The presence of gestational hypertension in a woman with progressive or excessive weight gain, generalized swelling, and new-onset or persistent headache that does not respond to analgesics is an indication for immediate hospitalization for evaluation and management. Other signs that merit hospitalization in a patient with gestational hypertension are listed on page 36.
A stepwise approach to managing eclampsia and other hypertensive emergencies

Avert eclamptic seizures by paying close attention to the patient’s history, risk factors, vital signs, and symptoms. Here, an expert outlines a systematic response to eclampsia—one that can help preserve the health of both mother and infant.

Baha M. Sibai, MD

**CASE**  Missed preeclampsia?

At her first prenatal visit at 14 weeks’ gestation, a 41-year-old woman (G2P1) presents with a dichorionic twin gestation, blood pressure (BP) of 105/68 mm Hg, and a body mass index (BMI) of 40 kg/m². The pregnancy was achieved through in vitro fertilization. Ten years earlier, the patient’s first pregnancy was complicated by preeclampsia, requiring preterm delivery at 33 weeks’ gestation.

By 28 weeks’ gestation, the patient has gained 26 lb. Her BP is 120/70 mm Hg, with no proteinuria detected by urine dipstick. By 30 weeks, she has gained an additional 8 lb, her BP is 142/84 mm Hg, and no proteinuria is detected. At 32 weeks, her BP is 140/92 mm Hg, she has gained another 8 lb, and no proteinuria is present. She also reports new-onset headaches that do not respond to over-the-counter analgesics. She is sent to the obstetric triage area for BP monitoring, blood testing for preeclampsia and nonstress test fetal monitoring.

During the 2-hour observation period, the patient continues to report headaches, and swelling of her face and hands is present. Her systolic BP values range from 132 to 152 mm Hg, and diastolic values range from 80 to 96 mm Hg. No proteinuria is detected, blood testing results for preeclampsia (complete blood count, liver enzymes, serum creatinine, and uric acid) are normal, and the nonstress tests are reactive in both fetuses.

The patient is given a diagnosis of gestational hypertension, along with a prescription for oral labetalol 200 mg daily and two tablets of acetaminophen with codeine for the headaches (to be taken every 6 hours as needed). She is sent home with instructions to return to her physician’s office in 1 week.

Two days later, she wakes in the middle of the night with a severe headache, blurred vision, and vomiting. Her husband calls the obstetrician’s answering service and is instructed to call 911 immediately. While waiting for an ambulance, the patient experiences a grand mal eclamptic convulsion. A second convulsion occurs during her transfer to the ED.

This scenario could have been avoided.

The obstetrician in this case was negligent for failing to recognize preeclampsia in a patient who had two clear risk factors.
Autoimmune or connective tissue disease is a risk factor for eclampsia and other hypertensive emergencies

Defining eclampsia
Eclampsia is marked by the onset of convulsions (during pregnancy or postpartum) in association with gestational hypertension alone, proteinuria, preeclampsia, or superimposed preeclampsia. Although it is rare, eclampsia is potentially life-threatening. For that reason, obstetricians, anesthesiologists, ED physicians, neurologists, and critical-care physicians should be well versed in its diagnosis and management. In this article, I focus on management.

A few preliminary points
Eclampsia can develop any time during the antenatal period (>16 weeks’ gestation), during labor and delivery, and as long as 6 weeks after delivery. Therefore, we should be vigilant for preeclampsia whenever a pregnant patient visits our office, as well as when she makes unscheduled visits to the ED or obstetric triage area or is hospitalized.

Early recognition of women at high risk for preeclampsia and eclampsia may allow for prompt intervention, including early hospitalization for close observation prior to delivery and postpartum.1,2,4–10

Hospitalization of high-risk women allows for use of antihypertensive agents to treat severe BP, administration of magnesium sulfate to prevent convulsions, and timely delivery of the infant. It also allows for intensive maternal support during and after an eclamptic seizure.

