

Hypertensive Disorders of Pregnancy

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The National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy has defined four categories of hypertension in pregnancy: chronic hypertension, gestational hypertension, preeclampsia, and preeclampsia superimposed on chronic hypertension. A maternal blood pressure measurement of 140/90 mm Hg or greater on two occasions before 20 weeks of gestation indicates chronic hypertension. Pharmacologic treatment is needed to prevent maternal end-organ damage from severely elevated blood pressure (150 to 180/100 to 110 mm Hg); treatment of mild to moderate chronic hypertension does not improve neonatal outcomes or prevent superimposed preeclampsia. Gestational hypertension is a provisional diagnosis for women with new-onset, nonproteinuric hypertension after 20 weeks of gestation; many of these women are eventually diagnosed with preeclampsia or chronic hypertension. Preeclampsia is the development of new-onset hypertension with proteinuria after 20 weeks of gestation. Adverse pregnancy outcomes related to severe preeclampsia are caused primarily by the need for preterm delivery. HELLP (i.e., hemolysis, elevated liver enzymes, and low platelet count) syndrome is a form of severe preeclampsia with high rates of neonatal and maternal morbidity. Magnesium sulfate is the drug of choice to prevent and treat eclampsia. The use of magnesium sulfate for seizure prophylaxis in women with mild preeclampsia is controversial because of the low incidence of seizures in this population. (*Am Fam Physician*. 2008;78(1):93-100. Copyright © 2008 American Academy of Family Physicians.)

► **Patient information:**
A handout on high blood pressure during pregnancy is available at <http://familydoctor.org/online/famdocen/home/women/pregnancy/complications/695.html>.

Hypertensive disorders represent the most common medical complication of pregnancy, affecting 6 to 8 percent of gestations in the United States.¹ In 2000, the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy defined four categories of hypertension in pregnancy: chronic hypertension, gestational hypertension, preeclampsia, and preeclampsia superimposed on chronic hypertension.¹

Chronic Hypertension

Chronic hypertension is defined as a blood pressure measurement of 140/90 mm Hg or more on two occasions before 20 weeks of gestation or persisting beyond 12 weeks postpartum.¹ Treatment of mild to moderate chronic hypertension neither benefits the fetus nor prevents preeclampsia.²⁻⁴ Excessively lowering blood pressure may result in decreased placental perfusion and adverse perinatal outcomes.⁵ When a patient's blood pressure is persistently greater than 150 to 180/100 to 110 mm Hg, pharmacologic treatment is needed to prevent maternal end-organ damage.^{1,2,4,6}

Methyldopa (Aldomet; brand no longer available in the United States), labetalol, and nifedipine (Procardia) are oral agents

commonly used to treat chronic hypertension in pregnancy. Angiotensin-converting enzyme inhibitors and angiotensin-II receptor antagonists are not used because of teratogenicity, intrauterine growth restriction (IUGR), and neonatal renal failure.⁴ The beta blocker atenolol (Tenormin) has been associated with IUGR,³ and thiazide diuretics can exacerbate intravascular fluid depletion if superimposed preeclampsia develops. Women in active labor with uncontrolled severe chronic hypertension require treatment with intravenous labetalol or hydralazine.⁷

Morbidity occurs primarily from superimposed preeclampsia or IUGR.⁴ A sudden increase in blood pressure, new proteinuria, or signs and symptoms of severe preeclampsia indicate superimposed preeclampsia. Fetal growth may be assessed by serial fundal height measurements supplemented by ultrasonography every four weeks starting at 28 weeks of gestation.⁴

Gestational Hypertension

Gestational hypertension has replaced the term pregnancy-induced hypertension to describe women who develop hypertension without proteinuria after 20 weeks of gestation.¹ Gestational hypertension is a

