Complications arising in the third trimester often challenge the clinician to balance the concern for maternal well-being with the consequences of infant prematurity. The most serious and challenging antepartum issues relate to preterm labor and birth, hypertensive disorders, and bleeding events. The article guides the practitioner through decision-making and management of these problems.

PRETERM BIRTH

Background

Preterm birth (delivery <37 weeks’ gestation) is considered the leading cause of infant morbidity and mortality worldwide. The consequences of prematurity can be lifelong and include complicated medical conditions, developmental concerns, and poor performance in school. In addition, the cost of neonatal intensive care and management of chronic health conditions can be enormous. A 2006 Institute of Medicine report estimated the annual costs of evaluation and care associated with preterm birth to be approximately $26.2 billion in 2005, or $51,600 per infant born preterm.1

In 2009 there were more than 500,000 preterm deliveries in the United States with a rate of 12.18%, down from 12.3% in 2006.2 Approximately 23% of all preterm births were recurrent. There are clear racial disparities in the incidence of preterm birth in the United States. African American women experience the highest rates of preterm birth (17.5% in 2009) and Asian/Pacific Islander women the lowest (10.7%). The differences in preterm birth observed between racial groups have not been explained by socioeconomic factors or other risk factors, including tobacco or drug use. The greatest risk factor for preterm birth is a history of preterm birth in a prior pregnancy; approximately 15% of all preterm births occur in women with this history.3 Other risk

The author has nothing to disclose.
Department of Obstetrics and Gynecology, Contra Costa Regional Medical Center, 2500 Alhambra Avenue, Martinez, CA 94543, USA
E-mail address: dr.emily.newfield@gmail.com

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factors include multiple gestation and low prepregnancy weight (body mass index [BMI] <20, calculated as the weight in kilograms divided by height in meters squared).

Many clinical management strategies have been developed and tested to assist women at risk for preterm birth. The role of prenatal care providers is to identify patients at greatest risk for preterm birth, offer safe interventions to patients to prevent recurrent preterm birth, and identify early symptoms of preterm parturition to manage the pregnancy effectively and efficiently.

**Women with a History of Preterm Birth**

Any woman with a history of any delivery between 16 0/7 and 36 6/7 weeks is at high risk of recurrent preterm birth. Women with a history of more than 1 preterm birth and a history of preterm birth at less than 32 weeks’ gestation are at greatest risk of recurrence. Women with a history of stillbirth are also at increased risk of future preterm birth.

Understanding the sequence of events that led to a prior preterm birth is important for helping to establish the risk of recurrence. The physical changes and symptoms of preterm birth are different from those that occur at term. In most preterm births, the cervix shortens (rippens), followed by decidual-membrane activation (commonly detected by the fetal fibronectin test), followed by uterine contractions. Most women experience these changes as increasing vaginal and/or pelvic pressure, mild pelvic cramping or low back pain, and increasing vaginal discharge. These early changes and symptoms can precede preterm labor by 3 to 6 weeks.4

Determining the series of events that preceded a spontaneous preterm birth is critical for effective counseling regarding the risk of reoccurring preterm birth. For example, if the woman describes a history of vaginal spotting (from vanishing twin or diagnostic amniocentesis) followed by ruptured membranes and preterm delivery, this may represent a lower risk of recurrent preterm birth. Acute events, such as placental abruption or chorioamnionitis, may provoke preterm labor and birth and, depending on the cause of the event, may be less likely to repeat themselves.

Prior preterm delivery of twin pregnancy is associated with increased risk of recurrent preterm birth, but the degree of risk depends on the gestational age at time of preterm delivery. If the twins are delivered after 34 weeks the risk of recurrent preterm birth is small, but can be up to 40% if the twins are delivered earlier than 30 weeks’ gestation.

**Prevention Strategies**

**Counseling**

An in-depth review and discussion of the patient’s obstetric and gynecologic history and associated risk of preterm birth is recommended in the first trimester. Obtaining the obstetric records, including operative and pathology reports, regarding any preterm birth is paramount to appropriate counseling and management of the current pregnancy. Surgical procedures on the cervix, that is, loop electrosurgical excision procedure and cold knife cone, are risk factors for preterm birth. Because assisted reproductive technology (including ovulation induction using clomiphene citrate and in vitro fertilization) is associated with an increased risk of preterm birth, it is important to know the circumstances regarding conception of the current pregnancy.

**Primary prevention**

At present there are no recommended interventions for primary prevention of preterm birth, but support for smoking-cessation efforts can be influential. Women at high risk of preterm birth (African American, low prepregnancy weight) should be counseled
regarding the signs and symptoms of preterm labor. Patients who have a shortened cervix identified by midtrimester ultrasonography (US) may benefit from cerclage placement and/or vaginal progesterone (200 mg per vagina at bedtime).5

Prevention of recurrent preterm birth

**Progesterone** 17α-Hydroxyprogesterone caproate (250 mg weekly) intramuscular (IM) injections from 16 to 36 weeks’ gestation have been demonstrated in several large randomized controlled trials to reduce the rate of recurrent preterm birth by approximately 33%.6 Some practitioners do not suggest progesterone treatment if not already initiated before 24 to 26 weeks’ gestation, because patients in the original research trials were enrolled up to 26 6/7 weeks. Patients with a history of delivery between 16 and 20 weeks may benefit from weekly progesterone treatment and should be offered this care. Oral and vaginal preparations are considered less effective than the IM preparation in patients with a history of preterm birth at greater than 20 weeks’ gestation and should not be used. Progesterone treatment does not prevent preterm birth in twin gestations.7,8

**Smoking** Smoking-cessation programs have shown an effect in reducing recurrent preterm delivery.

**Cerclage** Prophylactic vaginal cerclage in patients with a history of 3 or more second-trimester losses reduces the risk of preterm birth. In patients with a history of preterm birth, serial transvaginal US examinations beginning at 16 weeks have been helpful in identifying patients with shortened cervix (measuring less than 25 mm); these women may also benefit from vaginal cerclage placement.4 If a patient has failed vaginal cerclage in the past, transabdominal (open or laparoscopic) cerclage can be placed in early pregnancy (Figs. 1 and 2).

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**Fig. 1.** Prenatal care algorithm for women with a prior preterm birth at 16 to 34 weeks. IUGR, intrauterine growth retardation. (Adapted from Iams JD, Berghella V. Care for women with prior preterm birth. Am J Obstet Gynecol 2010;203(2):89–100; with permission.)
Evaluation of Preterm Labor

Patients who present for evaluation of pelvic pressure, vaginal discharge, vaginal bleeding, or low-back pain between 16 0/7 and 36 6/7 weeks should be assessed for possible preterm labor. Many hospital systems require patients with pregnancies less than 20 weeks’ gestation to be evaluated in the emergency department, whereas rapid assessment of possible preterm labor and development of an intervention strategy may be delayed. A detailed history of symptoms can help differentiate between spontaneous and evoked preterm labor. A complete obstetric history is important to determine the risk for possible recurrent preterm birth.

1. Accurate calculation of gestational age. Many of the tests and interventions associated with the management of preterm labor and birth are gestational-age dependent. Thus, immediate determination of fetal gestational age assists one in identifying which patients require what particular tests and/or interventions. If no prior US has been performed in the pregnancy, consider informal or formal US to assist in calculation of gestational age, estimated fetal weight, and fetal position.

2. Maternal vital assessment. Evaluation of the maternal condition is essential. For example, fever could represent chorioamnionitis, pyelonephritis, appendicitis, or influenza. Hypertension could represent pain or preeclampsia.

3. Fetal vital assessment
   a. Documentation of normal fetal heart tones is sufficient for fetuses less than 23 6/7 weeks.
   b. Continuous external fetal heart rate monitoring should be performed for all viable fetuses (24 0/7 weeks and more).
   c. Continuous tocometry is recommended.
   d. Informal (bedside) US should be performed to document fetal position, estimated fetal weight, placental location, cervical length, and quantity and quality of amniotic fluid.

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Fig. 2. Care algorithm for asymptomatic women with multiple prior preterm births (PTBs) or second-trimester losses (STLs). (Adapted from Iams JD, Berghella V. Care for women with prior preterm birth. Am J Obstet Gynecol 2010;203(2):89–100; with permission.)
of amniotic fluid. Shortened cervical length (<2 cm) is strongly associated with risk of preterm delivery.\(^8\)

4. Physical examination. Complete physical assessment of the mother, including cardiovascular, pulmonary, abdominal, and neurologic examinations, is important. Pelvic examination is mandatory. Informal bedside US or review of prior US reports/images should be undertaken to determine placental location before sterile vaginal examination is performed. If placenta previa or vasa previa is present, avoid digital examination. Careful examination for the presence of blood is important because the presence of blood may represent abruptio placentae, a known promoter of preterm labor.
   a. Sterile speculum examination should be performed first, before any sterile vaginal examination, because the fetal fibronectin test is invalid if the cervix has been manipulated in the prior 24 hours.
   b. Visual inspection of the vaginal vault and cervix can determine the presence of significant discharge or cervical shortening. Prolapsed membranes should not be manipulated digitally until confirmation of gestational age and determination of fetal presentation. Cervical bleeding can originate from the ectocervix (hemangioma, polyp, laceration, lesion) or from the cervical os itself.
   c. Tests
      - Fetal fibronectin: a glycoprotein present at the maternal-fetal interface, absent between 24 and 34 weeks’ gestation. The presence of fetal fibronectin in symptomatic patients is the most effective predictor of preterm labor. The negative predictive value of the test approximates to 99%; symptomatic patients with a negative result of fetal fibronectin test are very unlikely to deliver in the following 7 days. The positive predictive value ranges from 75% to 85%. The test is invalid if performed before 24 0/7 or after 34 0/7 weeks, and cannot be performed if the patient has had intercourse or sterile vaginal examination in the past 24 hours or if significant vaginal bleeding is present.
      - Evaluation of any pooled fluid should be performed to exclude spontaneous rupture of membranes.
      - *Neisseria gonorrhoeae* and *Chlamydia trachomatis* infections are the most common bacterial infections resulting in cervicitis and associated with preterm labor.
      - Microscopic evaluation of any vaginal discharge can determine the presence of bacterial vaginosis, trichomoniasis, or *Candida* infection. Bacterial vaginosis is associated with increased risk of preterm delivery, and although screening and treating asymptomatic patients has not been effective at preventing preterm delivery, treating symptomatic patients is recommended. Trichomoniasis should be treated, and the patient’s partner should be offered treatment as well. Microscopy can also determine if amniotic fluid is present in the vaginal vault, signifying preterm premature rupture of membranes (PPROM). Placental abruption and PPROM often coexist, with resultant uterine contractions because blood functions as a uterine irritant.
      - Urine analysis and culture is recommended. If patient cannot reliably perform a sterile urine sample, a catheter-obtained sample is appropriate.
      - Cocaine and amphetamines are associated with preterm labor, often secondary to placental abruption. Urine toxicology screening, with confirmatory testing on any positive result, should be obtained for all patients who present for evaluation of preterm labor.
• Group B Streptococcus (GBS) testing should be performed on all women at the time of sterile speculum examination. Patients allergic to penicillin have to inform the laboratory, so that appropriate sensitivity testing can be performed on any isolates.