Hospitalization is essential for women who exhibit features that suggest severe disease. More specifically, the presence of gestational hypertension with any of the following features is an indication for immediate hospitalization for evaluation and management:

- persistent severe hypertension (systolic BP ≥160 mm Hg or diastolic BP ≥110 mm Hg) for at least 1 hour
- gestational hypertension requiring oral antihypertensive therapy
- progressive and excessive weight gain (≥20 lb prior to 28 weeks’ gestation)
- generalized swelling (edema of hands or face)
- new-onset or persistent headaches despite analgesics
- persistent visual changes (blurred vision, scotomata, photophobia, double vision)

**TABLE 1 Risk factors for eclampsia and hypertensive emergencies**

- A history of early-onset (<37 weeks) or recurrent preeclampsia in a previous pregnancy
- A history of hemolysis, elevated liver enzymes, and low platelet count (HELLP syndrome) or eclampsia in a previous pregnancy
- Nulliparous with a body mass index >30 kg/m²
- Multifetal gestation
- Molar or partial molar pregnancy
- Diabetes mellitus (type 1 or 2)
- Chronic hypertension with persistent elevated blood pressure values while taking antihypertensive medications
- Renal disease with serum creatinine levels >1.1 mg/dL
- Autoimmune/connective tissue disease (lupus, antiphospholipid syndrome)
- Hyperthyroidism
- Gestational hypertension in current pregnancy
- New-onset gestational proteinuria in current pregnancy

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Managing eclampsia

Continued from page 36

- shortness of breath, dyspnea, orthopnea, or tightness in the chest
- persistent retrosternal chest pain, severe epigastric or right upper quadrant pain
- persistent nausea, vomiting, malaise
- altered mental state, confusion, numbness, tingling, or motor weakness
- platelet count below 100 × 10^3 μL
- aspartate aminotransferase (AST), alanine aminotransferase (ALT), or lactic acid dehydrogenase (LDH) levels more than twice the upper limit of normal
- serum creatinine level >1.1 mg/dL
- suspected abruptio placentae.

A stepwise approach to eclampsia

Eclampsia is an obstetric emergency. Inadequate preparation for it or an inappropriate response to maternal and fetal conditions during and after an eclamptic convolution can be detrimental to the mother and fetus. All obstetric units should have up-to-date protocols in place and should conduct mandatory drills to prepare nursing staff, obstetric providers, and anesthesia staff working in these units to manage eclampsia.

**Step 1: Let the seizure run its course**

During a seizure, resist the impulse to administer anticonvulsive drugs, including intravenous (IV) magnesium sulfate, because most eclamptic convulsions are self-limiting. Also abstain from administering medications such as IV phentoyin, diazepam, or midazolam, as these drugs are less effective than magnesium sulfate, and some can suppress the laryngeal reflex, increasing the risk of aspiration.

If the patient develops status epilepticus, initiate muscle paralysis and intubate her.

**Step 2: Support the maternal condition**

It is vital to support maternal respiratory and cardiovascular functions to prevent hypoxia, acidosis, and cardiorespiratory arrest.

Begin by establishing airway patency and maternal oxygenation during and after the convolution. Administer oxygen via a face mask, with or without a reservoir, at a rate of 8 to 10 L/min.

During the apneic period (see “Profile of an eclamptic seizure” on page 46), the patient will develop hypoxia. Use pulse oximetry to monitor oxygen saturation, with the goal of keeping it above 94%. Arterial blood gas analysis is required if oxygen saturation remains below 92% or if pulmonary edema or aspiration is suspected.
If the patient develops recurrent seizures, status epilepticus, florid alveolar pulmonary edema, or respiratory arrest, intubate her immediately.

**Step 3: Prevent maternal injury and aspiration**
Secure the side rails of the patient’s bed by elevating them to prevent a fall, and make sure they are padded to prevent trauma during convulsions and afterward, when some women become combative and agitated. Position the patient in a lateral decubitus position to minimize aspiration of oral secretions. If any secretions or vomitus are present, remove them via suction.

**Step 4: After the convulsion, give magnesium sulfate**
Magnesium is the drug of choice for seizure prophylaxis in women with preeclampsia and severe symptoms, and to prevent recurrent seizures in women with eclampsia.

