients, the vastly increased vascularization of the gravid uterus (possibly permitting formation of more adequate alternative blood supply), and the absence of leiomyomas in the gravid patients. It is also possible that arterial embolization of the gravid uterus is associated with an increased incidence of subsequent pregnancy loss above baseline, but there is a lack of literature supporting this.

Consider resuscitative endovascular balloon occlusion of the aorta — An ultrasound directed, aortic balloon placement technique has been used in trauma (military and civilian) and emergency room scenarios to decrease the amount of bleeding distal to the occluded site and provide a window of opportunity for resuscitation and definitive hemorrhage control [125-127]. Minimal data on the use of this technique in obstetrics are available, but suggest that in desperate situations, particularly in low-resource environments where interventional vascular specialists and blood banking are unavailable, resuscitative endovascular balloon occlusion of the aorta (REBOA) by appropriately trained personnel may offer a minimally invasive approach to resuscitation [128-131].

The aortic balloon has been designed to occlude the aorta in two of three aortic zones: zone 1 is the descending aorta at the level of the diaphragm, which is used in massive abdominal trauma, and zone 3 is in the infra-renal aorta, which is used in lower abdominal/pelvic hemorrhage, including postpartum hemorrhage (figure 4). The balloon is placed percutaneously into the common femoral artery using ultrasound guidance in the groin, and a Seldinger technique is employed to advance a 7F sheath into the artery. The balloon catheter is then advanced via the sheath into the required zone using the measured markings on the catheter. Reported complications are minimal given the smaller size of the sheath (7F) compared with aortic balloon catheters placed under fluoroscopy (12F to 14F) [132]. (See "Endovascular methods for aortic control in trauma", section on 'REBOA technique'.)

BLOOD LOSS >1500 ML WITH ONGOING EXCESSIVE BLEEDING REFRACTORY TO MEDICAL AND MINIMALLY INVASIVE INTERVENTIONS

Basic interventions

- Do all of the above, if possible. However, these patients are at high risk of hemodynamic instability despite these therapies.

In addition, apply the following interventions. These interventions may be indicated in some patients with less blood loss as well.

Maintain oxygenation — Maintain oxygen saturation >95 percent by administering oxygen (10 to 15 L/minute) by face mask and also transfuse to improve oxygen-carrying capacity and delivery. An anesthesiologist should assess the patient's airway and breathing, and intubate if indicated. A high-flow mask with the correct flow rate is important since a low oxygen flow rate may result in CO_2 retention and worsen the situation.

Avoid hypothermia and acidosis — Fluids and blood components should be normothermic to avoid hypothermia, which has been linked to coagulopathy in trauma patients [133,134]. Warming devices (blankets, devices for warming all IV fluids, insulation water mattresses, and/or upper- and lower-body forced-air warming devices) are employed to maintain normothermia (temperature ≥35.5°C).

Hypothermia results in sympathetic stimulation with increased myocardial oxygen consumption, particularly if shivering occurs, which may lead to myocardial ischemia. Other adverse consequences of hypothermia include sepsis, coagulopathy, decreased platelet function, and increased mortality. The combination of hypothermia and acidosis increases the risk of clinically significant bleeding despite adequate blood, plasma, and platelet replacement [133], so acidosis should be corrected, using bicarbonate for pH <7.1 if necessary. (See "Bicarbonate therapy in lactic acidosis".)

Consider external aortic compression — Aortic compression is a temporizing measure to reduce blood flow to the uterus and thus provide time to initiate and continue the measures described above to stabilize the patient [135,136]. Using a closed fist, firm downward pressure is applied above and slightly to the left of the patient's umbilicus to compress the abdominal aorta against the vertebrae just above the sacral promontory. This can be readily accomplished since the postpartum abdominal wall tends to be flaccid.

