



# Cutting the legal risks of hypertension in pregnancy

■ BY BAHAM. SIBAI, MD

What conditions, assumptions, and oversights increase an Ob/Gyn's vulnerability to legal claims involving hypertensive gravidas? An expert zeroes in and offers steps to reduce liability.

**H**ypertensive disorders are the most common medical complications of pregnancy, with a reported incidence in the United States of 6% to 8%.<sup>1,2</sup> These disorders are associated with an increased risk of

maternal and perinatal mortality and morbidity (TABLE 1).<sup>3</sup> Not surprisingly, they are a major cause of litigation against physicians and hospitals, most of it alleging misdiagnosis and/or mistreatment. In this article, I outline

**TABLE 1****Adverse outcomes in hypertensive disorders of pregnancy**

• Maternal complications
- Abruptio placentae
- Disseminated intravascular coagulopathy
- Eclampsia
- Renal failure
- Liver hemorrhage or failure
- Intracerebral hemorrhage
- Hypertensive encephalopathy
- Pulmonary edema
- Death
• Fetal-neonatal complications
- Severe IUGR
- Preterm delivery
- Hypoxia-acidosis
- Neurologic injury
- Death
IUGR=intrauterine growth restriction

precautions that can reduce or prevent the risk of medicolegal claims involving hypertension in pregnancy. Among the issues covered are terminology, diagnosis, management, and complications of the disorders, based on the literature, a review of legal claims, and personal experience.

**Definitions and terminology**

**H**ypertension in pregnancy is the term typically used to describe a wide spectrum of patients whose disorders range from mild elevations in blood pressure (BP) to severe hypertension and organ dysfunction. Hypertension may be present before pregnancy, or it may become evident before 20 weeks' gestation, in the second trimester, at term, during labor and delivery, or in the immediate (48 hours or less after delivery) or late (3 to 28 days after delivery) postpartum period.

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Unfortunately, the terminology used to describe these disorders is confusing and inconsistent. For example, "pregnancy-induced hypertension" is vague and broad and should not be employed in clinical practice. In the medical record, it is more advisable to describe exactly what the findings are, e.g., chronic hypertension, gestational hypertension (new-onset hypertension after 20 weeks' gestation), new-onset proteinuria (1+ or greater on at least 2 occasions), or a combination of findings.

The physician also should document whether the hypertension is mild or severe, as well as the presence or absence of associated symptoms such as persistent headache, visual changes, mental changes, epigastric or right

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**KEY POINTS**

- All gravidas are at risk for hypertension and preeclampsia in the antepartum, intrapartum, and postpartum periods.
- In a patient who was previously normotensive, the diagnosis of hypertension should be based on blood-pressure criteria rather than threshold-increase criteria, since a gradual increase in blood pressure from the first to third trimesters is seen in 67% to 75% of normotensive pregnancies.
- The ultimate goals of therapy for hypertensive disorders must always be safety of the mother first and then delivery of a live, mature infant who requires no intensive and prolonged neonatal care.
- Antihypertensive medications should not be used to control blood pressure on an outpatient basis in women with preeclampsia.
- Common themes in medicolegal claims are the assumption by health-care providers that a patient's proteinuria resulted from sample contamination or urinary tract infection, and a failure to appreciate the clinical significance of patient complaints during telephone calls.
- Complications related to HELLP syndrome are a major cause of litigation claiming failure to diagnose preeclampsia or failure to diagnose syndromes that can mimic preeclampsia.

**TABLE 2**

**Classification**

I. Gestational hypertension
Mild:
- Systolic, 140-160 mm Hg or
- Diastolic, 90-110 mm Hg
Severe:
- Systolic >160 mm Hg or
- Diastolic >110 mm Hg
II. Gestational proteinuria
Mild (≥1+ on dipstick and <5 g/24 hr)
Severe (≥5 g/24 hr)
III. Preeclampsia (hypertension + proteinuria)
Onset >20 weeks' gestation
Mild:
Mild hypertension and mild proteinuria
Severe:
Severe hypertension and proteinuria
Mild hypertension and severe proteinuria
Persistently severe cerebral symptoms
Thrombocytopenia
IV. Chronic hypertension
Hypertension before pregnancy
Hypertension before 20 weeks' gestation
V. Superimposed preeclampsia
Hypertension and new-onset proteinuria

