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Hypertensive disorders of pregnancy range from chronic pre-existing disease to life-threatening conditions such as HELLP syndrome (hemolysis, elevated liver enzymes, low platelets) and eclampsia. They often represent a continuum from bad to worse. The emergency department (ED) physician is likely to evaluate a pregnant patient for many conditions unrelated to the pregnancy itself, and knowledge of abnormalities that warrant further assessment and follow-up is essential. Abnormal vital signs must be addressed even if the patient's chief complaint is a sprained ankle or sore throat. This article will identify those conditions related to hypertensive disorders of pregnancy and their evaluation and treatment, specifically preeclampsia, eclampsia, and the HELLP syndrome. Although the ED physician often does not provide definitive care for these disorders, a working knowledge of abnormalities that may portend adverse outcomes for the mother and/or the fetus is essential. The number of times an ED physician diagnoses the HELLP syndrome, preeclampsia, or eclampsia may be small, but a missed opportunity for diagnosis may be catastrophic for all involved. Management of the variety of hypertensive pregnancy disorders is similar in many respects. Where appropriate, diagnostic pitfalls likely to present to the ED will be discussed.

—The Editor

Hypertensive Disorders of Pregnancy

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Introduction

The mechanism by which pregnancy-induced hypertension (PIH) occurs is unclear,[1,2] and the primary complication of this disorder is the later development of preeclampsia or eclampsia, both of which have significant fetal and maternal morbidity and mortality.[1,3-6] Hypertensive disorders of pregnancy affect 7-10% of all pregnancies in the United States and are responsible for 15% of all maternal deaths, most commonly from cerebral hemorrhage.[1,4,5,7,8] They are the second most common cause of maternal death in the United States, behind only thromboembolic disease.[9] Preeclampsia complicates 2-3% of pregnancies and may complicate up to 30% of pregnancies if the woman has pre-existing insulin dependent diabetes mellitus, hypertension, or kidney disease.[1,3,10-12] Hypertensive disorders of pregnancy cause significant

perinatal morbidity and mortality as well, including abruptio placentae, fetal growth restriction, and pre-term delivery.[6]

Currently, as defined by the American College of Obstetrics and Gynecology (ACOG), PIH exists if the blood pressure is equal to or greater than 140/90 in the seated patient, using the appropriate size cuff, and defining the diastolic as the point at which no more sound is heard (Korotkoff Phase V).[13] In the pregnant patient with pre-existing hypertension, a rise of 30/15 above her baseline blood pressure is cause for concern.[14,15]

Blood pressure measurements may be inaccurate for a variety of reasons, including operator error, improper cuff size, and failure to calibrate equipment. Ambulatory high blood pressure monitoring may be useful in determining "white coat hypertension" and for ongoing follow-up in the patient with hypertension, but is not without error, either. Two readings six hours apart that are elevated are necessary to support the diagnosis of hypertension.[16-18]

The diagnosis of hypertensive disorders of pregnancy and preeclampsia has varied over the years and begs for a standard definition.[19] Currently, hypertensive pregnant patients are divided into two subgroups: those with pre-existing disease, and those who develop hypertension in the second half of pregnancy. (*See Table 1.*) Those who are found to be hypertensive in the first 20 weeks probably have pre-existing undiagnosed disease (chronic hypertension) because it is usual for the blood pressure to fall during the first 20 weeks.[20] Blood pressure measurements that are elevated and occur after the gestational age of 20 weeks are part of the continuum of PIH. These include:

- hypertension without proteinuria or edema (gestational hypertension) (PIH);
- hypertension with associated proteinuria and/or edema (preeclampsia);
- hypertension with seizures (eclampsia);
- new onset proteinuria in the hypertensive patient (superimposed preeclampsia on chronic hypertension); and
- chronic hypertension.[15] (*See Table 1.*)

Non-proteinuric hypertension, now termed gestational hypertension or previously termed PIH, is estimated to occur in 8-10% of nulliparous women; preeclampsia in 2-3%; and eclampsia in 5-6/10,000 pregnancies that progress beyond 20 weeks.[4,5]

It is important for the ED physician to keep in mind that in the normal gravid patient, the blood pressure drops during the second trimester of pregnancy. Any reading over 140 mmHg systolic or 90 mmHg diastolic is abnormal. Close attention should be paid to blood pressure readings in any pregnant patient presenting to the ED, regardless of the reason for the visit. All abnormal values should be rechecked, and if persistent elevation is noted, contact should be made with the patient's obstetrician. Significant blood pressure elevation, especially in the absence of proteinuria, should prompt a urine drug screen for cocaine.[14]

It is not uncommon for an ED physician to encounter a pediatric patient who is pregnant; therefore, a brief mention of definitions is given, as they differ from those in adults. In females younger than 18 years, hypertension is defined by the American Academy of Pediatrics as a blood pressure consistently greater than the 95th percentile of pressures, corrected for age and height. These levels often are much lower than the 140/90 pressure used for adults. For example, in females 16 years of age, the 95th percentile is 125-132/83-86 mmHg.[21]

Many women choose to delay pregnancy until their 30s or 40s, which allows for a concomitant rise in pre-existing hypertension or chronic hypertension. Chronic hypertension also causes an increase in the prevalence of abruptio placenta, which occurs in approximately 10% of such pregnancies.[22] Pregnancies with pre-existing hypertension also are at increased risk for superimposed preeclampsia, with a reported rate of 25% in one

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Table 1. Definition of Hypertensive Disorders of Pregnancy in Adult Patients[15,22,34,35]

CHRONIC HYPERTENSION	Blood pressure (BP) > 140 mmHg systolic or > 90 mmHg diastolic diagnosed before 20 weeks gestation or persists > 12 weeks post-partum
GESTATIONAL HYPERTENSION AND/OR TRANSIENT HYPERTENSION OF PREGNANCY	BP >140/90 develops after mid-pregnancy without proteinuria. If preeclampsia doesn't develop in the woman with gestational hypertension and BP returns to normal within 12 weeks post-partum then the term is transient hypertension of pregnancy (a retrospective diagnosis)
PREECLAMPSIA	BP > 140/90 after 20 weeks, with associated proteinuria > 300 mg/24 hours, (or > 100 mg/dL on two random collections), or generalized edema
SUPERIMPOSED PREECLAMPSIA	Proteinuria > 300 mg/24 hours that occurs past 20 weeks in the patient with chronic hypertension without pre-existing proteinuria, or increasing proteinuria, or sudden rise in BP, or development of thrombocytopenia or elevated liver enzymes in the patient with hypertension and pre-existing proteinuria
ECLAMPSIA	Grand mal seizure that occurs in the pregnant patient past 20 weeks, not attributable to other etiologies, associated with preeclampsia

study.[23] The risk for small for gestational age infants, premature delivery, and perinatal death also increases.[24]

Blood Pressure Control in Pregnancy, Preeclampsia, Eclampsia, and HELLP

Identification of hypertension in a pregnant patient visiting the ED for any reason warrants re-evaluation of the vital signs, a urine dip for protein, a complete physical examination, and obstetrical consult. (*See Figure 1.*) Patients who have pre-existing disease, are currently receiving antihypertensive therapy, do not have proteinuria and who come back to their baseline on recheck after lying down for 30 minutes, may be managed for their initial presenting problem without further attention to the hypertension. However, if the patient's blood pressure remains elevated on recheck, even in the absence of proteinuria, adjustment should be made to her antihypertensive regimen in consult with her obstetrician. The management of the patient with a hypertensive disorder of pregnancy varies with the disorder being treated. Patients should not be discharged from the ED if their blood pressure is not controlled to less than 140/90. (*See Figure 1.*)

If the patient presents to the ED without a prior history of hypertension and has a painful condition, pain management may be all that is necessary to control the blood pressure. Unless otherwise indicated, these patients may be discharged from the ED.

Over the years, numerous recommendations have been put forth for the management of the pregnant hypertensive patient so as to decrease maternal morbidity and mortality and allow for the delivery of a viable newborn as close to maturity as possible. The threshold for beginning antihypertensive therapy is a diastolic blood pressure of 100 mmHg or greater. Antihypertensive choices in pregnancy are predicated on fetal safety and maintenance of uteroplacental blood flow. Studies have demonstrated that treatment of diastolic blood pressures greater than 110 mmHg with medication decreases the incidence of maternal cerebral and cardiac events.[25] Blood pressure control, however, does not prevent or cure preeclampsia.[18]

Bed rest is the primary non-pharmacologic treatment of PIH, which results in lower blood pressure and often diuresis. Strict bed rest may place the patient at increased risk for thromboembolic events, and should be avoided.[26] In addition, avoidance of alcohol and tobacco may help.[10] The choice of an antihypertensive agent depends on the urgency with which the blood pressure must be controlled. For patients who purely have hypertension, there are a variety of medications that may be used. (*See Table 2.*)[10,14,27-29]

Treatment of mild hypertension (less than 150/100) has not been shown to produce significant maternal or fetal benefits in most studies, and referral to obstetrics for further monitoring, once preeclampsia has been ruled out by the absence of proteinuria, is appropriate.[10] For moderate hypertension (diastolic 100-110 mmHg) that does not resolve with 30 minutes of rest without proteinuria or evidence of end organ involvement, methyldopa (Aldomet) 250-500 mg PO may be used to begin therapy. An alternative would be nifedipine (Procardia, Adalat) 10 mg PO, repeated in 30-60 minutes if blood pressure is greater than 150/100. Labetolol (Normodyne, Trandate) also may be used with a starting dose of 100 mg twice daily. When the blood pressure has been controlled to less than 150/100 and the patient's obstetrician has been consulted for follow-up within 24 hours, the patient may be discharged from the ED from a hypertensive standpoint with a prescription for the antihypertensive agent used. (*See Table 2.*)

Once preeclampsia develops (see next section) there often is a need to prevent the development of eclampsia, and initially hospital admission or in-patient observation in an obstetrical special care unit is warranted. An initial dose of dexamethasone (Decadron) 10 mg should be given if the patient is fewer than 34 weeks gestation.

For oral use, methyldopa has a very long track record of safety and is the only agent whose effects have been studied in children exposed in-utero. If this agent cannot be tolerated or is ineffective, alternatives should be chosen based on mechanism of action and age of the pregnancy. In all cases, angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor antagonists are contraindicated. If a patient becomes pregnant while on these agents, she should have her medication changed.[10,15] As many pregnancies are first diagnosed in the ED, once this diagnosis is made, a review of the patient's current medications should be undertaken for safe use in pregnancy, with modifications made in consultation with the patient's primary care physician or obstetrician.