Hypertension in Pregnancy

SORT: KEY RECOMMENDATIONS FOR PRACTICE

<i>Clinical recommendation</i>	<i>Evidence rating</i>	<i>References</i>
In women without end-organ damage, chronic hypertension in pregnancy does not require treatment unless the patient's blood pressure is persistently greater than 150 to 180/100 to 110 mm Hg.	C	1, 2, 4, 6
Calcium supplementation decreases the incidence of hypertension and preeclampsia, respectively, among all women (NNT = 11 and NNT = 20), women at high risk of hypertensive disorders (NNT = 2 and NNT = 6), and women with low calcium intake (NNT = 6 and NNT = 13).	A	26
Low-dose aspirin (75 to 81 mg daily) has small to moderate benefits for the prevention of preeclampsia (NNT = 72), preterm delivery (NNT = 74), and fetal death (NNT = 243). The benefit of aspirin is greatest (NNT = 19) for prevention of preeclampsia in women at highest risk (previous severe preeclampsia, diabetes, chronic hypertension, renal disease, or autoimmune disease).	B	27
For women with mild preeclampsia, delivery is generally not indicated until 37 to 38 weeks of gestation and should occur by 40 weeks.	C	7
Magnesium sulfate is the treatment of choice for women with preeclampsia to prevent eclamptic seizures (NNT = 100) and placental abruption (NNT = 100).	A	42
Intravenous labetalol or hydralazine may be used to treat severe hypertension in pregnancy because neither agent has demonstrated superior effectiveness.	B	1, 46
For managing severe preeclampsia between 24 and 34 weeks of gestation, the data are insufficient to determine whether an "interventionist" approach (i.e., induction or cesarean delivery 12 to 24 hours after corticosteroid administration) is superior to expectant management. Expectant management, with close monitoring of the mother and fetus, reduces neonatal complications and stay in the newborn intensive care nursery.	B	47-49
Magnesium sulfate is more effective than diazepam (Valium; NNT = 8) or phenytoin (Dilantin; NNT = 8) in preventing recurrent eclamptic seizures.	A	39, 54-56

NNT = number needed to treat.

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, see <http://www.aafp.org/afpsort.xml>.

provisional diagnosis that includes women eventually diagnosed with preeclampsia or chronic hypertension, as well as women retrospectively diagnosed with transient hypertension of pregnancy. Fifty percent of women diagnosed with gestational hypertension between 24 and 35 weeks develop preeclampsia.⁸ Expectant management of mild gestational hypertension can reduce the increased rate of cesarean delivery associated with the induction of nulliparous women who have an unripe cervix.⁹ Women who progress to severe gestational hypertension based on the degree of blood pressure elevation have worse perinatal outcomes than do women with mild preeclampsia, and require management similar to those with severe preeclampsia.¹⁰

Preeclampsia

Preeclampsia is a multiorgan disease process of unknown etiology¹¹ characterized by the development of hypertension and proteinuria after 20 weeks of gestation. *Table 1* lists

Table 1. Preeclampsia: Etiology and Risk Factors

Theories of pathogenesis	Risk factors^{7,12}
Abnormal placental implantation (defects in trophoblasts and spiral arterioles) ^{13,14}	Antiphospholipid antibody syndrome
Angiogenic factors (increased sFlt-1, decreased placental growth factor levels) ^{15,16}	Chronic hypertension
Cardiovascular maladaptation and vasoconstriction	Chronic renal disease
Genetic predisposition (maternal, paternal, thrombophilias) ¹⁷⁻²⁰	Elevated body mass index
Immunologic intolerance between fetoplacental and maternal tissue ⁷	Maternal age older than 40 years
Platelet activation	Multiple gestation
Vascular endothelial damage or dysfunction ⁷	Nulliparity
	Preeclampsia in a previous pregnancy (particularly if severe or before 32 weeks of gestation)
	Pregestational diabetes mellitus

NOTE: Previously, young maternal age was considered a risk factor, but this was not supported by a systematic review.²¹

sFlt-1 = soluble fms-like tyrosine kinase 1.

