



Obstetrics

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17.1 Introduction

When dealing with a patient who is pregnant, health-care providers are, for all practical purposes, taking care of two patients. A guiding principle for their care is “If momma isn’t doing well, then no one is doing well.” Care for an obstetrical patient has to take into account the physiologic changes of pregnancy that varies from the first trimester to the third trimester along with the effects of treatment on the fetus and the placenta.

During embryologic development, certain treatments and medication can affect formation of organs and tissues. Adverse events can damage the placenta leading to growth restriction of the fetus and even intrauterine fetal demise (IUFD).

Through an understanding of the bodily changes to the female parturient, the embryologic development of the fetus, and development of the placenta, management of these surgical patients can become less intimidating and help avoid perioperative complication.

Certain conditions exclusive to obstetrics can significantly alter normal pregnancy physiology and increase the risks of intraoperative complications. Surgical care of gravid women should not be withheld just because they are pregnant. This leads to the need for anesthesia/surgical providers to understand the physiologic changes of pregnancy to modify their care for this unique group.

17.2 Maternal Physiology

Changes to the female body systems begin almost immediately with conception. The early embryologic tissue produces hormones and tissues, factors that have an effect on almost every body system. Mechanical changes occur as the result of the growth of the pregnant uterus, enlarging breasts, organ hypertrophy, and tissue edema. The average gestation is 280 days, and although we discuss these effects based on trimesters, it is a continuum that peaks at 40 weeks (full-term pregnancy 37–42 weeks) and then gradually resolves usually by 6–8 weeks postpartum.

Beta human chorionic gonadotropin (BHCG) is the most common hormone measured to determine a pregnancy. It typically increases by 33–49% every 2 days and peaks around 10 weeks of gestation. Most qualitative pregnancy tests will turn

positive around 25 IU. Ovulation typically occurs around cycle day number 14 during the average menstrual cycle of 28 days. Conception usually occurs around 1–2 days later. This means pregnancy changes are occurring even before a patient realizes they are pregnant by cessation of menses. This also can lead to a false negative pregnancy test until someone is 2 or more days late for menstruation.

Normal maternal weight gain with pregnancy is between 22 and 37 pounds (10–16.8 kg) [1] with the fetus, placenta, and amniotic fluid only responsible for 35–59% of this weight. Prior recommendations for weight gain of 30 pounds in pregnancy were based on this, but because of the current obesity rates, it has now been decreased to 15–20 pounds. The rest of the weight gain in pregnancy is attributed to changes in organ systems to meet the physiologic needs associated with pregnancy.

17.2.1 Cardiac

Some of the most dramatic changes occur in the cardiovascular system to maximize oxygenation to the fetus along with increased blood flow to the uterus, which in turn is passed on to the fetus via the placenta. The heart enlarges and rotates slightly which can increase its silhouette in radiologic studies. This enlargement along with hypertrophy results in a remarkable increase in stroke volume (SV). The heart rate (HR) increases by 15–20 beats per minute during pregnancy. Preload is increased by the upsurge in blood volume. Reduced vascular resistance through vasodilatation decreases afterload. All together this works to increase cardiac output (CO) very early pregnancy by 20% and peaks at around 50% by 32–34 weeks. Remembering $SV \times HR = CO$, we can see the normal value of 4.88 increase to 7.34 L/min in the third trimester [2, 3]. Multi-gestations can result in an additional 20% increase in CO. This increase in CO can result in larger blood loss amounts in a shorter period of time with hemorrhage with masking of tachycardia until significant blood loss has occurred. CO is greatly affected by patient position and can be reduced by mechanical compression to the vena cava by the gravid uterus as early as 24 weeks. This reduces blood flow back to the heart. Placing the patient supine instead of a left lateral tilt can reduce CO by 25–30% [4].

Blood pressure (BP) in pregnancy is variable based on trimester. Initial drops in BP can be noted around 8 weeks of gestation and will gradually increase in midpregnancy and return to normal levels by term gestation. With this early decrease in BP around the time that pregnancy is diagnosed, many women with underlying hypertension may not be recognized. This can lead to diagnoses of preeclampsia when indeed the patient has chronic hypertension instead. Mean arterial pressure (MAP) on average is decreased by 5–10 mmHg and is mainly due to a decrease in systemic vascular resistance (SVR) leading to increased blood flow to the gravid uterus. During labor, there is a significant increase in CO and MAP caused by 300–500 ml increase in venous blood by a uterine contraction along with pain and anxiety increasing the heart rate. CO typically peaks immediately postpartum and will return to prepregnancy levels within 2–4 weeks after delivery [5].

Venous pressure increases in the lower extremities, as the pregnancy progresses, to as much as 25 cmH₂O. This increases the risks of edema and varicose veins and, coupled with stasis, leads to deep vein thrombosis. Additionally, decreased colloid osmotic pressure means pregnant patients are at increased risk of developing pulmonary edema with preeclampsia or fluid volume overload (increased cardiac preload). Brain natriuretic peptide (BNP) is still a reliable test for pulmonary edema/congestive heart failure in pregnancy [6].

Dyspnea is a normal feature of pregnancy beginning around 20 weeks of gestation and is usually mild in nature. It can imitate heart disease, but it usually doesn't occur at rest and isn't associated with additional symptoms such as chest pain with exertion, syncope, orthopnea, or paroxysmal nocturnal dyspnea [7]. If these additional symptoms occur, a further cardiac evaluation is warranted. Troponin is preferred over CK-MB when patients are in labor as uterine contractions can increase CK-MB [8].

Most pregnant women will exhibit a flow murmur with a S3 and systolic ejection murmur along the left sternal boarder from increased flow through both the pulmonic and aortic valves.

Cardiac rhythm is usually limited to a mild tachycardia and increased rate of benign isolated premature atrial and ventricular contractions thought to be associated with cardiac enlargement [9].

17.2.2 Respiratory

Upper airway edema and increased secretions can occur due to the increased estrogen levels during pregnancy. This can increase risks of anesthesia complications and difficulty in intubation. Coupled with increased vascularity of the nasal mucosa, epistaxis frequently occurs [10].

The thoracic region undergoes significant changes beginning in the first trimester. Considerable increase in breast size and weight can apply pressure to the chest wall. Respiratory muscle function and maximum inspiratory and expiratory pressures are unchanged. Elevation of the diaphragm from an enlarging gravid uterus will decrease the resting lung volume. This reduces total lung capacity (TLC), functional residual capacity (FRC), expiratory reserve volume (ERV), and residual volume (RV). Patients during pregnancy have an increased progesterone level which leads to an increased respiratory rate. This coupled with an increase in tidal volume (TV) results in a rise in minute ventilation (MV). With these effects, the gravid women will have an increase in PaO₂ and decrease in PaCO₂ resulting in respiratory alkalosis. Oxygen consumption is increased, and with apnea associated with intubation, desaturation can occur in as little as 3 min [2].