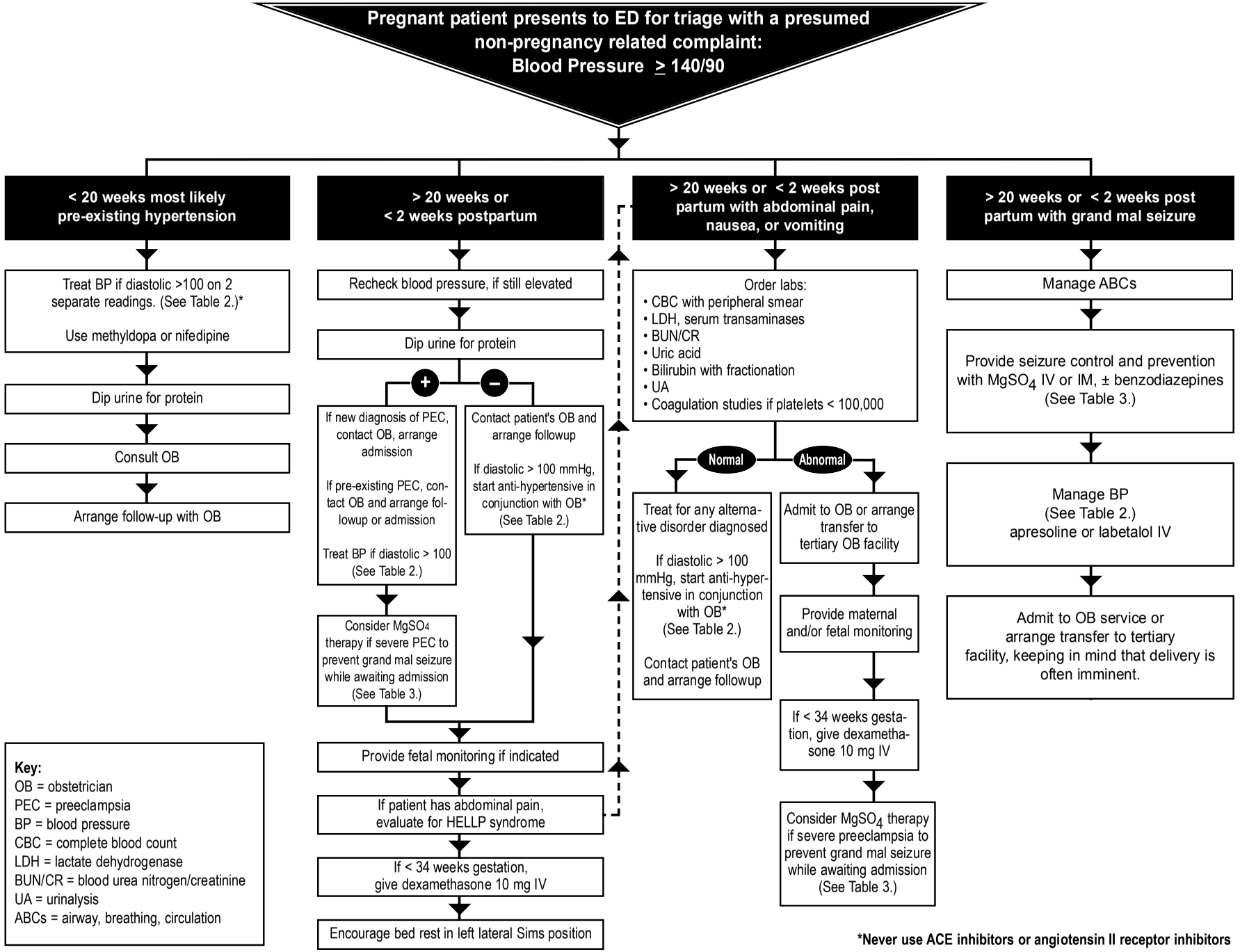


Figure 1. The Emergency Department Management of the Pregnanct Patient Noted to be Hypertensive

Table 2. Antihypertensive Medications and their Usage in Pregnancy[10,14,27-29]

MEDICATION	DOSAGE	POTENTIAL MATERNAL (M) OR FETAL (F) COMPLICATIONS
Methyldopa (Aldomet)	0.5-2.0 g/day, divided Q12 hours	M: Sedation, orthostatic hypotension, rarely fever and abnormal liver functions and Coombs' + hemolytic anemia
Clonidine (Catapres)	0.1-0.7 mg/day Generally start with 0.2 mg and give 0.1 mg/hour to a total of 0.7 mg	M: Drowsiness, xerostomia, rebound hypertension if medication is stopped abruptly
Labetolol* (Normodyne, Trandate)	5-20 mg IV, maximum of 300 mg/day No reflex tachycardia, headache, or fluid retention	M: Not used in patients with asthma or chronic lung disease, heart blockage F: Readily crosses placenta, causing bradycardia. Neonatal complications include hypoglycemia, respiratory problems, and hyperbilirubinemia.
Hydralazine (Apresoline)**	5 mg IV, repeat in 10 min, then 10 mg Q 30 min until BP controlled. If 30 mg doesn't control BP, switch agents.	M: Fluid retention, reflex tachycardia, and headache
Thiazide diuretics	12.5-25 mg PO of hydrochlorothiazide—may maintain chronic therapy, don't add after 20 weeks unless CHF	M: Limit use secondary to decreased uterine blood flow secondary to volume depletion
Loop diuretic	20-40 mg IV or PO, especially for pulmonary edema	M: Limit use secondary to decreased uterine blood flow secondary to volume depletion
Nifedipine (Procardia, Adalat)	30-60 mg/day (generally 10 mg every 30-60 min until diastolic < 110 mmHg)	M: Constipation
Verapamil (Isoptin, Calan, Verelan)	120-360 mg/day	M: Constipation
Enalapril, (Vasotec) lisinopril (Zestril, Prinivil), and captopril (Capoten)	**Contraindicated in pregnancy. Change to different medication.**	Causes IUGR, oligohydramnios, congenital malformations
Losartan (Cozaar) Valsartan (Diovan)	**Contraindicated in pregnancy. Change to different medication.**	Causes IUGR, oligohydramnios, congenital malformations

Key:
CHF = congestive heart failure; IUGR = Intrauterine growth restriction

*Newer agent used for hypertensive emergency while in labor and delivery (L&D) with severe preeclampsia. Also drug of choice for Marfans and coarctation patients. Risky in patients with lung disease. Fewer side effects than hydralazine
** Longstanding drug of choice for hypertension control in L & D

If the patient has severe hypertension, (greater than 180/110) or evidence of preeclampsia or end organ involvement, intravenous medications such as hydralazine (Apresoline) 5 mg IV, followed in 10 minutes by a repeat dose of 5 mg and then 10 mg every 30 minutes to a total dose of 30 mg, should be used. Fluid loading with 500-1000 cc normal saline may be necessary to prevent placental hypoperfusion. An alternative in the non-asthmatic patient would be the use of IV labetolol, beginning with 20 mg IV, followed by 40-80 mg IV every 10 minutes to a total dose of 300 mg or a blood pressure less than 140/90. Labetalol also can be administered as a continuous infusion of 0.5-2 mg/minute. This does not require fluid loading. In addition, loading with magnesium sulfate to prevent the development of eclampsia in the severely pre-eclamptic patient should be started in the ED if

Table 3. Anticonvulsant Dosage Regimens for Preeclampsia and Eclampsia[28,56-64,67]

IM MAGNESIUM SULFATE

- 4 g IV of $MgSO_4$ USP ($MgSO_4$ > 7 H_2O) as a 20% solution IV at a rate not to exceed 1 g/min
- 5 g deep IM of $MgSO_4$ (50% solution) each buttock immediately after IV dose (may be mixed with 1 cc of 2% lidocaine to decrease pain)
- If seizures persist > 15 minutes, give an additional 2-4 g IV over 2-4 minutes depending on maternal size.
- If seizures persist or recur following this, add 250 mg of amobarbital slow IV
- 5 g deep IM of $MgSO_4$ (50% solution) every 4 hours, alternating buttocks provided: patellar reflexes are present, respiratory rate is > 16/min and urine output is >100 cc in the previous 4 hours

IV MAGNESIUM SULFATE

- 4-6 g IV of $MgSO_4$ USP ($MgSO_4$ > 7 H_2O) as a 20% solution IV at a rate not to exceed 1 g/min, preferably diluted to 100 cc and administered over 15-20 minutes
- 20 g of $MgSO_4$ diluted in 1000 cc of IV fluid given at a rate of 2 g/hour (100 cc/hr)
- Use half of the maintenance dose/hour if renal insufficiency is present
- If seizures persist > 15 minutes, give an additional 2-4 g IV over 2-4 minutes depending on maternal size.
- If seizures persist or recur following this, add 250 mg of amobarbital (Amytal) slow IV

MAGNESIUM SULFATE MONITORING

- Monitor patient reflexes, respiratory rate, and urine output on an hourly basis, halting therapy if patellar reflexes are absent, respiratory rate is < 16/min, or urine output is < 100 cc/4-hour period
- Monitor serum magnesium levels at 4-6 hours and adjust dose accordingly to maintain levels between 4-7 mEq/l (4.8-8.4 mg/dL; 2-4 mmol/L)
- Discontinue 24 hours after delivery or the last seizure

ANTIDOTE TO MAGNESIUM SULFATE

At all times that $MgSO_4$ is being administered, a 10 cc ampule of calcium gluconate 10% solution should be at the bedside, and given slowly IV when signs of toxicity are present. The dose may have to be repeated if signs/symptoms of toxicity recur despite halting magnesium therapy. Any time respiratory impairment occurs, prompt intervention with intubation and mechanical ventilation will be lifesaving.

Contraindications to $MgSO_4$ include myasthenia gravis and cardiac conduction defects in the setting of cardiac muscle damage. Caution should be use in patients on calcium channel blockers or with renal insufficiency.

PHENYTOIN DOSING

- Phenytoin (Dilantin) should be used cautiously if at all in the myasthenic patient, and is recommended only for prophylaxis of seizures; therefore, diazepam 10 mg IV is given to immediately control seizures then:
  - 1 g of phenytoin, or phenytoin equivalents should be administered IV over 20 minutes.
  - This is followed by any one of the following regimens:
    - 100 mg IV every 6 hours for 24 hours ***OR*** 500 mg orally 10 hours later ***OR*** after bolus start infusion of 500 mg diluted in 20 0cc saline over 4 hours, and repeat infusion 12 hours after the initial therapy.
- Phenytoin therapy should be continued for 24 hours after delivery or after the last seizure, whichever is later.

DIAZEPAM DOSING

- 10 mg IV over 2 minutes, repeated if seizure recurs, followed by a 40-80 mg/liter in 1000 cc D5%/W IV infusion over 24 hours, followed by a 20-40 mg/liter infusion over 24 hours

rapid admission is not possible. (*See discussion on magnesium sulfate therapy.*) (*See Tables 2 and 3.*) Alternatively, 1 g of methyldopa may be given orally, followed by 250-500 mg every 6 hours. The maximum dose is 4 g/day.[29]

Table 2 lists several antihypertensives that may be of use in pregnancy-related hypertension. Both IV hydralazine and IV labetolol have been used parenterally to manage the acute hypertensive emergency in pregnancy. Hydralazine IV has been associated with decreased blood flow in the placenta causing fetal distress, especially if the mother is not pre-treated with volume expansion. Women with preeclampsia often are at risk for pulmonary edema, and excessive fluid loading can be problematic.[30] Labetolol has become more popular as an IV agent to control severe hypertension because it hasn't been shown to require fluid loading, but it is problematic and often contraindicated in patients with underlying lung disease such as asthma.[10,31]

Although not directly related to the management of hypertension in pregnancy, the ED physician often is queried as to the safety of medications while breast-feeding. Occasionally blood pressure control must be initiated, or often maintained, once the patient has delivered the baby. This may have a direct impact on the breast-feeding mother and her infant. All antihypertensives studied have been found in breast milk.[31] Long-term neonatal effects have not been studied.[24] Methyldopa as a first-line oral agent is reasonable unless contraindicated, and then labetolol may become first-line therapy. If the patient has renal disease, then calcium channel blockers are the drugs of choice. ACE inhibitors and angiotensin II receptor antagonists should not be used due to neonatal renal effects. Diuretics may decrease milk production.[24] Certain beta blockers are concentrated in breast milk (atenolol [Tenormin] and metoprolol [Lopressor, Toprol]) while others are not (labetolol and propranolol [Inderal]).[32]

Table 4. Risk Factors for Preeclampsia[24,43,44]

- Primigravida
- Primipaternity (first child from this father, especially if he has fathered another preeclampsia pregnancy or barrier contraception was used prior to this pregnancy)
- Age > 35 years
- Multiple gestations, Hydrops fetalis, Hydatidiform mole
- Family history of preeclampsia
- Underlying medical conditions such as hypertension, diabetes, insulin resistance, obesity, or hyperhomocysteinemia
- Familial thrombophilic conditions such as Factor V Leiden, Protein S deficiency
- Sickle cell trait
- Employment at a high stress job

Table 5. Signs, Symptoms, or Lab Values Associated with Severe Preeclampsia[10,18,22]

- CNS dysfunction including CVA, headache, vision changes, or seizures (eclampsia)
- Right upper quadrant abdominal pain due to liver capsule distention
- Blood pressure >160-180 mmHg systolic or 110 mmHg diastolic on two occasions at least 6 hours apart
- Proteinuria > 3.5-5 g/24 hours
- Oliguria, renal failure, or elevated creatinine
- Pulmonary edema
- HELLP syndrome or any of its laboratory components separately (hemolysis, elevated liver enzymes, low platelets)