• The sterile vaginal examination is critical to the diagnosis of preterm labor, provided the fetal membranes are intact and fetal fibronectin testing has been performed. Patients who present with cervical dilation of 3 cm or more with 80% cervical effacement are in active preterm labor and should be admitted for management. Patients with dilation of 2 to 3 cm and less than 80% cervical effacement are likely in preterm labor and should have the sterile vaginal examination repeated in 30 to 60 minutes. Patients with cervical dilation of less than 2 cm and less than 80% cervical effacement should be monitored for the following 1 to 2 hours to determine the rate of cervical change. If fetal fibronectin test result is negative in these patients, the likelihood of preterm delivery in the subsequent 7 days is less than 2%, and patients can be monitored in the outpatient setting.

Management of Preterm Labor

Steroids
If a patient shows evidence of preterm labor between 23 5/7 and 34 0/7 weeks' gestation, she requires immediate administration of steroids to promote fetal lung maturity. Treatment options include:

a. Betamethasone (12 mg IM x 2 doses at 24-hour intervals)
b. Dexamethasone (6 mg IM x 4 doses at 12-hour intervals).

Tocolytics
Medications to slow or stop contractions are the mainstay of treatment of preterm labor, although no data exist to suggest that these treatments prolong pregnancy for greater than 48 hours. These medications, including nonsteroidal anti-inflammatory drugs (NSAIDs), β-mimetics, magnesium sulfate, and calcium-channel blockers, can be used to prolong the pregnancy in order to complete the steroid administration and if necessary, to allow for transport to an appropriate clinical setting. All medications are equally efficacious, although calcium-channel blockers appear to have the lowest maternal safety profile. NSAIDs are associated with fetal oliguria and premature closure of the ductus arteriosus. Magnesium sulfate is associated with maternal respiratory distress and should not be used in patients with renal insufficiency. β-Mimetics can provoke maternal cardiovascular events and metabolic derangements. All tocolytics should be used cautiously and not in combination.9 Tocolytic therapy should be discontinued when labor is halted or after 48 hours, and multiple agents should not be used simultaneously. Tocolytics should not be used in patients with preterm labor preceded by ruptured membranes.

Magnesium sulfate for neuroprotection
Current evidence suggests that 24 hours of magnesium sulfate exposure in fetuses less than 32 weeks gestational age reduces the risk of cerebral palsy. This treatment should be offered to patients in preterm labor presenting between 24 0/7 and 32 0/7 weeks' gestation who appear to be at high risk for imminent delivery. Use of magnesium sulfate for neuroprotection and a separate agent for tocolysis is not recommended. Based on the data from available randomized controlled trials, the recommended dose for magnesium sulfate for neuroprotection is 4 g intravenous (IV) loading dose followed by a maintenance dose of 1 g IV per hour for a total of...
24 hours. The medication is discontinued if no delivery has occurred within 24 hours or following delivery. There is no recommendation for readministration of magnesium sulfate for neuroprotective purposes.\textsuperscript{10,11}

**Penicillin**

Although antibiotic therapy has not been demonstrated to stall progression of preterm labor, penicillin for GBS prophylaxis should be administered if delivery appears imminent. If the patient has experienced an allergic reaction to penicillin or cephalosporins that includes anaphylaxis, angioedema, respiratory distress, or urticaria, the 2010 *Guidelines for Prevention of Perinatal Group B Streptococcal Disease* by the Centers for Disease Control and Prevention recommends the use of clindamycin or vancomycin for GBS prophylaxis (refer to the guidelines at http://www.cdc.gov/groupbstrep/guidelines/guidelines.html).

**Transfer of care**

Depending on the level of care offered by a pediatric staff and the services available in the hospital, the patient may be served by transfer to a facility that can accommodate her preterm baby. Consultation with a perinatologist can be useful while managing a patient in preterm labor.

**Mode of delivery**

Vaginal delivery is appropriate for preterm infants, provided there is no evidence of fetal distress with contractions. Cesarean delivery may be considered for preterm infants who demonstrate evidence of fetal growth restriction or if serious maternal conditions coexist, such as severe preeclampsia or eclampsia.\textsuperscript{12}

**Postpartum management**

Mothers of preterm infants should be encouraged to breastfeed provided there are no contraindications. Patients who experience preterm birth require counseling in the immediate postpartum period regarding their risk for recurrent preterm birth. The recommended pregnancy interval for patients with a history of preterm birth is greater than 18 months, to limit recurrent adverse outcomes.\textsuperscript{13}

**HYPERTENSIVE DISORDERS**

Hypertensive disorders complicate approximately 6% of all pregnancies\textsuperscript{14} and account for 15% of all maternal deaths, being the second leading cause of maternal mortality in the United States. Women with hypertensive disorders in pregnancy are at risk for significant complications, including placental abruption, cerebral hemorrhage, hepatic dysfunction, renal insufficiency, and disseminated intravascular coagulation (DIC). The fetal consequences of hypertension include growth restriction, premature birth, and fetal demise.

There are 4 categories of hypertensive disorders in pregnancy: chronic hypertension, preeclampsia/eclampsia, gestational hypertension, and preeclampsia superimposed on chronic hypertension.

**Chronic Hypertension**

Hypertension is defined as a systolic blood pressure (SBP) of 140 mm Hg or more, a diastolic blood pressure (DBP) of 90 mm Hg or more, or both. It is important to differentiate hypertension that precedes pregnancy (chronic hypertension) from hypertension that develops during pregnancy (gestational hypertension or preeclampsia). According to the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy, hypertension that is identified
before 20 weeks’ gestation is chronic hypertension. Chronic hypertension is classified as mild (blood pressure >140/90 mm Hg) or severe (blood pressure >180/110 mm Hg). It may be challenging to identify women with chronic hypertension if they initiate prenatal care after 20 weeks’ gestation, because the physiologic decrease in blood pressure that occurs in the second trimester may delay accurate diagnosis.

Women with chronic hypertension are at increased risk of severe pregnancy complications, including intrauterine growth retardation (IUGR), preterm delivery, placental abruption, and fetal demise. This risk seems higher when patients have evidence of significant proteinuria (≥300 mg protein on 24-hour urine collection in early pregnancy). These women may also have end-organ damage (left ventricular hypertrophy, cardiomegaly, ischemic heart disease, chronic renal insufficiency) from the effects of long-standing hypertension. Cardiac disease can result in cardiac decompensation during the third trimester and parturition.

The current available data suggest that antihypertensive medications do not improve perinatal outcomes in patients with chronic hypertension. It is unclear whether patients treated with antihypertensive medication at the start of pregnancy should continue their medication, because continued use does not seem to prevent poor outcomes and may contribute to decreased placental perfusion. The current recommendation is to withhold antihypertensive medication in patients with mild hypertension unless blood pressures exceed 150/100 mm Hg, and to continue or initiate medication in patients with severe chronic hypertension (blood pressure ≥180/110 mm Hg). Target blood pressure in patients without evidence of end-organ damage (chronic renal insufficiency, cardiac disease) should be 140 to 150/80 to 90 mm Hg and less than 140/90 mm Hg if end-organ damage exists.

Although methyldopa has been used historically and is proven as effective in pregnancy with no issues of fetal safety, it is often poorly tolerated by women and can be unpredictable. Labetalol is a combined α-blocker and β-blocker, which is effective and well tolerated and is suggested as a first-line agent to control hypertension in pregnancy. Nifedipine can also be used safely in pregnancy. Diuretics have been studied and appear safe unless there is documented uteroplacental insufficiency, whereby diminished blood volume may compromise fetal well-being. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are contraindicated in the second and third trimesters because of severe fetal consequences including underdeveloped calvarium, anuria, renal dysgenesis, IUGR, and fetal death.

Antepartum management

Initial evaluation of a pregnant patient with chronic hypertension should include a baseline 24-hour urine collection and blood tests of renal function. Renal dysfunction in early pregnancy is associated with poor pregnancy outcomes; patients with a serum creatinine level of more than 2.5 at the start of pregnancy have a 40% risk of developing end-stage renal disease requiring dialysis during pregnancy or in the postpartum period. Determining whether patients have baseline renal dysfunction can also help interpret third-trimester changes that may represent superimposed preeclampsia. Women with chronic hypertension have a 10% to 25% risk of developing preeclampsia superimposed on baseline hypertension.

Because of the increased risk of intrauterine growth restriction, patients with chronic hypertension should receive a baseline US at 18 to 22 weeks’ gestation with US being repeated at 28 to 32 weeks, and then monthly until delivery. Twice-weekly fetal testing should begin at 32 weeks and continue until delivery. There is little consensus as to whether patients with mild chronic hypertension require induction at gestational age less than 40 weeks. Given that patients with chronic hypertension are at increased
risk for stillbirth and significant likelihood of developing superimposed preeclampsia, the American Congress of Obstetricians and Gynecologists Practice Bulletin on Chronic Hypertension in Pregnancy recommends induction of labor after 37 weeks for patients with mild chronic hypertension.\textsuperscript{15} Patients with severe hypertension often deliver prematurely or require early intervention because of superimposed preeclampsia. Patients with severe preeclampsia superimposed on chronic hypertension who are at greater than 28 weeks’ gestation should undergo delivery. If patients remain pregnant after 37 weeks’ gestation, those with severe chronic hypertension should undergo delivery. Cesarean delivery should be reserved for maternal or fetal indications; the diagnosis of hypertension does not require cesarean delivery.