17.2.3 Hematologic

A rise in blood volume starts at 6 weeks of gestation and increases during pregnancy with a climax at 40–50% by 30–34 weeks of gestation. Plasma volume increases by 50%, while erythrocyte development is at a slower rate and averages 400 ml. This results in an increase in hemoglobin (Hgb) but a dilution of the hematocrit (Hct). This is in anticipation for the significant blood loss that can occur at delivery.

White blood cell (WBC) also rises mildly as the pregnancy progresses. Adding in the stress associated with labor, some patients can develop leukocytosis (20,000 or higher WBC counts) at delivery. WBC counts alone shouldn't be used to diagnose infection. Clinical correlation is necessary to make an infection determination.

Pregnancy is associated with hypercoagulation with a significant increased risk of venous thromboembolic events (VTE), but most laboratory

values remain unchanged such as prothrombin time (PT) and partial thromboplastin time (PTT). Fibrinogen levels are increased, and values in the normal adult range can be associated with active bleeding such as placental abruption. Fibrin degradation products (FDP) are decreased and can be used in the evaluation of DIC, but D-dimer is elevated and cannot reliably predict or rule out VTE events [2].

17.2.4 Urologic

Kidney enlargement is noted during pregnancy along with dilation of the calyces, pelvis, vasculature, and ureters. The right ureter is typically dilated more than the left and on imaging can appear as hydronephrosis. Frequent urination is a common issue in pregnancy. This is related to increased production along with decreased bladder capacity from the enlarging uterus.

Increase in renal blood flow occurs in pregnancy leading to a 50% increase in glomerular filtration rate (GFR) by the end of the first trimester. This also results in an increase in creatinine clearance. Blood creatine, BUN, and uric acid are decreased in pregnant patients. Significant increases in total body water (8.5 L by term) result in blood volume expansion by 1.5 L. Additional extravascular accumulation of fluid is noted in the tissue. This results in edema and a hemodilutional anemia. Additionally there is a slight decrease in serum potassium and calcium levels along with increased excretion of protein, glucose, and albumin. Plasma osmolality is decreased because of these changes mediated by the kidneys [2, 11].

17.2.5 Gastrointestinal

The average caloric increase needed for pregnancy and breastfeeding is between 200 and 300 kcal/day. Morning sickness is a common complaint early in pregnancy peaking around 8 weeks of gestation and usually gone by 14 weeks. Many patients also experience increased production of saliva (ptyalism). The tone of the gastroesophageal sphincter along with decreased motility of the stomach can increase the rates of reflux irritation of the esophagus. The data about increased risk of aspiration is mixed in the literature. Decreased motility of the intestine, increased water absorp-

tion, and compression of the intestines by the gravid uterus are noted. These factors, along with increased iron intake from prenatal vitamin therapy, can produce significant constipation.

The appendix changes position toward the right upper quadrant in pregnancy as the uterus enlarges. Significant increases in portal vein pressure increase the incidents of hemorrhoids. Increased progesterone production slows the emptying of the gallbladder. This along with increased production of cholesterol can increase the risk of gallstone and sludge formation.

Liver size is unchanged in pregnancy, but a few liver lab values are affected such as increase in alkaline phosphatase and fibrinogen. Bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), and lactate dehydrogenase (LDH) are unaffected [2].

17.2.6 Endocrine

A small increase in the size of the thyroid is noted, but hormone production for the most part remains close to non-pregnancy levels. Thyroid-stimulating hormone (TSH) levels are close in structure to HCG, so it is not unusual for the level to decrease in the first trimester as HCG levels peak around 8 weeks of gestation. Increased levels can be noted in the presence of hyperemesis gravidarum. Free T4 levels rise slightly in the first trimester and then decrease slightly during the remainder of the pregnancy remaining slightly lower than expected in non-gravid women [12].

Adrenal size increases during pregnancy leading to the increased production of aldosterone, corticosteroid-binding globulin, adrenocorticotropic hormone (ACTH), cortisol, and free cortisol as the pregnancy progresses. Cortisol levels can be 3 times higher by delivery.

The pituitary gland enlarges significantly during pregnancy with increased production of prolactin in anticipation for breast lactation after delivery. Decreased levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) are noted due to increased estrogen and progesterone levels causing a negative feedback on the pituitary gland. The increased size makes it vulnerable to hypotension that can occur during a postpartum hemorrhage. The infarction of the gland can lead to Sheehan syndrome. This syn-

drome is noted as postpartum amenorrhea and infertility. Oxytocin levels increase during pregnancy and peak during active labor leading to uterine contractions.

The pancreas undergoes significant increased production of insulin as part of the physiologic changes to increase glucose delivery to the placenta and fetus. Pregnant women typically have fasting hypoglycemia and postprandial hyperglycemia. Some patients are not able to keep up with the needed insulin production and demonstrate gestational diabetes. This is associated with increased risks of type 2 (non-insulin) later in their lives.

Increased lipid levels are noted in pregnancy as cholesterol is needed for steroid synthesis and amino acids are an energy source for the fetus. Significant increases are noted in triglycerides, cholesterol, and low-density lipids (LDL). Slight increase in high-density lipids (HDL) is also seen in the gravid patient [2].

17.2.7 Skeletal

Calcium levels are decreased in pregnancy associated with the decrease in serum albumin binding of the calcium. Additionally, increased need for calcium by the fetus and increased maternal kidney excretion are noted [13].

Significant lordosis (anterior curvature of the lumbar spine) occurs to offset the enlarging uterine weight that changes balance. This can contribute to increase rates of back pain that occur late in pregnancy along with need for lumbar support when sitting or supine [2].

17.2.8 Other Systems

Although other body systems are affected by pregnancy, they have minimal impact on the perioperative management of catastrophic complication.

Box 17.1 Physiologic Changes in Pregnancy

Key laboratory values that are different in pregnancy

- Hemodynamic variables
 - Increased cardiac output
 - Decreased systemic vascular resistance
 - Decreased blood pressure

- Increased heart rate
- Decreased pulmonary vascular resistance
- Respiratory variables
 - Decreased function residual capacity
 - Increased minute ventilation
- Laboratory variables
 - Increased PAO₂ and Pao₂
 - Decreased Paco₂
 - Decreased serum bicarbonate (Hco₃)
 - Decreased hemoglobin and hematocrit levels
 - Increased white blood cell count
 - Decreased protein S levels
 - Decreased coagulation factors XI and XIII levels
 - Increased coagulation factors I, VII, VIII, IX, and X levels
 - Increased fibrinogen levels
 - Increased D-dimer levels
 - Increased erythrocyte sedimentation rate
 - Decreased serum creatinine levels
 - Decreased blood urea nitrogen level (BUN)
 - Decreased uric acid level
 - Increased alkaline phosphatase level
 - Increased aldosterone level
 - Increased serum cortisol, free cortisol, cortisol-binding globulin, and adrenocorticotropic hormone level
 - Increased insulin level
 - Decreased fasting blood glucose level
 - Increased triglyceride level
 - Increased cholesterol, low-density lipoprotein, and high-density lipoprotein levels

Data from Gabbe et al. [41], American College of Obstetricians and Gynecologists [42]

17.3 Fetal Development and Placenta

One of the biggest concerns raised by those who deal with surgical care and anesthesia for pregnant patients is the effects on the fetus. While the care of the mother is similar to a nonpregnant woman, and provides familiarity to a typical case, the addition of a growing fetus brings with it some trepidation. This can lead to reluctance to provide surgery or anesthesia out of worry the treatment could lead to miscarriage, fetal demise, or development of a fetal anomaly. Withholding treatment during pregnancy may lead to a more

detrimental situation with decline of the mother from her condition, which subsequently affects the growth and development of the fetus. An understanding of the stages of fetal and placental development can assist with preoperative and intraoperative planning [14].