Key:
CNS = central nervous system; CVA = cerebrovascular accident

Preeclampsia and Hypertension with Superimposed Preeclampsia

Introduction. Preeclampsia is defined as the condition where hypertension and proteinuria, with or without generalized edema, develops after 20 weeks gestation.[33-34] (*See Table 1.*) This condition also may be superimposed on pre-existing hypertension, and thus the name superimposed preeclampsia. Preeclampsia complicates 2-3% to 2-8% of pregnancies, and 75-80% of preeclampsia patients are primigravidas.[4,5,33,35] Maternal complications of preeclampsia include eclampsia, but also disseminated intravascular coagulation (DIC), HELLP, pulmonary edema, renal failure, cerebral edema and hemorrhage, and liver failure with potential for hepatic rupture and hemorrhage.[10,25] Fetal complications are related to the severity of the decreased perfusion and may include intrauterine growth restriction, premature delivery, hypoxemia, acidosis, and death.[10]

Pathophysiology and Classification. The precise etiology of preeclampsia remains an enigma, with multiple unproven theories being offered.[36] Evidence of increased platelet and endothelial activation has been noted to precede preeclampsia in some women. Abnormal placentation causing ischemia of the placenta and release of toxic factors that result in endothelial damage is thought to cause vascular constriction, increased capillary leakage, and platelet aggregation leading to preeclampsia and, in more severe cases, the HELLP syndrome and eclampsia.[37,38] Immunologic factors also may be the instigator of preeclampsia and HELLP, with tumor necrosis factor (alpha) being cited as one possible cause of both disorders.[37] Oxidative stress, in which there is a preponderance of oxidants, has been postulated as yet another cause of endothelial damage in preeclampsia and HELLP. N-acetylcysteine has been proposed to help tip the balance in the direction of anti-oxidation, thus helping prolong the pregnancy and decreasing fetal and maternal morbidity and mortality.[39] Lastly, in normal pregnancies, cytotrophoblast cells invade the uterine spiral arteries and replace the normal endothelial lining of these vessels. This allows for the development of a low resistance arteriolar system to supply the growing fetus.[40] Due to unknown causes, this trophoblastic remodeling is limited to the proximal vessels in patients with preeclampsia, causing a decreased uteroplacental perfusion and leading to hypoxia of the placenta, causing regions of placental infarction.[40] Regardless of the underlying cause, patients with preeclampsia have increased vascular reactivity when compared to normal healthy pregnant controls or patients with HELLP syndrome.[41,42]

Preeclampsia and even eclampsia can occur under all socioeconomic conditions, and risk factors are listed in Table 4.[24,43,44] Preeclampsia is classified as either mild or severe; there is no moderate level. Severe is defined in a pregnant patient past 20 weeks gestation with new onset proteinuria, hypertension, and one or more of the following complicating signs, symptoms, or lab values: central nervous system (CNS) dysfunction, liver capsule distention, hypertension, proteinuria, oliguria or renal failure, pulmonary edema, HELLP syndrome or any of its lab components separately.[10,18,22] (*Table 5.*) Absence of these classifies the preeclampsia as mild.[33] However, preeclampsia by nature has an unpredictable course and, therefore, all patients, whether labeled mild or severe, should be managed cautiously. Rapid progression from mild disease to an eclamptic seizure is not uncommon.[18,45] HELLP syndrome is a form of severe preeclampsia, and approximately 4-12% of cases of preeclampsia manifest as HELLP syndrome.[46-49] (*See section on HELLP.*)

Clinical Presentation. The signs and symptoms of preeclampsia can be variable. Patients presenting with CNS dysfunction, including headache, visual disturbances, or evidence of a cerebrovascular accident (CVA), should be evaluated for preeclampsia. In addition, right upper quadrant pain may be the presenting complaint, and preeclampsia may be the final diagnosis when other etiologies are ruled out. Occasionally, preeclampsia patients will present in pulmonary edema or acute renal failure. At times, hypertension will be an incidental finding in the ED patient seeking care for another problem. Because preeclampsia may be abrupt in onset or the patient may be devoid of prior prenatal care, hypertension and/or peripheral edema noted on physical exam should prompt the treating physician to obtain a urine specimen to evaluate for proteinuria. If the

Table 6. Differential of Patients Presenting with Hypertension, Proteinuria, and Edema[10,17,50,73]

- Preeclampsia
- Eclampsia
- Cholecystitis
- Viral hepatitis
- Pancreatitis
- Encephalitis
- Pyelonephritis
- Glomerulonephritis
- Nephrolithiasis
- Lupus exacerbation
- Cerebral vein thrombosis
- Intracerebral aneurysm rupture
- Acute fatty liver of pregnancy

systolic blood pressure is greater than 160 mmHg or the diastolic blood pressure is greater than 100 mmHg, obstetrical consult should be sought from the ED and an antihypertensive agent begun. (*See discussion on management of hypertension.*) If preeclampsia is diagnosed based on hypertension, proteinuria, +/- peripheral edema, then hospital admission is the norm, at least for a period of observation to further evaluate and monitor the mother and fetus.

Imitators of preeclampsia may include hemolytic uremic syndrome (HUS), thrombotic thrombocytopenia (TTP), acute fatty liver of pregnancy, autoimmune diseases such as system lupus erythematosis (SLE), and sepsis.[32,50] The same organ systems can be affected in all diseases in this differential, and preeclampsia may be superimposed on any of them, making the final diagnosis difficult and one that takes many tests and often much time to delineate. In the ED, ordering the initial battery of tests, namely a complete blood count (CBC) with peripheral smear, prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen, blood urea nitrogen (BUN), creatinine, glucose, electrolytes, uric acid, and hepatic panel to include bilirubin, LDH, AST, and ALT as well as a urinalysis will help begin the process. Obstetrics should be involved early, and a hematology consult may be necessary for plasma exchange in the patient with TTP. TTP and HUS are less common than HELLP syndrome, and often present with bleeding, bruising, or epistaxis. Mental status changes including seizures may occur, and abdominal pain is frequent. Fever and elevated creatinine occur in half of patients, and most demonstrate microscopic proteinuria and hematuria. However, DIC is uncommon with TTP and HUS, and, therefore, fibrinogen, PT, and PTT are normal. In addition, more than 50% of cases of TTP occur before 24 weeks gestation, and plasma transfusions and exchanges markedly have increased the survival, thus the need for urgent hematology consultation if TTP or HUS is suspected.[50]

Acute fatty liver of pregnancy usually develops in the third trimester and carries a 90% mortality if untreated. Presenting symptoms include headache, nausea, vomiting, abdominal pain, or malaise, with 50% of patients demonstrating hypertension and thrombocytopenia. Hypoglycemia and hyperammonemia suggest the disease. Abnormal coagulation studies in the form of decreased fibrinogen and abnormal PT and PTT are present 70-80% of the time. Elevation of bilirubin and hepatic transaminases is typical.[50]

Autoimmune diseases will share common findings with preeclampsia, but serologic testing for disorders such as SLE and antiphospholipid antibody syndrome should help differentiate the disorders. Finally, sepsis may mimic preeclampsia and/or HELLP syndrome. Often the vital signs will help make the diagnosis. Blood cultures and early broad spectrum antibiotics are essential for treatment of sepsis.[50]

Prevention. In recent years, several attempts to prevent preeclampsia have given mixed results. Low-dose aspirin therapy, vitamin and mineral supplements, calcium supplements, nutritional supplements such as fish oil, and various antihypertensive regimens have been studied, with none proven to be effective in all groups.[1,51-54] Aspirin given at 100 mg/day ingested eight hours after arising demonstrated a significant decrease in blood pressure when compared to the effect when taken immediately upon arising, and an even stronger effect was noted if the aspirin was taken at bedtime.[54] Until the pathogenesis of preeclampsia is defined more accurately, no accurate prevention is likely.[52]

Management of Preeclampsia. Over the years, many management strategies have been used to treat the preeclampsia patient. Definitive therapy is delivery, with the first priority being the safety of the mother, and the second priority the delivery of a mature live newborn.[55] Initial evaluation of the preeclamptic patient usually is performed as an in-patient and, following a thorough evaluation of the mother and fetus, a variety of options may be chosen, including at-home monitoring, day units, or continued hospitalization.[15] Therapy with at-home monitoring by the mother and frequent out-patient clinic visits now has become the standard in reliable patients without co-morbid diseases.[26]

Despite the fact that delivery is the definitive treatment for preeclampsia, increased efforts have been instituted to manage severe preeclampsia remote from term to increase fetal viability and decrease long-term sequelae associated with premature birth. This should be done only in a tertiary center with the ability to monitor the mother and fetus appropriately. The precise age of the fetus is most important in making clinical decisions that may have long-ranging consequences. In selected patients, hospital admission, careful monitoring, corticosteroid administration to encourage fetal lung maturation, and prophylaxis with magnesium sulfate[26, 56-64] (*see Table 3*) to prevent seizures, along with oral antihypertensives to control blood pressure in the 130-150/80-100 mmHg range (*see Table 1*) has been used with some success.[65] The frequency of eclampsia was decreased in patients with severe preeclampsia by the use of magnesium sulfate.[15,66,67]

Eclampsia

Introduction. Eclampsia is a disorder confined exclusively to the pregnant or newly delivered patient up to two weeks postpartum and still accounts for significant fetal and maternal morbidity and mortality, with approximately 50,000 maternal deaths

Table 7. Signs and Symptoms of Magnesium Toxicity[28,58,64,77,79]

SIGNS/SYMPTOMS	LEVEL IN MG/DL	LEVEL IN MEQ/L	LEVEL IN MMOL/L
Therapeutic range	4-8	3.3-6.7	2-4
Nausea, warm feeling, somnolence, diplopia, dysarthria, weakness	9-12	7.5-10	3.5-5
Loss of patellar reflex	9-12	10	> 5 (3.5-7)
Respiratory depression	12	10	> 6
Muscle paralysis	14.1 (14-17)	11.75 (12.5-14.1)	6-7.5
Cardiotoxicity Prolonged PR and QRS	> 14.1	> 11.75	> 7.5
Respiratory arrest	14.4 (14-17)	12 (12-14.1)	5-7.5
Cardiac arrest	31.5 (30-35)	26.25 (25-29)	12.5

annually worldwide.[4] It is defined as cerebral involvement resulting in convulsions in association with pre-eclampsia. It is further divided into antepartum, intra-partum, and postpartum, depending on when convulsions occur in relation to labor.[26] Eclampsia complicates approximately 1 in 2000 pregnancies in developed countries, but may rise as high as 1 in 100 pregnancies in developing countries.[35,56,57,68] It complicates 0.2-0.5% of all deliveries.[10] Up to 30% of women with eclampsia will have HELLP syndrome, which results in a greater number of pre-term infants and higher mortalities than in patients with eclampsia alone.[69]

The frequency of eclamptic seizures in women with preeclampsia is fewer than 1%.[70,71] Approximately 40% of seizures occur antepartum before women receive medical attention, and nearly 50% occur at a gestational age fewer than 36 weeks. Sixteen percent of eclamptic seizures occur more than 48 hours post-partum. Unfortunately, this means only 45-50% of all seizures will occur during a time when the patient actually is in the hospital being monitored, severely limiting efforts at seizure prophylaxis.[71,72] Ten percent of women who suffer an eclamptic seizure will develop some form of blindness, which usually resolves within a week and often is due to retinal detachment or occipital lobe involvement with edema, ischemia, or infarct.[26] The pathophysiology of eclampsia is an extension of preeclampsia, and predicting which patients will progress to this most severe form remains a mystery.

Clinical Presentation and ED Management. Eclampsia should be the ED working diagnosis in any pregnant patient past 20 weeks who presents with a new onset grand mal seizure. Although the differential should be sufficiently broad to include underlying epilepsy, intracerebral events such as a CVA, tumor or subarachnoid hemorrhage, as well as drug ingestions, the initial work up needs to include an urgent dipstick urine analysis to rule in or out proteinuria. By definition, eclampsia is a seizure in conjunction with preeclampsia. However, the patient may not be aware of her preeclamptic state due to rapid onset of preeclampsia in some patients, rapid progression from preeclampsia to eclampsia in some, and lack of prenatal care prior to the onset of seizure in others. The ED physician also should be suspicious of this diagnosis in the newly post-partum patient (up to 2 weeks) who presents with a grand mal seizure. Although eclampsia often is a diagnosis of exclusion, the work-up is fairly straightforward and should include a thorough history and physical with attention to the neurologic exam and fetal monitoring for signs of distress. This is followed by basic metabolic studies, a urine analysis, non-contrast head CT scan, and a urine drug screen. The differential for the pregnant patient with hypertension, proteinuria, and edema is broad and includes many conditions other than preeclampsia or eclampsia.[10,17,50,73] (*See Table 6.*) However, with appropriate lab tests and a non-contrast head CT scan, the ED physician should be able to rapidly rule out most other causes of these disorders.

