REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

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<tr><td>CHAPTER
100</td><td># Maternal Emergencies After 20 Weeks of Pregnancy and in the Postpartum Period</td></tr>
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INTRODUCTION

This chapter examines the diagnosis and treatment of the most important maternal emergencies occurring after 20 weeks of pregnancy and during the postpartum period. The second half of pregnancy is often characterized as ≥20 weeks of gestation for simplicity, but until 24 weeks, the chances of fetal survival are less than 50%. The postpartum period is generally accepted as the 6 weeks after delivery. Vast physiologic shifts in maternal cardiovascular tone occur as pregnancy progresses, highlighting the need for maternal blood pressure recordings and fetal heart tones during any ED visit. Conditions discussed are thromboembolic disease; chest pain; disorders associated with elevated blood pressure (hypertension, preeclampsia and HELLP syndrome [hemolysis, elevated liver enzymes, and low platelet count], and eclampsia); vaginal bleeding in the second half of pregnancy; premature rupture of membranes; postpartum hemorrhage; amniotic fluid embolus; peripartum cardiomyopathy; and endometritis.

THROMBOEMBOLIC DISEASE OF PREGNANCY

Venous thromboembolism includes deep venous thrombosis (DVT) and pulmonary embolism (PE) and is the leading cause of maternal morbidity and mortality in industrialized nations. Compared with nonpregnant women, the risk of venous thromboembolism increases fivefold during pregnancy and is increased by 60-fold in the first 3 months after delivery.[1,2]

PATHOPHYSIOLOGY

Pregnancy-related hypercoagulability is due to increased levels of clotting factors, increased platelet and fibrin activation, and decreased fibrinolytic activity, all of which are adaptations to prevent maternal hemorrhage. Physiologic changes include venous stasis, decreased venous outflow, and uterine compression of the inferior vena cava and iliac veins (particularly the left common iliac and left leg veins). Clots tend to develop in the deep venous system of the legs and pelvis, which includes the internal iliac, femoral, greater saphenous, and popliteal veins. Up to 24% of DVTs are complicated by PE, so early DVT diagnosis is important.[1-6]

RISK FACTORS AND CLINICAL FEATURES

Physiologic signs and symptoms of thromboembolic disease, such as tachycardia, tachypnea, lower extremity edema, and dyspnea are nonspecific and also occur during normal pregnancy. Predictive scoring criteria, such as Wells criteria, have not been validated in pregnant women, but left leg symptoms, calf circumference difference ≥2 cm, and leg symptoms in the first trimester are associated with DVT. Iliac vein thrombosis often presents with unilateral swelling of the entire leg and groin or back pain.

A personal or family history of thrombosis is an important risk factor. Other major risk factors include thrombophilias (not identifiable at the first presentation), obesity, maternal age >35, smoking, sickle cell disease, diabetes, hypertension, immobility, in vitro fertilization (greater risk for twins than for singleton), and preeclampsia. Cesarean delivery and postpartum complications further increase the risk.[1,4,5]

DIAGNOSIS OF DEEP VENOUS THROMBOSIS

Compression or duplex US is the test of choice, with a reported sensitivity and specificity for detecting proximal DVT in nonpregnant patients of 89% to 96% and 94% to 99%, respectively.[7] Compression US is less accurate for isolated calf and iliac vein thrombosis. MRI, either with or without contrast venography, is highly sensitive and specific for the diagnosis of pelvic and iliac vein thrombosis. MRI without contrast is preferred with the addition of contrast only if absolutely needed.[8,9] Impedance plethysmography and CT scan of the pelvis are alternatives to diagnose iliac vein thrombosis if MRI is not available. Impedance plethysmography is not widely available and requires operator expertise. CT exposes the fetus to radiation, and iodinated contrast media may affect fetal thyroid tissue.[1] If imaging resources are limited, venography with pelvic shielding is another option.[10]

d-dimers are not useful to include or exclude DVT or PE because levels progressively increase throughout pregnancy, and venous thromboembolism has been reported with negative d-dimers.[11] See chapter 56, Venous Thromboembolism, for a detailed discussion of d-dimers.

DIAGNOSIS OF PULMONARY EMBOLISM

Pregnant women with symptoms suggestive of PE and compression US results positive for DVT should receive anticoagulation without waiting for further confirmatory diagnostic studies.

Women with normal findings on US with suspicion of PE require further diagnostic imaging. The major options for definitive imaging are chest CT–pulmonary angiography and pulmonary perfusion scanning. **As of this writing, the fetal and maternal radiation dose with either modality is felt to be within acceptable limits.**[1,12] Typically, in most institutions, a consensus is obtained between emergency physicians, obstetricians, and radiologists in deciding the imaging steps. **Table 100-1** lists advantages and disadvantages of different imaging modalities.

TABLE 100-1	Imaging Modalities for Diagnosis of Pulmonary Embolism in Pregnancy				
	Radiation	Limitations	Disadvantages	Advantages	Lactation
Chest Radiograph	Minimal	Nonspecific and nonsensitive	Results determine next imaging study, requiring more time to diagnosis	May identify another cause of pulmonary symptoms	No change
CT-PA	High maternal breast radiation; lower fetal radiation than V/Q scan	Contrast allergy and renal insufficiency	Hyperdynamic state in pregnancy can affect interpretation	High sensitivity and specificity; needed if abnormal chest radiograph	No need to discard breast milk
V/Q scan	Low breast radiation but higher fetal radiation than CT-PA	Not useful if abnormal chest radiograph, asthma, COPD, underlying pulmonary disease	If negative or inconclusive and suspicion for PE remains, will need a CT-PA; limited availability and time for isotope preparation, which delays diagnosis	Negative perfusion study effectively rules out PE	Discard breast milk for 12 h
MRI/MRV	No radiation	Gadolinium safety for fetus is unknown; do not use in maternal renal insufficiency	Limited availability	Can detect pelvic and iliac thrombosis	No need to discard breast milk

Abbreviations: COPD = chronic obstructive pulmonary disease; CT-PA = chest CT–pulmonary angiography; V/Q scan = ventilation-perfusion scan; MRV = magnetic resonance venography.