**Preeclampsia/Eclampsia**

This pregnancy-specific problem is defined as an increase in blood pressure of 140/90 mm Hg or more after 20 weeks’ gestation with associated proteinuria of 300 mg or more protein in 24 hours. The disease typically manifests in the late third trimester, and risk factors include chronic hypertension, diabetes, extremes of maternal age, and history of preeclampsia in prior pregnancy. Preeclampsia is associated with severe maternal and fetal complications, including seizure, cerebral hemorrhage, acute kidney injury, pulmonary edema, liver hemorrhage (maternal) and fetal growth restriction, placental abruption, and complications of prematurity (fetal). Delivery is the only definitive treatment for preeclampsia. There is always maternal benefit to delivery because the risk for severe complications diminishes significantly and rapidly with delivery. The struggle for the obstetrician is to determine when the fetal consequences of delivery, that is, prematurity, are greater than the risk of prolonging a pregnancy complicated by preeclampsia.

**Diagnosis**

**Hypertension** Blood pressure elevations of 140/90 mm Hg or more should be measured on two separate occasions more than 4 hours apart but within 1 week. Patients at present using antihypertensives (for treatment of chronic hypertension) can be diagnosed with preeclampsia, if a single DBP of 110 mm Hg or more is observed.

**Proteinuria** The gold-standard test for the presence of significant proteinuria is the 24-hour urine collection for protein. Some practitioners advocate using the spot urine protein-to-creatinine ratio test to help determine if random elevations in blood pressure suggest the diagnosis of preeclampsia; a protein-to-creatinine ratio of greater than 0.19 is correlated with greater than or equal to 300 mg of protein in a 24-hour collection.\textsuperscript{18,19} Clinical studies addressing perinatal outcomes related to preeclampsia have assigned the diagnosis of preeclampsia to women with blood pressure measurements of more than 140/90 mm Hg on two occasions and 2+ protein or more (>100 mg/dL) on urinalysis on two separate occasions at least 4 hours apart without evidence of urinary tract infection. At this time there is no recommendation for replacement of the 24-hour urine collection as the definitive diagnostic test for the presence of significant proteinuria, but the spot urine protein-to-creatinine ratio may help screen patients who have a hypertensive disorder without proteinuria. Once the diagnosis of preeclampsia is made, there is no benefit to repeating the 24-hour urine collection because worsening proteinuria without other signs or symptoms of severe disease is not associated with adverse perinatal outcomes.

Preeclampsia is classified as mild or severe based on the blood pressure measurements, the degree of proteinuria, and evidence of laboratory abnormalities. The criteria are shown in Box 1.
In the past, the presence of edema (lower extremity, hands, face) was considered part of the diagnostic criteria for preeclampsia, but it has become discarded, as these symptoms are common in the third trimester and a significant number of women with preeclampsia do not develop edema.

The HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets) is considered a variant of severe preeclampsia but may present independently of hypertension and proteinuria. Patients may also exhibit only part of the syndrome, that is, low platelet count and transaminitis without evidence of hemolysis. DIC does not appear to coexist except in patients with abruptio placentae or subcapsular hematoma of the liver. Pregnancies complicated by HELLP are at risk for severe maternal and fetal complications; perinatal mortality has ranged from 7% to 60% and maternal mortality is high. Attempts to prolong the pregnancy by increasing plasma volume and correcting laboratory abnormalities using high-dose steroids have had limited success; most patients deteriorate within 1 week of initiation of conservative management. The general recommendation is immediate delivery, although delay for administration of betamethasone to promote fetal lung maturity is acceptable.

Patients who develop atypical preeclampsia with onset of proteinuria and edema (capillary leak syndrome) or multisystem organ dysfunction, without coexistent hypertension (Fig. 3) should be managed in a similar way to patients with typical severe preeclampsia.

Several conditions mimic severe preeclampsia/HELLP, including acute fatty liver of pregnancy, viral hepatitis, idiopathic thrombocytopenic purpura, thrombotic thrombocytopenic purpura (TTP), gallbladder disease, diabetes mellitus, pyelonephritis, and systemic lupus erythematosus. When patients present with multiorgan dysfunction, consultation with a perinatologist is very helpful, and these patients often require

<table>
<thead>
<tr>
<th>Box 1</th>
<th>Criteria for mild and severe preeclampsia</th>
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<tr>
<td><strong>Mild preeclampsia</strong></td>
<td></td>
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<tr>
<td>● SBP $\geq$ 140 mm Hg</td>
<td></td>
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<tr>
<td>● DBP $\geq$ 90 mm Hg</td>
<td></td>
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<tr>
<td>● Proteinuria $\geq$ 300 mg/24-hour urine collection</td>
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<tr>
<td><strong>Severe preeclampsia</strong></td>
<td></td>
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<tr>
<td>● SBP $\geq$ 160 mm Hg</td>
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<tr>
<td>● DBP $\geq$ 110 mm Hg</td>
<td></td>
</tr>
<tr>
<td>● Proteinuria $\geq$ 5000 mg/24-hour urine collection</td>
<td></td>
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<tr>
<td>● Oliguria &lt;400 mL urine output in 24 hours</td>
<td></td>
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<tr>
<td>● Cr $\geq$ 1.2</td>
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<tr>
<td>● Aminotransferases (ALT and AST) 2 times the upper limit of normal</td>
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<tr>
<td>● Thrombocytopenia &lt;100,000</td>
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<td>● Hemolysis</td>
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<td>● Epigastric pain, nausea, and vomiting</td>
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<td>● Pulmonary edema</td>
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<td>● Visual disturbance</td>
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Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; Cr, creatinine.
admission to the intensive care unit and may necessitate large-volume transfusion or plasmapheresis; these problems are often best managed in a tertiary care referral hospital.

Eclampsia is the presence of seizure activity in a patient with preeclampsia and no other known cause of seizure. Anyone who develops new-onset seizures during pregnancy should be presumed to have eclampsia, but the differential diagnosis of seizures in pregnancy includes cerebrovascular accidents, brain tumor, seizure disorder, drug ingestion, metabolic diseases, TTP, and reversible posterior leukoencephalopathy syndrome.

**Evaluation**

There is some evidence that the hypertension and symptoms associated with preeclampsia are late findings of a disorder that has been manifesting for days to weeks before diagnosis. Patients evaluated in the clinic with new-onset hypertension should undergo physical examination for signs or symptoms of preeclampsia. Laboratory evaluation for the evidence of severe disease should be ordered: complete blood count (CBC), blood urea nitrogen, creatinine, alanine aminotransferase, aspartate aminotransferase, uric acid, and L-lactate dehydrogenase. Patients should then complete a 24-hour urine collection for the diagnosis of proteinuria, differentiating gestational hypertension from preeclampsia and from atypical preeclampsia. Patients should be referred for immediate fetal testing to evaluate evidence of preterm labor, fetal distress, or oligohydramnios. Assessment of fetal growth should be obtained, as well as umbilical artery Doppler velocimetry to evaluate placental function.

Women with a diagnosis of mild preeclampsia at 37 weeks’ gestation or more can be considered for delivery. If patients are at less than 37 weeks’ gestation, most guidelines recommend delivery for evidence of severe disease, fetal growth restriction, oligohydramnios, or nonreassuring fetal testing.\(^{14}\)

Patients with severe preeclampsia well before term (28–32 weeks’ gestation) can be managed expectantly but require hospitalization and a daily assessment of blood pressure, laboratory values, amniotic fluid volume, and umbilical artery Doppler velocimetry. If less than 34 weeks’ gestation at the time of diagnosis, these patients should receive betamethasone to enhance fetal lung maturity given the high likelihood of

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**Fig. 3.** Overlapping role of hypertension, capillary leak, maternal symptoms, and fibrinolysis/hemolysis in the spectrum of atypical preeclampsia. CNS, central nervous system. (Adapted from Sibai BM, Stella CL. Diagnosis and management of atypical preeclampsia-eclampsia. Am J Obstet Gynecol 2009;200(5):481.e1–7; with permission.)
preterm delivery caused by the worsening disease. Patients with severe preeclampsia at greater than 34 weeks’ gestation should undergo delivery immediately.

**Intrapartum management**

**Mode of delivery** The diagnosis of preeclampsia does not require cesarean delivery, although patients with severe preeclampsia and compromised hepatic, renal, or hematologic function are best served by rapid delivery. Given the high risk of adverse maternal and fetal outcomes, many practitioners are uncomfortable with prolonged induction of labor in patients with severe disease and limit the time for cervical ripening to 24 hours. Correction of severe coagulopathy should be considered before cesarean delivery, and magnesium sulfate infusion can be halted before surgery and restarted in the immediate postpartum period. If concern exists for liver hemorrhage, alerting the available gynecologic oncologist or general surgeon/trauma surgeon in one’s facility is wise.

**Magnesium sulfate** Magnesium sulfate is used for seizure prophylaxis in patients with preeclampsia; its mechanism of action seems to be stabilization of the cerebrovascular endothelium and reduction of the cerebral perfusion pressure in patients with high baseline perfusion pressure. Magnesium sulfate therapy is associated with the risk of pulmonary edema and respiratory depression; it is particularly dangerous in patients with compromised renal function, as in severe preeclampsia. Although its efficacy is proven in patients with severe preeclampsia, its use as an anticonvulsant in patients with mild preeclampsia is controversial because of the high risk-to-benefit ratio. The dose of magnesium sulfate is 4 g IV loading dose followed by 1 to 2 g/h IV based on renal function. Strict evaluation of fluid status should be maintained; although a Foley catheter is not required, patients receiving magnesium therapy should not ambulate. Some practitioners advocate for fluid restriction to 1 L per day to prevent pulmonary edema from developing, particularly in patients receiving magnesium therapy. Care should be taken to avoid generating acute kidney injury from volume depletion, because most patients with preeclampsia suffer from a capillary leak syndrome and are typically total body volume overloaded with a larger volume of fluid in the extravascular space.