upper quadrant (RUQ) pain, shortness of breath, or vaginal bleeding. The term “preeclampsia” should be used only to describe patients who have persistent hypertension and new-onset proteinuria or associated symptoms. The term “HELLP syndrome” (hemolysis, elevated liver proteins, and low platelets) should be used only when a woman with suspected or confirmed preeclampsia has documented evidence of hemolysis (elevated lactate dehydrogenase [LDH] or bilirubin) plus elevated liver enzymes (aspartate aminotransferase [AST] or alanine aminotransferase [ALT]) and thrombocytopenia (platelet count below 100,000).

Unless all of these elements are present, the medical record should describe only preeclampsia with either elevated liver enzymes or thrombocytopenia.

**Diagnosing hypertension**

Hypertension is the hallmark of these disorders. In a woman who was previously normotensive, its diagnosis should be based on BP criteria, e.g., at least 140 mm Hg systolic and/or 90 mm Hg diastolic. Because the hypertensive readings must be present on 2 occasions at least 6 hours apart, it is important to obtain and record BP at each visit during pregnancy.

The threshold-increase criteria (i.e., relative hypertension) are inadequate to diagnose gestational hypertension or preeclampsia, since a gradual increase in BP from the first to third trimesters is seen in 67% to 75% of normotensive pregnancies.<sup>4</sup> However, these criteria signal the need for close observation of the patient, particularly when they are accompanied by generalized edema, proteinuria, and other symptoms such as headaches, blurred vision, or epigastric pain. In fact, the presence of these symptoms has a greater bearing on pregnancy outcome than the absolute level of blood pressure.

A simple clinical classification system for hypertensive disorders of pregnancy is listed in TABLE 2.

**Proteinuria**

Proteinuria is usually detected by urine dipstick or the sulfosalicylic acid cold test in random urine samples. The concentration of protein in random samples is highly variable and influenced by several factors, particularly vaginal secretions, urinary tract infection, and activity. Several clinical studies define abnormal proteinuria as at least 1+ on dipstick on 2 occasions 6 or more hours apart.<sup>1,2</sup>

A common theme in medicolegal claims is the assumption by health-care providers that proteinuria resulted from sample con-

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**TABLE 3**

**Risk factors for preeclampsia**

• Nulliparity
• Obesity
• Multiple partners
• Multifetal gestation
• Preeclampsia-eclampsia in previous pregnancy
• Chronic hypertension/renal disease
• Insulin-dependent diabetes mellitus
• Evidence of fetal growth retardation in current pregnancy
• Presence of thrombophilia
• Persistent proteinuria in current pregnancy
• Relative hypertension in association with symptoms
• Abruptio placentae in current pregnancy

tamination or urinary tract infection. To avoid this problem, dipstick proteinuria should be confirmed by catheterized urine sample, urine culture, or—if necessary—by 12- to 24-hour urine collection.

An abnormal proteinuria of 2+ on dipstick or more than 300 mg per 24-hour collection in association with hypertension establishes the diagnosis of preeclampsia. The physician should document this finding and explain to the patient that it increases the likelihood of convulsions, abruptio placentae, fetal growth retardation, and preterm delivery. The risks for these complications will depend on gestational age at onset, as well as the severity of the abnormalities.

**Identifying women at risk**

All pregnant women are at risk for hypertension and preeclampsia in the antepartum, intrapartum, and postpartum periods. Therefore, the clinician should measure BP, urine protein, and weight at each prenatal visit. He or she also should measure BP and urine protein during any visits to triage or the emergency room. In addition, BP should be monitored during labor and postpartum.

Among the risk factors for preeclampsia are nulliparity, obesity, chronic hypertension, and multifetal gestation (TABLE 3). These risk factors also should be used to identify women at risk for severe hypertension or preeclampsia in the early or late postpartum period. Just as important is educating and instructing women to report the symptoms of preeclampsia in the antepartum and postpartum periods.

**Antepartum management**

The ultimate goals of therapy for these disorders must always be safety of the mother first and then delivery of a live, mature infant who requires no intensive and prolonged neonatal care.<sup>5</sup> The choice between outpatient management or hospitalization and expectant management or consideration for delivery usually depends on 1 or more of several factors (TABLE 4). Thus, it is important to document these findings and discuss management options with the patient.