Consider laparotomy — Laparotomy is indicated in patients with massive bleeding and those who are unstable after the initial interventions described above since it is unlikely that ongoing replacement of blood products will match blood loss in these patients. Ideally, the clinician should correct hemostatic defects prior to laparotomy, but surgery should not be delayed if bleeding cannot be controlled promptly.

In patients with atony who have had a vaginal birth, laparotomy is generally a last resort when less invasive interventions have failed. The need for laparotomy is rare in this setting, as the combination of uterotonic therapy, uterine tamponade, and uterine artery embolization can be used to control bleeding in virtually all cases. (See 'Consider uterine or hypogastric artery embolization' above.)

Because the abdomen is already open in patients with atony at cesarean delivery, surgical procedures for control of hemorrhage are performed sooner and are successful in 85 to 90 percent of cases [137]. In postcesarean delivery patients with ongoing bleeding, the author has found that reopening the patient and washing out any collected blood and blood breakdown products and inspecting pedicles is best done earlier rather than later. The lax abdomen of a postpartum patient will not tamponade bleeding until very late in the process, and a large volume of blood can be lost without any increase in girth. In addition, the accumulation of clotted and unclotted blood in the abdominal cavity may activate the fibrinolytic system, with increased release of tissue-type plasminogen activator and possibly fibrinolytic shutdown with increased plasminogen activator inhibitor-1. This may potentiate any coagulopathy and interfere with efforts to reverse disseminated intravascular coagulation [138,139]. (See "Postpartum hemorrhage: Management approaches requiring laparotomy".)

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

- The key to management of postpartum hemorrhage (PPH) is to recognize excessive bleeding before it becomes life-threatening, identify the cause, and initiate appropriate interventions (table 1). (See 'Introduction' above.)
- Initial basic interventions include:
  - Obtain assistance, monitor vital signs and quantify blood loss, move unstable patients to an operating room, and perform a clot observation test (see 'Basic interventions' above)
  - Establish adequate intravenous access (see 'Establish adequate intravenous access' above)
  - Resuscitate with crystalloid (see 'Resuscitate with crystalloid' above)
  - Provide adequate analgesia (see 'Provide adequate anesthesia' above)
  - Examine for lacerations, atony, uterine inversion, retained products of conception, and uterine rupture (see 'Examine the lower genital tract and uterus to determine the cause of bleeding' above)
  - Treat the cause of bleeding (see 'Begin treatment of the cause of bleeding' above)
- Atony is the most common cause of PPH. Treatment involves:
  - Perform fundal massage and manual uterine compression. (See 'Perform uterine massage and compression' above.)
  - Increase oxytocin dose. (See 'Increase oxytocin infusion' above.)
  - Administration of tranexamic acid. When PPH is diagnosed within three hours of delivery, we recommend administration of tranexamic acid (**Grade 1B**). When more than three hours have elapsed since delivery, there is no clear evidence of benefit. (See 'Administer tranexamic acid' above.)

- If hemorrhage is not controlled, add either carboprost tromethamine or methylergonovine. Misoprostol (PGE1) is useful for reducing blood loss in settings where injectable uterotonics are unavailable or contraindicated, but has bothersome side effects. (See 'Administer additional uterotonic drugs' above.)

● Patients with blood loss >1500 mL with ongoing excessive bleeding require all of the above, and consideration of the following:

- Laboratory tests to evaluate blood loss and coagulopathy (see 'Laboratory evaluation' above)
- Placement of an intrauterine balloon for tamponade, after excluding cervical and vaginal lacerations, retained placenta, uterine inversion, and uterine rupture (see 'Perform uterine tamponade in patients with atony or lower segment bleeding' above)
- Transfusion of red cells and correction of coagulopathy (see 'Transfuse red blood cells, platelets, plasma' above and 'Correct clotting factory deficiencies' above)
- Selective arterial embolization if less invasive measures fail, the patient is hemodynamically stable, and volume and blood product replacement can compensate for the rate of blood loss (see 'Consider uterine or hypogastric artery embolization' above)
- Resuscitative endovascular balloon occlusion of the aorta by appropriately trained personnel can decrease the amount of bleeding distal to the occluded site and provide a window of opportunity for resuscitation and definitive hemorrhage control. (See 'Consider resuscitative endovascular balloon occlusion of the aorta' above.)