Information from references 7, and 12 through 21.

proposed etiologies and risk factors for preeclampsia.^{7,12-21} Prevention through routine supplementation with calcium, magnesium, omega-3 fatty acids, or antioxidant vitamins is ineffective.²²⁻²⁵ Calcium supplementation reduces the risk of developing preeclampsia in high-risk women and those with low dietary calcium intakes.²⁶

Low-dose aspirin (75 to 81 mg per day) is effective for women at increased risk of preeclampsia. Treating 69 women prevents one case of preeclampsia; treating 227 women prevents one fetal death.²⁷ For women at highest risk from previous severe preeclampsia, diabetes, chronic hypertension, or renal or autoimmune disease, only 18 need to be treated with low-dose aspirin to prevent one case of preeclampsia.²⁷

DIAGNOSIS

Blood pressure should be measured at each prenatal visit with an appropriately sized cuff and the patient in a seated position.^{28,29} Diagnostic criteria for preeclampsia are systolic blood pressure of 140 mm Hg or more or a diastolic blood pressure of 90 mm Hg or more on two occasions at least six hours apart.^{12,28,29} An increase of 30 mm Hg systolic or 15 mm Hg diastolic from baseline is no longer diagnostic for preeclampsia¹² because similar increases are common in uncomplicated pregnancies.

The diagnostic threshold for proteinuria is 300 mg in a 24-hour urine specimen. A 24-hour determination is most accurate because urine dipsticks can be affected by variable excretion, maternal dehydration, and bacteriuria.⁷ A random urine protein/creatinine ratio of less than 0.21 indicates that significant proteinuria is unlikely with a negative predictive value of 83 percent; however, confirmatory 24-hour urine protein determination is recommended.³⁰ Generalized edema (affecting the face and hands) is often present in patients with preeclampsia but is not a diagnostic criterion.¹

Severe Preeclampsia. Preeclampsia is characterized as mild or severe based on the degree of hypertension and proteinuria, and the presence of symptoms resulting from involvement of the kidneys, brain, liver, and cardiovascular system (*Table 2*).¹² Severe headache, visual disturbances, and hyperreflexia may signal impending eclampsia. Increased peripheral vascular resistance and pulmonary edema may occur. A decreased glomerular filtration rate may progress to oliguria and acute renal failure. The increased glomerular filtration rate of pregnancy lowers serum creatinine, and levels greater than 0.9 mg per dL (80 μ mol per L) are abnormal in pregnancy. Liver manifestations include elevated transaminase levels, subcapsular hemorrhage with right upper quadrant pain, and capsular rupture with life-threatening

intraabdominal bleeding. Obstetric complications include IUGR, placental abruption, and fetal demise.¹²

HELLP Syndrome. The acronym HELLP describes a variant of severe preeclampsia characterized by hemolysis, elevated liver enzymes, and low platelet count.³¹ HELLP syndrome occurs in up to 20 percent of pregnancies complicated by severe preeclampsia.³² The clinical presentation of HELLP syndrome is variable; 12 to 18 percent of affected women are normotensive and 13 percent do not have proteinuria.³³ At diagnosis, 30 percent of women are postpartum, 18 percent are term, and 52 percent are preterm.³² Common presenting complaints are right upper quadrant or epigastric pain, nausea, and vomiting. Many patients have a history of malaise or nonspecific symptoms suggesting an acute viral syndrome.³³ Any patient with these symptoms or signs of preeclampsia should be evaluated with complete blood count, platelet count, and liver enzyme determinations.³⁴

Laboratory tests are used to diagnose HELLP syndrome (*Table 3*³³⁻³⁵); a decreasing platelet count and an increasing L-lactate dehydrogenase level (indicative of both hemolysis and liver dysfunction) reflect disease severity.^{33,35} When the platelet count is less than 50,000 per mm³ (50×10^9 per L) or active bleeding occurs, coagulation studies (i.e., prothrombin time, partial thromboplastin time, and fibrinogen level) should be performed to rule out superimposed disseminated intravascular coagulation.