**Mild gestational hypertension.** In clinical practice, most women with hypertensive disorders have mild gestational hypertension, and pregnancy outcomes are usually good. However, about 25% of these patients may progress to preeclampsia and be at slightly increased risk for fetal growth retardation (5% to 10%), abruptio placentae (0.06% to 0.08%), HELLP syndrome (1%), and eclampsia (0.1%).<sup>6</sup> Thus, those who have a Bishop cervical score of more than 5 at or near term should undergo induction of labor for delivery. Even if conditions for induction are unfavorable, the pregnancy should not continue past 40 weeks' gestation. Cervical ripening and induction of labor should be performed.<sup>5</sup>

For patients remote from term (i.e., less than 37 weeks' gestation), management should include restricted activity at home, close observation of maternal BP and urine protein, weekly checks of platelet count and liver enzymes, and evaluation of fetal status with ultrasonography and nonstress testing. Ultrasound assessment of fetal growth should

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**TABLE 4****Clinical factors to be considered in management**

• Diagnosis of the condition
• Severity of the disease process
• Fetal gestational age
• Fetal growth and well-being
• Maternal condition
- Results of blood tests
- Presence of symptoms
• Presence of labor
• Bishop cervical score

be performed at the time of diagnosis and repeated every 3 weeks. Nonstress testing should be performed at the same time and repeated if there is a change in maternal condition or evidence of abnormal fetal growth. In addition, as mentioned, the patient should be instructed to remain alert for symptoms of preeclampsia.

The frequency of subsequent office visits, as well as the need for fetal testing, will depend on initial clinical findings and the ensuing progression. If the maternal condition remains stable (i.e., no excessive weight gain, proteinuria, or significant change in blood pressure; appropriate fundal height growth; and a stable fetal kick count), weekly visits are appropriate. Onset of maternal symptoms, a sudden increase in BP, or development of proteinuria requires more frequent evaluation, preferably in the hospital.<sup>1,2</sup>

Most medicolegal claims involving these patients concern issues such as patient selection for outpatient management, documentation of instructions and patient options, and a failure to appreciate the clinical significance of patient complaints during telephone calls.

Outpatient or home management should be considered only for patients who are highly reliable. (Reliable patients keep their appointments and have immediate transportation, phone access, and family members readily available.) Patients should be coun-

seled to rest at home and to avoid operating a car, as well as to immediately report symptoms such as headache, blurred vision, epigastric or abdominal pain, nausea or vomiting, contractions, and vaginal bleeding.

One way to avoid communication problems is to instruct the patient to mention that she was diagnosed with gestational hypertension when talking with office or hospital nursing or clerical staff, another doctor in the same practice who may not be familiar with her case, or personnel at the answering service.<sup>5</sup> A policy should be implemented whereby such calls are communicated to the physician as soon as possible. The presence of the aforementioned symptoms—particularly if they are persistent—is an indication for immediate evaluation at the office or hospital.

**Severe gestational hypertension.** Women with severe gestational hypertension are at increased risk for adverse maternal and perinatal outcomes.<sup>7</sup> These women should be managed similarly to those who have severe preeclampsia (see page 56). Therefore, a diagnosis of severe gestational hypertension requires immediate hospitalization.

Antihypertensive medications should not be used to control BP on an outpatient basis

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**TABLE 5****Indications for delivery in mild preeclampsia**

• Gestational age $\geq 37$ weeks with ripe cervix
• Onset of labor and/or membrane rupture $\geq 34$ weeks
• Onset of persistent headaches or visual symptoms
• Epigastric or RUQ pain
• Abdominal tenderness or vaginal bleeding
• Thrombocytopenia (platelets $< 100 \times 10^3/\text{mm}^3$ )
• Severe oligohydramnios or IUGR by ultrasound
• Abnormal FHR testing*
FHR=fetal heart rate; IUGR=intrauterine growth restriction; RUQ=right upper quadrant
* Nonreactive nonstress test confirmed by biophysical profile or contraction test.