Just like perfusion pressure and oxygenation can affect other organs and tissues in the body; the placenta with subsequent flow to the umbilical cord can be affected. Besides its ability to provide for exchange of oxygen and carbon dioxide, the placenta allows for the diffusion of nutrients such as glucose, proteins, and lipids to allow for fetal growth. The placenta acts as a selective barrier to prevent certain substances from crossing into the fetal circulation and potentially lead to adverse exposures. While the details of how the placenta acts as a selective barrier are beyond the scope of this chapter, certain general principles can be discussed. Lipid-soluble compounds cross the placenta easier than water-soluble. Larger molecules with higher molecular weight have a more difficult time crossing the placenta. Binding to plasma proteins can impact the amount of a substance that can pass through the placenta.

Fetal development can be simplified into stages upon which an exposure to a drug or therapy can have a different effect. Very few therapies are known teratogens. Few drugs have clinical trials in pregnant patients that can demonstrate safety of use during pregnancy. Most drugs are either felt to have little risk to the pregnancy or consideration of their use has to show maternal benefits outweigh the risks. Consultation with web based or books discussing the effects of drugs on pregnancies listed in the box will provide information about specific risks with medications [15].

Box 17.2 Drug Teratogen Resources

- Micromedex, Inc.
 - ▶ www.micromedex.com
- Reproductive Toxicity Center (REPROTOX)
 - ▶ www.reprotox.org
- Drugs in Pregnancy and Lactation 11th edition, 2017
By Briggs GG, Freeman RK, Towers CV, Forinash AB
Wolters Kluwer

The first stage is cellular division of the embryo. This is typically less than 31 days from LMP. Exposure to a substance typically results in an all or nothing effect on the fetus. During this phase the risk of miscarriages is highest with approximately 5–20% of pregnancies miscarrying. This makes it difficult to qualify if a therapy was the cause of the pregnancy loss or unrelated.

The next stage is organogenesis. From days 31–71 from the LMP, critical organs and tissues are developing and exposures to substances can potentially cause malformations. The incidence of major malformation is 2–3% in the general population, is usually polyfactorial in nature, and cannot be tied to a single therapy.

The third stage is the growth. This is where organs and tissues grow and the fetus enlarges in size. Exposure during this stage may lead to organ damage, restriction in function, or growth restriction of the fetus [16].

17.4 Fetal Monitoring

One of the guiding principles of fetal monitoring when surgical care of a pregnant patient is necessary is the willingness to act on the information. For this reason, the degree of fetal monitoring is varied according to gestational age of the pregnancy. The need for monitoring in a previable pregnancy typically is more basic, whereas the monitoring for a term pregnancy would need to be more advanced [17].

Another aspect of fetal monitoring has to deal with type of monitoring depending on access to the area of the uterus. Abdominal surgical procedures with a large incision may make continuous external fetal monitoring nearly impossible. Consideration for intermittent ultrasound assessment of the fetal heart rate may be an alternative. Consultation with an obstetrician preoperatively can help with anesthesia and surgical planning for the case [18].

Typically fetal cardiac activity is difficult to see on ultrasound prior to 7–8 weeks and auscultation with a Doppler prior to 10 weeks. The gravid uterus doesn't rise out of the pelvis until 12 weeks making continuous external fetal monitoring unreasonable until later in the second trimester.

The debate for fetal monitoring in pregnancy has to also take into account that the fetal monitoring could potentially pick up maternal condi-

tions that result in either decreased oxygenation or perfusion to the uterus and thereby the fetus. This gives the surgical team the opportunity to correct these issues. On the opposite side of the debate is that anesthesia affects the fetus and fetal monitoring may be unreliable. It isn't uncommon for the fetal heart rate baseline and variability to decrease with anesthesia and falsely give the impression of a need to intervene. This could potentially lead to an unnecessary emergency C-section. Because of these issues, the American College of Obstetricians and Gynecologists has published the recommendations in the box below.

Box 17.3 ACOG Guidelines for Fetal Monitoring During Surgery

- If the fetus is considered previable, it is generally sufficient to ascertain the fetal heart rate by Doppler before and after the procedure.
- At a minimum, if the fetus is considered to be viable, simultaneous electronic fetal heart rate and contraction monitoring should be performed before and after the procedure to assess fetal well-being and the absence of contractions.
- Intraoperative electronic fetal monitoring may be appropriate when all of the following apply:
 - The fetus is viable.
 - It is physically possible to perform intraoperative electronic fetal monitoring.
 - A health-care provider with obstetric surgery privileges is available and willing to intervene during the surgical procedure for fetal indications.
 - When possible, the woman has given informed consent to emergency cesarean delivery.
 - The nature of the planned surgery will allow the safe interruption or alteration of the procedure to provide access to perform emergency delivery.

In select circumstances, intraoperative fetal monitoring may be considered for previable fetuses to facilitate positioning or oxygenation interventions.

The decision to use fetal monitoring should be individualized and, if used, should be based on gestational age, type of surgery, and facilities available. Ultimately, each case warrants a team approach (anesthesia and obstetric care providers, surgeons, pediatricians, and nurses) for optimal safety of the woman and the fetus.

American College of Obstetricians and Gynecologists [14]

Considerations for fetal monitoring should at least involve a check of the heart rate prior to and after the conclusion of the surgical procedure and anesthesia [12]. In the first and early second trimesters, this may be all that is necessary. As the pregnancy reaches viability around 22–24 weeks, this can incorporate expansion of the monitoring to intraoperative evaluation with either intermittent or continuous monitoring depending on the type of surgery and access available to the lower abdomen. The level of monitoring should be done in consultation with an obstetrician who can base the decision on gestational age, surgery type, and available resources at the facility to act on any abnormal findings. Besides counseling the operative team, they can also counsel the patient and/or family.

17.5 Anesthesia for Pregnant Patients

The majority of the research related to the use of general anesthesia in pregnant patients is restricted to retrospective studies and registries making the conclusions limited. Most studies show that surgical anesthesia doesn't increase the risk of miscarriages or fetal anomalies [14].

The optimal timing of surgery and anesthesia for pregnant patients is in the second trimester where the risk of spontaneous miscarriages has decreased significantly and organogenesis is complete [19].