Consensus documents based on expert opinions of pulmonologists and radiologists recommend a plain chest radiograph first.[13] If the chest radiograph is abnormal, or the patient has chronic pulmonary disease, asthma, or chronic obstructive pulmonary disease, chest CT–pulmonary angiography is preferred.[13] If the chest radiograph is normal, a negative perfusion scan can be relied upon to exclude the diagnosis of PE, but an inconclusive perfusion scan will then require a chest CT.[1,12-14]

Magnetic resonance angiography (MRA) can detect PE, but its use in pregnancy has not been well studied. Institutions in which MRA of the pulmonary vasculature is performed routinely have demonstrated a sensitivity of 78% and specificity of 99% when the study is qualified as technically adequate.[15]

◼ TREATMENT OF DEEP VENOUS THROMBOSIS AND PULMONARY EMBOLISM

Venous thromboembolism during pregnancy is treated with either unfractionated heparin (UFH) or low-molecular-weight heparin (LMWH) (**Table 100-2**).[1,16,17] UFH and LMWH do not cross the placental barrier. UFH is preferred over LMWH in patients in a hemodynamically unstable condition with PE, patients who are likely to bleed, patients with renal insufficiency, patients in labor, those receiving regional anesthesia, and patients undergoing cesarean delivery. Monitor activated partial thromboplastin times when using UFH.[11] Dosing requirements of UFH and LMWH increase due to the physiologic changes of pregnancy. Adverse effects of UFH include uteroplacental hemorrhage, heparin-induced thrombocytopenia, and heparin-induced osteopenia. LMWH has fewer adverse effects and fewer bleeding episodes than UFH, and monitoring with anti–factor Xa levels is needed only in special circumstances. See chapter 239, Thrombotics and Antithrombotics, for further discussion of heparins.

Fondaparinux is used in the United States for the prevention and treatment of venous thromboembolism in heparin-allergic or heparin-intolerant pregnant patients.[1] However, fondaparinux is transported across the placenta in low concentrations, and minimal data exist on maternal-fetal safety.[1,17]

Do not prescribe warfarin (Coumadin□) in pregnancy because it crosses the placental barrier, causes CNS abnormalities, and causes warfarin embryopathy (bone and cartilage abnormalities and nasal and limb hypoplasia). Warfarin increases the risk of maternal and fetal hemorrhage, especially during delivery. Warfarin is only considered for women with mechanical heart valves who demonstrate a continued risk of venous thromboembolism despite treatment with UFH or LMWH.[1]

An inferior vena cava filter is indicated when anticoagulation is contraindicated, when an acute embolic event occurs despite anticoagulation, or when acute venous thromboembolism occurs with impending delivery of the fetus.[18]

Treatment of Life-Threatening Pulmonary Embolism Treatment options include systemic thrombolysis, catheter-guided thrombolysis, and surgical or catheter-guided embolectomy.[19-22] Data regarding maternal-fetal outcomes in conditions of maternal extremis are limited to case reports, and catheter-guided thrombolysis and embolectomy require precious time for preparation. **Recombinant tissue plasminogen activator** (10-milligram bolus, 90-milligram infusion over 2 hours) does not cross the placenta and has a lower rate of hemorrhagic complications and lower mortality rate than do streptokinase and urokinase in the nonpregnant population. **Streptokinase** (250,000-unit bolus, 100,000 units/h infusion for 24 hours) is also used but with a higher rate of subchorionic hemorrhage, allergic complications, and longer infusion duration than recombinant tissue plasminogen activator. Catheter-directed thrombolysis allows for earlier reperfusion and likely improves long-term pulmonary function compared with systemic therapy.[1,19] Fetal loss subsequent to surgical embolectomy is higher than with thrombolysis.[20,22]

CHEST PAIN

The differential diagnosis of chest pain in pregnant women is similar to that of nonpregnant women, but disorders such as aortic dissection and cardiomyopathy are associated with pregnancy. Advances in reproductive technology resulting in pregnancies in older women may result in an increase in **coronary artery disease** in this population. **Coronary artery dissection** and **coronary vasospasm** are more likely in women who smoke and those with migraine. Coronary artery disease is more likely in those >35 years old, diabetics, and hypertensives.[23] Treat **acute myocardial infarction** with low-dose aspirin, heparin, and percutaneous coronary intervention rather than with thrombolytics.[24,25] **Aortic dissection**, although rare, is usually encountered in the third trimester and the postpartum period. Risk factors are pregnancy, bicuspid aortic valve, connective tissue disorders (e.g., Marfan's syndrome), syncope, hypertension, and a family history of aneurysm.[26] Chest radiograph may not demonstrate a widened mediastinum, and diagnosis is made by MRI or CT scan.[23]

Peripartum cardiomyopathy is a dilated cardiomyopathy that can occur at any stage of gestation, but is classically defined as occurring in the last month of gestation or within the first 5 months after delivery, without an apparent cause or preexisting history of cardiac disease. The cause is unknown. Risk factors include cardiomyopathy during prior pregnancies, multiparity, maternal age >40 years old, chronic hypertension before pregnancy, gestational hypertension, preeclampsia, and HELLP syndrome. Symptoms and signs of peripartum cardiomyopathy are dyspnea, orthopnea, cough, palpitations, chest pain, edema, rales, and jugular venous distention. Diagnose and treat congestive heart failure and pulmonary edema with standard modalities (see chapter 53, Acute Heart Failure) **except that nitroprusside is relatively contraindicated in pregnancy because it can cause thiocyanate and cyanide accumulation in the fetus**. In the postpartum patient, angiotensin-converting enzyme inhibitors may be given. Anticoagulate with heparins because of increased risk of thromboembolism. **Do not use warfarin during pregnancy. Warfarin can be given in the postpartum period.[27,28]**

TABLE 100□2	Initial Treatment for Venous Thromboembolism during pregnancy[1,16,17]
Antithrombotic Agent	**Initial Dose**
Recommended: LOW MOLECULAR WEIGHT HEPARINS (LMWH)	
Enoxaparin (Lovenox®)	1 milligram/kg SC every 12 h
Dalteparin (Fragmin®)	100 units/kg SC every 12 h
Tinzaparin (Innohep®)	175 units/kg SC every 24 h
UNFRACTIONATED HEPARIN (LMWH)	10,000 units SC every 8-12 h to achieve aPTT 2-2.5 times base 6 hrs after dose
For heparin allergy or heparin-induced thrombocytopenia	
Fondaparinux (Arixtra®)	50-100 kg, 7.5 milligrams SC every 24 h; > 100 kg, 10 milligrams SC every 24 h
Danaparoid	removed from US market

DISORDERS ASSOCIATED WITH ELEVATED BLOOD PRESSURE: HYPERTENSION, PREECLAMPSIA AND HELLP SYNDROME, AND ECLAMPSIA

■ CHRONIC AND GESTATIONAL HYPERTENSION

The decrease in systemic vascular resistance results in a decrease in maternal blood pressure, and blood pressure reaches its nadir at 16 to 18 weeks of pregnancy. Blood pressure returns to prepregnancy values near the end of the second trimester.

Chronic hypertension in pregnancy is defined as a systolic blood pressure of ≥140 mm Hg or a diastolic blood pressure of ≥90 mm Hg that existed prior to pregnancy, is diagnosed before the 20th week of gestation, or persists longer than 12 weeks after delivery. Severe chronic hypertension is systolic blood pressure >160 mm Hg or diastolic pressure >110 mm Hg. Women with chronic hypertension are at increased risk for placental abruption, preeclampsia, low birth weight, cesarean delivery, premature birth, and fetal demise.[29]

Gestational hypertension is hypertension present only after the 20th week of pregnancy or in the immediate postpartum period but without proteinuria.