Patients with eclamptic seizures are at risk for status seizures. In antepartum eclampsia, labor may ensue and delivery may be rapid, at times before the post-ictal period is resolved.[57] Continuous fetal monitoring in the ED is warranted, with emergent obstetric consultation. Patients may not present to the hospital of planned delivery with their eclamptic seizure; therefore, regardless of the level of obstetrical care available at the institution, the ED physician must be adept at making this diagnosis and begin rapid emergent treatment of the mother and monitoring of the fetus. (*See below.*) Overall, 6.5% of patients with eclampsia develop multiple neurological complications, including cortical blindness, aphasia, paresis, psychosis, coma, or cerebrovascular accidents.[74] CT scans in patients with eclampsia often yield relevant diagnoses. Approximately 43% are normal, 44% have hypodensities, 6% reveal intracerebral hemorrhages, and 6% hydrocephalus.[74] There are two

reported cases in the literature of eclamptic seizures precipitated by the administration of the anticholinergic drug scopolamine for epigastric pain in the patient with HELLP syndrome. The proposed mechanism is unopposed sympathetic nervous system activity due to inhibition of the parasympathetic system, exacerbating vasospasm, and hypertension.[75]

Management of Eclampsia. Management of eclampsia is multifaceted and must include management of hypertension (*see previous section*), prevention of further seizures, and safe delivery of the fetus once the mother is stabilized. The majority of eclamptic seizures will resolve in 60-90 seconds, and delivery should be planned as soon as the mother's condition permits.[70] Magnesium therapy is designed to prevent further seizures and has been established as the most effective drug to prevent recurrent seizures in the eclamptic patient.[72] Intravenous and IM regimens exist and are effective for magnesium sulfate therapy, but the IM route is more painful, associated with abscess formation in 0.5% of cases, and very risky in patients with HELLP syndrome and a platelet count of fewer than 100,000.[76] (*See discussion below.*)

Use of Magnesium Sulfate. Magnesium sulfate first was administered intrathecally in 1906 to prevent eclamptic seizures. It first was used IM in 1926 to prevent recurrent seizures, and in 1933 it was given IV to women with preeclampsia and eclampsia.[59] Worldwide, the use of magnesium sulfate for eclampsia is variable. The Collaborative Eclampsia Trial has established magnesium sulfate as superior to diazepam or phenytoin for the prevention of recurrent seizures in eclampsia. In addition, it may have benefits to the fetus, and in preeclamptic females may reduce the risk of first convulsions.[57,67,77,78] Both the IM and IV regimens begin with 4 g of a 20% solution of magnesium sulfate IV over 4 minutes. In the IM regimen, this immediately is followed by 5 g IM of a 50% solution of magnesium sulfate in each buttock, followed by 5 g IM every 4 hours, alternating buttocks. In the IV regimen, the initial loading dose is followed by a continuous infusion of 2 g/hour—generally mixed as 20 g in a liter of IV solution and run at 100 cc/hour. The IV maintenance dose should be cut in half in the presence of renal insufficiency. In either case, if seizures persist after 15 minutes, an additional load of 2-4 g of magnesium sulfate is given IV, depending on maternal size. If the seizure persists, 250 mg of amobarbital (Amytal) should be given slowly IV. The patient receiving magnesium therapy must be monitored closely. Patellar reflexes, respiratory rate, and hourly urine output should be monitored in each patient, and magnesium therapy should be halted in the absence of patellar reflexes, a respiratory rate less than 16/min, or urine output less than 100 cc/4-hour period. Magnesium levels should be monitored every 4-6 hours, and the dose adjusted to maintain levels between 4 and 7 mEq/liter (4.8 - 8.4 mg/dL; 2-4 mmol/L). (*See Table 3.*)[26,56-64] The conversion of magnesium levels from mEq/L to mg/dL is as follows: 1.2 times magnesium level in mEq/L = magnesium level in mg/dL.[64]

Table 7 lists the common signs/symptoms of magnesium toxicity. Early signs and symptoms include nausea, somnolence, a sensation of warmth, diplopia, dysarthria, and weakness. Further increases in levels lead to loss of patellar reflexes, followed by respiratory depression, muscle paralysis, prolonged PR and QRS on ECG, respiratory arrest, followed by cardiac arrest and death.[26,58,59,64,77,79] In addition to being cautious with magnesium sulfate in patients on calcium channel blockers, the ED physician needs to give consideration to other drug/drug interactions that may occur, especially with paralytics. When performing rapid sequence intubation, the ED physician should not give a defasciculating dose of a non-depolarizing blocker. A single dose of succinylcholine (Anectine) 1 mg/kg may be used without difficulty. If paralytics need to be continued, the succinylcholine should be allowed to wear off, and then the dose of the non-depolarizing neuromuscular blocking agent should be decreased due to its potentiation with magnesium sulfate.[80,81]

Use of Other Anticonvulsants. In the patient in whom magnesium therapy is contraindicated (those with myasthenia gravis, cardiac conduction defects in the setting of cardiac muscle damage), or in those in whom it should be used cautiously if at all (those on calcium channel blockers or with renal insufficiency), phenytoin (Dilantin) or diazepam (Valium) may be used. It should be noted that in the myasthenic patient, phenytoin is not without risk, and therefore diazepam is probably the drug of choice. Initially diazepam should be used to control the seizure, beginning with 10 mg IV over 2 minutes, repeated if seizure recurs. Prevention of further seizures may continue with diazepam via infusion 40-80 mg/liter of D5/W over 24 hours titrated to seizure control and restlessness, followed by 20-40 mg/ liter D5W over the second 24 hours. Alternatively, once the seizure has been controlled, prevention may begin with phenytoin (or phenytoin equivalents) 1 g IV over 20 minutes, followed by one of the following: 100 mg IV every 6 hours for 24 hours; 500 mg orally 10 hours after initial dose; or an infusion of 500 mg in 200 cc of NS over four hours beginning immediately after the bolus, and repeated once 12 hours after the bolus. Phenytoin should be continued for 24 hours after delivery or after the last seizure, whichever is later.

HELLP Syndrome

Introduction. The HELLP syndrome is a severe form of preeclampsia that occurs in 0.2-0.8% of all pregnancies,[46,73,82,83] carries a 1-2% maternal mortality rate when care is provided in a tertiary center,[84,85] and an 11% perinatal mortality.[86] It occurs in 20% of women with severe preeclampsia and 10% of women with eclampsia.[84] The development of liver disease and a coagulopathy in association with hypertension portend a poorer fetal and maternal outcome in preeclampsia, as does a platelet count of fewer than 50,000.[46,69] In contrast to non-HELLP preeclampsia, HELLP syndrome is more common in multiparous women who are Caucasian.[84]

HELLP syndrome has been further classified as follows:

Class I: maternal platelet count < 50,000/microliter

Class II: maternal platelet count >50,000 and < 100,000/microliter

Class III: maternal platelet count > 100,000 and < 150,000/microliter

In one study, all groups with HELLP syndrome had higher maternal and perinatal morbidity than a control group with severe preeclampsia without HELLP.[69]

Maternal morbidity may include premature delivery, an increased cesarean section rate, more use of blood products, multisystem organ failure, sepsis, adult respiratory distress syndrome (ARDS), pulmonary edema, ascites, pleural effusion, cerebral edema, retinal detachment, hemorrhage (both spontaneous and postpartum), DIC, or hepatic rupture. Given the severe nature of HELLP complications, HELLP can be fatal for both mother and fetus.[46,84] The pathophysiology of HELLP is similar to preeclampsia. The reason certain patients go on to develop the HELLP syndrome while most do not is still an enigma and hinders definitive therapy.[69]

Clinical Presentation. The most common presenting complaints of this disorder are epigastric or right upper quadrant pain (65-90% of patients) believed to be secondary to liver capsule distention, often associated with nausea and vomiting.[46,73,69] These symptoms worsen as the severity of HELLP worsens.[83] The mother often has a shallow respiratory pattern due to diaphragmatic irritation, which may predispose her to atelectasis and pneumonia.[25] In Class I HELLP syndrome, 35% reported nausea with or without vomiting, 50% reported epigastric pain, and 61% reported a headache. Mean blood pressures were lower in patients with Class I or II HELLP syndrome when compared to patients with severe preeclampsia alone. Although HELLP is a form of preeclampsia, be aware that the presence of proteinuria and hypertension are variable, with up to 14% of patients with HELLP having no proteinuria, and nearly 20% being normotensive.[83,87] HELLP syndrome occurs most often before term, although up to one-third of cases may present post-partum, with development up to seven days post-partum reported.[84,88] The differential is similar to preeclampsia and is listed in Table 6. (*See previous discussion under pre-eclampsia section.*) Coagulation studies will help differentiate acute fatty liver of pregnancy and DIC from the other disorders. Most other labs tests are somewhat variable, and the patient condition as well as clinical experience will be necessary to reach a final diagnosis.

The intensity of HELLP generally reaches a peak 24 hours after delivery, and lab values return to normal following this in most patients.[73] Patients who do not demonstrate an increase in platelet count by 96 hours after delivery often have severe systemic, non-compensated disease.[69] Patients with more severe HELLP have shorter times from admission to delivery and longer lengths of stay overall.[83]

ED Evaluation. The ED physician evaluating any pregnant or recently delivered woman with complaints of upper abdominal pain should consider and work up the patient for the HELLP syndrome. Even though this is a preeclampsia syndrome, not all patients have hypertension or proteinuria.[84,89] This variability in patient presentation makes the possibility of misdiagnosis by the ED physician more likely. As with other hypertensive disorders of pregnancy, the criteria used to define HELLP vary somewhat. The most useful lab tests are a CBC with a peripheral smear, LDH and transaminases, creatinine, uric acid, fractionated bilirubin, and a urine analysis. Coagulation studies are useful in patients with platelets fewer than 100,000.[69,73] Thrombocytopenia is defined as a platelet count fewer than 150,000, and liver enzymes (LDH and the transaminases) are mildly to moderately elevated in most cases. Evidence of hemolysis may be seen on a peripheral smear, or the hematocrit may be low without another etiology.[46,49,82,90] A low threshold for admission to obstetrics from the ED should be maintained by the treating ED physician.

Misdiagnoses of HELLP syndrome include gastritis, cholelithiasis, appendicitis, pancreatitis, nephrolithiasis, pyelonephritis, viral gastroenteritis, and hepatitis. In the converse, severe diseases misdiagnosed as HELLP syndrome include dissecting aneurysms, cardiomyopathy, chronic hypertension with concomitant renal disease, acute fatty liver, alcoholic liver disease, cholecystitis, glomerulonephritis, and acute cocaine intoxication. As misdiagnosis may result in catastrophic maternal and fetal consequences, one should consider HELLP syndrome in patients diagnosed with the above conditions.[69]

In addition, it has been determined that on admission nausea, vomiting, or epigastric pain are all independent risk factors for increased maternal morbidity in patients with severe preeclampsia or HELLP syndrome. Laboratory values on admission that portend a high risk for significant maternal morbidity include; LDH greater than 1400 IU/L, AST greater than 150 IU/L, ALT greater than 100 IU/L, uric acid greater than 7.8 mg/dL, urinary protein of 4+, serum creatinine greater than 1.0 mg/dL. The LDH, AST, and uric acid have the strongest predictive value and are risk additive with worsening thrombocytopenia.[91]

Management of HELLP. Once the diagnosis of the HELLP syndrome has been established, consideration should be given to

Table 8. Pitfalls in the ED Management of Patients with Preeclampsia, HELLP, and Eclampsia

- Failure to rule out HELLP syndrome or preeclampsia in the patient presenting with a non-pregnancy related complaint, when their blood pressure is noted to be elevated.
- Failure to rule out HELLP syndrome in the evaluation of a pregnant patient past 20 weeks gestation with upper abdominal pain, nausea, or vomiting.
- Failure to consider HELLP syndrome in the newly delivered patient who presents to the ED with upper abdominal pain, nausea, or vomiting.
- Failure to administer dexamethasone early based on clinical suspicion in the pregnant patient past 20 weeks who may have HELLP syndrome.
- Failure to consider eclampsia as the etiology of a grand mal seizure in the patient fewer than 2 weeks post-partum.
- Failure to avoid anticholinergic medications in the patient with severe preeclampsia, especially in the face of upper abdominal pain.
- Failure to search current medication lists for ACE inhibitors and angiotensin receptor antagonists and change them in consultation with obstetrical or primary care consultation in the newly pregnant patient.

transfer to a tertiary care facility if available. Urgent delivery is the usual treatment, although under certain circumstances in a tertiary facility prepared for delivery 24 hours/day, expectant management may be considered to allow for steroid administration, especially if the gestational age is fewer than 34 weeks. In addition to fetal lung maturation, antenatal steroids and/or postpartum steroids have been shown to stabilize the maternal condition.[85,92,93] The only contraindication to antenatal steroids is intrauterine infection or maternal tuberculosis.[58,85,92,93] The current recommended protocol is dexamethasone 10 mg IV every 12 hours through delivery and into the postpartum period until the maternal blood pressure has stabilized, urine output is adequate without fluid boluses or diuretics, platelets are greater than 100,000, and the patient is clinically stable. Following this, two additional doses of 5 mg dexamethasone are given every 12 hours.[85,92,93] Isler, in a double blind study, demonstrated that dexamethasone 10 mg IV every 12 hours was superior to 12 mg betamethasone IM every 24 hours for the stability of HELLP syndrome.[94] Dexamethasone IV often allows a 48-hour window in the fetus fewer than 34 weeks gestational age for fetal lung maturation.[95] The ED physician faced with a pregnant patient fewer than 34 weeks gestation who presents with preeclampsia or HELLP should administer the initial dose of dexamethasone, providing contraindications are absent (i.e., intrauterine infection, maternal TB).

