

Collaborative Strategies for Management of Obstetric Hemorrhage

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KEYWORDS

- Obstetric hemorrhage • Transfusion • Hypovolemic shock • Maternal mortality
- Postpartum hemorrhage

KEY POINTS

- Obstetric hemorrhage is a leading preventable cause of perinatal morbidity and mortality requiring a rapid, coordinated, multidisciplinary response to management to promote optimal outcomes.
- Although blood loss is anticipated at birth, early recognition of excessive blood loss and initiation of pharmacotherapy, medical, and surgical interventions offer progressive management options.
- Goals of management include recognition and management of bleeding, maintaining tissue oxygenation and perfusion, and ongoing monitoring for coagulopathies and complications.

Hemorrhage remains the primary cause of maternal mortality worldwide and the sixth leading cause of death in the United States.¹ Postpartum hemorrhage is the number 1 cause of severe morbidity during hospitalization for birth, despite state and national initiatives.² In addition, studies show that more than 90% of maternal deaths related to obstetric hemorrhage are preventable.^{1,3,4} The purpose of this article is to review relevant physiologic changes of pregnancy that may have an impact on hemorrhage management, summarize causes of obstetric hemorrhage, and describe collaborative approaches for management of hemorrhage in this unique population.

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CAUSES OF OBSTETRIC HEMORRHAGE

The exact incidence of obstetric hemorrhage is unknown, but risk is present during every pregnancy. In the United States, the incidence of obstetric hemorrhage is 2.9% of all births. The incidence of hemorrhage requiring transfusion has increased by 114%.² Although definitions vary, there are some common themes. Obstetric hemorrhage can be viewed as cumulative estimated blood loss of greater than 1000 mL for either vaginal or cesarean birth, associated with signs and symptoms of hypovolemia,⁵ but providers should escalate observation and prepare for rapid intervention if blood loss is more than 500 mL to 1000 mL without symptoms. Diagnosis can be problematic due to the typically subjective and inaccurate nature of determining blood loss. Regardless of diagnosis, the following should be considered clinical triggers for heightened observation and action⁶:

1. Heart rate greater than or equal to 110 beats per minute
2. Blood pressure less than or equal to 85/45 mm Hg (>15% drop)
3. Oxygen saturation less than 95%

Causes of obstetric hemorrhage are varied, with the potential for bleeding complications at every stage of pregnancy. Numerous factors associated with hemorrhage in pregnancy, such as body mass index, length of labor, and factors associated with overdistention of the uterus, such as multiple gestation, polyhydramnios, and fetal macrosomia influence risk. Risk assessment is not static, performed only on admission. Risk status for obstetric hemorrhage can change significantly and rapidly, and modifications in the plan of care may be warranted based on ongoing risk assessment. Causes of obstetric hemorrhage relative to timing and risk are presented in **Table 1**. Antepartum and intrapartum hemorrhage is usually associated with abnormal placentation, uterine rupture, or placental abruption. Hemorrhage during the third stage of labor, the first 2 hours of the postpartum period, is the most common time for obstetric hemorrhage to occur. Related postpartum causes can be easily remembered in this manner: tone (70%), trauma (20%), tissue (10%), and thrombin (<1%).⁷

HEMOSTATIC/HEMATOLOGIC ADAPTATIONS OF PREGNANCY

Profound, protective hemodynamic and hematologic changes occur during pregnancy to provide reserve for anticipated blood loss during childbirth. These changes can alter the expected and clinically observed signs of compromise in a hemorrhaging obstetric patient. During pregnancy, blood volume increases by 1600 mL above nonpregnant values.⁸ Plasma volume increases 10% to 15% at 6 weeks' to 12 weeks' gestation and plateaus at 40% to 45% at approximately 30 weeks' to 34 weeks' gestation. Later in pregnancy, red blood cell mass increases by 30% to support higher metabolic requirements for oxygen.⁹ Hypervolemia in pregnancy further increases with the number of fetuses.⁸ Increased blood volume is responsible for an increase in maternal cardiac output of 40% to 50% (6–10 L/min at rest in term gestation). Hormonal changes mediate a decrease in peripheral vascular resistance. Alterations in coagulation factors and the fibrinolytic cascade result in a hypercoagulable state.¹⁰ Thus the mother is prepared with compensatory responses for significant blood loss at birth. Protective physiologic adaptations also result, however, in a mother's ability to experience significant blood loss before tachycardia and hypotension develop. In pregnant women, hypotension and tachycardia are late signs that manifest after blood loss is greater than 15% of circulating blood volume.¹¹ Physiologic adaptations of the hematologic and hemostatic systems are summarized in **Table 2**.

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