Safe treatment options for hypertensive women who are pregnant are labetalol and methyldopa.[30] **All antihypertensive drugs cross the placenta.** Labetalol is the first-line agent for chronic hypertension in pregnancy.[29] The starting dose is 100 milligrams PO twice a day, and the usual maintenance dose is 200 to 400 milligrams PO twice a day. Methyldopa, used safely in pregnancy for decades, is started at 250 milligrams every 6 hours PO and titrated to achieve the desired blood pressure. The usual daily dose is 500 milligrams to 3 grams divided in two to four doses per day, with a maximum of 3 grams per day.

Long-acting nifedipine may be added if blood pressure is not controlled with methyldopa or labetalol. Long-acting nifedipine is started at 30 milligrams PO once a day and can be increased up to 120 milligrams per day slowly if needed. For acute management of **hypertensive emergencies**, hydralazine 5 milligrams IV or IM, labetalol 20 milligrams IV, or nifedipine 10 to 30 milligrams PO (not a Food and Drug Administration–approved indication) may be used during pregnancy.[29-31] **Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are contraindicated because of their teratogenic effects on fetal scalp, lungs, and kidneys.**[29]

■ PREECLAMPSIA

Preeclampsia, or gestational hypertension with proteinuria, is characterized by hypertension before 20 weeks of gestation with either new-onset proteinuria, sudden increase in proteinuria, or development of **HELLP syndrome**.

The cause of preeclampsia is unknown. The histologic hallmark lesion of preeclampsia is acute atherosis of decidual arteries. Atherosis and thrombosis are thought to lead to placental ischemia and infarctions. Poor placental perfusion is presumed to lead to the formation of free radicals, to oxidative stress, and to inflammatory responses that may influence the mechanistic development of preeclampsia.[30]

Preeclampsia is associated with intrauterine growth retardation, premature labor, low birth weight, abruptio placentae, and future risk of maternal cardiovascular disease.[30,32]

Preeclampsia during an initial pregnancy increases the chances of recurrence in future pregnancies. Other important risk factors for preeclampsia include maternal age >40 years old, hypertension, diabetes, renal disease, collagen vascular disease, and multiple gestation. Low-dose aspirin therapy can prevent preeclampsia and its complications.[33,34]

Diagnosis of Preeclampsia Diagnostic criteria for preeclampsia are listed in **Table 100-3**, and laboratory evaluation is outlined in **Table 100-4**.[35]

HELLP Syndrome The **HELLP syndrome (Table 100-5)** is an important clinical variant of preeclampsia. HELLP is more common in the multigravid patient than in the primigravida. Hypertension may not be present initially or at all. This fact, combined with the usual complaint of epigastric or right upper quadrant pain, makes it easy to misdiagnose HELLP

TABLE 100–3	Diagnostic Criteria for Preeclampsia
Criteria for *mild* preeclampsia	
Systolic blood pressure ≥140 mm Hg OR diastolic blood pressure ≥90 mm Hg	
AND	
Proteinuria >0.3 grams in a 24-h collection	
AND	
>20-wk gestation	
AND	
No other systemic signs or symptoms	
Criteria for *severe* preeclampsia	
Blood pressure ≥160 mm Hg systolic or ≥110 mm Hg diastolic measured on two occasions at least 6 h apart with the patient at rest	
AND	
Visual disturbances or mental status disturbances	
OR	
Pulmonary edema or cyanosis	
OR	
Epigastric or right upper quadrant pain; abnormal liver function studies	
OR	
Thrombocytopenia	
OR	
Oliguria (<500 mL in 24 h)	
OR	
Proteinuria of ≥5 grams in a 24-h collection or ≥3+ on two random urine samples collected at least 4 h apart	
Impaired fetal growth	

syndrome for other causes of abdominal pain, such as gastroenteritis, cholecystitis, hepatitis, pancreatitis, or pyelonephritis. **A pregnant woman at >20 weeks gestation or up to 7 days postpartum with abdominal pain should be evaluated for HELLP syndrome.**

Complications of severe preeclampsia, HELLP syndrome, and eclampsia include disseminated intravascular coagulopathy, spontaneous hepatic and splenic hemorrhage, end-organ failure, abruptio placentae, intracranial bleeding, maternal death, and fetal death.[35]

Treatment of Preeclampsia For **mild eclampsia**, outpatient management is an option after consultation with the obstetrician, as long as arrangements are made for frequent clinical and laboratory evaluation and close fetal surveillance.[35] Headache, scintillating scotomata or other visual changes, abdominal pain, vaginal bleeding, and decreased fetal movement require immediate reevaluation. Treat **severe preeclampsia** (blood pressure >160 mm Hg) with antihypertensive agents **(Table 100-6)**

TABLE 100–4	Laboratory Evaluation for Preeclampsia
Test	**Comments**
CBC with differential	May see hemoconcentration or falling hematocrit. Thrombocytopenia suggests severe disease.
Creatinine	Elevation suggests severe disease.
Alanine and aspartate aminotransferase concentrations	Elevation suggests severe disease.
Lactate dehydrogenase level	Elevation suggests microangiopathic hemolysis.
Protein in urine	3+ proteinuria; 24-h collection may be done by obstetric service. >5 grams/24 h suggests severe disease.
Protein/creatinine ratio	0.1–0.3 indicates need for 24-h collection[29]
Uric acid level	Level ≥5.5 milligrams/dL may suggest superimposed preeclampsia on chronic hypertension[29]

TABLE 100–5	Laboratory Abnormalities in HELLP Syndrome
Test	**Findings**
CBC and test of peripheral smear	Schistocytes
Platelet count	<100,000/μL <150,000/μL suspicious for syndrome
Liver function tests (alanine aminotransferase, aspartate aminotransferase levels)	Elevated but below levels usually seen in viral hepatitis (<500 U/L)
Renal function tests	Normal or elevated blood urea nitrogen and creatinine levels
Coagulation profile	Abnormal
Lactate dehydrogenase	>600 U/L suspicious for hemolytic anemia
Total bilirubin	>1.2 milligrams/dL

and IV magnesium sulfate.[35-37] Consult with the obstetrician for admission or transfer to a center that manages high-risk pregnancy especially in the presence of HELLP syndrome. **The only *definitive* resolution for preeclampsia is delivery.**

The initial management of **HELLP syndrome** is similar to that of **severe preeclampsia** or **eclampsia**: IV magnesium, blood pressure control, and hospital admission for stabilization. Correct coagulopathy. If HELLP syndrome is suspected and obstetric care is not available locally, stabilize the patient as best as possible and transfer to a tertiary care center with high-risk obstetrics facilities. The definitive treatment is delivery, especially if the patient is ≥34 weeks of gestation. Corticosteroid administration can help delay delivery and improve fetal outcome in pregnancies <34 weeks of gestation.