**Blood pressure control** Patients with severe preeclampsia are at risk for hemorrhagic stroke. This risk seems to be related primarily not only to SBP but also to DBP and mean arterial pressure (MAP). Patients with SBP of persistently more than 160 mm Hg should receive medication to gradually lower SBP and MAP. Patients with symptoms of cerebrovascular irritability (headache, vision changes, somnolence, confusion) should be treated at lower thresholds with an SBP of 150 mm Hg or more and a DBP of 95 to 100 mm Hg or more. The recommended antihypertensives for parenteral treatment are labetalol and hydralazine. Acute changes in blood pressure can result in fetal distress caused by decreased placental perfusion pressure, and fetal cardiac monitoring should be continuous when adjusting antihypertensives.

**Postpartum management**

If patients have received magnesium sulfate therapy for seizure prophylaxis intrapartum, this treatment should continue for 24 hours postpartum because 25% of eclamptic seizures occur during the first 24 to 48 hours postpartum. Women may require continued blood pressure control in the postpartum period. If the patient is breastfeeding, care should be taken to avoid medications contraindicated in the newborn. β-Blockers and combined α-β-blockers (metoprolol, atenolol, and labetalol) have the least transfer to maternal milk, and calcium-channel blockers (nifedipine,
diltiazem) are also acceptable for breastfeeding women. If patients remain persistently volume overloaded, they may benefit from diuretic administration (furosemide) to acutely lower blood pressure, but care should be avoided to prevent volume depletion, which can interfere with milk production.

Postpartum preeclampsia may be diagnosed in women who were previously normotensive and asymptomatic. Often these patients require readmission to the hospital for blood pressure control, and if there is evidence of severe hypertension, magnesium sulfate therapy for 24 hours for seizure prophylaxis is provided. Care should be taken to ensure accurate diagnosis in postpartum patients. For example, severe preeclampsia can manifest as pulmonary edema caused by capillary leak syndrome, but pulmonary edema may represent postpartum cardiomyopathy in which magnesium sulfate therapy is contraindicated. Neurologic signs may be symptomatic of severe preeclampsia but may also represent complications, such as reversible posterior leukoencephalopathy syndrome. Patients who present to the emergency department in the postpartum period with symptoms consistent with severe preeclampsia should receive consultation and guidance from the available obstetrics team.

**Eclampsia**

Eclampsia is presence of seizures in a patient without other neurologic issues, with signs and symptoms consistent with preeclampsia. Women with eclampsia may demonstrate markedly high blood pressure and severe laboratory abnormalities, or minimal blood pressure changes and no symptoms of neuroirritability before seizure activity. This range of presentation makes it complicated to predict those at the greatest risk for seizure. Eclamptic seizures may occur at any point during the pregnancy; 50% of seizures occur antepartum, 25% intrapartum, and 25% postpartum.

Eclamptic seizures are controlled with magnesium sulfate; the treatment is a loading dose of 6 g IV over 15 to 20 minutes followed by a maintenance rate of 2 g IV per hour. Serum magnesium level should be checked 4 hours after the loading dose. If patients continue to seize after administration of the loading dose, another dose of 2 g IV may be given. Benzodiazepines should not be administered, as they may contribute to decreased respiratory effort and risk of aspiration. Care should be instilled to avoid maternal injury (padded guard rails on bed, padded tongue blade to bedside). Laboratory evaluation should be performed to evaluate for metabolic acidosis and abnormalities related to severe preeclampsia or HELLP.

Fetal outcome is generally good after an eclamptic event, although placental abruption may occur after eclamptic seizure, and the fetus may be affected by transient hypoxia associated with convulsions. Patients with eclampsia who are less than 32 weeks’ gestation with an unfavorable cervix should undergo cesarean delivery; these infants are at risk for complications related to prematurity as well as possible growth restriction. Patients at greater than 32 weeks’ gestation are induced after stabilization, provided there are no fetal indications for emergent cesarean delivery. Continuous fetal monitoring during labor induction is essential. Maternal mortality from eclampsia is associated with antepartum seizure activity, fetal prematurity, and advanced maternal age. It is appropriate to consider transfer to a tertiary care center for management of the complicated fetal and maternal issues associated with eclampsia.

**Future complications**

Although most women with preeclampsia do not experience recurrent preeclampsia in future pregnancies, 15% develop preeclampsia in their second pregnancy. Women
with preeclampsia in 2 prior pregnancies have a recurrence risk of 30%.\textsuperscript{23,24} Women with a history of severe preeclampsia resulting in a preterm delivery are at greatest risk for recurrence, particularly if the prior pregnancy was complicated by abruptio placentae, and should receive frequent surveillance and fetal monitoring. Although several interventions, including low-dose aspirin, vitamin C, calcium, and magnesium, have been tested for the prevention of preeclampsia in patients at high risk of disease, there is no evidence that any of these treatments are effective in disease prevention.\textsuperscript{14}

Women with a history of preeclampsia are at risk for development of chronic hypertension, ischemic heart disease, and end-stage renal disease.\textsuperscript{24–26} Given that a woman’s obstetric history is an important component to her risk assessment for future cardiovascular and renal disease, it is important to ensure adequate documentation of pregnancy-related complications for effective counseling and evaluation by her primary care physician.

**Gestational Hypertension**

**Diagnostic criteria**

This diagnosis is assigned to women who develop hypertension after 20 weeks’ gestation without associated proteinuria or laboratory abnormalities. Women with blood pressure measurements of 140/90 mm Hg or more have mild gestational hypertension, and those with blood pressure of 160/110 mm Hg or more have severe gestational hypertension. Approximately 20% to 50% of patients with mild gestational hypertension develop preeclampsia during pregnancy.\textsuperscript{27,28} If laboratory abnormalities arise or if the woman develops symptoms consistent with preeclampsia (headache, vision changes, right upper quadrant discomfort) without proteinuria, the diagnosis of atypical preeclampsia should be considered.\textsuperscript{29}

**Management**

Blood pressure control should be initiated to prevent maternal stroke when SBPs are persistently 160 mm Hg or more and DBPs are 100 mm Hg or more. The target blood pressure is an SBP of 130 to 150 mm Hg and a DBP of 80 to 100 mm Hg. Patients with severe gestational hypertension require inpatient management.

Fetal monitoring, fetal growth assessment via US, and evaluation of umbilical artery Doppler velocimetry should be performed consistent with recommendations for evaluation and treatment of patients with preeclampsia, because the risk of adverse perinatal outcomes is similar. Patients with severe gestational hypertension (without proteinuria) have similar outcomes to patients with severe preeclampsia and worse outcomes than patients with mild preeclampsia, including increased risk of preterm delivery and fetal growth restriction (Table 1).\textsuperscript{30}

Given these risks, patients with mild gestational hypertension should undergo delivery after 37 weeks’ gestation or sooner if there is evidence of severe disease, fetal growth restriction, oligohydramnios, or abnormal umbilical artery Doppler results. Women with severe hypertension before 34 weeks’ gestation should be offered betamethasone for fetal lung maturity and, if less than 32 weeks’ gestation, magnesium sulfate for fetal neuroprotection. These patients often require hospital-based blood pressure management to titrate oral medications for adequate control. If blood pressure persists in the severe range despite maximization of oral medications, delivery is recommended. Otherwise patients with severe gestational hypertension should undergo delivery after 34 weeks, similar to management recommendations for severe preeclampsia.

Gestational hypertension is not a contraindication for vaginal delivery, and cesarean delivery should be reserved for maternal or fetal indications. Intrapartum blood
pressure control should follow the same recommendations as for patients with severe preeclampsia. There is no consensus as to whether patients with severe gestational hypertension require magnesium sulfate intrapartum for seizure prophylaxis.

If the hypertension resolves within 3 months postpartum, the patient retains the diagnosis of gestational hypertension. If her hypertension persists after the post-partum period, the patient is assigned the diagnosis of chronic hypertension.

THIRD-TRIMESTER VAGINAL BLEEDING

The differential diagnosis of vaginal bleeding in the third trimester includes labor (preterm and term), placenta previa, vasa previa, abruptio placentae, uterine rupture, and vaginal or cervical trauma. Given the significant consequences of each of these problems, patients should be encouraged to present for care for any sign of vaginal bleeding and should be assessed immediately by a physician.

The workup is as follows.

1. Determination of gestational age and prior obstetric history (ie, prior cesarean delivery or uterine surgery, history of abruption).
2. Vital signs (maternal and fetal). Hypertensive disorders are associated with abruptio placentae. Maternal hypotension and tachycardia may represent significant blood loss. Fetal bradycardia is concerning for abruptio placentae or uterine rupture, and frequent contractile activity is associated with both abruptio placentae and labor.
3. History. Traumatic injury in pregnancy, including minor falls or motor vehicle accidents, can provoke placental abruption. Patients with suspicious falls or injuries should be screened for intimate partner violence. Any signs or symptoms of the past several days that may reflect labor or preeclampsia should be reviewed. A history of prior uterine surgery conveys an increased risk of placenta previa.
5. Physical examination. If placenta previa suspected, do not perform vaginal examination. If there is no evidence of placenta previa, sterile speculum examination is

<table>
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<th>Table 1</th>
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<tr>
<td><strong>Relative risk of adverse outcomes (severe gestational hypertension vs severe preeclampsia)</strong></td>
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<tr>
<td><strong>Outcome</strong></td>
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<tr>
<td>Delivery at &lt;37 wk</td>
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<tr>
<td>Delivery at &lt;35 wk</td>
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<tr>
<td>SGA infant</td>
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<tr>
<td>Abruptio placentae</td>
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<tr>
<td>LGA infant</td>
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<tr>
<td>NICU admission</td>
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<tr>
<td>Respiratory distress syndrome</td>
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</table>

The severe gestational hypertension group is the reference group.

*Abbreviations: CI, confidence interval; LGA, large for gestational age; NICU, neonatal intensive care unit; SGA, small for gestational age.*

performed to determine the source of bleeding. Fetal fibronectin testing is invalid if a significant amount of blood is present or if the patient has had recent sexual intercourse. Evaluation for possible rupture of membranes is done; PPROM can provoke abruptio placentae. Speculum examination followed by sterile vaginal examination is done to examine for cervical changes consistent with labor.