Table 2. Diagnostic Criteria for Severe Preeclampsia*

Blood pressure \geq 160 mm Hg systolic or 110 mm Hg diastolic on two occasions at least six hours apart during bed rest
Proteinuria \geq 5 g in a 24-hour urine specimen or 3+ or greater on two random urine specimens collected at least four hours apart
Any of the following associated signs and symptoms:
Cerebral or visual disturbances
Epigastric or right upper quadrant pain
Fetal growth restriction
Impaired liver function
Oliguria $<$ 500 mL in 24 hours
Pulmonary edema
Thrombocytopenia

*—One or more of the criteria are present.

Adapted with permission from American College of Obstetricians and Gynecologists. ACOG Committee on Practice Bulletins—Obstetrics. ACOG Practice Bulletin No. 33, January 2002. *Diagnosis and management of preeclampsia and eclampsia*. *Obstet Gynecol.* 2002;99(1):160.

Table 3. Criteria for Laboratory Diagnosis of HELLP Syndrome

Hemolysis	Abnormal peripheral blood smear (evidence of damaged erythrocytes, such as schistocytes and burr cells) Serum bilirubin ≥ 1.2 mg per dL (21 μ mol per L) LDH > 600 U per L (10.02 μ kat per L)
Elevated liver enzymes	AST (SGOT) elevated* ALT (SGPT) elevated*
Low platelet count	$< 100,000$ per mm^3 (100×10^9 per L) or Class 1: $\leq 50,000$ per mm^3 (50×10^9 per L) Class 2: $> 50,000$ but $\leq 100,000$ per mm^3 Class 3: $> 100,000$ but $< 150,000$ per mm^3 (150×10^9 per L)

ALT = alanine transaminase; AST = aspartate transaminase; HELLP = hemolysis, elevated liver enzymes, and low platelet count; LDH = L-lactate dehydrogenase; SGOT = serum glutamic-oxaloacetic transaminase; SGPT = serum glutamic-pyruvic transaminase.

*—There is no standard definition for degree of transaminase elevation to be diagnostic for HELLP syndrome.³³ Some criteria use any elevation, whereas others use a twofold elevation in either AST or ALT levels.

Information from references 33 through 35.

Table 4. Expectant Management of Mild Preeclampsia*

Maternal monitoring

Measure blood pressure twice weekly
Obtain laboratory tests weekly: CBC, platelet count, ALT, AST, LDH, uric acid, creatinine
Assess for proteinuria: screen with dipstick or spot protein/creatinine ratio and obtain periodic 24-hour urine collections

Fetal monitoring

Obtain nonstress test twice weekly
Measure amniotic fluid index once or twice weekly
Biophysical profile may be done weekly in place of one of the twice-weekly nonstress tests and amniotic fluid index
Perform ultrasonography for fetal growth every three to four weeks

ALT = alanine transaminase; AST = aspartate transaminase; CBC = complete blood count; LDH = L-lactate dehydrogenase.

*—This is one possible regimen for managing mild preeclampsia before term. Women with stable, mild preeclampsia are generally delivered after 37 but before 40 weeks of gestation.

Information from references 1 and 7.