◼ ECLAMPSIA

Eclampsia is the development of new-onset seizures, superimposed upon preeclampsia, in a woman between 20 weeks of gestation and 4 weeks postpartum.

Eclampsia should be suspected and treated in any pregnant woman who is at >20 weeks of gestation or <4 weeks postpartum who develops seizures, coma, or encephalopathy. Occasionally, eclampsia can present with seizure in the absence of blood pressure elevation and proteinuria.[30] **Management of eclampsia includes treatment of seizures, treatment of hypertension, and emergent obstetric consultation to facilitate urgent delivery of the fetus.** Treat seizures with **magnesium sulfate**, 4 to 6 grams IV in 100-mL aliquot given over 20 to 30 minutes followed by an infusion of 2 grams per hour for at least 24 hours.[35-37] Magnesium is renally excreted, and in women with renal insufficiency, reduce the dose to 2 grams IV bolus and obtain a serum magnesium level before increasing the dose. The main side effects of high levels of magnesium are flushing, diaphoresis, hypothermia, hypotension, flaccid paralysis, and respiratory depression. When levels approach toxicity, patellar reflexes diminish

and respiratory rate slows. Administer antihypertensives as suggested in Table 100-6. Replace coagulation factors if there is coagulopathy. Obtain emergency obstetric consultation for prompt delivery. If obstetric services are not available, stabilize the patient as best as possible and transfer to a center with facilities for advanced obstetric care.

VAGINAL BLEEDING IN THE SECOND HALF OF PREGNANCY

The causes of serious vaginal bleeding in the second half of pregnancy include abruptio placentae, placenta previa, and vasa previa. All can cause severe hemorrhage. **Do not perform a digital or speculum pelvic examination to assess vaginal bleeding until a transvaginal US is performed to determine the location of the placenta. Mechanical disruption of the placenta by speculum or digital examination may precipitate catastrophic hemorrhage.** When transvaginal US is properly and carefully performed by those experienced in transvaginal US (vaginal probe is angled against the anterior lip of the cervix, probe is not advanced to contact the placenta, probe is not inserted into the cervix), the technique does not cause hemorrhage.[40] If there is no evidence of placenta previa or vasa previa, then a sterile speculum examination may be performed to determine if premature rupture of membranes or abruption is present.

◼ ABRUPTIO PLACENTAE

Abruptio placentae is the premature separation of a normally implanted placenta from the uterine lining (**Figure 100-1**). The incidence of spontaneous abruption is highest between 24 and 28 weeks of gestation.[41] Abruption can cause uteroplacental insufficiency and fetal distress or demise. Maternal complications include coagulopathy, hemorrhagic shock, uterine rupture, and multiple organ failure. Abruption usually occurs spontaneously but is also associated with trauma, even minor trauma. See chapter 256, Trauma in Pregnancy. Risk factors for abruption include abdominal trauma, cocaine use, oligohydramnios, chorioamnionitis, advanced maternal age or parity, eclampsia, and chronic or acute hypertension.

Clinical features depend on the degree of placental abruption. Mild abruption is characterized by mild uterine tenderness, no or mild vaginal bleeding, normal maternal vital signs, no coagulopathy, and fetal distress. Signs and symptoms of severe abruption are no or heavy vaginal bleeding, fetal distress, coagulopathy, severe uterine pain or tenderness, continuous or repetitive uterine contractions, and maternal hypotension or shock. Nausea, vomiting, and back pain may also be present. **Consider placental abruption in pregnant women with acute, painful vaginal bleeding or with acute abdominal/uterine pain.**

Diagnosis is made by the clinical features. Electronic fetal monitoring (cardiotocodynamometry) is very sensitive for identifying fetal distress as a sign of placental abruption and has a 100% negative predictive value for adverse outcomes when monitoring is reassuring.[42] Transvaginal US is fairly specific for the diagnosis, but is not sensitive for the detection of

| TABLE 100–6	Antihypertensive Drugs for Treatment of Acute, Severe Hypertension in Preeclampsia and Eclampsia[38,39]			
Generic Name	**Mechanism of Action**	**Onset of Action**	**Dosage**	**Comment**
Labetalol	Selective α and nonselective β antagonist	5 min	20 milligrams IV, then 40–80 milligrams IV every 10 min as needed (maximum, 300 milligrams); IV infusion 1–2 milligrams/min titrated	Less hypotension and reflex tachycardia than hydralazine. Higher doses cause neonatal hypoglycemia. Longer use associated with fetal growth restriction.
Hydralazine	Arterial vasodilator	20 min	5 milligrams IV or 10 milligrams IM, repeat at 20-min intervals; consider other drug if no response at maximum of 20 milligrams IV or 30 milligrams IM	Maternal hypotension, fetal distress; must wait 20 min for response between IV doses.
Nifedipine	Calcium channel antagonist	10–20 min	10 milligrams PO, repeat in 30 min if necessary	Food and Drug Administration does not approve short-acting nifedipine for treatment of hypertension.
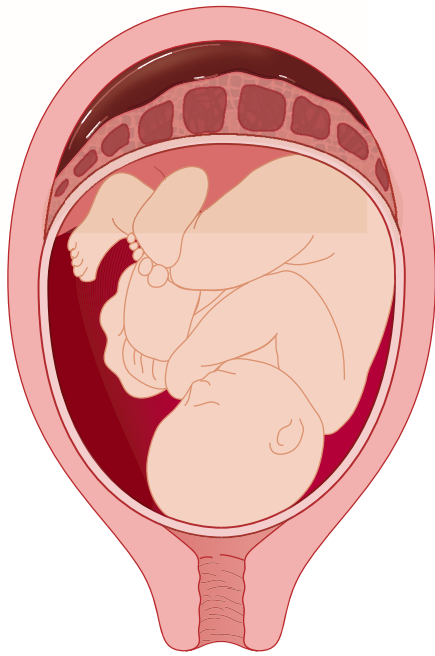
FIGURE 100-1. Abruptio placentae. The placenta has separated from the superior pole of the uterus.

FIGURE 100-2. Complete placenta previa. Placenta overlies the internal os.

retroplacental clot because the appearance of clotted blood evolves in echotexture over time.[41] MRI is diagnostic but requires the transport of a potentially unstable patient out of the ED or intensive care unit for imaging.

Treatment consists of maternal stabilization, cardiotocographic monitoring to detect fetal distress, and emergency obstetric consultation. Place two large-bore IVs; obtain a CBC, metabolic panel, coagulation panel, fibrin degradation product, and fibrinogen levels; and type and cross-match maternal blood. Administer RhoGAM□ if the mother is Rh negative. For disseminated intravascular coagulation, replace coagulation factors. Immediate delivery is indicated for severe abruption.

