



## Obstetric Hemorrhage Prevention, Recognition, and Treatment

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### Keywords

- Postpartum hemorrhage • Obstetric hemorrhage • Massive transfusion
- Maternal morbidity

### Key points

- The obstetric population remains difficult to assess when critically ill.
- Vital sign changes generally considered foreboding may be normal in pregnancy, often leading to delayed diagnosis.
- Obstetric hemorrhage remains a significant source of maternal morbidity and mortality. Although many conditions convey increased risk, uterine atony remains the major cause of postpartum hemorrhage.
- Preparation for hemorrhage should be commonplace in all obstetric units and should include the components of the National Partnership for Maternal Safety hemorrhage bundle. Individual patients should undergo risk assessment starting in the antenatal period and should be optimized if possible.

## INTRODUCTION AND EPIDEMIOLOGY

The obstetric population remains difficult to assess when critically ill. Vital sign changes generally considered foreboding may be normal in pregnancy, often leading to delayed diagnosis. Furthermore, obstetric hemorrhage can be complicated by difficult identification of cause, whereas quantification of blood loss is often grossly inaccurate or impossible [1]. Resuscitation and transfusion endpoints may be less distinct and resuscitation must account for ongoing blood loss.

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Despite significant medical advances over the last century, bleeding remains the leading cause of death among parturients worldwide [2] and a major cause of mortality in the United States [3]. According to a 2014 World Health Organization analysis, the risks of maternal deaths from postpartum hemorrhage (PPH) in developed and developing regions are approximately 16% and 27%, respectively [4]. In the United States, there are approximately 650 maternal deaths per year from hemorrhage [2], accounting for about 11% of all peripartum mortality between 2006 and 2011 [3].

The definition of PPH varies worldwide; in the United States, greater than 500 mL blood loss (EBL) after vaginal delivery (VD) and greater than 1000 mL after cesarean delivery (CD) are accepted [5]. More pertinent than a definition, specialists agreed that women with greater than 1000 mL EBL with bleeding refractory to usual measures in the first 24 hours postpartum are at increased risk of hemorrhage-related morbidity [6]. Several obstetric-specific abnormalities contribute to PPH in addition to general population risk factors, such as surgical intervention or trauma [7]. Significant baseline systemic diseases can be major contributing factors in the incidence of hemorrhage. That being said, uterine atony accounts for up to 80% for PPH, and a large portion of patients with clinically significant bleeding is devoid of risk factors [5].

## **BLEEDING RISK ASSESSMENT IN PREGNANCY AND SPECIFIC CAUSES**

As noted, it may be difficult to predict if women will have significant PPH. Thankfully, most parturients can tolerate acute EBL of less than 1000 mL with little or no clinical significance [7]. As such, it may be more important to identify patients at risk for morbidity and mortality to improve resource utilization, readiness, and even survival.

Uterine rupture, placenta previa, and placental abruption can all result in antepartum hemorrhage with implications for both fetal and maternal well-being. Postpartum bleeding is generally related to uterine atony, retained or invasive placenta, genital tract trauma, and surgical complications. However, antepartum and postpartum bleeding causes often overlap. For example, uterine rupture often causes antepartum hemorrhage but will also make postpartum hemostasis more complicated. The next section discusses PPH abnormalities, risk factors, and obstetric, surgical, and anesthesiologist management considerations.

### **Uterine atony**

The gravid uterus blood supply typically comes from 2 uterine arteries and 2 ovarian arteries in addition to vast collateralization [8]. Total arterial blood flow term uterus is between 500 and 900 mL/min [9], around 12% of total cardiac output [10]. Thus, unabated uterine bleeding could cause rapid maternal exsanguination. The key steps in hemostasis are uterine contraction and vasoconstriction, which occur postpartum in response to high levels of endogenous

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