6. Laboratory tests. Confirm maternal blood type and administer RhoGam, 300 µg for Rh-negative patients; Rh-negative women should also undergo Kleihauer-Betke testing to determine the degree of transplacental hemorrhage. CBC, blood type and screening, and coagulation studies should be collected if there is concern for significant blood loss. Tests for evaluation of severe preeclampsia are necessary if the patient is hypertensive. Anyone with new-onset hypertension and possible abruptio placentae should receive urine toxicology screening, to reveal any association with cocaine and amphetamine use.

Labor

Preterm labor with bleeding may represent abruptio placentae or a bloody show with cervical change. Cervical blood vessels may also become exposed and lacerated during labor. Patients with a cerclage in place who present with vaginal bleeding and suspicion of preterm labor may require removal of cerclage to prevent cervical injury from continued dilation. If preterm labor is complicated by extensive bleeding, forestalling labor with tocolytics to allow steroid administration may not be possible. Assessment of quantity and quality of vaginal bleeding every hour is recommended.

Placenta Previa

Placenta previa, commonly diagnosed during routine US in the second trimester, refers to a placenta that completely or partially obstructs the internal cervical os, and complicates approximately 0.5% of all pregnancies. Transvaginal US is superior to abdominal US in characterizing the position of the placenta in relation to the cervix, and transvaginal US does not increase the risk for vaginal bleeding.31 Risk factors for placenta previa include prior uterine surgery (including termination of pregnancy and myomectomy), smoking, cocaine, and advanced maternal age. The frequency of placenta previa increases with each subsequent cesarean delivery; the relative risk of previa is 4.5 with 1 prior cesarean delivery and increases to 45% with 4 prior cesarean deliveries. The risk of abnormal placental invasion (accreta, increta, percreta) increases dramatically with the presence of previa and increasing number of prior cesarean deliveries; 11% of patients with placenta previa and 1 prior cesarean delivery have placenta accreta, whereas 61% of patients with placenta previa and 3 prior cesarean deliveries have placenta accreta.31 US is the best way to determine the presence of accreta in patients with placenta previa.

The classic presentation of placenta previa is painless vaginal bleeding, but patients may develop painful contractions or experience no bleeding for the entire pregnancy. Patients with placenta previa and acute bleeding should be hospitalized and observed; it is reasonable to place 1 to 2 large-bore IV catheters and obtain CBC, and blood type and screen. Women between 24 and 34 weeks’ gestation with bleeding should receive betamethasone to promote fetal lung maturity. Uterine contractions in patients with placenta previa can provoke continued bleeding, and several studies suggest that tocolytics may be of benefit in prolonging pregnancy in preterm patients.30 If bleeding ceases, outpatient management may be acceptable if the patient has excellent family support and transportation and lives close to the hospital. If these things cannot be assured, the patient is best served by inpatient
management for the remainder of the pregnancy. In patients with placenta previa, transvaginal sonogram is repeated in the third trimester; cesarean delivery is recommended for all patients if the placental edge is less than 2 cm from the internal os.\textsuperscript{31} Cesarean delivery under regional anesthesia is typically scheduled at 37 weeks’ gestation. Delivery may be complicated by excessive bleeding, and 2 units of packed red blood cells should be available at the time of surgery.

Patients with known placenta accreta have the same risks of bleeding in the third trimester but require cesarean hysterectomy at the time of delivery. Thus, obstetric care should be managed in a hospital with appropriate surgical and pediatric staff and in a facility with a blood bank that can manage large volume resuscitation.\textsuperscript{32} Patients with placenta accreta may have evidence of placenta percreta at the time of delivery; the most commonly affected organ is the bladder, and patients may require partial bladder resection at the time of hysterectomy. Surgical planning with a multidisciplinary team, including the surgical staff of obstetrics and gynecology, urology, and vascular surgery, as well as the staff from anesthesia, neonatology, interventional radiology, and the blood bank, is recommended.

**Vasa Previa**

Vasa previa is a condition that occurs when the fetal vessels course through the amniotic sac and over the cervix, without the protection of the placenta. Vasa previa can result from a velamentous cord insertion or from vessels traversing separate placental lobes. Vasa previa is seen in approximately 1 in 2500 deliveries. Risk factors include multiple pregnancies and resolved low-lying placentas, as well as pregnancies known to have multiple placental lobes. Diagnosis of vasa previa can be made antepartum by identifying the placental cord insertion. Significant consequences can result following amniotomy, including heavy vaginal bleeding and fetal distress. Emergent delivery is often necessary. Because of the risk of fetal exsanguination following rupture of membranes, many patients with vasa previa are monitored as inpatients through the 35th week and delivered by cesarean delivery at 36 weeks without preoperative amniocentesis (to avoid provoking spontaneous rupture of membranes).\textsuperscript{31}

**Abruptio Placentae**

Placental abruption is the premature separation of the placenta from the uterus and complicates 1% of all deliveries. The consequences of abruption can be severe; complete abruption can result in massive obstetric hemorrhage with DIC and renal failure. Large placental separation deprives the fetus of adequate oxygenation and can result in fetal injury or death. This is a particularly severe problem in preterm gestations. Risk factors for abruption include prior pregnancy complicated by abruption, hypertensive disorders, trauma, cocaine and methamphetamine use, and premature rupture of membranes. Fetal growth restriction and oligohydramnios also pose a risk of abruption.

Clinical presentation of abruption ranges from absence of signs or symptoms to complete hypovolemic shock with DIC and fetal death. Patients may have severe preeclampsia, which is masked because of hypotension and volume depletion and only identified after resuscitation. Management depends on gestational age and maternal/fetal well-being. If fetal death has occurred, patients can be offered vaginal delivery with intensive monitoring for evidence of hemorrhage or oliguria. Cesarean delivery may be considered if maternal condition worsens or if labor arrests, but any coagulopathy should be corrected before surgery to minimize further blood loss. Women with live fetuses greater than 34 weeks’ gestation should be delivered immediately, and if less than 34 weeks’ gestation and maternal/fetal well-being has been
established, patients may receive steroids and tocolytics and be expectantly managed. Women who have experienced trauma and are at greater than 24 weeks’ gestation should be monitored for 8 hours; if there are persistent contractions on tocol-ometry or if vaginal bleeding is evident, continued monitoring should be performed. If fetal well-being cannot be assured, patients should be delivered.

**Cervical/Vaginal Trauma**

Patients may present with cervical bleeding following intercourse; evaluation for cervicitis or a cervical lesion is important. Although cervical cancer is the most commonly identified gynecologic cancer in pregnancy, it remains a rare finding, with an incidence of 1 in 1200 to 10,000 pregnancies. Sexual trauma may result in vaginal lacerations; all patients with evidence of vaginal or vulvar trauma should be screened for intimate partner violence. Effective evaluation and repair of any traumatic injury may require examination under anesthesia.

**REFERENCES**


Complications in Late Pregnancy

David Meguerdichian, MD\textsuperscript{a,b,*}

KEYWORDS
- Late pregnancy
- Placenta previa
- Abruptio placenta
- Amniotic fluid embolus
- Preterm pregnancy
- Braxton Hicks contractions
- Preterm premature rupture of membranes
- Uterine rupture

KEY POINTS
- Managing complications of late pregnancy requires prompt maternal and fetal evaluation as well as an understanding of hospital protocols so patients can have appropriate triage and treatment.
- Abruptio placentae classically presents as painful, third-trimester bleeding in contrast to the often painless bleeding of placenta previa.
- Ultrasound imaging is most useful in ruling out placenta previa and should not delay care in unstable third-trimester bleeding patients.
- In the intrapartum or early postpartum period, evidence of hypotension, respiratory distress, or disseminated intravascular coagulation suggests amniotic fluid embolism and requires aggressive resuscitative measures.
- Corticosteroid administration is the most beneficial antenatal intervention in preterm labor.
- Uterine rupture should be suspected in patients with a history of uterine surgery who present with abdominal pain, vaginal bleeding, and cessation of uterine contractions.
- Appropriate management of late pregnancy complications requires prompt treatment of hemodynamic instability, evaluation of maternal and fetal welfare, and early obstetric consultation.

INTRODUCTION

Complications of late pregnancy are seen with less frequency in the emergency department (ED) than those of the first trimester (eg, spontaneous abortion and ectopic pregnancy). In many hospitals, the lower frequency is the result of an institutional policy that triages many of these patients directly to the labor and delivery unit. Nonetheless, emergency medicine (EM) physicians must be comfortable in identifying...
and addressing these complications, given the profound impact they can have on maternal and fetal health. The emergencies of late pregnancy include abruptio placentae, placenta previa, amniotic fluid embolism, preterm labor, preterm premature rupture of membranes, and uterine rupture. This article discusses the clinical presentations, the risk factors, and the most current diagnostic approaches and management strategies for the key complications that EM physicians will encounter in late-pregnancy patients.