Consideration for options such as spinal or epidural anesthesia can reduce exposure of the fetus to agents. Care needs to be taken to avoid hypotension with adequate hydration to avoid hypotension, which can reduce uterine blood flow to the fetus.

Discussion of the physiologic changes associated with pregnancy earlier in this chapter should encourage the anesthesiologist to plan ahead for certain aspects of the surgical case. Theoretical delays in gastric emptying with relaxation of the gastroesophageal sphincter can potentially increase the risk of aspiration during intubation [20]. Treating pregnant patients with the notion that even if fasting they can aspirate may be prudent. Cricoid pressure, metoclopramide, and antacids should be considered. Edema of the face and neck associated with pregnancy, along with mild thyroid enlargement, may increase the challenges of intubation. Some studies show almost one third of term gravid patients may

have a class IV Mallampati airway [10, 21]. Increased rate of desaturation with apnea (in as little as 3 min), coupled with the airway changes, should encourage ready accessibility to airway tools such as glide scopes and alternatives to endotracheal tubes.

17.6 Perioperative Care for Pregnant Patients

Because of significant compression to the vena cava and aorta by the gravid uterus, pregnant patients should be placed in a left lateral tilt if possible. If not, at least a tilt of the hips with a fully padded 1 L IV bag, semicircular gel pad, or rolled-up blanket under the right buttocks can offer a tilt to the left. This will prevent decreased preload and cardiac output, which translates into uteroplacental hypoperfusion. Hypercoagulability in pregnancy can increase the risks of venous thrombotic events (VTE) including deep vein thrombosis (DVT). At a minimum, serial compression devices (SCD) should be applied. Because of the higher molecular weight of heparins (including low molecular weight heparin), the ability to cross the placenta is limited [16]. For higher-risk cases, additional VTE prophylaxis can be used in pregnancy.

Typically antibiotic prophylaxis for most types of procedures can be used with the exception of fluoroquinolones and tetracyclines [16]. Penicillin-, cephalosporin-, erythromycin-, and vancomycin-based prophylaxis are felt to be safe (consult teratogenicity databases or your hospital pharmacist about specific agents).

Maintenance of normal body temperature is important to prevent peripheral vasoconstriction, which could affect blood flow to the uterus with hypothermia. Care should be exercised to avoid increased body temperature as febrile illnesses have been discussed as a potential risk factor for miscarriage and congenital anomalies early in pregnancy.

17.7 Obstetrical Physiologic Changes Affecting Perioperative Care

Specific perioperative complications are discussed in detail in other chapters of this book. Some of these complications are managed via similar means as the nonpregnant patient. Because of

physiologic changes discussed earlier in this chapter, certain complications in normal pregnancy require alterations in their management. Particular conditions exclusive to obstetrics can increase the risks of complications and/or require significant modifications to their management.

In normal pregnancy, airway management has to take into account that gravid women have increased edema, increased oral secretions, increased reflux, and increased gag reflex. Partnered with the potential to desaturate in a quicker manner, efficient placement of an airway is a concern. Maternal desaturation can quickly lead to fetal desaturation if the situation isn't rectified quickly.

During induction, edema can lead to a class IV Mallampati airway requiring additional tools since visualization of the epiglottis and vocal cords may not be possible [10, 21]. Edema especially late in pregnancy can affect placement of oral airways while trying to establish the airway. Additionally, this edema may prevent passage of the usual diameter of endotracheal tube. Smaller tubes may be necessary which can indirectly affect gas exchange and pressures needed to ventilate. Having the usual tools used for difficult airways and suction readily available prior to induction can prevent prolonged intubation and maternal desaturation. Because of the increased secretions and reflux, cricoid pressure and the use of anesthesia protocols to reduce the risks of aspiration should be considered.

Loss of an airway can quickly lead to maternal desaturation with little notice prior to the rapid drop of O₂ saturation. Re-establishment of the airway, ventilation, and oxygenation can be complicated by the decrease in total lung capacity. During an emergency, the tendency to provide increased volume and pressure while bagging with a facemask, along with relaxation of the gastroesophageal sphincter, can rapidly lead to stomach hyperinflation and aspiration of contents. Although placement of oral gastric or nasogastric tubes can deflate this hyperinflation, it is best to avoid this issue by carefully adjusting the volume and pressure while bagging until the endotracheal tube can be replaced.

There are progressive changes in cardiac and respiratory physiology (as discussed earlier in the chapter) as the pregnancy advances that can make ventilation complications unusual. Issues with bronchospasm and constriction from inflamma-

tion can be managed in the same fashion as with nonpregnant women. Typically the immune system is downregulated during pregnancy to prevent rejection of the fetus. This means that certain types of asthma may improve during pregnancy. The use of bronchodilators and glucocorticoids is typically safe in pregnancy (consult teratogenicity databases or your hospital pharmacist about specific agents). Ventilation-perfusion mismatch can be seen during pregnancy associated with pulmonary embolism and in some cases amniotic fluid embolism. Pregnancy is a time of hypercoagulation so there is an increased rate of venous thrombotic event (VTE). Management of VTE is unchanged by pregnancy with anticoagulation by heparin or low molecular weight heparin. Amniotic fluid embolism will be discussed later in this chapter.

Pulmonary edema can be associated with certain conditions such as preeclampsia caused by endovascular leakage. Treatment will be addressed under the preeclampsia pregnancy-associated hypertension heading.

Obstetrical hemorrhage is one of the leading causes of maternal/fetal morbidity and mortality [22]. Average blood loss for a vaginal delivery is 500 ml, a C-section is 1000 ml, and a cesarean hysterectomy is 1500 ml [23]. Blood and fluid loss in pregnancy usually is masked until a significant loss has occurred because of the increase intravascular volume and vasodilatation that occurs in pregnancy to increase blood flow to the uterus. This is coupled with the increase in cardiac output and slight baseline tachycardia in pregnant women. Signs of significant blood loss may not appear in the form of considerable tachycardia and hypotension until 25% of the total blood volume has been lost.

The delay of the customary signs of hypovolemia, joined with the rapid nature of blood loss that can occur with pregnancy, places a high emphasis on the need to anticipate potential blood loss and preemptively arrange for treatment [24]. Early identification of bleeding and communication with the rest of the OR team that bleeding is apparent should trigger treatment prior to the physiologic changes occurring. This leads to the need to proactively anticipate the conditions that can lead to rapid loss of blood and have protocols in place for massive transfusion to obtain necessary blood products in a timely fashion [24]. Adequate diameter IV access has to be obtained

in gravid patients prior to the start of procedures, and additional IV access or central access may be needed in patients at high risk for blood loss.

Close monitoring of urinary output is an integral part of screening circulatory function and treatment response in hemorrhage via blood flow to the kidneys and production of urine. Foley cauterization with a closed drainage system should be considered for any procedure at high risk for blood loss in pregnant patients. Urine output of at least 0.5 ml/kg/h should be maintained during the operative course.