● Patients with blood loss >1500 mL and ongoing excessive bleeding refractory to medical and minimally invasive interventions require consideration of all of the above, and should receive oxygen to maintain oxygen saturation >95 percent and receive normothermic fluids and blood to avoid hypothermia. Acidosis should be corrected using bicarbonate, if necessary. Aortic compression is a temporizing measure to reduce blood flow to the uterus and thus provide time to initiate and continue other measures. These interventions may be indicated in some patients with less blood loss as well. (See 'Blood loss >1500 mL with ongoing excessive bleeding refractory to medical and minimally invasive interventions' above.)

● Laparotomy is indicated in patients with massive bleeding and those who are unstable after the initial interventions described above since it is unlikely that ongoing replacement of blood products will match blood loss in these patients. Ideally, the clinician should correct hemostatic defects prior to laparotomy, but surgery should not be delayed if bleeding cannot be controlled promptly. (See 'Consider laparotomy' above.)

ACKNOWLEDGMENT — The author and UpToDate would like to acknowledge Dr. Allan J Jacobs, who contributed to earlier versions of this topic review.

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GRAPHICS

Potential interventions for treatment of postpartum hemorrhage

Pharmacologic interventions	
Drug	**Dosing**
Oxytocin	10 to 40 units in 500 to 1000 mL normal saline infused at a rate sufficient to control atony or 10 units IM
Tranexamic acid	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	Methylergonovine 0.2 mg IM every two to four hours or ergometrine 0.5 mg IV or IM or ergonovine 0.25 mg IM or IV every two hours
Carboprost	0.25 mg IM every 15 to 90 minutes up to eight doses or 500 mcg IM incrementally up to 3 mg or 0.5 mg intramyometrial
Misoprostol	800 to 1000 mcg rectally
Dinoprostone	20 mg vaginally or rectally every two hours
Recombinant human Factor VIIa	50 to 100 mcg/kg every two hours
Surgical interventions	
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	
Tamponade	
Intrauterine tamponade with an intrauterine balloon or alternative device (eg, bladder catheter bulb, Sengstaken-Blakemore tube)	
Uterine packing (eg, 4 inch gauge packing)	
Consultations	

General surgery
Trauma surgery
Anesthesia team
Interventional radiology
Gynecologic oncology
Urology

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

Data from: 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.