■ PLACENTA PREVIA

Placenta previa is a placenta that extends near, partially over, or beyond the internal cervical os. Normal placental implantation is in the corpus or fundal region, whereas in placenta previa, implantation is lower in the uterus. The cause is unknown. Although low-lying or partial placenta previa is not uncommon early in pregnancy, the placenta usually migrates to a normal position as the pregnancy nears term.

Risk factors for placenta previa include cesarean delivery, multiple uterine surgeries, advanced maternal age, minority group status, cigarette smoking, and cocaine use. Patients with symptomatic placenta previa present with painless bright-red vaginal bleeding, which should be differentiated from the normal passage of blood-stained mucus that occurs near the onset of labor. There are three subclasses of placenta previa: marginal placenta previa, which reaches the internal os but does not cover it; partial placenta previa, where the placenta partially covers the internal os; and complete placenta previa, which completely covers the internal os (**Figure 100-2**).

While proceeding with further patient assessment, place two large-bore IVs for fluid resuscitation; obtain CBC and coagulation parameters; and type and cross-match blood. **Do not perform a digital or speculum vaginal examination until normal placental position is confirmed by US, as disruption of the cervical-placental junction could precipitate catastrophic hemorrhage.** Carefully perform transvaginal US (see earlier). Once placenta previa is identified, consult obstetrics for management options. A double setup, in which two teams of staff are available in the operating room during a vaginal examination, may be indicated in cases where the placenta lies within 1 to 2 cm of the cervical os and labor is imminent. Otherwise, women in the second half of pregnancy with
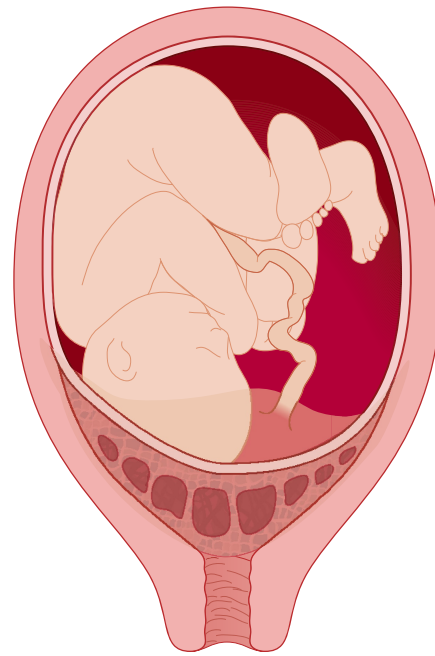
placenta previa are usually admitted to the hospital for observation and fetal monitoring

■ VASA PREVIA

Vasa previa is a rare cause of late-pregnancy bleeding. In vasa previa, umbilical vessels course in the amniotic membrane at the level of the cervical os, so that when the cervix begins to dilate in labor with membrane rupture, the blood vessels tear. Fetal distress or fetal demise may result from fetal exsanguination or vessel compression. Risk factors associated with vasa previa are placenta previa, in vitro fertilization, velamentous insertion of the umbilical cord, and bilobed placenta. Vasa previa can sometimes be diagnosed by Doppler color US performed early in pregnancy. Otherwise, it is seldom recognized prior to catastrophic vessel disruption during labor. Treatment is rapid operative delivery.

PREMATURE RUPTURE OF MEMBRANES, PRETERM LABOR, AND PRETERM BIRTH

Premature rupture of membranes is rupture of membranes prior to onset of contractions. **Membrane rupture before 37 weeks of gestation is known as preterm premature rupture of membranes.** The time from membrane rupture to delivery, known as the latent period, is usually inversely proportional to the gestational age when preterm premature rupture of membranes occurs. For pregnancies between 25 and 32 weeks, 33% had latent periods longer than 3 days. For pregnancies at 33 to 34 weeks and 35 to 36 weeks, 16% and 4.5% had latencies greater than 3 days, respectively.[43] The latent period allows for pharmacologic intervention to improve fetal lung maturity and increase fetal survival.

Preterm (premature) labor is labor prior to 37 weeks of gestation and is often preceded by premature rupture of membranes. Preterm labor is thought to be a syndrome initiated by multiple mechanisms, including infection, inflammation, uteroplacental ischemia or hemorrhage, uterine overdistention secondary to multiple gestation, stress, and other immunologically mediated processes.[44] Several non–genital tract infections, including pyelonephritis, asymptomatic bacteriuria, pneumonia, appendicitis, and periodontal disease, are also implicated in preterm labor.

Untreated *Chlamydia*, gonorrhea, *Trichomonas vaginalis*, and bacterial vaginosis infections increase the risk of preterm premature rupture

of membranes and preterm labor. With appropriate patient selection, vaginal progesterone gel or suppository or IM progesterone can reduce the likelihood of preterm delivery.[45]

Preterm birth is birth before 37 weeks of gestation, and may occur spontaneously or as a result of premature rupture of membranes or placental abruption or may be induced for medical reasons. Surviving preterm neonates are at risk for sepsis, neurologic defects, feeding problems, blindness, deafness, and respiratory distress. Infants weighing <1500 grams or born at <32 weeks of gestation are at greatest risk for these problems, but even infants born at between 34 0/6 and 36 6/7 weeks gestation (late preterm infants) may require intensive care admission for IV fluids, sepsis, hyperbilirubinemia, and mechanical ventilation.[41,43]

■ DIAGNOSIS OF PREMATURE RUPTURE OF MEMBRANES

Details of the history taking and physical examination that aid in the diagnosis of premature rupture of membranes are listed in **Table 100-7**. **Avoid digital cervical examination because it decreases the latent period and may increase the likelihood of infection. Perform speculum examination and visually examine the cervix to identify dilation and test vaginal fluid (Table 100-7).** The combination of history, nitrazine paper, and fern testing (**Figure 100-3**) are reported to diagnose 90% of cases of premature rupture of membranes.[46] US assessment of amniotic fluid volume correctly identifies premature rupture of membranes when a low amniotic fluid index (≤10 cm) is present.[47]

Premature labor or premature rupture of membranes requires obstetric consultation. If obstetric services are unavailable, transfer the patient to a center where such services are available. Obstetric treatment includes corticosteroids and antibiotics to treat group B *Streptococcus*.