**TABLE 6**

**Characteristics of patients eligible for outpatient management of mild preeclampsia**

• Systolic pressure $\leq$ 150 mm Hg or diastolic pressure $\leq$ 100 mm Hg
• Proteinuria $\leq$ 1,000 mg/24 hr or $\leq$ 2+ on dipstick
• No labor
• Highly reliable
• Absent cerebral signs and symptoms
• Absent epigastric or upper quadrant pain
• Absent fetal growth retardation by ultrasound
• Normal antepartum fetal testing
• Normal liver enzymes and platelet count

in these women. Following hospitalization, women with severe gestational hypertension should undergo evaluation of maternal and fetal status, including a platelet count, liver enzymes, and 24-hour urine protein. Fetal evaluation should include ultrasound examination of fetal weight and fluid and nonstress testing or a biophysical profile, as indicated. As discussed, subsequent management depends on the factors listed in TABLE 4.

**Mild preeclampsia.** Pregnancies complicated by mild preeclampsia, particularly those at less than 36 weeks' gestation, are associated with increased rates of fetal growth retardation (10% to 15%), preterm delivery (50%),

**TABLE 7**

**Antepartum management of mild preeclampsia**

• Relative bed rest (hospital or home)
• Blood pressure measurement (daily)
• Dipstick for urine protein (daily)
• Evaluation of symptoms (daily)
• Platelet count and liver enzymes (2x/week)
• Fetal movement counts (daily)
• Antepartum fetal testing (2x/week)
• Ultrasound for fetal growth (every 3 weeks)

abruptio placentae (1% to 2%), and eclampsia (1%).<sup>6</sup> The first step in managing these patients is prompt evaluation of maternal and fetal conditions. A decision can then be made regarding the need for delivery, expectant management, or hospitalization.

Maternal evaluation should include serial measurements of blood pressure, urine dipstick evaluation for proteinuria, 24-hour urine protein measurements, a platelet count, liver enzymes, and documentation of any symptoms. Fetal evaluation should include ultrasound assessment of the amniotic fluid and estimated fetal weight, as well as a nonstress test.

Indications for delivery include persistent headaches or visual symptoms, epigastric or RUQ pain, and thrombocytopenia (TABLE 5). Some women may be eligible for outpatient management with rest at home if they satisfy the criteria listed in TABLE 6. Subsequent management is outlined in TABLE 7.

If a woman is managed as an outpatient, prompt hospitalization is indicated if there is any evidence of disease progression (e.g., significant changes in BP or proteinuria, excessive weight gain, new symptoms) or abnormal fetal growth. Antihypertensive medications should be avoided during outpatient management. Women should be counseled to report the same symptoms and changes in fetal movement described for patients with gestational hypertension. When calls are made to office or hospital personnel and the on-call physician, patients should explain that they have been diagnosed with preeclampsia. Finally, women should be given specific phone numbers to call and be advised when to report immediately to the hospital.

**Severe preeclampsia.** Pregnancies complicated by severe preeclampsia usually are marked by a significant reduction in uteroplacental and fetoplacental blood flow. This reduction is particularly pronounced in women who develop severe preeclampsia at less than 32 weeks' gestation. Thus, these



**TABLE 8****Indications for delivery during expectant management of severe preeclampsia**

• Fetal indications:
- 34 weeks' gestation
- 33-34 weeks with documented lung maturity or after steroid use
- Estimated fetal weight below the fifth percentile by ultrasound
- Evidence of severe oligohydramnios
- Abnormal fetal testing
- Rupture of membranes
• Maternal indications:
- Preterm labor or vaginal bleeding
- Eclampsia or encephalopathy
- Pulmonary edema or renal failure
- Persistent oliguria despite therapy
- Persistent thrombocytopenia
- Severe epigastric pain or cerebral symptoms
- Maternal desire
- Severe hypertension unresponsive to maximum drug therapy

pregnancies are associated with high rates of perinatal mortality and morbidity, mainly because of severe fetal growth retardation and preterm delivery.<sup>7</sup> They also may be associated with increased rates of maternal morbidity, such as HELLP syndrome, disseminated intravascular coagulopathy (DIC) (from severe abruptio placentae), pulmonary edema, eclampsia, or acute renal failure. Consequently, the onset of severe preeclampsia requires immediate hospitalization and intensive monitoring of maternal and fetal conditions.<sup>8</sup>