In summary, if the ED physician suspects the HELLP syndrome, plans should be made to admit or transfer the patient to a tertiary care facility and, if the patient is fewer than 34 weeks gestation, dexamethasone 10 mg IV should be given. In addition, as eclamptic seizures frequently develop in the HELLP patient, magnesium sulfate therapy is indicated as a prophylactic measure to prevent the development of this most serious complication, and may need to be continued 48 hours into the puerperium until evidence of recovery is apparent.[69] (*See Table 3.*) (*See Table 8 for a summary of pitfalls in ED management of patients with preeclampsia, HELLP, or eclampsia.*)

References

1. Wallenburg HC. Prevention of pre-eclampsia: Status and perspectives 2000. *Eur J Obstet Gynecol Reprod Biol* 2001;94:13-22.
2. Granger JP, Alexander BT, Bennett WA, et al. Pathophysiology of pregnancy induced hypertension. *AJH* 2001;14:178S-185S.
3. Davey DA, MacGillivray I. The classification and definition of the hypertensive disorders of pregnancy. *Am J Obstet Gynecol* 1988; 158:892-898.
4. Duley L. Maternal mortality associated with hypertensive disorders of pregnancy in Africa, Asia, Latin America and the Caribbean. *Br J Obstet Gynaecol* 1992;99:547-553.
5. Department of Health; Welsh Office; Scottish Home and Health Department; Department of Health and Social Services, Northern Ireland: Report on confidential enquiries into maternal deaths in the United Kingdom 1978-1988. HM Stationery Office, London, 1994: 22-33.
6. Wallenburg HC, Visser W. Pregnancy-induced hypertensive disorders. *Curr Opin Obstet Gynecol* 1994;6:19-29.
7. Granger JP, Alexander BT, Bennett WA, et al. Pathophysiology of pregnancy-induced hypertension. *Am J Hypertens* June 2001;14: 178S-185S.
8. Saftlas AF, Olson DR, Franks AL, et al. Epidemiology of preeclampsia and eclampsia in the United States, 1979-1986. *Am J Obstet Gynecol* 1990;163:460-465.
9. Rochat RW, Koonin LM, Atrash JF. The Maternal Mortality Collaborative. Maternal Mortality in the United States: Report from the Maternal Mortality Collaborative. *Obstet Gynecol* 1988;72:91-97.
10. Perloff D. Hypertension and pregnancy-related hypertension. *Cardiol Clin* 1998;16:79-101.
11. Das V, Bhargava T, Das SK, et al. Microalbuminuria: A predictor of pregnancy-induced hypertension. *Br J Obstet Gynaecol* 1996;103: 928-930.
12. Hsu, CD, Tan H-Y, Hong S-F, et al. Strategies for reducing the frequency of preeclampsia in pregnancies with insulin-dependent diabetes mellitus. *Am J Perinatol* 1996:13:265-268.
13. American College of Obstetricians and Gynecologists, Committee on Obstetrics. Hypertension in pregnancy. Washington, DC: Technical Bulletin Number 219, January, 1996.
14. Mabie WC. Management of acute severe hypertension and encephalopathy. *Clin Obstet Gynecol* 1999;42:519-531.
15. [No authors listed.] Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy. *Am J Obstet Gynecol* 2000; 183:s1-s22.
16. Brown MA, Simpson JM. Diversity of blood pressure recording during pregnancy: Implications for the hypertensive disorders. *Med J Aust* 1992;156:306-308.
17. Higgins JR, deSwiet M. Blood-pressure measurement and classification in pregnancy. *Lancet* 2001;357:131-135.
18. Garovic VD. Hypertension in pregnancy: Diagnosis and treatment. *Mayo Clin Proc* 2000;75:1071-1076.
19. Chappell L, Poulton L, Halligan A, et al. Lack of consistency in research papers over the definition of pre-eclampsia. *Br J Obstet Gynaecol* 1999;106:983-985.
20. Walker JJ. Pre-eclampsia. *Lancet* 2000;356:1260-1265.
21. Update on the Task Force (1987) on High Blood Pressure in Children and Adolescents: A working group from the National High Blood Pressure Education Program. *Pediatrics* 1996;98:649-658.
22. Norwitz ER. Robinson JN, Repke JT. Prevention of preeclampsia: Is it possible? *Clin Obstet Gynecol* 1999;42:436-454.
23. Sibai BM. Diagnosis and management of chronic hypertension in pregnancy. *Obstet Gynecol* 1991;78:451-461.
24. Sibai BM, Lindheimer M, Hauth J, et al. Risk factors for preeclampsia, abruptio placenta, and adverse neonatal outcomes among women with chronic hypertension. *N Engl J Med* 1998;339: 667-671.
25. Sibai BM, Anderson GD. Pregnancy outcome of intensive therapy in severe hypertension in first trimester. *Obstet Gynecol* 1986;67: 517-522.
26. Greer IA. Thrombosis in pregnancy: Maternal and fetal issues. *Lancet* 1999;353:1258-1265.
27. Linton DM, Anthony J. Critical care management of severe preeclampsia. *Intensive Care Med* 1997;23:248-255.

28. Hypertensive Disorders in Pregnancy. In: Cunningham FG, Gilstrap LC III, Gant NF, et al eds. *Williams Obstetrics,* 21st ed. New York: McGraw-Hill; 2001:567-618.
29. Khedun SM, Moodley J, Naicker T, et al. Drug management of hypertensive disorders of pregnancy. *Pharmacol Ther* 1997;74: 221-258.
30. Magee LA. Treating hypertension in women of child-bearing age and during pregnancy. *Drug Saf* 2001;24:457-474.
31. White WB. Management of hypertension during lactation. *Hypertension* 1985;6:297-300.
32. Atkinson H, Begg EJ. Concentrations of beta-blocking drugs in human milk. *J Pediatr* 1990;116:156.
33. Livingston JC, Sibai BM. Chronic hypertension in pregnancy. *Obstet Gynecol Clin North Am* 2001;28:447-463.
34. Redman CWG, Jeffries M. Revised definition of pre-eclampsia. *Lancet* 1988;1(8589):809-812.
35. World Health Organization Internal Collaborative Study of Hypertensive Disorders of Pregnancy. Geographic variation in the incidence of hypertension in pregnancy. *Am J Obstet Gynecol* 1988; 158:80-83.
36. Dekker GA, Sibai BM. Etiology and pathogenesis of preeclampsia: Current concepts. *Am J Obstet Gynecol* 1998;179:1359-1375.
37. Curtin WM, Weinstein L. A review of HELLP syndrome. *J Perinatol* 1999;19:138-143.
38. Van Beek E, Peters LLH. Pathogenesis of preeclampsia: A comprehensive model. *Obstet Gynecol Surv* 1998;53:233-239.
39. Roes EM, Raijmakers MTM, Zusterzeel PLM, et. al. Deficient detoxifying capacity in the pathophysiology of preeclampsia. *Med Hypotheses* 2000;55:415-418.
40. Granger JP, Alexander BT, Llinas MT, et al. Pathophysiology of hypertension during preeclampsia linking placental ischemia with endothelial dysfunction. *Hypertension* 2001;38[part 2]:718-722.
41. Fischer T, Schneider MP, Schobel HP, et al. Vascular reactivity in patients with preeclampsia and HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome. *Am J Obstet Gynecol* Dec 2000;183:1489-1494.
42. Cunningham FG, Lindheimer MD. Hypertension in pregnancy. *N Engl J Med* 1992;326:927-932.
43. Roberts JM, Cooper DW. Pathogenesis and genetics of pre-eclampsia. *Lancet* 2001;357:53-56.
44. Dekker GA. Risk factors for preeclampsia. *Clin Obstet Gynecol* 1999;42:422-435.
45. Page NM, Lowry PJ. Is "pre-eclampsia" simply a response to the side effects of a placental tachykinin? *J Endocrinol* 2000;167: 355-361.
46. Saphier CJ, Repke JT. Hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome: A review of diagnosis and management. *Semin Perinatol* 1998;22:118-133.
47. Reubinoff BE, Schenker JG. HELLP syndrome—a syndrome of hemolysis, elevated liver enzymes and low platelet count—complicating preeclampsia-eclampsia. *Int J Gynaecol Obstet* 1991; 36: 95-102.
48. Usta IM, Sibai BM. Emergent management of puerperal eclampsia. *Obstet Gynecol Clin North Am* 1995;22:315-335.
49. Sibai BM, Taslimi MM, el-Nazer A, et al. Maternal-perinatal outcome associated with the syndrome of hemolysis, elevated liver enzymes, and low platelets in severe eclampsia-pre-eclampsia. *Am J Obstet Gynecol* 1986;155:501-509.
50. Egerman RS, Sibai BM. Imitators of preeclampsia and eclampsia. *Clin Obstet Gynecol* 1999;42:551-562.
51. Heyborne KD. Preeclampsia prevention: Lessons from the low-dose aspirin therapy trials. *Am J Obstet Gynecol* 2000;183:523-528.
52. Duley L, Henderson-Smart D, Knight M, et al. Antiplatelet drugs for prevention of pre-eclampsia and its consequences: Systematic review. *BMJ* 2001;322:329-333.
53. Sibai BM. Prevention of preeclampsia: A big disappointment. *Am J Obstet Gynecol* 1998;179:1275-1278.
54. Hermida RC, Ayala DE, Fernandez JR, et al. Administration time-dependent effects of aspirin in women at differing risk for preeclampsia. *Hypertension* 1999;34:1016-1023.
55. Barton JR, Witlin AG, Sibai BM. Management of mild preeclampsia. *Clin Obstet Gynecol* 1999;42:455-469.
56. Ramin KD. The prevention and management of eclampsia. *Obstet Gynecol Clin North Am* 1999;26:489-503.
57. [No authors listed.] Which anticonvulsant for women with eclampsia? Evidence from the Collaborative Eclampsia Trial. *Lancet* 1995; 345:1455-1463.
58. Moran P, Davison JM. Clinical management of established pre-eclampsia. *Baillieres Best Pract Res Clin Obstet Gynaecol* 1999; 13:77-93.
59. Idama TO, Lindow SW. Magnesium sulphate: A review of clinical pharmacology applied to obstetrics. *Br J Obstet Gynaecol* 1998; 105:260-268.
60. Pritchard JA, Cunningham FG, Pritchard SA. The Parkland Memorial Hospital protocol for treatment of eclampsia: Evaluation of 245 cases. *Am J Obstet Gynecol* 1984;148:951-963.
61. Zuspan FP. Treatment of severe pre-eclampsia and eclampsia. *Clin Obstet Gynecol* 1966;9:945-972.
62. Dommisse J. Phenytoin sodium and magnesium sulfate in the management of eclampsia. *Br J Obstet Gynaecol* 1990;97:104-109.
63. Crowther C. Magnesium sulphate versus diazepam in the management of eclampsia: A randomized controlled trial. *Br J Obstet Gynaecol* 1990;97:110-117.
64. McCubbin, JH, Sibai BM, Abdella TN, et al. Cardiorespiratory arrest due to acute maternal hypermagnesaemia. *Lancet* 1981; I(8228):1058.
65. Friedman SA, Schiff E, Lubarsky SL, et al. Expectant management of severe preeclampsia remote from term. *Clin Obstet Gynecol* 1999;42:470-478.
66. Coetzee EF, Dommisse J, Anthony J. A randomised controlled trial of intravenous magnesium sulfate versus placebo in the management of women with severe pre-eclampsia. *Br J Obstet Gynaecol* 1998;105:300-303.
67. Lucas MJ, Leveno KJ, Cunningham FG. A comparison of magnesium sulfate with phenytoin for the prevention of eclampsia. *N Engl J Med* 1995;333:201-205.
68. Douglas KA, Redman CWG. Eclampsia in the United Kingdom. *BMJ* 1994;309:1395-1400.
69. Magann EF, Martin JN Jr. Twelve steps to optimal management of HELLP syndrome. *Clini Obstet Gynecol* 1999;42:532-550.