**ABRUPTIO PLACENTAE**

*Causes and Risk Factors*

Abruptio placentae is the separation of an implanted placenta after the twentieth week of pregnancy and before actual delivery.\(^1,2\) The separation can be complete or partial, with bleeding into the decidua basalis.\(^1\) Bleeding can remain concealed and undetected or track between the membranes, subsequently escaping through the cervix.\(^3\) This hemorrhage causes compression of the intervillous space and ultimately placental tissue damage.\(^2\) Mechanisms for abruption seem to be multifactorial and include impaired placentation, poor perfusion/hypoxia to the uteroplacental interface, and placental insufficiency.\(^4,5\) The symptoms of abruption range from none to severe hemorrhage with subsequent fetal death and maternal morbidity, making this diagnosis challenging for the EM physician. Risk factors associated with abruptio placentae are listed in Box 1.\(^2,6\)

**Clinical Presentation**

The most common presenting symptoms of placental abruption are vaginal bleeding, uterine contractions, abdominal pain, and uterine tenderness.\(^4\) Vaginal bleeding occurs in 80% of cases; the blood is usually dark, and the amount correlates poorly with the degree of abruption.\(^1\) Other symptoms may include back pain, nausea, vomiting, and reduced fetal movements. The maternal coagulation cascade can be activated at any time during this process and result in disseminated intravascular coagulation (DIC).\(^7\) In severe abruptio placentae, the uterus becomes severely contracted and painful, maternal hypotension ensues, and fetal death is possible.\(^2\)

<table>
<thead>
<tr>
<th>Risk factors associated with abruptio placentae</th>
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<tr>
<td>- History of abruptio placentae</td>
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<tr>
<td>- Maternal hypertensive disease (preeclampsia, chronic hypertension)</td>
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<td>- Cigarette smoking</td>
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<td>- Cocaine/vasoconstrictive drug use</td>
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<td>- Abdominal trauma</td>
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<td>- Multiple pregnancies</td>
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<td>- Chorioamnionitis</td>
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<td>- Placental abnormalities</td>
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<td>- Folate deficiencies</td>
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<td>- Premature rupture of membranes</td>
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<td>- Hyperhomocysteinemia</td>
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<td>- Thrombophilia</td>
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Cardiotocogram monitoring can demonstrate a variety of heart rhythms and contractions when abruptio placentae is present. Classically, the uterine contractions have a high-frequency, low-amplitude pattern with an elevated baseline tone. Fetal heart rates can show recurrent late or variable decelerations, bradycardia, or sinusoidal patterns.\(^2,7\)

**Classification**

Abruptio placentae has been categorized into 3 grades that describe the clinical findings\(^1,8\):

- Grade 1 (40% of cases): slight vaginal bleeding, uterine irritability, no maternal distress, no fetal distress
- Grade 2 (45% of cases): mild to moderate vaginal bleeding, uterine contractions, little maternal distress, presence of fetal distress
- Grade 3 (15% of cases): severe vaginal bleeding; painful, tetanic uterus; maternal hypotension and coagulopathy; fetal distress and, often, death

**Diagnosis**

**Clinical**

The diagnosis of abruptio placentae is a clinical one and should be considered in any late-pregnancy patient presenting to the ED with vaginal bleeding, abdominal pain, recent trauma, or signs of preterm labor. In moderate to severe cases, the diagnosis is often clear and made before delivery; in mild cases, the diagnosis is delayed until after delivery and based on the evaluation of a retroplacental clot.\(^2\) The main alternative diagnosis in women with late-pregnancy bleeding is placenta previa. The alternative considerations are clearly broader for late-pregnancy women who present with abdominal pain and include those listed below:

- Premature labor
- Complications of preeclampsia
- Hepatic and gallbladder disease
- Appendicitis
- Ovarian torsion

**Ultrasound**

Similar to its clinical presentation, abruptio placentae has a variety of ultrasonographic appearances. Factors, such as size of the bleed, location of the bleed, and duration between the abruption and time to evaluation, play a part in how well the abruption can be observed.\(^7,9\) Small and acute bleeds are difficult to appreciate because they can be isoechoic in comparison with the placenta; larger, concealed bleeds are usually more easily visualized.\(^10\) When a clot is identified on the ultrasound image, this finding has an 88% positive predictive value for abruption (Fig. 1).\(^10\) Ultrasound is a tool for monitoring expectant cases and excluding placenta previa, but it should never be used to examine unstable patients because it can delay definitive care.\(^2\)

**Kleihauer-Betke test**

The Kleihauer-Betke test is performed frequently but shows limited usefulness in the diagnosis of abruptio placentae. A retrospective cohort study using this test found no positive tests among placentas later identified as having undergone abruption on pathologic review.\(^11\) Thus, this test cannot be used to confirm or rule out abruption. The Kleihauer-Betke test is better applied for quantifying fetomaternal transfusion to guide the administration of Rh-immune globulin to an Rh-negative mother.\(^7\)
Treatment

The treatment strategies for abruptio placentae rely heavily on the presentation and state of the mother and fetus. Many institutions have protocols for triaging and evaluating obstetrics patients either in the ED or in the labor and delivery suite. The EM physician should use these protocols to guide initial management. If no obstetric services are available in the institution, the patient’s own obstetrician should be involved promptly. When the diagnosis is uncertain and the presentation is mild, the treating physician can rely on expectant management. At the other end of the spectrum, severe abruptio placentae calls for immediate hemodynamic stabilization of the mother and delivery of the baby.\(^2\) Overall, the care of abruptio placentae must be tailored on a case-by-case basis.

Stable mother and fetus

The initial evaluation of stable patients with abruption begins by placing them in a monitored bed and obtaining intravenous (IV) access with 2 large-bore catheters. If patients are at or near full term with imminent delivery, vaginal delivery is preferred if it can be tolerated by the mother and fetus. This delivery should occur in a setting capable of progressing to cesarean section. For mothers who are preterm and medically stable, expectant management is safe and appropriate.\(^12–14\) In fact, a retrospective study of 131 patients with abruption demonstrated that tocolysis is safe for use in preterm abruptio placentae.\(^12\) In all cases, the mother and fetus must be monitored and reevaluated continuously because a stable abruption can progress to an unstable abruption, causing patients to deteriorate rapidly.

Unstable mother or fetus

As with any unstable patient presenting to the ED, the initial management of patients with severe abruptio placentae involves placing them in a monitored bed, obtaining IV access with 2 large-bore catheters, and performing a primary survey. On their arrival in the ED, a complete blood count should be requested and a blood-bank specimen should be collected for type and crossmatch, coagulation studies, and Rh testing. Patients who are hypotensive should be treated initially with IV normal saline and transitioned to packed red blood cells if they are unresponsive to this initial intervention. Once the mother stabilizes, the fetus can be evaluated with ultrasound imaging to

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**Fig. 1.** Ultrasound image showing acute placental abruption with a retroplacental hematoma (arrow) lifting part of the placenta. (Courtesy of Carol Benson, MD, Brigham and Women’s Hospital Department of Radiology.)
rule out placenta previa and monitor the heart rate.\textsuperscript{2} If the fetus is viable, delivery should be expedited in the operating room with a double setup for delivery. Vaginal delivery is acceptable until fetal monitoring shows signs of distress or maternal surveillance shows evidence of deterioration.\textsuperscript{2,7} At this point, cesarean delivery should be performed because complete placental detachment is imminent and could occur at any time.\textsuperscript{7} Meticulous monitoring should be performed at all times for signs of hemorrhagic shock and DIC, which are 2 complications commonly associated with abruptio placentae. Hemorrhagic shock should be addressed with transfusion aimed at restoring appropriate circulating blood volume. DIC is more commonly seen after fetal death, and its management involves treating the underlying condition with delivery of the fetus and placenta.\textsuperscript{2,15} Vaginal delivery is the route of choice in such cases, but cesarean section might be indicated for maternal reasons and can be performed once DIC is reversed.\textsuperscript{2,7} Resolution of coagulopathy is rapid following delivery but can be augmented with the addition of fresh frozen plasma (FFP).

Placental abruption continues to be a key cause of perinatal morbidity and mortality. Prompt recognition of this condition is vital for the well-being of the fetus and the mother. Expectant management is appropriate in preterm cases with a stable patient and fetus, whereas emergent delivery is key to management for mature term or unstable cases.

**PLACENTA PREVIA**

*Causes and Risk Factors*

Placenta previa is the implantation of the placenta overlying or within 2 cm of the internal cervical os.\textsuperscript{16,17} In placenta previa, the placenta implants partially or fully in the lower uterine segment, as opposed to the upper uterine segment, as in normal pregnancy.\textsuperscript{18} Placenta previa has been classified into 4 categories based on the placenta’s location:\textsuperscript{18}

- Complete: placenta completely covers the internal os
- Partial: placenta partially covers the internal os
- Marginal: placenta just reaches but does not cover the internal os
- Low lying: placenta extends into the lower uterine segment, with no approximation to the internal os

Placenta previa occurs in 0.3% to 0.5% of pregnancies.\textsuperscript{19,20} The exact cause of implantation of the placenta in the lower uterine segment is unclear. Uterine scarring is one proposed predisposing factor.\textsuperscript{18} In many cases, low-implanting placetas move away from the cervix and toward the better-vascularized fundus as the pregnancy progresses.\textsuperscript{18} Factors that seem to increase the risk of placenta previa are listed in Box 2.\textsuperscript{19,21}

**Box 2**

<table>
<thead>
<tr>
<th>Risk factors for placenta previa</th>
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<tbody>
<tr>
<td>Advanced maternal age</td>
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<tr>
<td>Multiparity</td>
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<tr>
<td>Cigarette smoking</td>
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<tr>
<td>Previous cesarean sections</td>
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<tr>
<td>Chronic hypertension</td>
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<td>Multiple gestations</td>
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<td>Uterine surgery</td>
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**Clinical Presentation**

Placenta previa is a common incidental finding seen in 4% of regular second-trimester ultrasound studies and only 0.4% by the time all pregnancies reach term.\textsuperscript{19,21–23} In contrast to the usual painful presentation of abruptio placentae, symptomatic placenta previa is characterized by painless, bright-red vaginal bleeding at or after the end of the second trimester.\textsuperscript{18} The initial sentinel bleed rarely causes hemodynamic instability unless the placenta is further disrupted by cervical instrumentation or digital examination, which can cause severe hemorrhage.\textsuperscript{17} Despite being classified as painless, placenta previa bleeding can occur in women in active labor who are experiencing pain from contractions.

**Diagnosis**

Ultrasound is the diagnostic modality of choice for localizing and identifying placenta previa. Transvaginal ultrasonography is the most accurate means of visualizing 2 key structures, the placenta and cervical os, which are often obscured on the transabdominal approach by the fetus and the maternal bony pelvis.\textsuperscript{22} In one study, crucial landmarks were identified poorly on 50% of transabdominal ultrasound images; this series also showed 26% fewer cases of placenta previa when a transvaginal approach was used in suspected cases.\textsuperscript{24} This approach, when performed by properly trained personnel, is safe and does not lead to an increased risk of bleeding.\textsuperscript{25,26}

**Treatment**

Placenta previa must be considered in all women in late pregnancy presenting to the ED with vaginal bleeding. The initial assessment includes maternal evaluation, with emphasis on identifying abnormal vital signs, performing a primary survey, and establishing IV access with 2 large-bore catheters. A complete blood count, type and cross-match, Rh-status study, and coagulation studies should be requested. Continuous fetal monitoring should also be prioritized during this initial management period. An ultrasound study can be performed to locate the placenta and diagnose placenta previa.