The first steps in management are administering magnesium sulfate to prevent convulsions<sup>9</sup> and antihypertensive drugs to control extreme levels of hypertension, as well as evaluation of the patient for the presence of symptoms and/or hemolysis, elevated liver enzymes, elevated serum creatinine, and thrombocytopenia.<sup>8</sup>

In general, delivery is considered appropriate for the mother regardless of gestational age. Unfortunately, it may not be appropriate for the

fetus, particularly when the gestational age is less than 33 weeks. Consequently, expectant management—in an attempt to prolong pregnancy in the interest of fetal viability—may be appropriate for gestations between 24 and 32 weeks. However, it is appropriate only in a select group of patients and should be practiced only in a tertiary-care center with adequate maternal and neonatal facilities. Patients should be informed of the risks and benefits of such management, which requires close daily monitoring of maternal-fetal conditions. It is recommended that such patients be managed in consultation with a perinatologist.

Antepartum fetal testing and maternal liver enzymes and platelet counts should be performed daily. Indications for delivery during expectant management are listed in **TABLE 8**.

**HELLP syndrome.** Hemolysis (H), elevated liver proteins (EL), and low platelets (LP)—or a combination of these (HELLP)—are well-recognized complications of preeclampsia-eclampsia. Several of the signs, symptoms, and laboratory abnormalities that constitute HELLP syndrome may be confused with similar findings that are usually present in a number of distinct medical and surgical disorders (**TABLE 9**). This is particularly true when they manifest remote from term. Diagnosis often is delayed, and management frequently is complicated by inappropriate medical and surgical treatments that may be dangerous to both mother and fetus. Not surprisingly, complications related to this syndrome are a major cause of litigation claiming failure to diagnose preeclampsia or failure to diagnose syndromes that can mimic preeclampsia.

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**TABLE 9**

**Imitators of HELLP syndrome**

• Acute fatty liver of pregnancy
• Thrombotic thrombocytopenia purpura
• Hemolytic uremic syndrome
• Exacerbation of lupus nephritis
• Thrombophilias
• Disseminated herpes simplex
• Acute glomerulonephritis
• Immune thrombocytopenia
• Septic shock
• Gall bladder disease/pancreatitis
• Hypertensive encephalopathy
• Intracerebral hemorrhage
HELLP=hemolysis, elevated liver proteins, and low platelets

The typical HELLP patient is white and complains of epigastric or RUQ pain at less than 36 weeks' gestation. Some women experience nausea and vomiting (40% to 50%) or diarrhea (5% to 10%), and others have non-specific viral syndrome-like symptoms. Most patients report a history of malaise for the few hours or days leading up to presentation. Some of these women may complain of flank or shoulder pain, hematuria, bleeding from the gums, or jaundice. Hypertension or pro-

**Patients with preeclampsia are at increased risk for convulsions during labor and for the first 12 to 24 hours postpartum.**

teinuria may be slight or absent. Physical examination will show RUQ tenderness, significant weight gain (ascites, generalized edema), petechiae, or purpura.

Some patients may have a variety of signs and symptoms, none of which are diagnostic of classic preeclampsia. For this reason, all gravidas beyond 20 weeks' gestation having any of these symptoms should undergo a platelet count and liver enzyme determinations, regard-

less of maternal BP. Since 30% of HELLP syndrome cases develop postpartum, similar evaluation should be performed in all women having these complaints within 2 weeks of delivery.

**Intrapartum management**

Some of the medicolegal claims involving intrapartum management of hypertensive disorders concern the failure to give magnesium sulfate to prevent convulsions, the failure to distinguish bleeding of a "bloody show" from abruptio placentae, the failure to use antihypertensive drugs to treat a certain BP level, the mode of delivery, and the association of abnormal fetal-heart-rate (FHR) tracings with neonatal outcome.

In some women, hypertension first develops in labor. These patients should undergo urine protein and blood tests to confirm the presence or absence of preeclampsia and should then be managed accordingly.

**Preventing convulsions.** Patients with preeclampsia are at increased risk for convulsions during labor and for the first 12 to 24 hours postpartum. The risks are slight in those with mild disease (1%), but may reach 3% to 10% in women with severe disease at less than 32 weeks' gestation and in patients with HELLP syndrome.<sup>9</sup> Therefore, all patients with diagnosed preeclampsia should receive parenteral magnesium sulfate during labor and for at least 12 hours postpartum; in patients with severe preeclampsia or HELLP syndrome, magnesium sulfate should be continued for at least 24 hours postpartum. A typical regimen is a loading dose of 6 g given over 20 minutes, followed by a maintenance dose of 2 g per hour as a continuous intravenous (IV) solution (5% dextrose in lactated Ringer's solution).