70. Witlin AG. Prevention and treatment of eclamptic convulsions. *Clin Obstet Gynecol* 1999;42:507-518.
71. Witlin AG, Sibae BM. Magnesium sulfate therapy in preeclampsia and eclampsia. *Obstet Gynecol* 1998;92:883-889.
72. Lubarsky SL, Barton JR, Friedman SA, et al. Late postpartum eclampsia revisited. *Obstet Gynecol* 1994;83:503-505.
73. Jones SL. HELLP! A cry for laboratory assistance: A comprehensive review of the HELLP syndrome highlighting the role of the laboratory. *Hematopathol Mol Hematol* 1998;11:147-171.
74. Thomas SV. Neurological aspects of eclampsia. *J Neurol Sci* 1998;155:37-43.
75. Kobayashi T, Sugimura M, Tokunaga N, et al. Anticholinergics induce eclamptic seizures. *Semin Thromb Hemost* 2002;28:511-514.
76. Anumba DOC, Robson SC. Management of pre-eclampsia and haemolysis, elevated liver enzymes and low platelets syndrome. *Curr Opin Obstet Gynecol* 1999;11:149-156.
77. Sibai BM. Magnesium sulfate is the ideal anticonvulsant in pre-eclampsia-eclampsia. *Am J Obstet Gynecol* 1990;162:1141-1145.
78. Nelson KB, Grether JK. Can magnesium sulfate reduce the risk of cerebral palsy in very low birth weight infants? *Pediatrics* 1995;95: 263-269.
79. Lu JF, Nightingale CH. Magnesium sulfate in eclampsia and pre-eclampsia. *Clin Pharmacokinet* 2000;38:305-314.
80. James MFM, Cork RC, Dennett JE. Succinylcholine pretreatment with magnesium sulfate. *Anesth Analg* 1986;65:373-376.
81. Baraka A, Yazigi A. Neuromuscular interaction of magnesium with succinylcholine-vecuronium sequence in the eclamptic parturient. *Anesthesiology* 1987;67:806-808.
82. Weinstein L. Syndrome of hemolysis, elevated liver enzymes, and low platelet count: A severe consequence of hypertension in pregnancy. *Am J Obstet Gynecol* 1982;142:159-167.
83. Martin JN, Rinehart BK, May WL, et al. The spectrum of severe preeclampsia: Comparative analysis by HELLP (hemolysis, elevated liver enzyme levels, and low platelet count) syndrome classification. *Am J Obstet Gynecol* 1999:180:1373-1384.
84. Sibai BM, Ramadan MK, Usta I, et al. Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes and low platelets (HELLP Syndrome). *Am J Obstet Gynecol* 1993;169:1000-1006.
85. Magann EF, Martin JN. Critical care of the HELLP syndrome with corticosteroids. *Am J Perinatol* 2000;17:417-422.
86. Egerman RS, Sibai BM. HELLP Syndrome. *Clin Obstet Gynecol* 1999;42:381-389.
87. Sibai BM. The HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets): Much ado about nothing? *Am J Obstet Gynecol* 1990;162:311-316.
88. Martin JN, Stedman CM. Imitators of preeclampsia and HELLP syndrome. *Obstet Gynecol Clin North Am* 1991;18:181-198.
89. Schroder W, Heyl W. HELLP syndrome—difficulties in diagnosis and therapy of a severe form of preeclampsia. *Clin Exp Obstet Gynecol* 1993;20:88-94.
90. Martin JN Jr, Files JC, Blake PG, et al. Plasma exchange for preeclampsia. I. Postpartum use for persistently severe pre-eclampsia with HELLP syndrome. *Am J Obstet Gynecol* 1990;162: 126-137.
91. Martin JN, May WL, Magann EF, et al. Early risk assessment of severe preeclampsia: Admission battery of symptoms and laboratory tests to predict likelihood of subsequent significant maternal morbidity. *Am J Obstet Gynecol* 1999;180:1407-1414.
92. Magann EF, Bass D, Chauhan SP, et al. Antepartum corticosteroids: Disease stabilization in patients with the syndrome of hemolysis, elevated liver enzymes, and low platelets (HELLP). *Am J Obstet Gynecol* 1994;171:1148-1153.
93. Magann EF, Perry KG Jr, Meydrech EF, et al. Postpartum corticosteroids: Accelerated recovery from the syndrome of hemolysis, elevated liver enzymes, and low platelets (HELLP). *Am J Obstet Gynecol* 1994;171:1154-1158.
94. Isler CM, Barrilleaux PS, Magann EF, et al. A prospective, randomized trial comparing the efficacy of dexamethasone and betamethasone for the treatment of antepartum HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome. *Am J Obstet Gynecol* 2001;184:1332-1339.
95. O'Brien JM, Milligan DA, Barton JR. Impact of high-dose corticosteroid therapy for patients with HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome. *Am J Obstet Gynecol* 2000;183:921-924.

Physician CME Questions

101. In the normal gravid patient, blood pressure drops during the second trimester of pregnancy.
    A. True
    B. False

102. Which of the following statements is true of blood pressure in females younger than 18 years?
    A. The definition of hypertension is the same as for adult women.
    B. Hypertension in these patients is defined as those above 140/90.
    C. Hypertension is defined as blood pressure consistently greater than the 95th percentile of pressures, corrected for age and height.
    D. The levels for hypertension usually are higher than for adult women.

103. Which of the following is an indication that magnesium therapy should be stopped?
    A. The absence of patellar reflexes
    B. A respiratory rate less than 16/min.
    C. Urine output less than 100 cc/4-hour period
    D. All of the above

104. Which of the following is the definition of preeclampsia?
    A. Blood pressure greater than 140 mmHg systolic or greater than 90 mmHg diastolic diagnosed before 20 weeks gestation
    B. Blood pressure greater than 140/90 after 20 weeks with associated proteinuria greater than 300 mg/24 hours or generalized edema
    C. Blood pressure greater than 140/90 that develops after mid-pregnancy without proteinuria
    D. Grand mal seizure that occurs in the pregnant patient past 20

weeks and that is not attributable to other etiologies

105. Which of the following is/are risk factor(s) for preeclampsia?
    A. Primigravida
    B. Age greater than 35 years
    C. Employment at a high stress job
    D. Multiple gestations
    E. All of the above

106. Which antihypertensive agents are contraindicated in patients who are pregnant?
    A. ACE inhibitors
    B. Labetolol
    C. Clonidine
    D. Hydralazine

107. Which of the following is/are maternal complications of preeclampsia?
    A. Disseminated intravascular coagulation
    B. Pulmonary edema
    C. Renal failure
    D. Cerebral edema and hemorrhage
    E. All of the above

108. What is the definitive therapy for preeclampsia?
    A. At-home monitoring
    B. Continued hospitalization
    C. Day units
    D. Delivery

109. Magnesium sulfate has significant toxicity. Which of the following correctly shows the order of the signs and symptoms of toxicity from lowest magnesium level to highest magnesium level?
    A. Loss of patellar reflexes, nausea and diplopia, muscle weakness, cardiac arrest
    B. Nausea and scotomas, respiratory arrest, muscle weakness, loss of patellar reflexes

Emergency Medicine Reports
CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

C. Loss of patellar reflexes, cardiac arrest, muscle paralysis, respiratory arrest
D. Nausea and diplopia, loss of patellar reflexes, respiratory arrest, cardiac arrest

110. The presenting symtpom(s) of HELLP syndrome is/are:
A. epigastric or right upper quadrant pain.
B. nausea
C. vomiting.
D. chest pain.
E. A, B, and C.

In Future Issues:

Acute Thrombotic Disorders

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

CME Answer Key

101. A	106. A
102. C	107. E
103. D	108. D
104. B	109. D
105. E	110. E

Prepare Your Hospital for a Very Unusual Flu Season

Vaccine Shortages May Wreak Havoc with Hospital EDs, Absenteeism

With the unprecedented shortage of influenza vaccine this flu season, hospitals are scrambling to prepare for what may be a record number of flu patients presenting to their already overcrowded emergency departments (EDs) and for staff shortages due to record absenteeism. After almost half of the U.S.'s planned vaccine supply was contaminated, high-risk candidates—including the very young, the elderly, those with chronic illnesses, pregnant women, the immunocompromised, and health care workers with direct patient care—have been identified as those to receive the vaccine.

In response to the national shortage of vaccine, Thomson American Health Consultants has developed an influenza sourcebook to ensure you and your hospital are prepared for what you may face this flu season. Hospital Influenza Crisis Management will provide you with the information you need to deal with ED overcrowding, potential liability risks, staff shortages, and infection control implications for staff and patients.

This sourcebook will address the real threat of a potential pandemic and the proposed response and preparedness efforts that should be taken in case of such an event. Major guidelines and recommendations for influenza immunization and treatment are included, along with recommendations for health care worker vaccination and the efficacy of and criteria for using the live attenuated influenza vaccine.

Don't miss out on this valuable resource in preparing your hospital for this most unusual flu season. Hospital Influenza Crisis Management will also offer readers continuing education credits. For information, or to reserve your copy at the pre-publication price of $149 (a $50 discount off the regular price), call our customer service department at (800) 688-2421. Please reference code 64462.