If hypotension or severe hemorrhage is identified, the mother and fetus can be resuscitated initially with IV normal saline boluses and ultimately with crossmatched packed red blood cells, as for abruptio placentae. Once the mother is stabilized, she should be transferred to the obstetric unit for further management; if this resource is not available, she should be transferred, accompanied by a high-acuity transfer team, to an appropriate receiving hospital.

Following stabilization of the patient, the goal of the EM physician is to prolong pregnancy until the fetal lungs mature.\textsuperscript{17} Under the guidance of an obstetrician, various strategies can be used to achieve this goal. Tocolytic agents safely prolong pregnancy in patients with vaginal bleeding and preterm contractions.\textsuperscript{27} Administration of corticosteroids can be considered to promote lung maturation in the fetus of women who present with placenta previa bleeding at 24 to 34 weeks’ gestation.\textsuperscript{28,29} These patients should have a neonatology consultation to better understand the management of the child following premature birth.\textsuperscript{18}

Cervical cerclage has been proposed as an intervention for prolonging pregnancy in patients with placenta previa. This intervention has shown mixed results; one small study showed improved outcomes, including mean birth weight and greater gestational age at delivery, whereas another small study showed no statistically significant difference between the outcomes in the cerclage group versus controls.\textsuperscript{30,31}
Cochrane meta-analysis demonstrated that cerclage may lessen the risk of delivery before 34 weeks, yet the investigators argued for further studies before recommending that this practice be made the standard of care.32

Following any or all of these interventions, patients should be admitted for close monitoring to allow fetal maturation and a safe, successful delivery. Women who are stable, asymptomatic, and reliable and who can rapidly return to labor and delivery can be managed as outpatients at the discretion of their obstetricians.18

**AMNIOTIC FLUID EMBOLISM**

*Causes and Risk Factors*

Amniotic fluid embolism (AFE) is a rare, catastrophic, and often fatal complication of late pregnancy. The pathogenesis and disease process remain poorly understood, even though AFE is one of the main causes of maternal mortality in the United States.33 The pathogenesis of AFE is thought to be the introduction of amniotic fluid and debris into the systemic maternal circulation, with resultant physical obstruction in the pulmonary vasculature. However, autopsy studies and the inability to reproduce the disease in animal models have sparked other theories to explain the mechanism for this maternal complication.34 Some investigators have proposed that the hemodynamic manifestation of AFE is comparable with that of anaphylactic shock and hypothesized that an immunologic mechanism drives the process.35–38 More recently, complement activation has been proposed as playing a role in the pathogenesis of AFE, with more promising serologic and histologic evidence than prior hypotheses.35,38,39 Despite the lack of a definitive understanding, it does seem more convincing that the introduction of amniotic fluid into the maternal circulation results in the release of several endogenous mediators that trigger the physiologic presentation of this disease.34 Risk factors for AFE were elucidated in 2 recent retrospective studies (Box 3).40,41

**Clinical Presentation**

AFE usually occurs intrapartum or during the initial postpartum period. The symptoms are typically sudden in onset and can occur as late as 48 hours after delivery.34,42 The most common signs and symptoms of AFE are the following34,43:

- Hypotension
- Fetal distress
- Pulmonary edema/respiratory distress
- Cardiac arrest

**Box 3**

*Risk factors for amniotic fluid embolism*

- Maternal age more than 35 years
- Cesarean delivery
- Forceps or vacuum-assisted delivery
- Abruptio placentae
- Placenta previa
- Eclampsia
- Fetal distress
Cyanosis
Coagulopathy/DIC

Initially, patients experience the release of vasoactive substances, causing vaso-
spasm, pulmonary hypertension, and subsequent cardiovascular collapse. Surviv-
ors of this initial offense are usually confronted with the challenges of DIC, left
ventricular dysfunction, and pulmonary edema. Maternal death from AFE is typi-
cally caused by acute cardiac arrest, severe hemorrhage from DIC, or the develop-
ment of acute respiratory distress syndrome with subsequent multiorgan failure
following survival of the initial event.

Diagnosis
The diagnosis of AFE is based on the clinical presentation and should be suspected
in women displaying hypotension, respiratory distress, or evidence of DIC during the
intrapartum period or the first 48 hours after delivery. The diagnosis is essentially
made by excluding other medical conditions or explanations of patients’ symptoms
and is usually made with certainty only at autopsy. Intravascular fetal material, tryp-
tase, and complement can be elevated in patients with AFE. These diagnostic
markers could be promising, but further investigation is required. General laboratory
and imaging studies are nonspecific but should be used by the EM physician to
guide ED management of these critically ill patients. These studies include a complete
blood count, coagulation studies, arterial blood gas measurements, cardiac markers,
an electrocardiogram, a chest radiograph, and possible bedside echocardiography.

Treatment
Proper management of AFE requires early recognition and prompt initiation of neces-
sary supportive and resuscitative measures. The initial treatment involves identifying
abnormal vital signs, commencing cardiorespiratory monitoring, and rapidly perform-
ing a primary survey.

Following the primary evaluation, treatment of AFE should focus on airway control,
hemodynamic stability, and correction of coagulopathy. Control of the airway from
the onset is key because some form of hypoxia is present with AFE. Early tracheal
intubation with 100% oxygen administration and positive-pressure ventilation should
be achieved without delay. Two large-bore IV catheters should be placed to allow
rapid resuscitation with normal saline and packed red blood cells if hypotension or
severe bleeding ensues. Vasopressors, such as dopamine and norepinephrine, and
inotropes, such as dobutamine and milrinone, can be used to address refractory
hypotension, with a goal systolic blood pressure of greater than 90 mm Hg. Progress-
sion to vasopressors should prompt central venous catheter placement, which also
allows closer central pressure monitoring and sampling. Other blood products, such
as FFP, platelets, and cryoprecipitate, should be available for administration if
coagulopathy develops. The EM physician’s main goal, as with all cases of cardio-
pulmonary collapse, should be rapid protection of the airway, correction of hemody-
amic instability, and prompt admission to the intensive care unit for further monitoring
and management.

FALSE LABOR/BRAXTON HICKS CONTRACTIONS
Braxton Hicks contractions, named after the physician who first described this
phenomenon in 1872, are classified as irregular, uncoordinated, painful, or painless
contractions, which can be easily confused with true labor. After 30 weeks, the
intensity of these contractions can increase and patients may describe a greater
firmness in their lower abdomen. Contrary to true labor, these contractions are not associated with demonstrable cervical effacement and dilatation.\textsuperscript{46}

**Diagnosis**

False labor is usually a clinical diagnosis. Examination of the cervix, with care to not disrupt the intact membrane, will show minimal dilation.\textsuperscript{47} External tocometric monitoring can be performed and will show no increase in the frequency or duration of the uterine contractions, as is seen in true labor.\textsuperscript{47}

**Treatment**

Braxton Hicks contractions are typically managed with hydration, bed rest, and analgesia as needed. An obstetrician can be consulted when the distinction between true and false labor remains unclear.

**PRETERM LABOR**

**Causes and Risk Factors**

Preterm labor (PTL) is defined by the World Health Organization as the onset of delivery at or before 37 weeks’ gestation.\textsuperscript{48,49} Preterm delivery occurs in roughly 12% of all live births in the United States and remains a major cause of perinatal morbidity and mortality.\textsuperscript{50,51} Because of the often-unexpected nature of PTL, the EM physician should be comfortable in evaluating and managing patients with this condition in the ED.

The causes of PTL are multifactorial and closely tied to several clinical factors\textsuperscript{49,52}:

- Psychosocial factors
  - Low socioeconomic status
  - Extremes of age (<18 or >40 years)
  - Tobacco use
  - Substance abuse (cocaine)
  - Nonwhite race
- Maternal reproductive and gynecologic factors
  - History of preterm labor
  - Low pregnancy weight
  - Prior second-trimester abortion
  - Multiple gestations
  - Uterine anomalies
  - Cervical incompetence
  - History of placental abruption or previa
- Infections of the genital and urinary tract

**Clinical Presentation**

Understanding and identifying the signs and symptoms of PTL will allow the EM physician to diagnose and treat this condition expeditiously. Typical presentations include uterine activity with frequent contractions of more than 4 per hour, cramping abdominal pain, pelvic pressure, an increase or change in vaginal discharge, back pain, and vaginal bleeding.\textsuperscript{52,53}

**Diagnosis**

PTL can be diagnosed on clinical grounds when patients are between 20 and 36 weeks’ gestation and demonstrates the following\textsuperscript{48}:

- Painful contractions lasting longer than 30 seconds and occurring at least 4 times every 20 minutes
- A change in the position, length, or dilation of the cervix, usually with effacement of at least 80% and dilation greater than 2 cm

Useful ED studies include a complete blood count, urinalysis, and pelvic ultrasonography. Emergent ultrasonography, which has been shown to be far superior to the vaginal digital examination, can allow assessment for cervical shortening—a finding that places patients at a higher risk for preterm delivery.\(^{54-56}\) Aside from assessing cervical length, ultrasound examination can identify fetal anomalies, fetal presentation, placenta previa, abruptio placentae, or fetal demise, giving the obstetrics team information that can be used to counsel patients on expected outcomes.\(^{57}\) A growing body of obstetrics literature is showing that the finding of fetal fibronectin in cervical or vaginal fluid augments imaging in identifying patients at risk for PTL.\(^{58-60}\) All of the aforementioned studies can be done in the ED or labor and delivery, depending on hospital protocols and urgency of delivery.

**Treatment**

In the presence of a viable fetus and healthy mother, the management of preterm labor relies on several medical modalities. The hallmarks of management are the use of tocolytics to prolong pregnancy and the administration of medications to promote fetal maturity.