Graphic 73412 Version 8.0

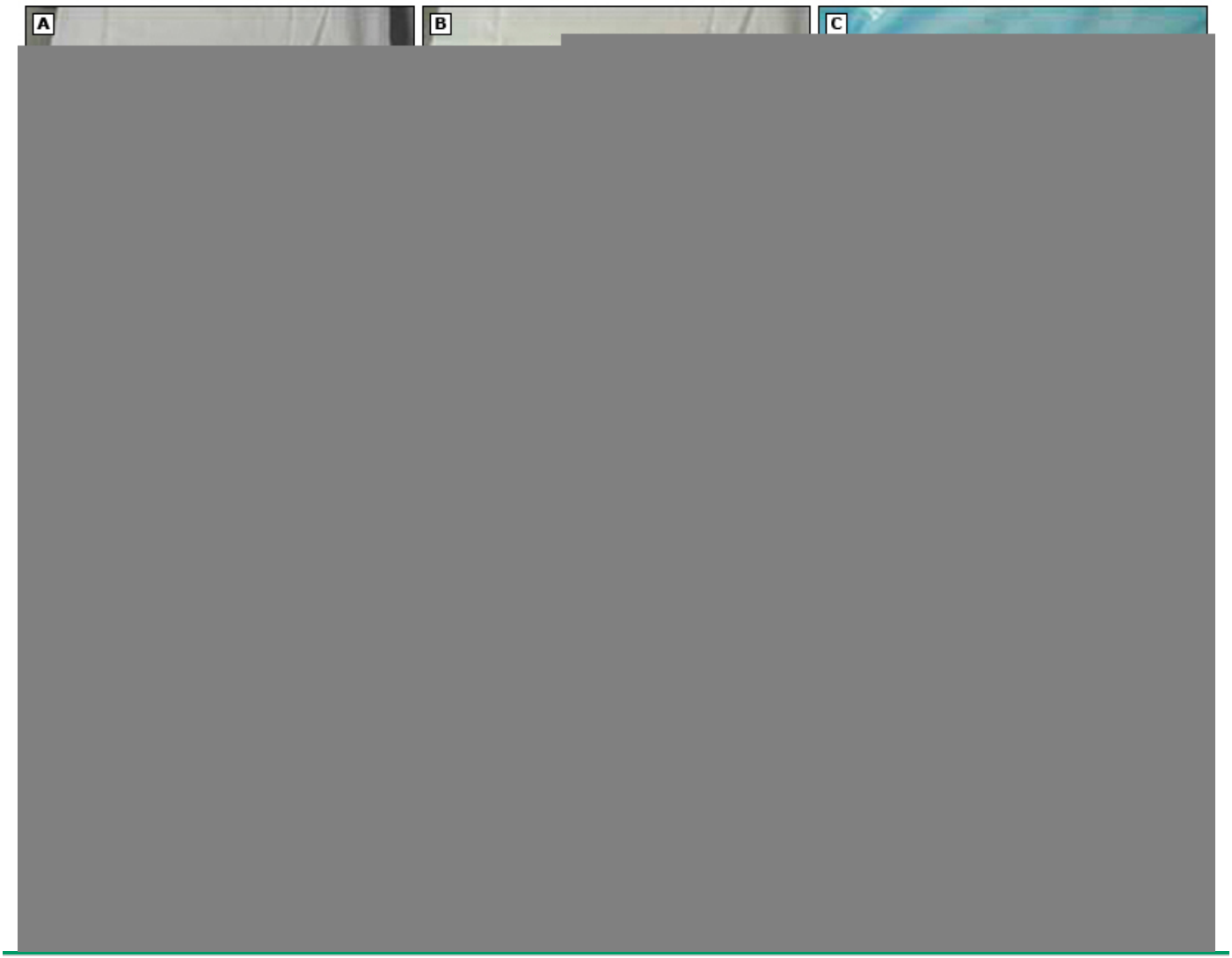
Symptoms related to blood loss with postpartum hemorrhage

Blood loss, % (mL)	Blood pressure, mmHg	Signs and symptoms
10 to 15 (500 to 1000)	Normal	Palpitations, lightheadedness, mild increase in heart rate
15 to 25 (1000 to 1500)	Slightly low	Weakness, sweating, tachycardia (100 to 120 beats/minute)
25 to 35 (1500 to 2000)	70 to 80	Restlessness, confusion, pallor, oliguria, tachycardia (120 to 140 beats/minute)
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.

Graphic 56885 Version 4.0

Visual aid for estimating intrapartum blood loss



Visual aid. Pocket card with images of measured volumes of artificial blood.

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. Reproduced with permission from Lippincott Williams & Wilkins. Copyright © 2014 American College of Obstetricians and Gynecologists. Unauthorized reproduction of this material is prohibited.