Magnesium sulfate should be initiated at the beginning of labor induction or, in women scheduled to undergo elective cesarean section, at least 1 hour prior to surgery.

Excessive doses of magnesium sulfate can lead to toxicity that could prove fatal to the

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mother or fetus. Therefore, specific protocols should be used to monitor all women receiving this drug. All such patients should have their reflexes checked every hour, urine-out measured hourly, and respiratory rate monitored closely. Magnesium sulfate should never be given as an IV bolus. It is recommended that the loading and maintenance doses be prepared premixed in an IV solution to ensure the proper concentration. The solution should be infused using a standard infusion pump. Serum magnesium levels should be obtained on a regular basis only in women with abnormal renal function (serum creatinine of 1.2 mg or more per deciliter), reduced urine output (less than 100 mL in 4 hours), or absent reflexes. In these cases, the infusion of magnesium sulfate should be discontinued or reduced according to maternal serum level.

In general, magnesium will not reach toxic levels until 4 hours or more after a maintenance dose. If the patient develops muscle weakness (double vision, inappropriate speech, respiratory difficulty) or shallow or absent respiratory effort or cardiac arrest, discontinue magnesium sulfate immediately, administer 1 g calcium gluconate in an IV (slowly, over 5 to 10 minutes), and intubate and ventilate the patient artificially. Subsequent management depends on the magnesium level.

**Diagnosis of abruptio placentae.** The slightly increased risk of abruptio placentae in patients with hypertensive disorders is not related to hypertension, but rather to the presence of preeclampsia. The highest risk usually is seen in patients who are suffering or just suffered from convulsions or in women with HELLP syndrome. Thus, it is important to document that patients were notified to report the onset of abdominal tenderness or vaginal bleeding. Also, instruct nursing personnel to report these findings to the physician as soon as they occur. The physician should evaluate the patient immediately.

Early in labor, bleeding may signify a “bloody show,” particularly when it is mini-

mal, brief, and not associated with uterine contractions or FHR patterns. Abruptio placentae should be suspected in the presence of tetanic contractions, more than 5 uterine contractions in 10 minutes, or repetitive late decelerations or fetal bradycardia. Also suspect abruptio placentae if bleeding is persistent in the absence of placenta previa.

### Antihypertensive therapy

The objective of treating severe hypertension is to prevent cerebrovascular accidents without compromising uteroplacental blood flow, which is already reduced in severe preeclampsia-eclampsia. There is considerable confusion regarding what BP level to treat and the desired level to achieve and maintain during treatment. Though there is little to no information regarding what systolic BP level to treat, most studies and textbooks recommend treatment if it exceeds 110 mm Hg.<sup>2</sup> Some require this elevation to persist for at least 6 hours, some for 1 hour, and

**There is considerable confusion regarding what BP level to treat and the desired level to achieve and maintain during treatment.**

others for 30 minutes. The general recommendation is to lower the diastolic level to below 100 mm Hg, keeping it between 90 and 100 mm Hg.<sup>1,2</sup> A recent consensus report and American College of Obstetricians and Gynecologists (ACOG) practice bulletin recommend treating diastolic blood pressure levels as low as 105 to 110 mm Hg, but no specific time period is described.<sup>1,2</sup> A review of all such recommendations—none of which are based on scientific data—found considerable inconsistencies. Nonetheless, these recommendations have prompted expert witnesses in several malpractice cases to testify that diastolic BP as low as 100 mm Hg and systolic