**Tocolytic therapy**

The primary aim of tocolytic therapy is to arrest premature labor and imminent delivery for up to 48 hours.\(^{61}\) Tocolytic medications should be administered in the ED in consultation with an obstetrician. Ultimately, a delay in delivery is sought to allow increased time for 2 key steps\(^{48,52,61}\):

- Administration of a complete course of antenatal glucocorticosteroids aimed at promoting fetal lung maturity
- Transfer to a tertiary obstetrics and pediatric center or unit that is capable of managing preterm labor and a premature infant

Several contraindications to tocolysis exist and should be considered before initiating therapy\(^{52}\):

- Fetal distress
- Preeclampsia or eclampsia
- Fetal demise
- Chorioamnionitis
- DIC
- Acute vaginal bleeding
- Preterm premature rupture of membranes

Evidence supports the use of β-adrenergic receptor agonists, calcium channel blockers, magnesium sulfate, and nonsteroidal antiinflammatory drugs (NSAIDs) as first-line agents for tocolysis.\(^{61}\) A recent meta-analysis showed benefit of these agents versus placebo alone for delaying delivery but also showed that NSAIDs might be the superior first-line agent because of their tolerability and fewer maternal/fetal side effects.\(^{62}\) The side-effect profiles of each agent (Box 4) should be reviewed and discussed with the consulting obstetrician before initiating therapy.\(^{52,61}\)

Titratable IV tocolytics should be administered first, while coordinating transfer to a labor and delivery unit, to identify the dose necessary for uterine contraction cessation. It is critical that every patient undergoing tocolytic therapy have external fetal heart monitoring to ensure prompt identification of fetal distress.
Corticosteroid therapy

Antenatal corticosteroid administration is the most beneficial intervention for improved neonatal outcomes in patients who deliver preterm.\textsuperscript{48,52,61} This intervention is beneficial regardless of the maternal membrane status. Its use results in lower severity and frequency of respiratory distress syndrome, intracranial hemorrhage, necrotizing enterocolitis, and death compared with those not receiving therapy.\textsuperscript{29} The most commonly used and studied corticosteroids are betamethasone and dexamethasone, administered via either of the 2 regimens listed here\textsuperscript{63}:

- Two 12-mg doses of betamethasone given intramuscularly 24 hours apart
- Four 6-mg doses of dexamethasone given intramuscularly every 12 hours

Antibiotics

Several maternal infections have been implicated as having a role in preterm labor, specifically at less than 32 weeks.\textsuperscript{61} Treatment with antibiotics has shown no benefit in prolonging pregnancy or avoiding preterm delivery.\textsuperscript{61} Nonetheless, women should be treated for sexually transmitted, urinary tract, respiratory, and vaginal infections if they are identified on testing. Group B streptococci screening should be performed, and positive or high-risk mothers should be treated with penicillin G (5 million units IV followed by 2.5 million units every 4 hours until delivery) to prevent vertical transmission of this infection to the newborn. In cases of preterm labor with rupture of membranes, a 48-hour course of ampicillin and erythromycin, followed by 5 days of amoxicillin and erythromycin, should be used to prolong pregnancy and reduce the risk of neonatal morbidity from infection transmission.\textsuperscript{64}
PRETERM PREMATURE RUPTURE OF MEMBRANES

Causes and Risk Factors

Premature rupture of membranes (PROM) is the rupture of the amniotic membrane before the onset of labor. This outcome is classified as preterm PROM (PPROM) if it occurs before 37 weeks’ gestation. PROM complicates close to 8% of pregnancies. At term, normal weakening of the membranes is caused by physiologic changes and the force of uterine contractions. The weakening seen with PROM has been associated with the risk factors listed in Box 5. However, PROM can occur even in patients with none of the identified risk factors.

Diagnosis

Rupture of membranes typically presents as a large release of clear vaginal fluid or a regular trickle. From the outset, PROM can be diagnosed with an accurate history and physical examination. Special care must be taken with the physical examination to avoid the introduction of infection. A digital cervical examination should be avoided in particular because it provides little information over a speculum examination and raises the risk of infection or rupture of intact membranes. With a sterile speculum examination, the ED physician can diagnose membrane rupture by visualizing the passage of fluid from the cervix. If in doubt, the additional diagnostic strategies listed here can be used:

- pH measurement (Amniotic fluid has a basic pH of 7.1–7.3 compared with the more acidic vaginal secretions.)
- Applying posterior fornix fluid to a microscope slide and observing ferning under a microscope

Management

The initial evaluation of patients presenting to the ED with PROM requires an accurate determination of gestational age, an assessment of maternal/fetal risk factors, and prompt evaluation and counseling of patients in concert with their obstetrician. If gestational age is uncertain from the menstrual history or previous imaging, an ED ultrasound study can be performed to provide an estimate of gestational age. All patients with PROM should be evaluated for infection in similar fashion to patients

<table>
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<th>Box 5</th>
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<tr>
<td>Risk factors for PROM</td>
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<tr>
<td>• Intra-amniotic infection</td>
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<tr>
<td>• Poor socioeconomic status</td>
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<tr>
<td>• Late pregnancy bleeding</td>
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<tr>
<td>• Low body mass index</td>
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<tr>
<td>• Tobacco use</td>
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<td>• Amniocentesis</td>
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<td>• Copper and ascorbic acid nutritional deficiencies</td>
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<td>• Connective tissue disorders</td>
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<tr>
<td>• Previous preterm birth</td>
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<td>• Short cervical length</td>
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in preterm labor. The incidence of infection in PROM increases with decreased gestational age; 13% to 60% of patients with PPROM have clinically evident intra-amniotic infection. Testing for *Chlamydia*, *Neisseria gonorrhoeae*, urinary tract infections, and group B streptococci should be requested and, if present, treated to avoid the onset of overt infectious symptoms and to prevent vertical transmission to the fetus. Electronic fetal heart rate monitoring should be initiated to assess for occult umbilical cord compression and fetal distress.

Term patients with PROM generally experience the prompt onset of labor and delivery. Oxytocin induction can be considered to decrease the period between PROM and delivery as well as the frequency of chorioamnionitis. Patients with PPROM, similar to PTL management, require transfer or admission to an obstetrics unit to initiate preterm treatments, including the following:

- Antenatal glucocorticoid therapy to promote fetal lung maturity
- Tocolytic drugs to delay delivery for up to 48 hours to allow maximum effect of the glucocorticoids
- A 48-hour course of IV ampicillin and erythromycin followed by 5 days of amoxicillin and erythromycin to prolong pregnancy and decrease infection in cases remote from term
- Chemoprophylaxis to prevent vertical transmission of group B streptococci

Unlike some cases of PTL, women with PROM and PPROM cannot be managed with home care because of the usual brevity of the latent period, the rapidity with which fetal infection may occur, and the need for close fetal monitoring.

**UTERINE RUPTURE**

*Causes and Risk Factors*

Rupture of the gravid uterus is a rare, life-threatening complication for both the mother and fetus. It is defined as direct communication between the uterine and peritoneal spaces following disruption of the uterine wall. Uterine ruptures can be classified as either partial or complete and by their cause:

- Traumatic
  - Obstetric (forceps use, fundal pressure)
  - Nonobstetric (violence, car crashes)
- Spontaneous
  - Previous uterine surgery
  - Unscarred uterus

The main risk factor for uterine rupture is a scarred uterus, which is usually secondary to a previous cesarean section. A fair amount of recent literature focuses on uterine rupture as a result of women attempting vaginal birth after previous cesarean delivery. Risk factors are listed in Box 6.

**Clinical Presentation**

Maternal manifestations of uterine rupture usually include the acute onset of abdominal pain, cessation of uterine contractions, and various degrees of vaginal bleeding. As in any severe intra-abdominal catastrophe, the patient can show signs of shock, including hypotension and tachycardia. Fetal manifestations of uterine rupture include bradycardia, variable or late decelerations, and fetal demise.
Diagnosis

The diagnosis of uterine rupture can be challenging. A history of uterine surgery should place this diagnosis high on the differential for the EM physician treating symptomatic second- or third-trimester gravid patients. Uterine rupture is quite rare among women with unscarred uteri. As with any patient with abdominal pain and possible shock, basic laboratory studies, such as a complete blood count, coagulation studies, and a type and crossmatch for blood products, should be performed. Imaging studies, such as ultrasonography, might identify a uterine wall defect, a fetal anatomy protruding from the uterus, or signs of free fluid in the pelvis, which will help the EM physician care for these critically ill patients.75

Management

On initial presentation, patients with suspected uterine rupture should be placed in a monitored bed, have 2 large-bore IV catheters inserted for access, and undergo a primary survey, with attention to abnormal vital signs and evidence of shock. Patients who are hypotensive and those with evidence of severe hemorrhage should be stabilized with IV normal saline boluses and ultimately with crossmatched packed red blood cells, if indicated. Once the mother is safely stabilized, she should be transferred expeditiously to the obstetric unit or operating room for cesarean delivery. Early transfer and involvement of an obstetrician is key because it allows maximal fetal outcome and repair of the injured uterus. Smaller ruptures can undergo primary repair; women with large ruptures and those complicated by severe hemorrhage should undergo hysterectomy.76 Future hopes of childbearing are taken into consideration, but the mother’s condition and the extent of the injury usually dictate whether she should undergo repair or definitive hysterectomy.

SUMMARY

Complications of late pregnancy are managed infrequently in the ED and, thus, can pose a challenge when encountered as an acute presentation by the EM physician. Emergency care of these conditions requires rapid assessment, identification of hemodynamically unstable patients, and prompt intervention. Care must be taken to evaluate and manage both maternal and fetal well-being. Aside from addressing hemodynamic instability, focused care should include strategies to promote fetal

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<th>Box 6</th>
<th>Risk factors for uterine rupture</th>
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<td>- Previous cesarean delivery</td>
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<tr>
<td>- Uterine surgery</td>
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<td>- Congenital uterine malformations/abnormal uterine anatomy</td>
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<td>- Labor-induction agents</td>
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<td>- Trauma</td>
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<td>- Elective abortion</td>
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<td>- Advance maternal age</td>
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<td>- Multiple gestations</td>
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<td>- Abnormal placentation</td>
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maturity and prevent vertical transmission of infection if delivery is imminent. Early obstetrics consultation and involvement, as well as an understanding of one’s own hospital triaging policies for third-trimester pregnancy, will allow appropriate and timely disposition plans for these patients.

REFERENCES