In the latter group, once the eclamptic convulsion has ended, give a loading dose of

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**TABLE 2** Potential maternal and perinatal complications in eclampsia and hypertensive emergencies

<table>
<thead>
<tr>
<th>Maternal</th>
<th>Perinatal</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Death</td>
<td>• Fetal or neonatal death</td>
</tr>
<tr>
<td>• Cerebral ischemia or hemorrhage</td>
<td>• Fetal growth restriction</td>
</tr>
<tr>
<td>• Seizures</td>
<td>• Hypoxia or acidosis</td>
</tr>
<tr>
<td>• Residual long-term neurologic deficits</td>
<td>• Preterm birth</td>
</tr>
<tr>
<td>• Pulmonary edema/congestive heart failure</td>
<td>• Long-term deficits</td>
</tr>
<tr>
<td>• Aspiration pneumonia</td>
<td></td>
</tr>
<tr>
<td>• Myocardial ischemia/ventricular arrhythmias</td>
<td></td>
</tr>
<tr>
<td>• Hepatic ischemia/hemorrhage</td>
<td></td>
</tr>
<tr>
<td>• Acute renal failure</td>
<td></td>
</tr>
<tr>
<td>• Abruptio placentae/disseminated intravascular coagulation</td>
<td></td>
</tr>
</tbody>
</table>

Continued on page 42
Suspect pulmonary edema if the patient has respiratory symptoms in association with tachypnea, tachycardia, or sustained oxygen saturation values below 93%.

IV magnesium (6 g/100 mL over 20 minutes), followed by a continuous infusion of 2 g/h for at least 24 hours. If the patient develops a second seizure during the maintenance infusion, administer another bolus of magnesium (2 g/100 mL over 3–5 minutes).

**Step 5: Treat severe hypertension**

If severe hypertension persists for 60 minutes or longer, it can lead to injury of the brain, heart, and kidneys. To avoid these complications, it is essential to reduce BP to a safe range and maintain that level without compromising cerebral perfusion pressure and uteroplacental blood flow (which already may be reduced in some patients).

**The goal of antihypertensive therapy is to keep systolic BP between 140 and 155 mm Hg and diastolic values between 90 and 105 mm Hg.**

Several agents are available for the treatment of severe hypertension during pregnancy and postpartum. The most commonly used IV medications for this purpose are labetalol and hydralazine. Another option is oral, rapidly acting nifedipine.

Several randomized trials have compared efficacy and side effects between IV bolus injections of hydralazine; IV labetalol; and oral, rapidly acting nifedipine. In general, the findings of these studies suggest that either IV hydralazine or labetalol or oral nifedipine can be used to treat severe hypertension in pregnancy, as long as the provider is familiar with the dose to be used, the expected onset of action, and potential side effects (TABLE 3).

Women who develop generalized swelling or hemoconcentration (hematocrit ≥40%), or both, usually experience markedly reduced plasma volume. For this reason, these women will benefit from treatment with labetalol. If this is ineffective, then add IV hydralazine. However, delay administration of a rapidly acting vasodilator such as hydralazine to prevent an excessive hypotensive response and a secondary reduction in tissue perfusion and uteroplacental blood flow. Rather, administer a bolus infusion of 250 to 500 mL of isotonic saline before giving a vasodilator.

Additional details about the use of antihypertensive drugs are given in the section on other hypertensive emergencies, which begins on page 44.

**Step 6: Evaluate the patient for complications**

**Pulmonary edema** can develop in patients with eclampsia or another hypertensive emergency. Suspect it if the patient has respiratory symptoms in association with tachypnea, tachycardia, or sustained oxygen saturation values below 93%, as well as when the patient exhibits basal rales during auscultation of the lungs. Treatment involves the
Rapid reduction of systolic BP can lead to marked reductions in uteroplacental blood flow and a nonreassuring FHR tracing.

Administration of oxygen and IV furosemide (20–40 mg push), repeated as needed.

Some women with eclampsia may develop severe cerebral edema, hemorrhage, or both. The edema can be vasogenic or cytotoxic, leading to increased intracerebral pressure. Suspect edema or hemorrhage if the patient remains unresponsive, continues to experience convulsions despite therapy, or exhibits sensory or motor neurologic deficits. In such cases, neuroimaging is indicated, and the patient should be managed in consultation with neurology or neurosurgery.

Step 7: Begin the process of induction and delivery
Once the patient has been stabilized—and not before—initiate the induction process. Be aware that during and after the convulsion, changes in fetal heart rate (FHR) and uterine monitoring will usually be evident:
- prolonged deceleration or bradycardia (3–10 minutes)
- compensatory tachycardia, decreased beat-to-beat variability
- transient recurrent decelerations
- increased uterine tone and greater frequency of uterine activity.

These changes in FHR and uterine activity usually last 3 to 15 minutes. For this reason, it is important to avoid rushing the patient for cesarean delivery, as FHR and uterine activity are likely to return to normal after maternal resuscitation and stabilization. If not, consider other causes, such as abruptio placentae.