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**TABLE 10**

**Indications for antihypertensive therapy**

<b>I. Antepartum and intrapartum</b>
<ul style="list-style-type: none"> <li>• Persistent elevations for at least 1 hour:             <ul style="list-style-type: none"> <li>• Systolic BP <math>\geq</math>180 mm Hg or</li> <li>• Diastolic BP <math>\geq</math>110 mm Hg or</li> <li>• MAP <math>\geq</math>130 mm Hg</li> </ul> </li> <li>• Persistent elevations for at least 30 minutes:             <ul style="list-style-type: none"> <li>• Systolic BP <math>\geq</math>200 mm Hg or</li> <li>• Diastolic BP <math>\geq</math>120 mm Hg or</li> <li>• MAP <math>\geq</math>140 mm Hg</li> </ul> </li> <li>• Thrombocytopenia or congestive heart failure*             <ul style="list-style-type: none"> <li>• Systolic BP <math>\geq</math>160 mm Hg or</li> <li>• Diastolic BP <math>\geq</math>105 mm Hg or</li> <li>• MAP <math>\geq</math>125 mm Hg</li> </ul> </li> </ul>
<b>II. Postpartum**</b>
<ul style="list-style-type: none"> <li>• Systolic BP <math>\geq</math>160 mm Hg or</li> <li>• Diastolic BP <math>\geq</math>105 mm Hg or</li> <li>• MAP <math>\geq</math>125 mm Hg</li> </ul>
<p>MAP=mean arterial pressure            * persistent for at least 30 minutes            ** persistent for at least 1 hour</p>

BP as low as 160 mm Hg should be treated.

Preeclampsia is characterized by endothelial cell injury as well as vasospasm. These vascular pathologic changes have been described in cerebral blood vessels using Doppler velocimetry, angiography, computerized tomography (CT) scanning, magnetic resonance imaging (MRI), and autopsy. Some of these patients are predisposed to cerebral ischemic changes and stroke as a result of the disease process itself. Also, some patients with severe preeclampsia-eclampsia will have thrombocytopenia and DIC because of either the illness's progression or abruptio placentae. These patients also are predisposed to intracerebral hemorrhage at normal or mildly elevated blood pressures. Finally, some

patients with preeclampsia have a propensity to develop cerebral complications because of the aforementioned abnormalities. In such cases, the presence of hypertension (usually fluctuating with wide pulse pressure) is the result of intracerebral hemorrhage, rather than the cause. Consequently, any aggressive or rapid reduction in maternal BP may result in reduced cerebral blood flow, producing cerebral ischemia to normal brain tissue.

Cerebral tissue perfusion is directly related to mean arterial pressure (MAP), which is calculated from both systolic and diastolic pressures:

$$MAP = \frac{1(\text{systolic}) + 2(\text{diastolic})}{3}$$

In the nonpregnant state, there is a loss of cerebral autoregulation when MAP exceeds 150 mm Hg. This results in cerebrovascular injury leading to hypertensive encephalopathy or hemorrhage. The upper limit of cerebral autoregulation in pregnancy is unknown, and most recommendations for treating blood pressure have focused on diastolic BP values. The maximum duration of sustained hypertension before starting therapy is unknown.

In view of these inconsistencies, and to avoid potential medicolegal claims, the following steps are recommended:

- Antihypertensive treatment should be started in accordance with the information in TABLE 10. The aim of therapy is to keep MAP below 125 mm Hg (but not below 105 mm Hg) and diastolic pressure below 105 mm Hg (but not below 90 mm Hg). This can be achieved with a 5-mg bolus dose of hydralazine, to be repeated as needed every 15 to 20 minutes for a maximum total dose of 20 mg per hour. Blood pressure should be recorded every 15 minutes during therapy and every hour once the desired values are achieved. If hydralazine does not lower BP sufficiently and/or if maternal side effects such as tachycardia or

**TABLE 11****Symptoms patients should report in the postpartum period**

- Persistent headaches
- Visual changes
- Altered mental status
- Epigastric pain ± nausea or vomiting
- Chest pain or tightness
- Shortness of breath
- Calf tenderness or leg swelling

headache develop, another drug such as labetalol (20-mg IV bolus doses) or nifedipine (10-mg oral tablets) can be used.

- If recurrent hypertension (as previously defined) develops, antihypertensive drugs should be repeated as needed. Therefore, it is important to document serial measurements of BP during labor and postpartum in all patients with severe preeclampsia-eclampsia.