Corticosteroids given at <34 weeks of gestation speed fetal lung maturity, decrease the incidence of intraventricular hemorrhage, reduce the duration

FIGURE 100-3. Ferning of amniotic fluid. [Photo contributed by Robert Buckley, MD.]

of mechanical ventilation, and reduce the incidence of necrotizing enterocolitis without increasing maternal or fetal infection. Betamethasone (12 milligrams IM every 24 hours for 2 days) or dexamethasone (6 milligrams IM every 12 hours for 2 days) can be used for gestations between 24 and 34 weeks.[48]

Antibiotics can decrease neonatal infection, prolong latency, and reduce the incidence of postpartum endometritis, chorioamnionitis, neonatal infections, and intraventricular hemorrhage. Antibiotic choices are penicillin G (5 million units IV then 2.5 to 3 million units every 4 hours until delivery), ampicillin (2 grams IV then 1 gram every 4 hours until delivery), and cefazolin (2 grams IV). Clindamycin or vancomycin

TABLE 100-7	Keys to Diagnosis of Premature Rupture of Membranes
Information Obtained	**Comments**
History	
Gush of fluid and continued leakage of fluid	Ask patient to perform a Valsalva maneuver while speculum is in place to look for gush of fluid, or apply fundal pressure.
Details of contractions	Determine if active labor is in process.
Date of last menstrual period	Use to calculate estimated date of delivery and gestational age. Gestational age is number of weeks from first day of last menstrual period.
Vaginal bleeding	Raises concern for placenta previa.
Recent intercourse	
Fever	Infection raises fetal and maternal risk.
Physical Examination	
Measurement of fundal height	
Auscultation of fetal heart tones	
Sterile speculum examination	Check for: Cervical dilatation and effacement. Pooling in vagina of fluid leaking from cervix. If no fluid is noted, apply fundal pressure or ask patient to perform a Valsalva maneuver or cough.
Laboratory Evaluation	
Vaginal fluid	Test with nitrazine paper. **Blue color indicates pH >6.5, signaling presence of amniotic fluid.** Note: Blood, semen, bacterial vaginosis, trichomoniasis, soap, and antiseptics may cause false positive; false negative, about 7%.[46]
Swab of vaginal walls or posterior fornix; do not swab cervical mucus; cervical mucus produces thick, dark, wide arborization pattern, not delicate ferning	Examine glass slide preparation for ferning. **Ferning indicates presence of amniotic fluid.** Blood may obscure ferning. Mucus results in a false-positive fern test finding. Test for *Chlamydia*, *Neisseria gonorrhoeae*, group B streptococci, and bacterial vaginosis.

are alternatives for those allergic to penicillins. Macrolides are not recommended.[49] Do not give amoxicillin-clavulanate because it is associated with necrotizing enterocolitis. There is no need for prophylactic antibiotics if membranes are intact.[50]

Tocolytic therapy using magnesium sulfate, β-mimetics, indomethacin, or calcium channel blockers is controversial and is relatively contraindicated in the patient with preterm premature rupture of membranes. Tocolysis may allow time for antenatal administration of corticosteroids and antibiotics and transfer of the patient to an appropriate neonatal center. Because there are no widely accepted protocols for tocolytic management, consult the obstetrician, who can explain the pharmacologic risks, benefits, and alternatives to the patient. Magnesium sulfate may reduce the risk and severity of cerebral palsy in infants when birth is anticipated before 32 weeks of gestation.[51]

Management guidelines for expedited delivery in the near-term gestation vary. Expedited delivery was historically recommended in patients with preterm premature rupture of membranes at >34 weeks of gestation to avoid the complications of chorioamnionitis and neonatal sepsis, but recent trials suggest that induction of labor does not reduce the risk of neonatal sepsis.[52]

MATERNAL EMERGENCIES DURING LABOR AND DELIVERY

The most important maternal emergencies of labor and delivery are postpartum hemorrhage, uterine rupture, and amniotic fluid embolism. Difficult deliveries are discussed in chapter 101, Emergency Delivery.

■ POSTPARTUM HEMORRHAGE

Postpartum hemorrhage usually occurs within the first 24 hours of delivery and is referred to as **primary postpartum hemorrhage**. The main causes of primary postpartum hemorrhage are **uterine atony**, **retained placental fragments**, **lower genital tract lacerations**, **uterine rupture**, **uterine inversion**, and **hereditary coagulopathy**. **Table 100-8** lists the most common risk factors. **Secondary postpartum hemorrhage** occurs after the first 24 hours and up to 6 weeks postpartum. The most common causes of secondary postpartum hemorrhage are failure of the uterine lining to subinvolute at the former placental site, retained placental tissue, genital tract wounds, and uterogenital infection.[53,54]

Excessive blood loss in the postpartum period is defined as a 10% drop in the hematocrit, a need for transfusion of packed red blood cells, or volume loss that causes symptoms of hypovolemia. Normally, plasma volume increases by 40% and red blood cell volume by 25% at the end of the third trimester. The hematologic changes of pregnancy can mask the typical symptoms of hemorrhage, and the first sign may be only a mild increase in pulse rate. Up to a 30% loss in total blood volume may be required before blood pressure drops.

Most cases of postpartum hemorrhage are due to uterine atony. Another 20% result from cervical, vaginal, or perineal lacerations. Retention of placental tissue may account for another 10%, and underlying

TABLE 100–8	Risk Factors for Postpartum Hemorrhage
Primiparity or grand multiparity	
Previous postpartum hemorrhage	
Preeclampsia	
Prior cesarean section	
Placenta previa or low-lying placenta	
Marginal umbilical cord insertion	
Transverse fetal lie	
Labor induction or augmentation	
Cervical or uterine trauma	
Fetal age <32 weeks of gestation	
Fetal birth weight >4500 grams	
Prolonged third stage of labor	

coagulopathy is uncommon but potentially treatable.[55] **The initial steps are to begin aggressive fluid and blood resuscitation while simultaneously identifying and treating the underlying cause (Tables 100-9 and 100-10).** Nonpneumatic antishock garments can be applied in combination with fluid resuscitation and uterotonics, resulting in reduced blood loss and increased maternal survival in remote settings or with delayed transport.[56]

Uterine atony is the most common cause of postpartum hemorrhage. Risk factors include preeclampsia, prolonged use of uterotonics or tocolytics, prolonged labor, multifetal gestation or fetal macrosomia, multiparity, retained placenta, and uterine infection. Initiate bimanual uterine massage; place a fist in the anterior fornix and compress the uterine fundus against the hand in a suprapubic location (**Figure 100-4**).

Uterine inversion mostly results from previous cesarean section or overzealous attempts to remove the placenta to manage the third stage of labor.[53] Inversion can also occur in patients with connective tissue disorders and uterine structural anomalies. It can be a difficult diagnosis to make, especially if the fundus remains cephalad to the cervix. The diagnosis can be made with transvaginal or transabdominal ultrasound. Uterine inversion requires immediate manual replacement of the uterus. A Rüsch balloon catheter can be applied to correct uterine inversion.[57] Whatever technique is used, correction of uterine inversion is a very painful and difficult procedure that may require general anesthesia and tocolytic agents.

Retention of placental fragments or **abnormal placental implantation** (placenta accreta) may cause severe hemorrhage and may require emergency pelvic embolectomy, hemostatic brace sutures (B-Lynch sutures), or peripartum hysterectomy.