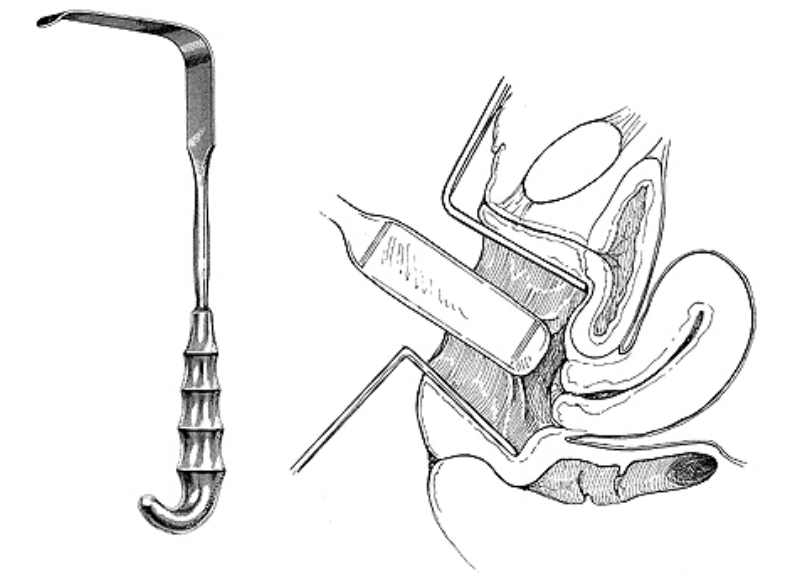
Graphic 103418 Version 1.0

Gelpi retractor

The tips are designed to limit tissue penetration.

Graphic 65767 Version 1.0

Heaney retractor



This retractor is especially designed for vaginal exposure.

Graphic 70162 Version 1.0

Breisky-Navratil vaginal retractors

Graphic 53830 Version 1.0

Sample massive transfusion algorithm

Texas Children's Pavilion for Women massive transfusion protocol.

MTP: massive transfusion protocol; PRBC: packed red blood cells; PCA: patient-controlled analgesia; RRT: rapid response team; BB: blood bank; Hg: hemoglobin; Hct: hematocrit; DIC: disseminated intravascular coagulation; PT: prothrombin time; INR: international normalized ratio; PTT: partial thromboplastin time; ABG: arterial blood gas; RBC: red blood cells; FFP: fresh frozen plasma; OB: Obstetrics; Anes: Anesthesia; OR: operating room; CRNA: certified registered nurse anesthetist; Chrg: charge; RN: registered nurse; Lab: laboratory; Tech: technician; MD: medical doctor; L&D: labor and delivery; iCa: ionized calcium; K: potassium; Glu: glucose; PCA: patient care assistant.
* Every two packages or based on lab results.

Graphic 91236 Version 4.0

Blood components: Indications and dosing in adults

Component (volume)	Contents	Indications and dose
Whole blood (1 unit = 500 mL)	RBCs, platelets, plasma	Rarely required. May be appropriate when massive bleeding requires transfusion of more than 5 to 7 units of RBCs (increasingly used in early trauma management).
RBCs in additive solution (1 unit = 350 mL)	RBCs	Anemia, bleeding. The increase in hemoglobin from 1 unit of RBCs will be approximately 1 g/dL; the increase in hematocrit will be approximately 3 percentage points.
FFP or other plasma product* (1 unit = 200 to 300 mL)	All soluble plasma proteins and clotting factors	Bleeding or expected bleeding (eg, emergency surgery) in individuals with deficiencies of multiple coagulation factors (eg, DIC, liver disease, massive transfusion, anticoagulation with warfarin or warfarin overdose if not corrected by vitamin K and/or PCC, depending on the clinical setting); therapeutic plasma exchange in TTP. FFP may be used to manage bleeding in individuals with isolated factor deficiencies (most often factor V) if a factor concentrate or recombinant factor is not available. In the rare event that FFP is used to replace a clotting factor, the dose is 10 to 20 mg/kg. This dose will raise the level of any factor, including fibrinogen, by close to 30%, which is typically sufficient for hemostasis.
Cryoprecipitate, also called "cryo" (1 unit = 10 to 20 mL)	Fibrinogen; factors VIII and XIII; VWF	Bleeding or expected bleeding with low fibrinogen: The increase in plasma fibrinogen from 1 unit of Cryoprecipitate per 10 kg body weight will be approximately 50 mg/dL. Bleeding or expected bleeding in individuals with deficiencies of factor XIII or factor VIII (hemophilia A) if a recombinant product or factor concentrate is unavailable. Bleeding or expected bleeding in individuals with VWD if DDAVP (desmopressin) is ineffective and recombinant VWF or a VWF concentrate is unavailable. Cryoprecipitate is generally provided in pools containing 5 units, and most patients receive one to two pools.
Platelets (derived from whole blood or apheresis) (1 unit of apheresis platelets or a 5 to 6 unit pool of platelets from whole blood = 200 to 300 mL)	Platelets	The platelet count increase from 5 to 6 units of whole blood-derived platelets or 1 unit of apheresis platelets will be approximately 30,000/microL in an average sized adult.