Recommendations for optimal blood product replacement for obstetrical patients have been modified from trauma protocols and are considered multicomponent [25]. Ratio of packed red blood cells/fresh frozen plasma/platelets is now 1:1:1 [26]. Hemorrhage in pregnancy can quickly lead to a consumptive coagulopathy with decreased fibrinogen. Fibrinogen levels are normally elevated above normal adult values in pregnancy. A normal value can be misleading. Disseminated intravascular coagulation (DIC) requires the addition of cryoprecipitate in pregnancy.

17.8 Obstetrical Conditions

17.8.1 Ectopic Pregnancies

Typically ectopic pregnancies occur in the fallopian tube and are diagnosed in the first trimester. Thanks to advancing ultrasound technology incorporated with BHCG levels, most ectopic pregnancies are diagnosed prior to rupture and bleeding. Patients may present emergently with acute abdominal pain, significant bleeding, and blood loss from their unrealized pregnancy. It is not unusual to find over a liter of blood in the pelvis from a ruptured ectopic. Hemodynamic instability can progress rapidly requiring preoperative and intraoperative volume resuscitation.

Certain types of ectopic pregnancies can result in even higher levels of blood loss or risks based on the site of implantation. Cornual ectopic pregnancy (implantation in the portion of the fallopian tube transversing the uterine myometrium or first portion of the fallopian tube) typically ruptures later in the first trimester or early second trimester. The amount of bleeding can be profuse and quickly become catastrophic [27].

This condition requires quick surgical intervention along with aggressive fluid/blood replacement. Again preoperative planning for the need of blood products and large-bore IV access along with rapid activation of massive transfusion protocol should be considered.

Seen more recently with the increased rates of C-sections is implantation of the pregnancy into the C-section scar. These can result in a scar dehiscence and perforation into the abdominal cavity or even the bladder resulting in a severe hemorrhage [28]. These patients may require emergent hysterectomy if they are actively bleeding. This requires planning for the need of blood products and potential coagulopathy that can occur with hemorrhage.

17.8.2 Molar Pregnancy

Hydatidiform moles are part of gestational trophoblastic disease (GTD) and are atypical pregnancies associated with placental hypertrophy. They are typically diagnosed during the first trimester by abnormally high BHCG and snowstorm pattern on ultrasound. When evacuation is indicated by suction D&C, there is a significant risk for blood loss and embolization of the tissue. Preparation for potential large blood loss with availability of blood products and oxytocin (Pitocin) to help the uterus to clamp down should be included in operative management. If embolization was to occur, significant hypoxia can happen along with an inflammatory reaction that can trigger a consumptive coagulopathy similar to amniotic fluid embolism (see management under that heading) [29].

17.8.3 Abnormal Placentation

This section includes issues with atypical locations of the placenta as well as invasion of placental tissue into the uterine myometrium. As the number of C-sections has increased for delivery, we are seeing increased numbers of patients with placental abnormalities typically related to scarring of the endometrial cavity.

A placenta previa is when part or the entire placenta covers the cervix. A vasa previa is when membranous umbilical vessels cover the cervix [30]. With labor significant bleeding can occur resulting in maternal/fetal distress and the need for emergent C-section. With a significant number of previa, the placenta may locate near the

incision point for a low transverse C-section (the most common type). This can result in additional bleeding and difficulty reaching the fetus [31]. Modification of the uterine incision (classical c-section) may be necessary and result in more blood loss. The need for crystalloid and blood products may be necessary; therefore, the preoperative planning should include adequate IV access and the availability of blood products.

Placenta previa sometimes will occur because of a placenta accreta, placenta increta, or placenta percreta. All three of these conditions result when there is a loss of the decidua and there is invasion of the placenta into the underlying myometrium causing the placenta not to separate after delivery. Accreta is the term for when the placenta superficially invades the myometrium. Increta indicates deep myometrial invasion of the placenta. Percreta is the most serious situation as the placenta has invaded through the myometrium and into adjacent tissues such as the bladder, bowel, abdominal wall, and vessels. Catastrophic bleeding can occur if not recognized preoperatively, and attempts are made to manually extract the placenta [31].

If diagnosed preoperatively, referral to a tertiary care center with a multidisciplinary team should be considered. Typically, the availability of neonatology, general/vascular surgery, urology, interventional radiology, and gyn oncology may be required. The perioperative team should choose a room large enough to accommodate a large team. General anesthesia should be considered to allow for muscle relaxation and placement of retractors. Massive transfusion protocols should be readied, and large amounts of blood products should be available in-house if not in the operating room.

Some institutions may have the availability for interventional radiology to place occlusion balloons or embolize vessels [32]. Urology may consider placement of ureteral stents. Cell salvage equipment should be readied if available. Rapid infusion devices, central venous access, and arterial lines may be needed.

Typically, a fundal or posterior uterine incision is utilized and followed by closure of the uterine incision. This is followed by a cesarean hysterectomy to prevent further hemorrhage. Some small series have demonstrated the options for conservative management with closure of the uterine incision with the placenta left in place. This may be considered if no significant bleeding is encountered and the facility can emergently deal with a secondary hemorrhage.

A very rare type of pregnancy is an intra-abdominal pregnancy with implantation outside of the uterus. The attachment of the placenta to bowel, peritoneal lining, omentum, or any other intra-abdominal structure is highly vascular and invades into the structure preventing normal separation. If the placental attachment is disturbed, significant bleeding that is difficult to control occurs. Packing of the abdomen, sewing the edge of the placenta in place, or use of hemostatic agents may be required. Perioperative preparation for significant bleeding is necessary as discussed above.

17.8.4 Placental Abruption

This is the premature separation of the placenta from the uterine wall prior to delivery of the fetus. Typically associated with pain and uterine contractions, it can occur with varied signs depending on the amount of bleeding that occurs. Most likely to occur in the third trimester, it can be a source of fetal distress and may require emergent C-section.

These gravid patients can have significant bleeding leading to hemodynamic instability and consumptive coagulopathy requiring treatment with little prior preparation in the face of an emergent delivery [31]. As discussed in other parts of this chapter, rapid assessment for blood loss amounts and preemptive planning for the need to infuse large amounts of fluids and/or blood products is the mainstay of management. Need for platelets and cryoprecipitate may become necessary on short notice [22].

17.8.5 Uterine Inversion

Uterine inversion is when the uterine fundus invertly prolapses to or through the cervix. This can result in significant hemorrhage and quickly has to be attended to for successful resolution. It can occur when the placenta fails to release and traction is placed on the umbilical cord either during vaginal delivery or C-section. It can also occur spontaneously but less likely. In order for the obstetrician to replace the uterus, relaxation of the uterus may be required. Use of tocolytics such as terbutaline 0.25 mg SQ, magnesium sulfate IV or IM, halogenated inhaled general anesthetics, or nitroglycerin SL has been shown to be effective in these cases. Some incidences may require lapa-

rotomy to resolve. Close monitoring of blood loss and hemodynamic stabilization may be required during the replacement of the uterus [22].