MANAGEMENT OF PREECLAMPSIA

A common regimen for expectant management of mild preeclampsia is outlined in Table 4.^{1,7} Nonstress tests, amniotic fluid index measurements, and biophysical profiles are used to monitor patients for uteroplacental insufficiency.^{1,7} Umbilical artery systolic/diastolic ratios measured by Doppler ultrasonography may detect early

Management of Mild Gestational Hypertension or Preeclampsia

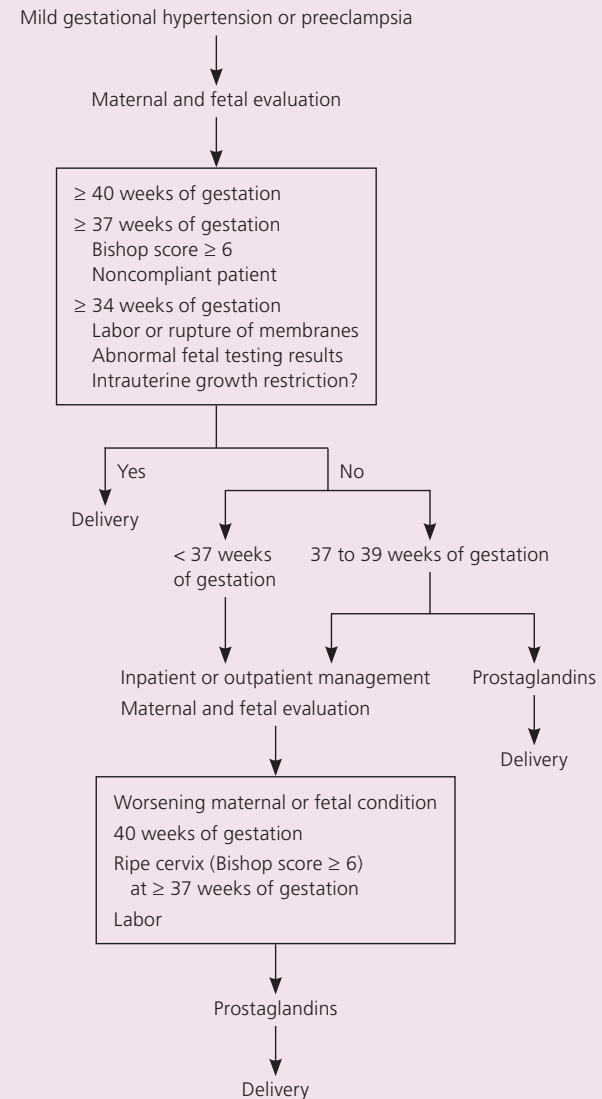


Figure 1. Recommended management of mild gestational hypertension or preeclampsia.

Adapted with permission from Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. *Obstet Gynecol.* 2003;102(1):186.

uteroplacental insufficiency.^{36,37} The decision to deliver involves balancing the risks of worsening preeclampsia against those of prematurity. Delivery is generally not indicated for women with mild preeclampsia until 37 to 38 weeks of gestation and should occur by 40 weeks^{1,7} (Figure 1⁷). Patients with severe preeclampsia are admitted to the hospital, placed on bed rest, and carefully monitored (Figure 2⁷ and Table 5^{1,7,12}). The goals of treatment are to prevent seizures, lower blood pressure to avoid maternal end-organ damage, and expedite delivery.

Magnesium Sulfate. The use of magnesium sulfate helps prevent seizures in women with preeclampsia.³⁸⁻⁴⁰ One eclamptic seizure is prevented for every

100 women treated.³⁸ The use of magnesium sulfate is controversial in women with mild preeclampsia because the incidence of eclamptic seizures is only 0.5 percent in these patients. Assuming one half of seizures are preventable with magnesium sulfate,³⁸ 400 women with mild preeclampsia would need to be treated to prevent one seizure.⁴¹ Magnesium sulfate has the additional benefit of reducing the incidence of placental abruption.⁴²

Magnesium sulfate slows neuromuscular conduction and depresses central nervous system irritability without significant effects on blood pressure. One fourth of women will experience adverse effects, especially flushing.⁴² Table 5 outlines the standard dosing regimen.^{1,7,12} Serum magnesium levels should be monitored in women with elevated serum creatinine levels, decreased urine output, or absent deep tendon reflexes.⁴³ Magnesium toxicity can lead to respiratory paralysis, central nervous system depression, and cardiac arrest. The antidote is calcium gluconate, 1 g infused intravenously over two minutes.⁴⁴