Uterine rupture is a rare complication but carries high maternal and fetal mortality. Previous cesarean section is the primary risk factor for uterine rupture, and single-layer surgical closure of the uterus, fetal size >3500 grams, and labor augmentation increase the rate of rupture during a trial of labor. Anatomic abnormalities such as a bicornuate uterus, grand multiparity, history of connective tissue disorders, and abnormal placentation are also associated with rupture. Clinical signs of uterine rupture are persistent abdominal pain, severe vaginal bleeding, loss of fetal station, and palpable uterine defect. Fetal monitors may show fetal distress and bradycardia. The diagnosis of uterine rupture must be made clinically and rapidly. Treatment is aggressive fluid and blood resuscitation and surgical delivery of the fetus.

■ AMNIOTIC FLUID EMBOLUS

Amniotic fluid embolus is a rare and often catastrophic complication of pregnancy that occurs when amniotic fluid and cells of fetal origin enter the maternal circulation during labor or delivery. Most cases occur before delivery. Fetal and maternal mortality rates are high. Amniotic fluid embolism is difficult to diagnose and is usually a diagnosis of exclusion. The onset of symptoms until cardiovascular collapse can range from seconds to over 4 hours. Presenting signs include respiratory distress, hypoxia, pulmonary edema, altered mental status, seizures, sudden maternal cardiovascular collapse, disseminated intravascular coagulation, and sudden onset of fetal distress.[58]

Postulated causes are antigenic stimuli or activation of the clotting cascade when amniotic fluid enters the maternal circulation. Physiologically, the hemodynamic changes shown on echocardiogram during confirmed cases of amniotic fluid embolism are due to the acute onset of severe pulmonary hypertension, right ventricular failure with leftward deviation of the septum, and the absence of pulmonary edema. Secondarily, left ventricular filling becomes impaired due to profound right heart failure, eventually resulting in myocardial ischemia.[58]

Death can occur rapidly. Treatment is supportive, and there are no specific interventions currently available; prevent and/or treat hypoxia, hypotension, and hypoperfusion. Place the woman in the left lateral decubitus position to minimize vena cava compression; give oxygen by nonrebreather mask or endotracheal tube; resuscitate with fluid and blood; and administer pressors to support maternofetal circulation until emergency delivery of the fetus is performed. Obtain emergency obstetric consultation. If the gravid patient cannot be resuscitated, perimortem cesarean delivery within 5 minutes of cardiac arrest increases the chances of neonatal survival.

TABLE 100–9	Drug Regimens for Postpartum Hemorrhage
Drug	**Comments**
Oxytocin (Pitocin®) 　10 units IM *or* 　20-40 units in 1 L NS; give 500 mL over 10 min, then 250 mL/h	First-line treatment. Uterotonic agent. Rapid administration may cause transient hypotension.
Carboprost (Hemabate®) 　0.25 mg IM, repeat as needed every 15-90 minutes for a total dose of 2 milligrams	Prostaglandin. Side effects: nausea, vomiting, diarrhea, hypertension, bronchospasm. Avoid in patients with hypertension or asthma.
Misoprostol (Cytotec®, Apo-Misoprostol®) 　1000 micrograms rectally	Prostaglandin. Side effects: nausea, vomiting, diarrhea. Not FDA approved for this indication; widely used internationally due to heat stability.
Methylergonovine (Methergine®) or Ergonovine® 　0.2 milligrams IM, repeat as needed every 2-4 h to maximum of 5 doses	Ergot. Contraindicated in patients with hypertension or preeclampsia.

Abbreviations: FDA = Food and Drug Administration; NS = normal saline; SL = sublingual.

TABLE 100–10	Common Causes and Treatment of Postpartum Hemorrhage
Tone	Perform bimanual uterine massage. Give drugs to improve uterine tone as outlined in Table 100-9.
Trauma	Examine for cervical, vaginal, or perineal lacerations or hematomas. Repair lacerations. Incise, drain, and appropriately ligate bleeding vessels causing a hematoma. Correct uterine inversion with manual replacement. Uterine rupture requires surgery.
Tissue	Inspect the placenta for missing fragments; if a portion is absent, manually evacuate the uterine cavity. Invasive placentation may require hysterectomy. Perform transvaginal or transabdominal US to identify abnormal fluid-filled uterus. Consider a balloon tamponade with either uterine-specific balloon device (Bakri or Rüsch) or an adaptation of a Foley catheter or condom as a temporizing measure.
Thrombin	Consider DIC in the setting of severe preeclampsia, sepsis, placental abruption, shock, or intrauterine fetal demise, although undiagnosed coagulopathies may rarely present in nulliparas. Replace coagulation factors.

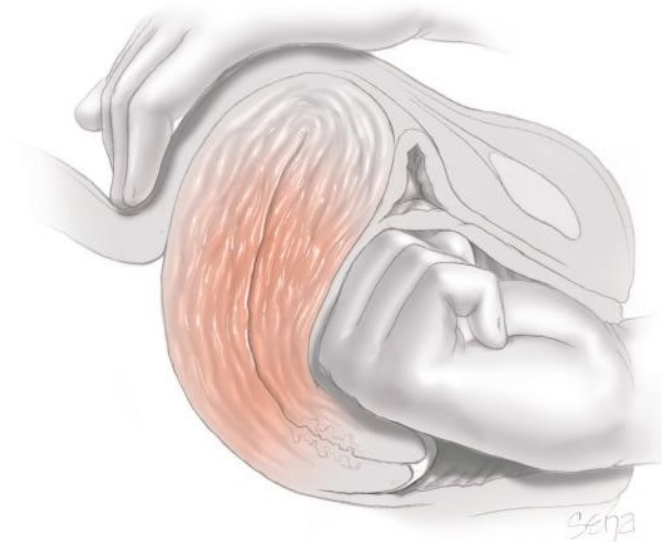*Abbreviation:* DIC = disseminated intravascular coagulation.

FIGURE 100-4. Bimanual method for uterine massage. [Reproduced, with permission, from Cunningham G, Leveno KL, Bloom SL, Hauth JC, Rouse DJ, Spong CY (eds): *Williams Obstetrics,* 23rd ed. © 2010. The McGraw-Hill Companies, Inc. New York, Figure 35-17.]

POSTPARTUM ENDOMETRITIS

Most postpartum infections are identified after hospital discharge. **In a postpartum woman with fever, assume pelvic infection until proven otherwise. Also consider respiratory tract infection, pyelonephritis, mastitis, thrombophlebitis, and appendicitis.**[59] Risk factors for postpartum endometritis are listed in **Table 100-11**.

The most common pathogens are gram-positive and gram-negative aerobes, anaerobes, *Mycoplasma hominis*, *Chlamydia trachomatis*, and *Neisseria gonorrhoeae. Gardnerella vaginalis* is isolated more often in younger women. Many infections are polymicrobial. Group A streptococcal infections are less common, but are increasing causes of postpartum endometritis.[60]

Symptoms of postpartum endometritis are fever, foul-smelling lochia, leukocytosis, tachycardia, pelvic pain, and uterine tenderness. Only scant vaginal discharge may be present, especially in patients infected with group B streptococci. In patients who are status post cesarean section, there may be surgical wound tenderness and purulent exudate.