17.8.6 Uterine Atony

Post delivery or post C-section, subinvolution of the uterus can occur leading to significant hemorrhage and hemodynamic instability. Certain risk factors such as prolonged use of oxytocin, high parity, infection, general anesthesia, multi-gestation, polyhydramnios, fetal macrosomia, fibroids, and uterine inversion can all contribute to this condition. This can occur immediately after delivery or can be delayed for hours or even days after delivery [22].

Quickly recognizing atony and a systematic protocol for its management can help limit the impact on the patient. Immediate uterine massage and emptying of the bladder are indicated. Anesthesia should increase oxytocin IV fluid rates and implement massive transfusion protocols or summon blood products for possible administration. Additional use of uterotonics is indicated in about 25% of cases. These include methylergonovine (Methergine) 0.2 mg IM but is contraindicated in cases of hypertension and preeclampsia. 15-Methyl prostaglandin F-2 alpha (Hemobate) 250 mcg IM or intramyometrial can be given every 15 min up to eight doses. This is contraindicated in patients with asthma. Misoprostol (Cytotec) 600–1000 mcg can be administered PO, SL, or PR once.

Additional surgical tamponade or vessel ligation can be employed. Use of intrauterine balloons such as the Bakri or Ebb can help when medications fail to resolve the atony. Alternatives include using several large (60 cc) Foley catheters or packing the uterus with Kerlix gauze [33].

If available, uterine artery embolization by the interventional radiologist may help reduce pulse pressure to the uterus. Vascular ligation by the surgeon with O'Leary stitches to the uterine vessels and/or utero-ovarian ligaments can have the same effect. Hypogastric (internal iliac) artery ligation has fallen out of favor because of limited success and risks. The obstetrician may employ uterine compression sutures such as B-lynch before the final option of hysterectomy is considered [34]. The perioperative management of these cases of postpartum hemorrhage is further discussed below.

Box 17.4 Uterotonic Medications for Postpartum Hemorrhage

Drug ^a	Dose and route	Frequency	Contraindications	Adverse effects
Oxytocin	IV: 10–40 units per 500–1,000 mL as continuous infusion or IM: 10 units	Continuous	Rare, hypersensitivity to medication	Usually none
				Nausea, vomiting, hyponatremia with prolonged dosing
				Hypotension can result from IV push, which is not recommended
Methylergonovine	IM: 0.2 mg	Every 2–4 h	Hypertension, pre-eclampsia, cardiovascular disease, hypersensitivity to drug	Nausea, vomiting, severe hypertension particularly when given IV, which is not recommended
15-methyl PGF	IM: 0.25 mg Intramyometrial: 0.25 mg	Every 15–90 min, eight doses maximum	Asthma, relative contraindication for hypertension, active hepatic, pulmonary, or cardiac disease	Nausea, vomiting, diarrhea, fever (transient), headache, chills, shivering hypertension, bronchospasm
Misoprostol	600–1,000 micrograms oral, sublingual, or rectal	One time	Rare, hypersensitivity to medication or to prostaglandins	Nausea, vomiting, diarrhea shivering, fever (transient), headache

Modified from Lyndon et al. [43], American College of Obstetricians and Gynecologists [22]

Abbreviations: *IV* intravenously, *IM* intramuscularly, *PG* prostaglandin

^aAll agents can cause nausea and vomiting

17.8.7 Postpartum Hemorrhage

Postpartum hemorrhage is a catastrophic perioperative event and a leading cause of peripartum morbidity and mortality. In addition to the disorders mentioned above, there are additional obstetrical conditions that can cause hemorrhage. Genital tract trauma in the forms of cervical, vaginal, or perineal lacerations can require perioperative management. Additionally uterine rupture can occur especially in patients with prior C-section and necessitate an emergent C-section complicated by large blood loss. Retained placental tissue can cause significant bleeding and

require operative intervention with dilation and curettage [22].

Certain comorbidities can also contribute to postpartum bleeding such as sepsis, inherited coagulation disorders (von Willebrand, hemophilia), conditions requiring anticoagulation, and thrombocytopenia. The thrombocytopenia can sometimes be gestational or related to obstetric disorders such as preeclampsia.

Successful management requires planning for these events. Having postpartum hemorrhage (PPH) protocols (with checklists) in place for rapid intervention should be considered for every obstetrical facility. These protocols should include

means to quickly summon needed or additional providers. Having necessary obstetrical and anesthesia supplies available on a PPH cart can increase the speed to treat [35].

Massive transfusion protocols should be developed in conjunction with the institutions blood bank so that anesthesia personnel and obstetricians can urgently obtain blood products when emergent needs arise [26]. Adequate anesthesia preoperative planning should consider the potential risk for hemorrhage with obstetrical conditions. Anticipatory use of rapid infuser lines, large-bore IV, and multiple IV access should be considered along with requests for blood products prior to induction of anesthesia.

Uterotonic agents should be rapidly available for administration in the OR, labor unit, and postpartum unit. Some recent studies have suggested the use of tranexamic acid 1 g IV as an adjuvant therapy to use for postpartum hemorrhage [36]. Consideration for availability of equipment such as cell salvage and personnel such as interventional radiologist can be proactively arranged.

17.8.8 Pregnancy-Associated Hypertension

Hypertension in pregnancy is a common disorder affecting between 5 and 10% of gravid females. Preeclampsia (hypertension with proteinuria) and gestational hypertension typically occur later in pregnancy but can occur any time after 20 weeks of gestation. It can sometimes be confused with underlying hypertension which can be masked by decrease in blood pressure early in a gestation when most patients are first seen for prenatal care. Preeclampsia is diagnosed by elevated blood pressure above 140/90 along with signs such as persistent headache, scotomata (spots in vision), right upper quadrant or epigastric pain, and non-dependent edema. Laboratory evidence of the disease can include proteinuria, hemoconcentration (increased hemoglobin and hematocrit), increased uric acid, increased LDH, and elevation of liver enzymes. Patients are felt to have severe preeclampsia when blood pressures are greater than 160/110. Although the etiology is uncertain, the treatment is delivery. Mild preeclampsia is typically observed until the patient is full term or develops severe preeclampsia signs. Severe preeclampsia warrants

immediate delivery. If untreated, preeclampsia can potentially lead to eclampsia (seizures). Variations of this condition can include HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets). HELLP syndrome is considered to be a severe form of these disorders. Sometimes patients can have preeclampsia superimposed on chronic hypertension.

Physiologically these patients have significant endovascular leakage leading to intravascular volume depletion, edema, ascites, and pulmonary edema. Additional effects can be renal and/or liver dysfunction along with decreased fetal blood flow. Additionally these women can develop DIC, placental abruptions, acute renal failure, liver hemorrhage (subcapsular hematoma), liver failure, acute respiratory distress (ARDS), strokes, and even death. These effects can vary between slow to rapid onset and progression [37].