Eclampsia itself is not an indication for cesarean delivery. The selection of mode of delivery should be based on the presence or absence of labor, the cervical Bishop score, fetal gestational age, fetal presentation, and overall fetal condition.

Choosing an anesthetic
Regional analgesia/anesthesia is the method of choice for most women with eclampsia. However, regional anesthesia is to be avoided in the presence of disseminated intravascular coagulation or thrombocytopenia (the threshold platelet count is usually less than $75 \times 10^3 \mu L$. In such a case, IV analgesia can be used during labor, and general anesthesia may be appropriate for cesarean delivery. Both spinal and epidural analgesia and anesthesia are appropriate for women with eclampsia.

How to manage other hypertensive emergencies
A hypertensive emergency during pregnancy or postpartum involves acute-onset, persistent (>15 minutes), severe systolic BP ($\geq 160$ mm Hg) or severe diastolic BP ($\geq 110$ mm Hg), or both. The first step in such an emergency is to ensure the accurate measurement of BP using standard techniques.

Patients with acute-onset, persistent, severe BP should be hospitalized promptly for evaluation and treatment to prevent organ damage. Once such a patient is hospitalized, BP should be recorded every 15 minutes, with continuous FHR monitoring to ensure fetal viability.

The timing of initiation of antihypertensive medications, as well as determination of the type of medication best suited for the patient, should be based on:
- systolic and diastolic BP levels
- maternal clinical and laboratory findings
- presence of associated symptoms
- preexisting medical comorbidities
- whether the patient is antepartum or postpartum.

For example, a sustained BP level of 200/120 mm Hg requires therapy after 15 minutes, whereas observation may be suitable for as long as 60 minutes for a sustained BP of 160/72 mm Hg during labor.

Rapid reduction of systolic BP can lead to marked reductions in uteroplacental blood flow and a nonreassuring FHR tracing. Moreover, a rapid reduction of severe systolic BP in patients who have constricted plasma volume can reduce perfusion to the kidney, brain, and placenta. However, sustained BP of 165/100 mm Hg in association with central nervous system signs or symptoms, congestive heart failure, thrombocytopenia, or
Profile of an eclamptic seizure

Witnessing an eclamptic convulsion can be a frightening experience for nurses and medical providers. The convulsion usually lasts 60 to 90 seconds and occurs in two phases:

- **Phase 1** (15–25 seconds) involves facial twitching, rolling of the eyes, and stiffening of the body, with generalized muscular contractions.
- **Phase 2** (20–50 seconds) involves alternate contraction and relaxation of the muscles of the body in rapid succession, starting in the face and spreading throughout the body. Foaming at the mouth also occurs, and the patient may bite her tongue if it isn’t protected.

Apnea develops during and immediately after the convulsion, lasting about 120 seconds. A period of hyperventilation follows to compensate for the respiratory acidosis during the apneic period.

A postictal state follows the convulsion, and the patient usually remembers nothing of the episode. Some patients also become restless, combative, and agitated, requiring sedation. Aspiration is possible during or after the convulsion.

Apnea develops during and immediately after an eclamptic convulsion, lasting about 120 seconds.

Postpartum status requires therapy within 1 hour.

In general, it is difficult to obtain accurate BP recordings using noninvasive electronic instruments during labor because of the effects of labor on systolic BP and the lack of standardized methods for positioning of the arm cuff and the patient.

For these reasons, the decision about when to start acute antihypertensive therapy, based on systolic or diastolic BP, or both, should be individualized. And the choice of antihypertensive agent should be based on maternal clinical findings.

Choosing an antihypertensive agent

Because both hydralazine and nifedipine are associated with tachycardia, avoid them in patients with a heart rate above 110 bpm, using labetalol instead.\(^\text{10}\)

In patients with bradycardia (heart rate <60 bpm), asthma, or congestive heart failure, however, labetalol should be avoided. In these populations, **hydralazine or nifedipine** is the drug of choice. Nifedipine is associated with improved renal blood flow and a resultant increase in urine output, making it preferable for patients with decreased urine output or severe postpartum hypertension.\(^\text{10}\)

One theoretical concern is that the combined use of nifedipine and magnesium sulfate can cause excessive hypotension and neuromuscular blockage. As a result, some experts recommend that nifedipine be avoided in patients receiving magnesium sulfate. However, a recent review of this subject concluded that combined use of these drugs does **not** increase the risks of excessive hypotension and neuromuscular blockage in patients with severe hypertension or preeclampsia.