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Hypertensive Disorders of Pregnancy

Antihypertensive Medications and Their Usage in Pregnancy

MEDICATION	DOSAGE	POTENTIAL MATERNAL (M) OR FETAL (F) COMPLICATIONS
Methyldopa (Aldomet)	0.5-2.0 g/day, divided Q12 hours	M: Sedation, orthostatic hypotension, rarely fever and abnormal liver functions and Coombs' + hemolytic anemia
Clonidine (Catapres)	0.1-0.7 mg/day Generally start with 0.2 mg and give 0.1 mg/hour to a total of 0.7 mg	M: Drowsiness, xerostomia, rebound hypertension if medication is stopped abruptly
Labetolol* (Normodyne, Trandate)	5-20 mg IV, maximum of 300 mg/day No reflex tachycardia, headache, or fluid retention	M: Not used in patients with asthma or chronic lung disease, heart blockage F: Readily crosses placenta, causing bradycardia. Neonatal complications include hypoglycemia, respiratory problems, and hyperbilirubinemia.
Hydralazine (Apresoline)**	5 mg IV, repeat in 10 min, then 10 mg Q 30 min until BP controlled. If 30 mg doesn't control BP, switch agents.	M: Fluid retention, reflex tachycardia, and headache
Thiazide diuretics	12.5-25 mg PO of hydrochlorothiazide—may maintain chronic therapy, don't add after 20 weeks unless CHF	M: Limit use secondary to decreased uterine blood flow secondary to volume depletion
Loop diuretic	20-40 mg IV or PO, especially for pulmonary edema	M: Limit use secondary to decreased uterine blood flow secondary to volume depletion
Nifedipine (Procardia, Adalat)	30-60 mg/day (generally 10 mg every 30-60 min until diastolic < 110 mmHg)	M: Constipation
Verapamil (Isoptin, Calan, Verelan)	120-360 mg/day	M: Constipation
Enalapril, (Vasotec) lisinopril (Zestril, Prinivil), and captopril (Capoten)	**Contraindicated in pregnancy. Change to different medication.**	Causes IUGR, oligohydramnios, congenital malformations
Losartan (Cozaar) Valsartan (Diovan)	**Contraindicated in pregnancy. Change to different medication.**	Causes IUGR, oligohydramnios, congenital malformations

Key:
CHF = congestive heart failure; IUGR = Intrauterine growth restriction

*Newer agent used for hypertensive emergency while in labor and delivery (L&D) with severe preeclampsia. Also drug of choice for Marfans and coarctation patients. Risky in patients with lung disease. Fewer side effects than hydralazine
** Longstanding drug of choice for hypertension control in L & D

Anticonvulsant Dosage Regimens for Preeclampsia and Eclampsia

IM MAGNESIUM SULFATE

- 4 g IV of $MgSO_4$ USP ($MgSO_4$ > 7 H_2O) as a 20% solution IV at a rate not to exceed 1 g/min
- 5 g deep IM of $MgSO_4$ (50% solution) each buttock immediately after IV dose (may be mixed with 1 cc of 2% lidocaine to decrease pain)
- If seizures persist > 15 minutes, give an additional 2-4 g IV over 2-4 minutes depending on maternal size.
- If seizures persist or recur following this, add 250 mg of amobarbital slow IV
- 5 g deep IM of $MgSO_4$ (50% solution) every 4 hours, alternating buttocks provided: patellar reflexes are present, respiratory rate is > 16/min and urine output is >100 cc in the previous 4 hours

IV MAGNESIUM SULFATE

- 4-6 g IV of $MgSO_4$ USP ($MgSO_4$ > 7 H_2O) as a 20% solution IV at a rate not to exceed 1 g/min, preferably diluted to 100 cc and administered over 15-20 minutes
- 20 g of $MgSO_4$ diluted in 1000 cc of IV fluid given at a rate of 2 g/hour (100 cc/hr)
- Use half of the maintenance dose/hour if renal insufficiency is present
- If seizures persist > 15 minutes, give an additional 2-4 g IV over 2-4 minutes depending on maternal size.
- If seizures persist or recur following this, add 250 mg of amobarbital (Amytal) slow IV

MAGNESIUM SULFATE MONITORING

- Monitor patient reflexes, respiratory rate, and urine output on an hourly basis, halting therapy if patellar reflexes are absent, respiratory rate is < 16/min, or urine output is < 100 cc/4-hour period
- Monitor serum magnesium levels at 4-6 hours and adjust dose accordingly to maintain levels between 4-7 mEq/l (4.8-8.4 mg/dL; 2-4 mmol/L)
- Discontinue 24 hours after delivery or the last seizure

ANTIDOTE TO MAGNESIUM SULFATE

At all times that $MgSO_4$ is being administered, a 10 cc ampule of calcium gluconate 10% solution should be at the bedside, and given slowly IV when signs of toxicity are present. The dose may have to be repeated if signs/symptoms of toxicity recur despite halting magnesium therapy. Any time respiratory impairment occurs, prompt intervention with intubation and mechanical ventilation will be lifesaving.

Contraindications to $MgSO_4$ include myasthenia gravis and cardiac conduction defects in the setting of cardiac muscle damage. Caution should be use in patients on calcium channel blockers or with renal insufficiency.

PHENYTOIN DOSING

- Phenytoin (Dilantin) should be used cautiously if at all in the myasthenic patient, and is recommended only for prophylaxis of seizures; therefore, diazepam 10 mg IV is given to immediately control seizures then:
  - 1 g of phenytoin, or phenytoin equivalents should be administered IV over 20 minutes.
  - This is followed by any one of the following regimens:
    - 100 mg IV every 6 hours for 24 hours ***OR*** 500 mg orally 10 hours later ***OR*** after bolus start infusion of 500 mg diluted in 20 0cc saline over 4 hours, and repeat infusion 12 hours after the initial therapy.
- Phenytoin therapy should be continued for 24 hours after delivery or after the last seizure, whichever is later.

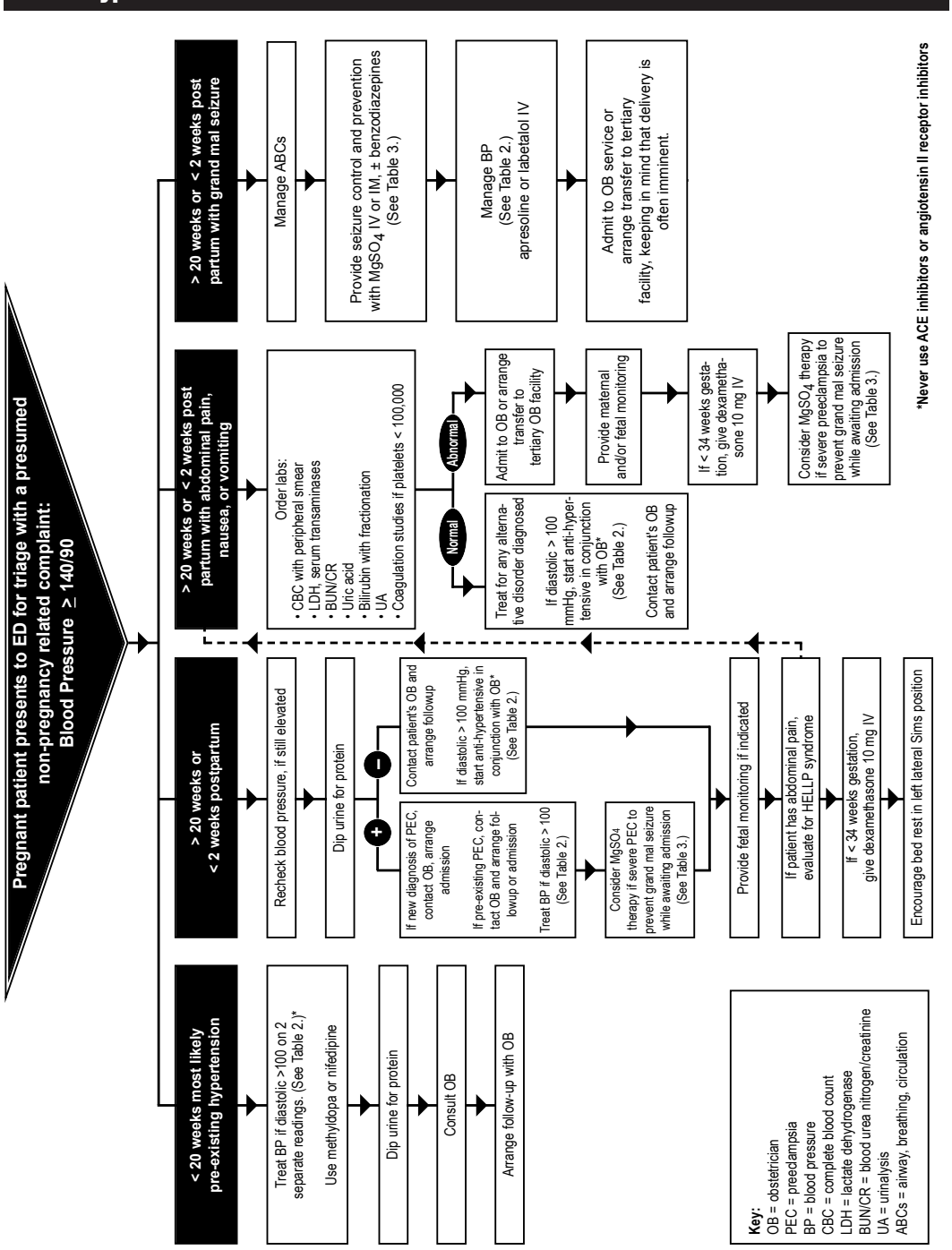
DIAZEPAM DOSING

- 10 mg IV over 2 minutes, repeated if seizure recurs, followed by a 40-80 mg/liter in 1000 cc D5%/W IV infusion over 24 hours, followed by a 20-40 mg/liter infusion over 24 hours

Signs and Symptoms of Magnesium Toxicity

SIGNS/SYMPTOMS	LEVEL IN MG/DL	LEVEL IN MEQ/L	LEVEL IN MMOL/L
Therapeutic range	4-8	3.3-6.7	2-4
Nausea, warm feeling, somnolence, diplopia, dysarthria, weakness	9-12	7.5-10	3.5-5
Loss of patellar reflex	9-12	10	> 5 (3.5-7)
Respiratory depression	12	10	> 6
Muscle paralysis	14.1 (14-17)	11.75 (12.5-14.1)	6-7.5
Cardiotoxicity Prolonged PR and QRS	> 14.1	> 11.75	> 7.5
Respiratory arrest	14.4 (14-17)	12 (12-14.1)	5-7.5
Cardiac arrest	31.5 (30-35)	26.25 (25-29)	12.5

The Emergency Department Management of the Pregnant Patient Noted to Be Hypertensive



Definition of Hypertensive Disorders of Pregnancy in Adult Patients

CHRONIC HYPERTENSION

Blood pressure (BP) > 140 mmHg systolic or > 90 mmHg diastolic diagnosed before 20 weeks gestation or persists > 12 weeks post-partum

GESTATIONAL HYPERTENSION AND/OR TRANSIENT HYPERTENSION OF PREGNANCY

BP >140/90 develops after mid-pregnancy without proteinuria. If preeclampsia doesn't develop in the woman with gestational hypertension and BP returns to normal within 12 weeks post-partum then the term is transient hypertension of pregnancy (a retrospective diagnosis)

PREECLAMPSIA

BP > 140/90 after 20 weeks, with associated proteinuria > 300 mg/24 hours, (or > 100 mg/dL on two random collections), or generalized edema

SUPERIMPOSED PREECLAMPSIA

Proteinuria > 300 mg/24 hours that occurs past 20 weeks in the patient with chronic hypertension without pre-existing proteinuria, or increasing proteinuria, or sudden rise in BP, or development of thrombocytopenia or elevated liver enzymes in the patient with hypertension and pre-existing proteinuria

ECLAMPSIA

Grand mal seizure that occurs in the pregnant patient past 20 weeks, not attributable to other etiologies, associated with preeclampsia

Differential of Patients Presenting with Hypertension, Proteinuria, and Edema

- Preeclampsia
- Eclampsia
- Cholecystitis
- Viral hepatitis
- Pancreatitis
- Encephalitis
- Pyelonephritis
- Glomerulonephritis
- Nephrolithiasis
- Lupus exacerbation
- Cerebral vein thrombosis
- Intracerebral aneurysm rupture
- Acute fatty liver of pregnancy

Signs, Symptoms, or Lab Values Associated with Severe Preeclampsia

- CNS dysfunction including CVA, headache, vision changes, or seizures (eclampsia)
- Right upper quadrant abdominal pain due to liver capsule distention
- Blood pressure >160-180 mmHg systolic or 110 mmHg diastolic on two occasions at least 6 hours apart
- Proteinuria > 3.5-5 g/24 hours
- Oliguria, renal failure, or elevated creatinine
- Pulmonary edema
- HELLP syndrome or any of its laboratory components separately (hemolysis, elevated liver enzymes, low platelets)

Key:
CNS = central nervous system; CVA = cerebrovascular accident

Risk Factors for Preeclampsia

- Primigravida
- Primipaternity (first child from this father, especially if he has fathered another preeclampsia pregnancy or barrier contraception was used prior to this pregnancy)
- Age > 35 years
- Multiple gestations, Hydrops fetalis, Hydatidiform mole
- Family history of preeclampsia
- Underlying medical conditions such as hypertension, diabetes, insulin resistance, obesity, or hyperhomocysteinemia
- Familial thrombophilic conditions such as Factor V Leiden, Protein S deficiency
- Sickle cell trait
- Employment at a high stress job

Supplement to *Emergency Medicine Reports*, November 15, 2004: "Hypertensive Disorders of Pregnancy." *Authors:* **Mary Hughes, DO, FACEP, FACOEP,** Professor, Emergency Medicine, Michigan State University College of Osteopathic Medicine (MSU-COM); Program Director, MSU-COM Emergency Medicine Residency, East Lansing, MI.