Antihypertensive Medications. The optimal level of blood pressure control in pregnancies complicated by hypertension is unknown.^{2,6} Less tight control may decrease the risk that the infant will be small for gestational age, but it may increase the risk of respiratory distress syndrome of the newborn, severe hypertension, and antenatal hospitalization.^{2,5} Although traditional recommendations are based on diastolic blood pressure, a retrospective review of 28 women with severe preeclampsia who experienced a cerebrovascular accident demonstrated that more than 90 percent had systolic blood pressure over 160 mm Hg, but only 12.5 percent had diastolic blood pressure over 110 mm Hg.⁴⁵

Intravenous labetalol and hydralazine are commonly used for the acute management of preeclampsia.^{1,46} A Cochrane review showed no evidence that one parenteral agent had superior effectiveness.⁴⁶ For women with severe preeclampsia undergoing expectant management remote from term, oral labetalol and nifedipine are acceptable options.⁷

Fluid Management. Excessive fluid administration can result in pulmonary edema, ascites, and cardiopulmonary overload, whereas too little fluid exacerbates

an already constricted intravascular volume and leads to further end-organ ischemia. Urine output should be greater than 30 mL per hour⁴⁴ and intravenous fluids limited to 100 mL per hour.^{35,44}

Delivery Decisions in Severe Preeclampsia. Delivery is the only cure for preeclampsia. Decisions regarding the timing and mode of delivery are based on a combination of maternal and fetal factors. Fetal factors include gestational age, evidence of lung maturity, and signs

Management of Severe Preeclampsia

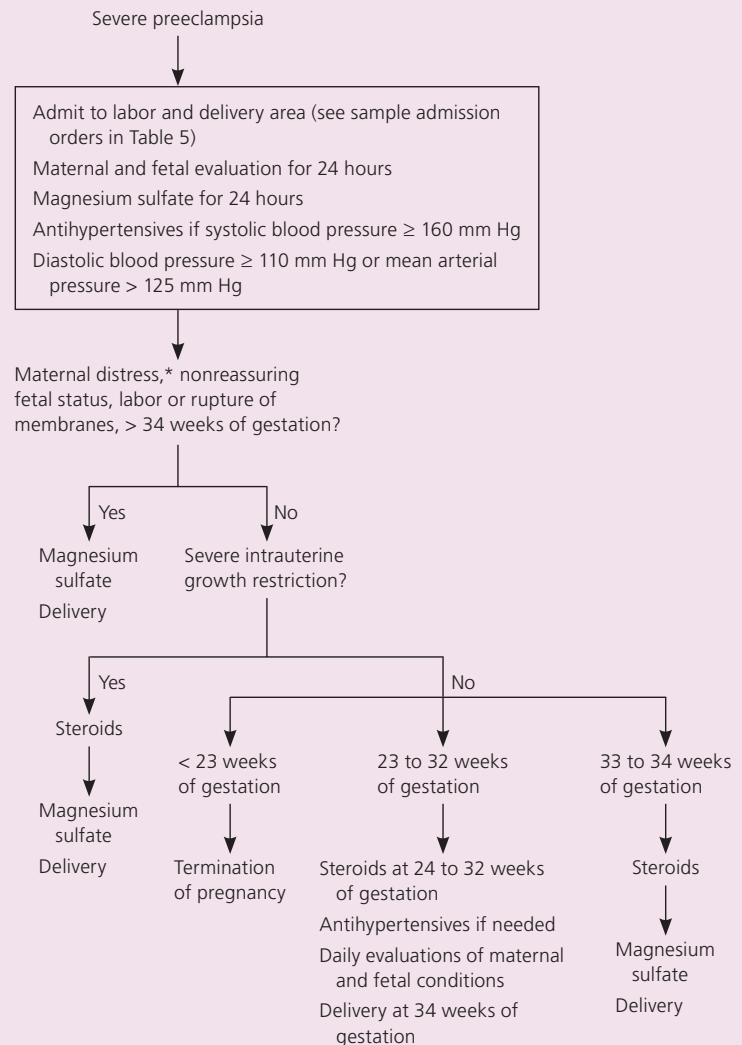


Figure 2. Recommended management of severe preeclampsia.