**The initial dose of labetalol**, when it is your chosen agent, is 20 mg IV, with BP measured 10 minutes later. If the target BP threshold is not achieved, administer 40 mg, 80 mg, and 80 mg at 10-minute intervals, as needed, again measuring BP 10 minutes after every dose. If, after a maximum dose of 240 mg, the desired BP threshold still has not been reached, give 5 to 10 mg IV hydralazine and measure BP 20 minutes later. If the target BP threshold still has not been achieved, it is essential to obtain consultation on the need for continuous infusion of labetalol, nicardipine, or sodium nitroprusside.

**The initial dose of hydralazine**, when it is your chosen agent, is 5 to 10 mg IV, with BP measured 20 minutes later. If needed, give another 10 mg and measure BP after another 20-minute interval. After a maximum dose of hydralazine 20 mg, switch to IV labetalol, using the regimen described above for labetalol, if the BP threshold still has not been achieved.

**Nitroglycerin may be helpful in carefully selected patients**

This drug is an arterial—but mostly venous—dilator. It is administered via IV infusion at an initial rate of 5 µg/min, with the rate gradually increased every 3 to 5 minutes (titrated to BP) to a maximum dose of 100 µg/min. It is the drug of choice in any hypertensive emergency associated with pulmonary edema and for control of hypertension associated with tracheal manipulation during intubation and extubation with general anesthesia.

Nitroglycerin is contraindicated in hypertensive encephalopathy because it increases cerebral blood flow and intracranial pressure. **This drug should be administered**
Sodium nitroprusside is the drug of choice in hypertensive encephalopathy, but should be administered only under the supervision of an experienced obstetric intensivist.

**Sodium nitroprusside: Only in an ICU**

This agent causes arterial and venous relaxation by interfering with the influx and intracellular activation of calcium. It is the drug of choice in hypertensive encephalopathy because it controls both afterload (vascular resistance) and preload (fluid status). It should be used only in the setting of intensive care. **The recommended dose** is IV infusion at a rate of 0.25 to 5.00 µg/kg/min. Sodium nitroprusside has an immediate onset of action and may continue to exert an effect 3 to 5 minutes after discontinuation. Any hypotension caused by the drug should subside within minutes after discontinuation of the drip, due to the drug’s short half-life.

Nitroprusside is metabolized into thiocyanate and excreted in the urine. Cyanide can accumulate with large doses (>10 µg/kg/min) or prolonged administration (>48 hours), or if the patient has renal insufficiency or decreased hepatic metabolism. Signs of toxicity include anorexia, disorientation, headache, fatigue, restlessness, tinnitus, delirium, hallucinations, nausea, vomiting, and metabolic acidosis. When infused at a rate of less than 2 µg/kg/min, however, cyanide toxicity is unlikely.

As is the case with nitroglycerin, this drug should be administered only under the supervision of an experienced obstetric intensivist.

**CASE Resolved**

Upon arrival at the ED, the patient exhibits shallow, rapid breathing and foaming from the mouth. She is placed in a lateral decubitus position, an oral airway is established, and all secretions are suctioned. Oxygen is administered via face mask at a rate of 8 L/min. Her initial oxygen saturation level is 92%. IV access is secured, and a loading dose of magnesium sulfate 6 g is given over 20 minutes. Oxygen saturation increases to 94% to 96%. Auscultation of both lungs is normal.

The patient remains in a postictal state for about 15 minutes, but then orients to name, place, and time. FHR monitoring of both fetuses reveals a normal baseline with moderate variability, as well as variable decelerations in the presenting twin.

A maintenance dose of magnesium sulfate is initiated at a rate of 2 g/h, with the BP level recorded every 15 minutes. Systolic values remain between 170 and 180 mm Hg, and diastolic values between 108 and 112 mm Hg for 60 minutes. The obstetrician administers IV labetalol (20 mg) over 2 minutes. About 15 minutes later, the BP level is 154/100 mm Hg, with values remaining in the range of 150 to 156 mm Hg systolic and 92 to 104 mm Hg diastolic. Ultrasonography reveals that the presenting twin is in a breech position, with estimated fetal weight below the 10th percentile and oligohydramnios. As a result, the obstetrician elects to proceed to cesarean delivery. The twins are delivered by cesarean section using spinal anesthesia. Although the infants are premature, there are no complications.

**References**