Refer to UpToDate topics on these products and on specific conditions for details of use. Frozen blood products (FFP, Cryoprecipitate) take 10 to 30 minutes to thaw. It may take the same amount of time to perform an uncomplicated crossmatch.

RBCs: red blood cells; FFP: Fresh Frozen Plasma; DIC: disseminated intravascular coagulation; PCC: prothrombin complex concentrate; TTP: thrombotic thrombocytopenic purpura; VWF: von Willebrand factor; VWD: von Willebrand disease.
* Other plasma products include Plasma Frozen Within 24 Hours After Phlebotomy (PF24) or Thawed Plasma. PF24 may be used interchangeably with FFP for all of the indications listed above, with the exceptions of factor VIII deficiency or protein C deficiency, which are treated with recombinant products or plasma-derived factor concentrates. In the rare event that specific factor concentrates are unavailable and these deficiencies must be treated with a plasma product, FFP should be used. Thawed Plasma may be used interchangeably with FFP for all of the indications listed above, with the exception of factor VIII deficiency without access to factor VIII concentrates, in which FFP should be used; or factor V deficiency, in which FFP or PF24 should be used.

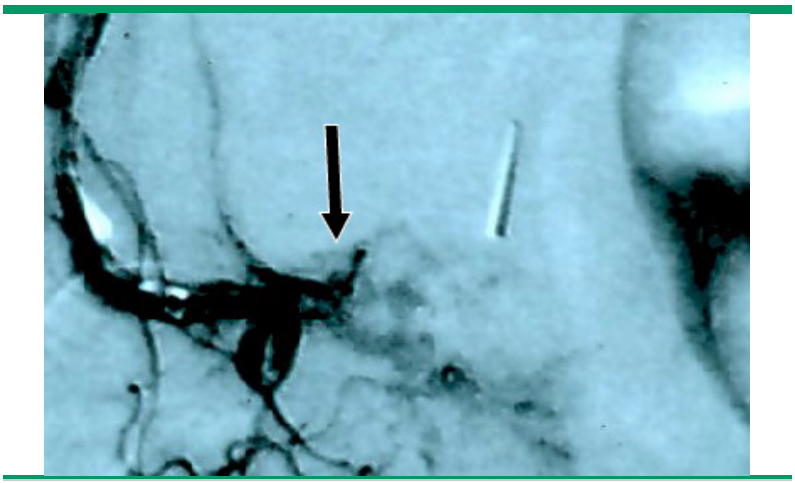
Graphic 53854 Version 18.0

Right hypogastric angiogram

Right hypogastric angiogram on a 34-year-old woman with postpartum hemorrhage shows an area of extravasation (arrow).