Vaginal samples are of little value because of contamination with local flora. Blood cultures are rarely positive. Culture any purulent material from surgical incisions. Creatine phosphokinase levels may be elevated in group B streptococcal endometritis. Consult the obstetrician for disposition decisions. Admission is needed for patients who appear ill, have had cesarean section, or have underlying comorbidities. Treatment consists of antibiotics (**Table 100-12**), abscess drainage, and debridement of necrotic tissue.

After consultation with and recommendation by the obstetrician, patients with mild illness and those for whom follow-up in 24 hours is assured can be treated as outpatients with oral antibiotics, such as clindamycin, 300 milligrams three times per day PO for 10 days, or doxycycline, 100 milligrams twice per day for 10 days. **Do not give doxycycline to women who are breastfeeding**.

Complications of endometritis include parametrial phlegmons; surgical, incisional, and pelvic abscesses; infected hematomas; septic pelvic thrombophlebitis; necrotizing fasciitis; and peritonitis.

TABLE 100–11	Risk Factors for Postpartum Endometritis
Cesarean section*	
Multiple gestation	
Younger maternal age	
Long duration of labor and membrane rupture	
Internal fetal monitoring	
Low socioeconomic level	
Digital examination after 37 wk of gestation	
Maternal human immunodeficiency virus infection	

*Most significant risk factor.

TABLE 100–12	Inpatient Treatment of Postpartum Endometritis*
Cefoxitin (2 grams IV every 6 h) and vancomycin 1 gram IV every 12 h	
OR	
Cefotetan (2 grams IV every 12 h) and vancomycin 1 gram IV every 12 h	
OR	
Cefotaxime (2 grams IV every 6 h) and vancomycin 1 gram IV every 12 h	
OR	
Clindamycin (500 milligrams IV every 6 h) *plus* gentamicin (4.2 milligrams/kg IV daily) and vancomycin 1 gram IV every 12 h	
OR	
Ampicillin (2 grams IV every 4 h) *plus* gentamicin (4.2 milligrams/kg IV daily) and vancomycin 1 gram IV every 12 h	
OR	
Metronidazole (500 milligrams IV every 8 h) *plus* ampicillin (2 grams IV every 4 h) *plus* an aminoglycoside and vancomycin 1 gram IV every 12 h	

*Consult the hospital pharmacist to determine if the antibiotic regimen selected is appropriate for breastfeeding.

TABLE 101–1	Equipment and Supplies for Emergency Delivery
Sterile gloves	
Sterile towels and drapes	
Povidone-iodine (Betadine) to cleanse the perineum	
Sterile lubricant gel	
Sterile scissors	
Kelly clamps	
Cord clamps	
Rubber suction bulb	
Towel or blanket for the infant	
Gauze sponges (4×4)	
Syringes (10 mL) and needles (22–24 gauge)	
Placenta basin	
Suture (3-0 chromic and 2-0 Vicryl) and needle driver	

Note: List excludes standard adult and neonatal resuscitation equipment.

TRANSFER OF THE PREGNANT PATIENT AND THE EMERGENCY MEDICAL TREATMENT IN ACTIVE LABOR ACT

There are times when a pregnant woman must be transferred to another hospital. Emergency physicians who work in a hospital that does not provide obstetric services should be familiar with the protocols in place for transfer and inpatient care of such patients. The **Emergency Medical Treatment and Active Labor Act** specifically addresses the care of pregnant women before and during transfer (see chapter 303, Legal Issues in Emergency Medicine).

A woman having contractions is considered to have an emergency medical condition if there is insufficient time for transfer before delivery or if the transfer may pose a threat to the health or safety of the child. In such a situation, the patient should not be transferred prior to delivery unless the patient requests transfer. If contractions are not present, an emergency medical condition is not automatically present, and the regular rules of providing medical care and determining the need for transfer of patients apply.

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REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

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CHAPTER 101

Emergency Delivery

Sarah Elisabeth Frasure

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INTRODUCTION AND EPIDEMIOLOGY

The thought of a woman presenting to the ED in active labor is justifiably a cause for anxiety—the emergency physician must contend not only with the often rusty recollection of the stages of normal delivery, but also with the knowledge that there are serious and even fatal complications associated with labor. Maternal and fetal survival may depend on the ability to successfully manage pre-eclampsia, eclampsia, hemorrhage, shoulder dystocia, malpresentation, cord prolapse, breech delivery, or fetal distress. Every ED should be prepared to take care of a woman in active labor. Tools include a basic delivery kit, an infant warmer or isolate, and medical supplies and equipment for neonatal resuscitation (see chapter 108, "Resuscitation of Neonates" and **Tables 101-1** and **101-2**).

Out-of-hospital births occurred in 1.36% of births in 2012, and out-of-hospital births in the United States had a lower risk profile than in-hospital births, so that fewer teen, preterm, low-birth-weight, and multiple births occurred out of hospital.[1] Occasionally, planned home deliveries experience medical complications and require rapid transport to the ED to assist with labor and delivery. In a prospective study of home births in the United States and Canada, nearly 12% of 5400 women who had planned a home delivery ultimately required urgent transfer to a hospital during the course of labor. The majority of *intrapartum* transfers were performed for failure to progress, need for pain management, and maternal exhaustion. *Postpartum* transfers encompassed a variety of obstetric and/or neonatal complications such as maternal hemorrhage, retained placenta, or newborn respiratory distress.[2]

Out-of-hospital deliveries may also occur due to inadequate or non-existent prenatal care, transportation difficulties, remote setting, or the onset of premature labor. Occasionally, a woman may also attempt to avoid the hospital/physician fees associated with pregnancy until the delivery of her child, presenting to a hospital for the first time when in active labor.

The development of obstetric centers for high-risk pregnancy has led to a significant decline in neonatal mortality in the United States, and transports to specialized units have increased. The most common reasons for transport include preterm labor (41%), premature rupture of membranes (21%), hypertensive disease (16%), and antepartum hemorrhage (13%).[3] Other indications for transport include eclampsia or pre-eclampsia, fetal distress, multiple gestation, fetal anomalies, and maternal health problems, including traumatic injuries. EMS units transporting an actively laboring patient should carry sterile delivery packs, relevant medical supplies (Table 101-1), and appropriate medications (Table 101-2). The transport team should be trained to assist in the precipitous delivery of an infant. Prehospital protocols regarding the complications of labor and delivery should be reviewed regularly to ensure that EMS personnel are adequately prepared for both normal delivery and potentially catastrophic pregnancy-related events.

For deliveries in an austere environment or in a disaster zone, the United Nations Population Fund provides a vaginal delivery kit for use during disaster relief, which includes a plastic sheet to lay on the ground, soap for washing hands and the perineum, string and a razor blade to tie and cut the umbilical cord, and a blanket to protect the newborn baby.[4]