These threshold values are empiric. There is no evidence to suggest any correlation between maternal BP values and the likelihood of stroke in women with preeclampsia. Indeed, in the absence of cerebrovascular disease (aneurysms, malformations, or cerebral venous thrombosis) or severe thrombocytopenia, the likelihood of stroke in patients with severe hypertensive disorders of pregnancy was less than 0.1%—even though most of these women were untreated despite a diastolic BP exceeding 110 mm Hg for at least 3 hours.<sup>10</sup> These data fail to suggest that an increment of 5 to 10 mm Hg in diastolic pressure is going to make a difference in the development of stroke.<sup>10</sup>

Finally, it is important to obtain arteriography or autopsy in all patients with cerebral hemorrhage in pregnancy or postpartum to rule out the presence of anatomic cerebrovascular malformations.

**Fetal distress and mode of delivery**

**S**ome of the typical medicolegal claims involving fetal distress and mode of deliv-

ery concern the issue of performing a cesarean section to prevent maternal and neonatal complications in patients with preeclampsia, HELLP syndrome, or eclampsia. Vaginal delivery is the best method in these patients. Cesarean section should be reserved for obstetric indications. No limit should be placed on the duration of labor if these patients are progressing normally.

As stated previously, preeclamptic pregnancies may be associated with reduced uteroplacental blood flow and higher than normal rates of fetal growth retardation, oligohydramnios, abruptio placentae, and preterm birth. As a result, these pregnancies are more likely to have abnormal FHR patterns than normotensive pregnancies. Some of these infants will be delivered with low Apgar scores and acidotic blood gases secondary to their antenatal complications rather than labor itself. Thus, it is important to document the presence of these complications before the onset of labor, and to utilize continuous FHR monitoring during labor in all such patients.

In some patients, the presence of severe abnormal FHR patterns (absent beat-to-beat variability with repetitive late or severe variable decelerations) during labor is evidence of

**Preeclamptic pregnancies are more likely to have abnormal FHR patterns than normotensive pregnancies.**

an already compromised fetus that has suffered cerebral injury during the prenatal period or prior to monitoring. This is particularly true when the pregnancy is complicated by severe fetal growth retardation, oligohydramnios, or abruptio placentae. Therefore, delivery of such fetuses by emergency cesarean section does not guarantee that the infant will escape neurologic deficits later in life.

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Occasionally, severe FHR abnormalities or ominous patterns can be found in patients who are either unconscious after eclamptic convulsions, in shock from ruptured liver hematoma, or who have experienced severe abruptio placentae and possible coagulopathy.

**All postpartum women should be educated about the signs and symptoms of preeclampsia and severe hypertension.**

In such cases, emergency cesarean section performed due to fetal considerations may lead to a catastrophic outcome for both mother and infant. In eclamptic patients, fetal bradycardia and late decelerations are common findings during and immediately after a convulsion. These abnormalities are reversible following correction of maternal hypoxia and acidosis. These patients should be oriented to name, place, and time before being subjected to anesthesia and surgery.

### Postpartum management

**H**ypertension or preeclampsia may first develop in the postpartum period or may worsen at this time. Thus, all postpartum women should be educated about the signs and symptoms of preeclampsia and severe hypertension. In addition, health-care providers and all personnel who answer patient phone calls should be educated about what information to report to physicians.<sup>10</sup>

It is important to remember that postpartum women—particularly those with chronic hypertension or with hypertension-preeclampsia in the current pregnancy—are at risk for HELLP syndrome, eclampsia, pulmonary edema, stroke, and thromboembolic disease. Women should be instructed to report any of the symptoms listed in TABLE 11.

In the immediate postpartum period, women with preeclampsia should have accu-

rate intake and output of fluids. These women usually receive large amounts of fluids during labor as a result of prehydration prior to epidural anesthesia and IV administration of oxytocin or magnesium sulfate. They also receive fluids after delivery because of oxytocin and magnesium sulfate administration. As a result, some of these women may have abnormal renal function and capillary leakage, putting them at risk for pulmonary edema. These patients should be monitored closely for this complication.

In the late postpartum period (more than 48 hours after delivery), some women with hypertension or preeclampsia are at increased risk for complications such as severe preeclampsia, eclampsia, HELLP syndrome, congestive heart failure, and stroke.<sup>10,11</sup> Because these women usually present to the emergency department, emergency room staff and physicians often are the first to evaluate them. It is vital that these health-care professionals are educated regarding prompt evaluation and recognition of signs and symptoms of preeclampsia in the postpartum period. ■

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