*—Signs of maternal distress are thrombocytopenia, imminent eclampsia, pulmonary edema, and hemolysis plus elevated liver enzyme levels.

Adapted with permission from Sibai BM. *Diagnosis and management of gestational hypertension and preeclampsia.* *Obstet Gynecol.* 2003;102(1):188.

Table 5. Labor and Delivery Sample Admission Orders for Severe Preeclampsia

Bed rest with seizure precautions

Vital signs (blood pressure, pulse, respiration); deep tendon reflexes; and mental status every 15 to 60 minutes until stable, then every 60 minutes while on magnesium sulfate

Accurate intake and output; Foley catheter if needed

Administer lactated Ringer's solution at 75 mL per hour IV to maintain urine output of 30 to 40 mL per hour; total intake (IV and oral) should not exceed 125 mL per hour or 3,000 mL per day

Continuous fetal heart rate monitoring⁷

Laboratory tests

- Dipstick urine collection for protein level on admission
- 24-hour urine collection for total protein level
- CBC with platelets, peripheral blood smear
- BUN, creatinine, uric acid
- AST, ALT, LDH

Fetal evaluation: nonstress test on admission; obstetric ultrasonography for estimated fetal weight, amniotic fluid volume, and umbilical artery Doppler measurements

Medications

Magnesium sulfate

- Loading dose of 4 to 6 g diluted in 100 mL of normal saline, given IV over 15 to 20 minutes, followed by a continuous infusion of 2 g per hour¹²
- Assess serum magnesium level if urine output is < 30 mL per hour or there is a loss of deep tendon reflexes, decreased respiratory rate, or altered mental status
- Therapeutic range for serum magnesium is 4 to 7 mg per dL

Corticosteroids (if between 24 and 34 weeks of gestation and not previously administered)

- Betamethasone (Celestone), 12 mg IM initially, then repeat in 24 hours
or
- Dexamethasone, 6 mg IM initially, then repeat every 12 hours for three additional doses

For systolic blood pressure > 160 mm Hg or diastolic > 110 mm Hg, one of the following should be given to achieve a systolic measurement of 140 to 155 mm Hg and/or a diastolic measurement of 90 to 105 mm Hg⁷:

- Hydralazine, 5 to 10 mg IV every 15 to 30 minutes (maximal dose: 30 mg)⁷
or
- Labetalol, 20 mg IV initially; if the initial dose is not effective, double the dose to 40 mg and then 80 mg at 10-minute intervals until target blood pressure is reached or a total of 220 mg has been administered¹⁷; the maximal dose of IV labetalol is 220 mg in a 24-hour period^{7,12}

Calcium gluconate, 1 g IV; keep at bedside in case of respiratory depression from magnesium sulfate use

ALT = alanine transaminase; AST = aspartate transaminase; BUN = blood urea nitrogen; CBC = complete blood count; IM = intramuscularly; IV = intravenously; LDH = L-lactate dehydrogenase.

Information from references 1, 7, and 12.