Treatment is usually centered on delivery at a center that can handle the maternal conditions and fetus after delivery especially if premature and needing neonatal intensive care. Magnesium sulfate 4-6 g load over 20 min followed by 2 g/h IV is usually employed to prevent eclampsia (phenytoin is used instead of magnesium in certain parts of the world). This is continued until 24 h after delivery. The dosage may need to be reduced in the presence of decreased renal function. Hypertension episodes are managed with labetalol IV, nifedipine PO, or hydralazine IV. Labetalol IV is given as slow push 10-40 mg every 10 min for a maximum of 300 mg. Nifedipine PO is administered 10-20 mg every 20 min for a maximum of 50 mg. Hydralazine IV is administered 5-10 mg every 20 min for a maximum of 25 mg [38].

Perioperative care should consider the significant physiologic changes associated with these patients. Even though these patients have significant edema, they are intravascularly constricted; therefore, diuretics have little effect and can result in decreased uterine blood flow and fetal distress. Fluid management intraoperatively should lean toward the conservative side as the endovascular leakage can cause worsening generalized and pulmonary edema. Anesthesia should take into account these patients may develop thrombocytopenia. Even in patients with normal platelet counts, the function of the platelets may be sub-optimal which may increase the risk of complications associated with spinal or epidural anesthesia. Bleeding

risks can increase because of preeclampsia, and preoperative planning should consider need for blood products including packed red blood cells, platelets, and cryoprecipitate. Intravascular constriction can mask anemia and true blood volume. Elevated blood pressures can hide hypotension normally noted with excessive blood loss. Airway management in general anesthesia can be complicated by laryngeal edema and facial edema.

17.8.9 Eclampsia

Eclampsia is the incidence of seizure activity typically associated with preeclampsia. It can occur even in patients with little or no preeclampsia signs. It usually will occur without warning and usually only lasts for a few minutes. Delivery is indicated, but emergent C-section is not required. Eclampsia can occur perioperatively, and treatment is geared toward protecting the patient during their seizure with padding and positioning to avoid risk of aspiration. Protection of the airway and supplemental oxygenation are important. Medical treatment is usually magnesium sulfate as mentioned above. Use of benzodiazepines should be reserved to patients nonresponsive to magnesium and have both IV access and availability for intubation. Fetal sedation can occur with their use. Lorazepam 2 mg can be used as a slow IV push [37]. In rare cases of status epilepticus, general anesthesia may be considered.

17.8.10 Amniotic Fluid Embolism

Amniotic fluid embolus (AFE) is a rare condition caused by fetal debris entering the maternal circulation, which then triggers abnormal activation of proinflammatory mediator response systems. Estimates of incidence and mortality rates vary widely due to a lack of established standardized criteria; however, maternal mortality is believed to occur in 30–90% of cases. The incidence of AFE ranges from 1:15,000 to 1:53,000 deliveries. Nearly 70% of AFE present suddenly at time of delivery or immediately postpartum and typically present with an otherwise unexplainable combination of clinical manifestations often characterized by hypotension, fetal distress, pulmonary edema, acute respiratory distress syndrome (ARDS), car-

diopulmonary arrest, hypoxia, coagulopathy, and/or seizure.

Although AFE can present in a multitude of ways, the rapid deterioration of a patient suffering from an AFE can be outlined in three progressive stages. In phase 1, pulmonary and systemic vasoconstriction leads to hypertension and severe O₂ desaturation. Phase 2 follows immediately and results in decreased systemic vascular resistance and cardiac output. In phase 3, sudden cardiac failure, ARDS, and coagulopathy via DIC cascade ensue. No rapid test for AFE exists; therefore, the diagnosis remains clinical, and quick recognition is paramount to successful treatment as most maternal mortality occurs within 30 min.

Management and treatment of AFE are supportive and require rapid simultaneous interdisciplinary cooperation between OB/GYNs, RNs, anesthesiologists, and critical care personnel. Establishing large-bore IV access with pulse oximetry, continuous vital sign, and cardiac monitoring is essential. Respiratory support by anesthesia typically requires endotracheal intubation and mechanical ventilation. The basics of CPR-ACLS and massive transfusion protocols must be immediately available and initiated. Hemodynamic support requires judicious use of fluids, vasopressors, inotropes, and pulmonary vasodilators. Laboratory studies such as CBC, BMP, PT/PTT/INR/fibrinogen, and ABGs are very useful to track treatment success; however, treatment should never be delayed awaiting these results [39, 40].

17.9 Summary

Perioperative care of pregnant patients has to take into account the physiologic changes that occur during the progression of the gestation. It has to consider the effects the condition and its treatment have on the fetus. Withholding or limiting treatment because of pregnancy can lead to a more detrimental situation and increase the risks for mother and fetus. Fetal monitoring has to consider the gestational age of the fetus, ability to intervene upon abnormalities noted, and treatment options available at the particular stage of pregnancy.

Understanding of obstetrical conditions can assist anesthesia and operative personnel in their pre- and intraoperative management. Most of the

critical complications associated with pregnancy have to do with hemorrhage. Proactively anticipating and quickly intervening are key to the optimal management.