Graphic 67041 Version 4.0

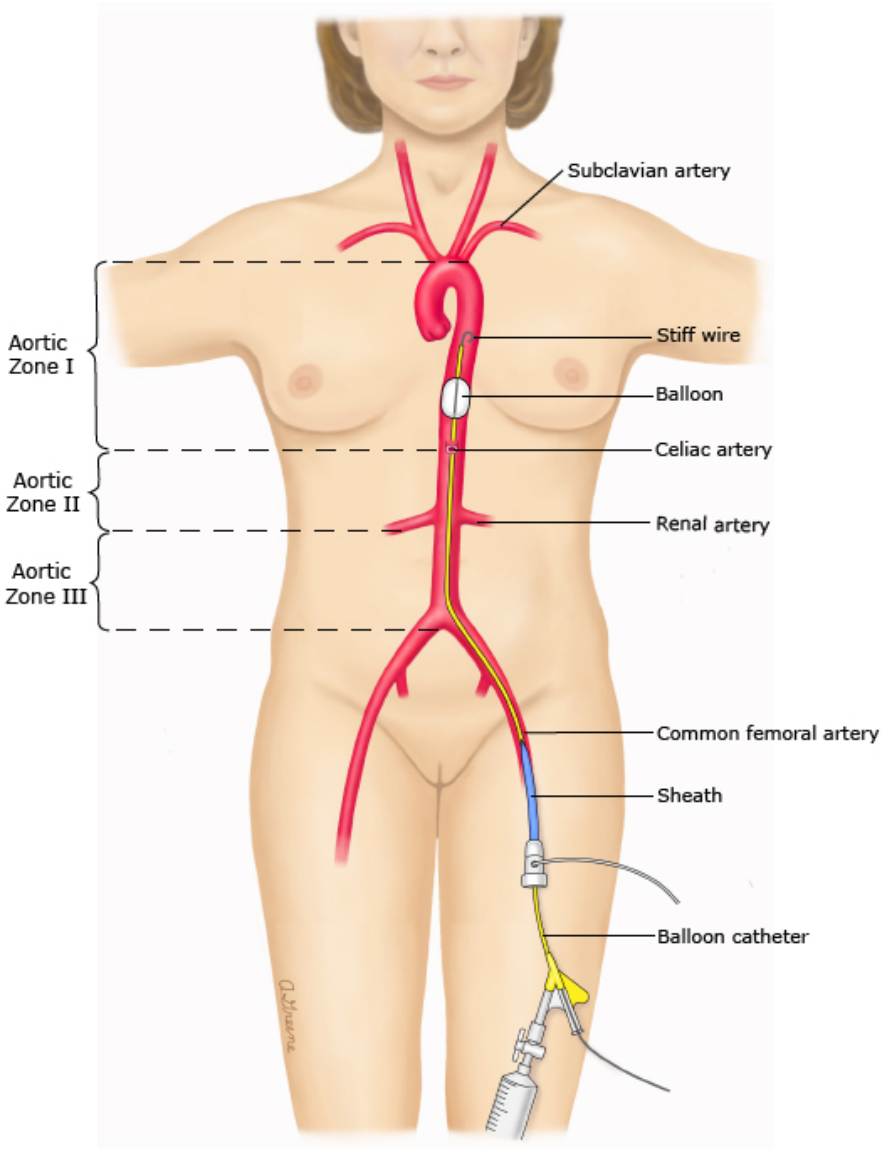
Right uterine angiogram



Right uterine angiogram reveals occlusion of the right uterine artery (arrow) after superselective uterine artery embolization. The procedure successfully stopped the bleeding.

Graphic 78839 Version 4.0

Common femoral arterial access for REBOA (female)



Graphic 117365 Version 1.0

Contributor Disclosures

Michael A Belfort, MBBCH, MD, PhD, FRCSC, FRCOG Patent Holder: Clinical Innovations [Postpartum hemorrhage (Balloon tamponade system for control of postpartum hemorrhage)]. **Charles J Lockwood, MD, MHCM** Nothing to disclose **Steven Kleinman, MD** Consultant/Advisory Boards: Cerus Corp [Pathogen reduction of blood components (Intercept Blood system)]. Equity Ownership/Stock Options: Cerus Corp. **Vanessa A Barss, MD, FACOG** Nothing to disclose

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