For patients with severe preeclampsia between 24 and 34 weeks of gestation, the data are insufficient to recommend “interventionist” versus expectant management.⁴⁷ Subspecialty consultation is indicated.^{48,49} Corticosteroids are administered to accelerate fetal lung maturity.⁷ Interventionist management advocates induction or cesarean delivery 12 to 24 hours after corticosteroid administration. Expectant management, with close monitoring of the mother and fetus, delays delivery when possible and reduces neonatal complications and length of stay in the newborn intensive care nursery.⁴⁷⁻⁴⁹ Contraindications to expectant management include persistent severe symptoms, multiorgan dysfunction, severe IUGR (i.e., estimated fetal weight below the 5th percentile), suspected placental abruption, or nonreassuring fetal testing.⁴⁹

In women with HELLP syndrome, the fetus is delivered at an earlier gestation; specifically, fetuses older than 28 weeks are routinely delivered 24 to 48 hours after the first maternal dose of corticosteroids is administered.³⁴ Conservative management of HELLP syndrome remains experimental and, for most women, the clinical course is too rapid to complete the steroid regimen before initiating delivery.³³

Vaginal delivery is recommended for women with severe preeclampsia if there is no evidence of maternal or fetal compromise or other obstetric contraindication.¹ Some experts recommend cesarean delivery for fetuses younger than 30 weeks when the cervix is not ripe, but a trial of induction may be considered.^{1,7} In patients with HELLP syndrome, cesarean delivery carries special risks, such as bleeding from thrombocytopenia and difficulty controlling blood pressure because of depleted intravascular volume.^{33,34}

Postpartum Management. Most patients with preeclampsia respond promptly to delivery with decreased blood pressure, diuresis, and clinical improvement. Eclampsia may occur postpartum; the greatest risk of postpartum eclampsia is within the first 48 hours.⁴³ Magnesium sulfate is continued for 12 to 24 hours, or occasionally longer if the clinical situation warrants. There are no reliable data on postpartum hypertensive management⁵⁰; however, oral nifedipine is commonly used.⁷

of fetal compromise on antenatal assessment. Patients with treatment-resistant severe hypertension or other signs of maternal or fetal deterioration should be delivered within 24 hours, irrespective of gestational age or fetal lung maturity. Fetuses older than 34 weeks, or those with documented lung maturity, are also delivered without delay.⁷

Eclampsia

An eclamptic seizure may be preceded by increasingly severe preeclampsia, or it may appear unexpectedly in a patient with minimally elevated blood pressure and no proteinuria. Blood pressure is only mildly elevated in 30 to 60 percent of women who develop eclampsia.⁴³ An eclamptic seizure usually lasts from 60 to 90 seconds, during which time the patient is without respiratory effort. A postictal phase may follow with confusion, agitation, and combativeness. The timing of an eclamptic seizure can be antepartum (53 percent), intrapartum (19 percent), or postpartum (28 percent).⁵¹ Late postpartum (more than 48 hours after delivery) onset of eclampsia was traditionally thought to be rare; however, a study of 29 cases of postpartum eclampsia demonstrated that 79 percent occurred in the late postpartum period.^{43,52}

MANAGEMENT OF ECLAMPSIA

Initial management of an eclamptic seizure includes protecting the airway and minimizing the risk of aspiration by placing the woman on her left side, suctioning her mouth, and administering oxygen. A medical professional skilled in performing intubations should be immediately available.⁵³ Close observation, soft padding, and use of side rails on the bed may help prevent trauma from falls or violent seizure activity. After the convulsion has ended and the patient is stabilized, plans should be made for prompt delivery. In rural or remote areas, physicians need to consider the risk of transfer versus the benefits of tertiary maternal and neonatal care.

It is important to avoid unnecessary interventions and iatrogenic complications.^{43,53} Magnesium sulfate is the drug of choice because it is more effective in preventing recurrent seizures than phenytoin (Dilantin) or diazepam (Valium).^{39,54-56} If a patient has already received a prophylactic loading dose of magnesium sulfate and is receiving a continuous infusion, an additional 2 g should be given intravenously. Otherwise, a 6-g loading dose is given intravenously over 15 to 20 minutes, followed by maintenance infusion of 2 g per hour. A total of 8 g of magnesium sulfate should not be exceeded over a short period of time.^{43,53}

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