References

- American College of Obstetricians and Gynecologists. Weight gain during pregnancy. ACOG Committee opinion no. 548. *Obstet Gynecol.* 2013;121:210–2.
- Gabbe SG, Niebyl JR, Simpson JL, Landon MB, Galan HL, Jauniaux ER, et al., editors. *Obstetrics: normal and problem pregnancies.* 7th ed. Philadelphia: Elsevier; 2017. p. 38–63.
- Van Oppen AC, Stigter RH, Bruinse HW. Cardiac output in normal pregnancy: a critical review. *Obstet Gynecol.* 1996;87:310–8.
- Sanghavi M, Rutherford JD. Cardiovascular physiology of pregnancy. *Circulation.* 2014;130:1003–8.
- Mahendru AA, Everett TR, Wilkinson IB, Lees CC, McEniery CM. A longitudinal study of maternal cardiovascular function from preconception to the postpartum period. *J Hypertens.* 2014;32:849–56.
- Hameed AB, Chan K, Ghamsary M, Elkayam U. Longitudinal changes in the B-type natriuretic peptide levels in normal pregnancy and postpartum. *Clin Cardiol.* 2009;32:E60–2.
- Crapo R. Normal cardiopulmonary physiology during pregnancy. *Clin Obstet Gynecol.* 1996;39:3–16.
- Roth A, Elkayam U. Acute myocardial infarction associated with pregnancy. *J Am Coll Cardiol.* 2008;52:171–80.
- Shotan A, Ostrzega E, Mehra A, Johnson J, Elkayam U. Incidence of arrhythmias in normal pregnancy and relation to palpitations. *Am J Cardiol.* 1997;79:1061–4.
- Pilkington S, Carli F, Dakin MJ, et al. Increase in Mallampati score during pregnancy. *Br J Anaesth.* 1995;74:638–42.
- Conrad KP, Stillman IE, Lindheimer MD. The kidney in normal pregnancy and preeclampsia. In: Taylor RN, Roberts JM, Cunningham FG, Lindheimer MD, editors. *Chesley's hypertensive disorders in pregnancy.* 4th ed. New York: Academic Press; 2014. p. 335–78.
- Glinioer D. The regulation of thyroid function in pregnancy: pathways of endocrine adaptation from physiology. *Endocr Rev.* 2014;18:404–33.
- Kovacs CS, Kronenberg HM. Maternal-fetal calcium and bone metabolism during pregnancy, puerperium, and lactation. *Endocr Rev.* 1997;18:832–72.
- American College of Obstetricians and Gynecologists. Nonobstetric surgery during pregnancy. Committee opinion no. 696. *Obstet Gynecol.* 2017;129:777–8.
- Gabbe SG, Niebyl JR, Simpson JL, Landon MB, Galan HL, Jauniaux ER, et al. *Obstetrics: normal and problem pregnancies.* 7th ed. Philadelphia: Elsevier; 2017. p. 2–25.
- Gabbe SG, Niebyl JR, Simpson JL, Landon MB, Galan HL, Jauniaux ER, et al. *Obstetrics: normal and problem pregnancies.* 7th ed. Philadelphia: Elsevier; 2017. p. 136–59.
- American College of Obstetricians and Gynecologists. Antepartum fetal surveillance. ACOG Practice Bulletin no. 145. *Obstet Gynecol.* 2014;124:182–92.
- Gabbe SG, Niebyl JR, Simpson JL, Landon MB, Galan HL, Jauniaux ER, et al., editors. *Obstetrics: normal and problem pregnancies.* 7th ed. Philadelphia: Elsevier; 2017. p. 550–64.
- Cohen-Kerem R, Railton C, Oren D, et al. Pregnancy outcome following non-obstetric surgical intervention. *Am J Surg.* 2005;190:467.
- Chiloiro M, Darconza G, Piccioli E, et al. Gastric emptying and orocecal transit time in pregnancy. *J Gastroenterol.* 2001;36:538.
- Mallampati SR, Gatt SP, Gugino LD, et al. A clinical sign to predict difficult tracheal intubation: a prospective study. *Can Anaesth Soc J.* 1985;32:429.
- American College of Obstetricians and Gynecologists. Postpartum hemorrhage. ACOG Practice Bulletin no. 183. *Obstet Gynecol.* 2017;130:e168–86.
- Dahlke JD, Mendez-Figueroa H, Maggio L, Hauspurg AK, Sperling JD, Chauhan SP, et al. Prevention and management of postpartum hemorrhage: a comparison of 4 national guidelines. *Am J Obstet Gynecol.* 2015;213:76e1–10.
- Combs CA, Murphy EL, Laros RK Jr. Factors associated with postpartum hemorrhage with vaginal birth. *Obstet Gynecol.* 1991;77:69–76.
- Borgman MA, Spinella PC, Perkins JG, Grathwohl KW, Repine T, Beekley AC, et al. The ratio of blood products transfused affects mortality in patients receiving massive transfusions at a combat support hospital. *J Trauma.* 2007;63:805–13.
- Burtelow M, Riley E, Druzin M, Fontaine M, Viele M, Goodnough LT. How we treat: management of life-threatening primary postpartum hemorrhage with a standardized massive transfusion protocol. *Transfusion.* 2007;47:1564–72.
- Luciano DE, Jain A, Roy G, Solima E, Luciano AA. Ectopic pregnancy—from surgical emergency to medical management. *J Minim Invasive Gynecol.* 2004;2:109–21.
- Maheux-Lacroix S, Li F, Bujold E, Nesbitt-Hawes E, Deans R, Abbott J. Cesarean scar pregnancies: a systematic review of treatment options. *J Minim Invasive Gynecol.* 2017;24(6):915–25.
- American College of Obstetricians and Gynecologists. Diagnosis and treatment of Gestational Trophoblastic disease. ACOG Practice Bulletin no. 53. *Obstet Gynecol.* 2004;103:1365–77.
- Oyelese Y, Smulian JC. Placenta previa, placenta accreta, and vasa previa. *Obstet Gynecol.* 2006; 107:927.
- Gabbe SG, Niebyl JR, Simpson JL, Landon MB, Galan HL, Jauniaux ER, et al., editors. *Obstetrics: normal and problem pregnancies.* 7th ed. Philadelphia: Elsevier; 2017. p. 395–424.
- Ballas J, Hull AD, Saenz C, et al. Preoperative intravascular balloon catheters and surgical outcomes in pregnancies complicated by placenta accreta: a management paradox. *Am J Obstet Gynecol.* 2012;207:216.
- Patacchiola F, D'Alfonso A, Di Fonso A, Di Febbo G, Kaliakoudas D, Carta G. Intrauterine balloon tamponade as management of postpartum haemorrhage and

- prevention of haemorrhage related to low-lying placenta. *Clin Exp Obstet Gynecol.* 2012;39:498–9.
34. Hayman RG, Arulkumaran S, Steer PJ. Uterine compression sutures: surgical management of postpartum hemorrhage. *Obstet Gynecol.* 2002;99:502–6.
 35. American College of Obstetricians and Gynecologists. Preparing for clinical emergencies in obstetrics and gynecology. Committee opinion no. 590. *Obstet Gynecol.* 2014;123:722–5.
 36. Simonazzi G, Bisulli M, Saccone G, Moro E, Marshall A, Berghella V. Tranexamic acid for preventing postpartum blood loss after cesarean delivery: a systematic review and meta-analysis of randomized controlled trials. *Acta Obstet Gynecol Scand.* 2016;95:28–37.
 37. Gabbe SG, Niebyl JR, Simpson JL, Landon MB, Galan HL, Jauniaux ER, et al., editors. *Obstetrics: normal and problem pregnancies.* 7th ed. Philadelphia: Elsevier; 2017. p. 661–705.
 38. American College of Obstetricians and Gynecologists. Emergent therapy for acute-onset, severe hypertension during pregnancy and the postpartum period. Committee opinion no. 692. *Obstet Gynecol.* 2017;129:e90–5.
 39. Conde-Agudelo A, Romero R. Amniotic fluid embolism: an evidence-based review. *Am J Obstet Gynecol.* 2009;201:445.e1–445.e13.
 40. Society for Maternal-Fetal Medicine (SMFM) with the assistance of, Pacheco LD, Saade G, et al. Amniotic fluid embolism: diagnosis and management. *Am J Obstet Gynecol.* 2016;215:B16–24.
 41. Gabbe SG, Niebyl JR, Simpson JL, Galan H, Goetzl L, Jauniaux ER, et al., editors. *Obstetrics: normal and problem pregnancies.* 5th ed. Philadelphia: Churchill Livingstone Elsevier; 2007.
 42. American College of Obstetricians and Gynecologists. Critical care in pregnancy. ACOG Practice Bulletin no. 170. *Obstet Gynecol.* 2016;128:e147–54.
 43. Lyndon A, Lagrew D, Shields L, Main E, Cape V, editors. *Improving health care response to obstetric hemorrhage version 2.0. A California quality improvement toolkit.* Stanford (CA): California Maternal Quality Care Collaborative; Sacramento (CA): California Department of Public Health; 2015.