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Trauma Reports

Vol. 5, No. 6

Supplement to *Emergency Medicine Reports*, *Pediatric Emergency Medicine Reports*, *ED Management*, and *Emergency Medicine Alert*

Nov./Dec. 2004

As an isolated injury or a frequent component in a multiple trauma patient, the knee is one of the most commonly injured joints. Although not usually a life-threatening injury, correct management may significantly affect the quality of life and mobility for a patient following a traumatic event. In addition, failure to make a timely and accurate diagnosis of a vascular injury may have significant detrimental effects for the trauma patient and result in permanent disability.

It is critical that emergency department (ED) physicians use the history of the injury to direct the physical examination and diagnostic testing, as well as provide prompt referral to an orthopedic specialist if the condition requires surgical intervention. The ED physician also must understand the limitations faced in making a definitive diagnosis in the first 24 hours following an acute injury, a critical time period.

The authors present a thorough review of the anatomy, critical features of the physical examination, and indications for further diagnostic testing in a patient who has sustained a traumatic knee injury.

— The Editor

Knee Trauma: Assessment, Diagnostic Evaluation, and Management

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Introduction

Traumatic knee injuries are a common reason for presentation to EDs following injury; approximately 1 million knee injuries are treated in EDs in the United States each year.[1,2] In addition to isolated knee injuries, the knee joint is frequently injured in association with other injuries in multiple trauma victims.

Because the knee is a complex joint, it is critical to understand the different patterns of injury that can occur: injuries to bone, ligaments, cartilage, and surrounding soft tissue.

The emergency physician (EP) must develop a rational imaging strategy to identify potential injuries and have a good understanding of the types of injuries to properly care for the patient with knee trauma. Many traumatic knee injuries require orthopedic consultation for operative management, and those injuries must be identified quickly and efficiently to expedite care of the patient.

Finally, there are many pitfalls in the evaluation of the knee, especially the failure to diagnose arterial injury, which makes a thorough understanding of the knee and its surrounding structures essential to the active clinician.

Anatomy of the Knee

An understanding of the anatomy of the knee is imperative to assessing injuries involving this complex joint. The knee is the largest articular joint in the body and consists of a hinge joint formed by the articulation of the femur and the tibia. The femur broadens out at the knee joint into two rounded articular prominences: the medial and lateral condyles. Those prominences articulate with the tibial plateaus on the proximal tibia. Small eminences from the condyles form the medial and lateral epicondyles, which serve as attachment points for the collateral ligaments. The anterior portion between both condyles contains a shallow depression that accommodates the patella.

The patello-femoral articulation is not involved in weight bearing.[2] The proximal tibia also contains medial and lateral condyles, which are flattened surfaces that support weight bearing. These surfaces are referred to as the medial and lateral tibial plateaus. Each plateau has approximately 10° of downward slope away from the center of the tibia. Centrally, the tibial plateau contains two superior projections: the anterior and posterior tibial spine. These projections serve as attachment points for the cruciate ligaments.

Also of interest is the tibial tuberosity, which is located on the anterior surface of the tibia below the knee. This bony prominence serves as the attachment of the quadriceps tendon. The proximal fibula, although not technically part of the knee, is included in this discussion. These structures combine to form the major bony articulations of the knee. (*See Figure 1.*)

The popliteal fossa is a depression found behind the knee joint. Several important structures are located in this recess, including the popliteal artery and vein, as well as the peroneal and tibial nerves. The popliteal artery represents the continuation of the femoral artery into the knee joint. This artery supplies a rich anasthomosis of blood vessels supplying the knee joint and then divides into the anterior and posterior tibial arteries. The artery is relatively fixed within the popliteal fossa, thus, making it prone to injury with any stress on the knee joint.[3,4]

The common peroneal nerve wraps around the proximal fibula. Injury to this nerve may result in a foot drop.[5] Intact dorsiflexion at the ankle must be documented with any significant trauma to the proximal fibula.

The static stability of the knee joint depends on its surrounding ligaments (*See Figure 1*). The anterior cruciate ligament (ACL) prevents anterior displacement of the tibia in relation to the femur. The posterior cruciate ligament (PCL) prevents posterior and rotational displacement. Both the ACL and PCL also contribute to proprioception. The medial collateral ligament (MCL) resists valgus stress, while the lateral collateral ligament (LCL) resists varus stress.

With the knee in full extension, all of the ligaments are taut, ensuring complete stability of the knee joint. The knee must be flexed to 20-40° before the joint is relaxed, and some rotational movement of the joint is possible. The medial and lateral menisci are cartilaginous structures that support the femorotibial articulation and also aid in weight bearing. (*See Figure 2.*) The menisci contribute to joint stability and provide both lubrication and cushioning to the knee. The medial meniscus is attached firmly to the MCL and joint capsule making it more susceptible to injury. Because of the absence of pain fibers, menisci injuries may have a delayed detection or go undetected.

The muscular structures of the knee provide dynamic stability to the joint. The knee extensors (quadriceps group) include the vastus medialis, vastus intermedius, vastus lateralis, and rectus femoris. They combine to form the quadriceps tendon, which inserts into and encompasses the patella. The quadriceps tendon then becomes the patellar tendon and inserts into the tibial tuberosity. The vastus medialis exerts a medial force on the patella, thus, preventing lateral subluxation. The knee flexors (hamstrings group) include the semimembranosus, semitendinosus, and biceps femoris. The knee adductors include the gracilis, sartorius, and a portion of the semitendinosus, combining to form the pes anserinus medially. The iliotibial tract, popliteus muscle, and a portion of the biceps femoris attach laterally.

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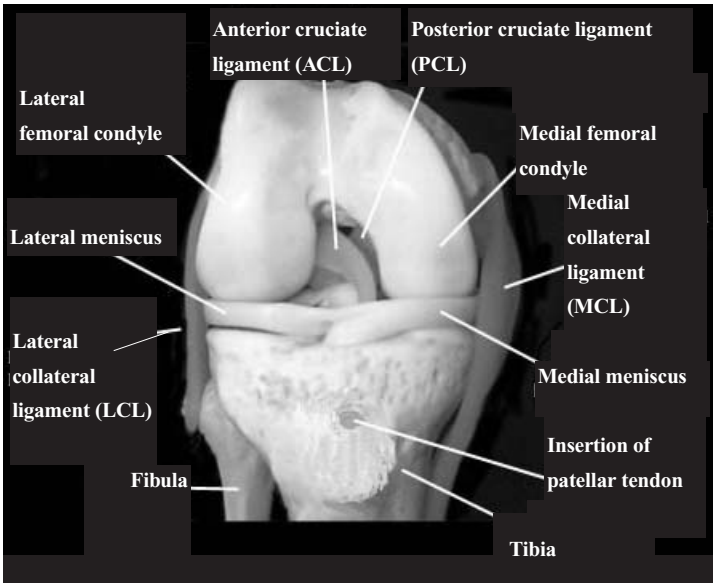
Figure 1. Knee Bony Anatomy



The bony anatomy of the knee consists of the articulation of the femur and the tibia, which meet to form a hinge joint. The joint is protected in front by the patella. The joint is cushioned by articular cartilage that is located at the ends of the tibia, femur, and under the patella. The lateral and medial meniscus are cartilaginous and further cushion the joint.

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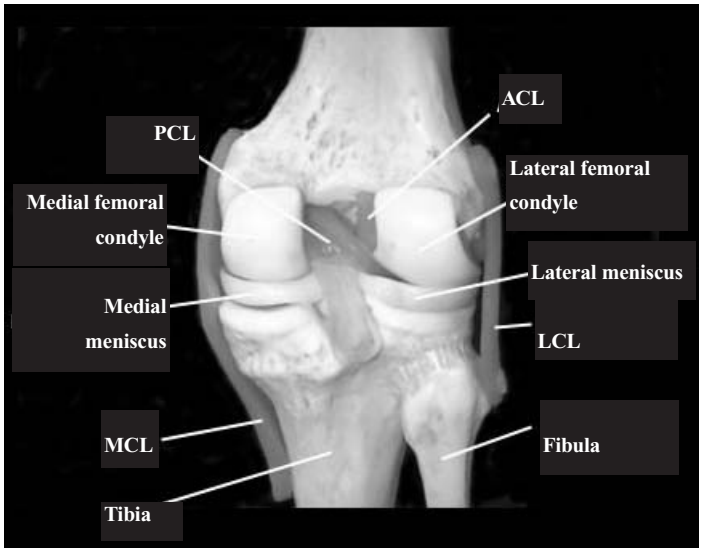
Figure 2. Knee Ligamentous Anatomy



The ligaments of the knee are designed to stabilize the knee. The collateral ligaments are located along the sides of the knee and limit lateral motion. The ACL is located at the center of the knee and connects the tibia to the femur. The ACL functions to limit rotation and forward motion of the tibia. The PCL limits backward motion of the tibia. The MCL restricts valgus stress, and the LCL resists varus stress.

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History and Physical Examination

A thorough history regarding the mechanism of injury is crucial to identification of the particular pattern of knee injury. For example, an anterior force applied to the tibia with the foot planted typically results in an ACL injury; the PCL frequently is damaged by a posterior force under the same conditions.[6,7]

It should be noted, however, that an accurate history of the injury may be absent in patients with multiple trauma; therefore, a careful examination of the knee is imperative and correlation with historical information appropriate in all patients. Information obtained should include the direction of the force that caused the injury and the weight-bearing status of the patient at the time of impact. The knee is commonly subjected to compression, distraction, flexion, extension, rotation, valgus or varus stress, and sliding forces.

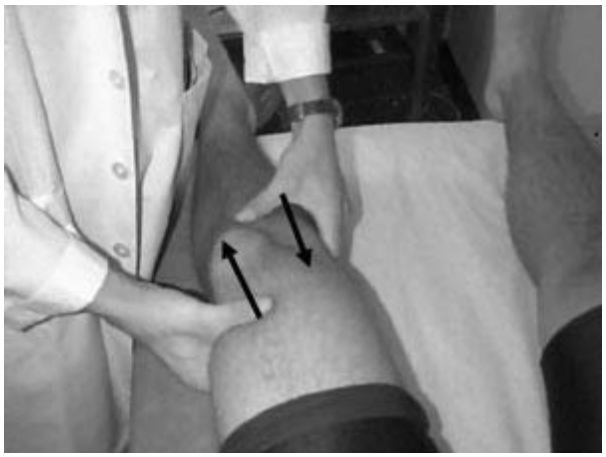
Additional information, including the position of the patient in an automobile (e.g., driver, front-seat or rear-seat passenger), anticipated impact, and damage to the dashboard, may be particularly important in association with motor vehicle trauma. The location of pain and the presence of a pop or snap (noted in up to 90% of cases associated with ACL rupture at the time of injury) are also very important.[8-10]

Patients with ACL or PCL injuries also may complain of the knee "giving out," especially with pivoting. Complaints of knee locking may indicate a torn meniscus, a loose body, rupture of the ACL or PCL, or an osteochondral fracture.[11-12]

An osteochondral fracture occurs when a portion of the articular surface, including both lining cartilage and underlying bone, becomes separated from either the femur or tibia. The time of injury and the amount of swelling or effusion that has developed can be helpful in distinguishing the injured structure. The cruciate ligaments are well vascularized, and a tear can produce a large effusion within a couple of hours.[8,9,13,14] Acute hemarthroses is a sign of intra-articular injury, with a high percentage of these injuries involving ACL tears. Osteochondral fractures also can produce large effusions quickly and account for as many as 67% of traumatic hemarthroses in children without apparent fracture on x-ray.[15] A smaller effusion that collects during a longer period of time is more indicative of a partial ligament tear or meniscal injury.

A detailed examination of the knee in flexion and extension can provide very specific information about the type of injury involved. The initial evaluation should include assessment of deformity, swelling, effusion, tenderness, and open wounds. It is also helpful to compare any findings to the uninjured knee. Location of tenderness on examination corresponds with the site of injury identified surgically in up to 76% of cases.[10] As noted above, the presence of a large effusion that is noted within 1-2

Figure. 3 The Lachman Test



The Lachman's test is more sensitive than the anterior drawer test for ACL rupture. As illustrated above, the knee should be flexed 20-30°. One hand should hold the distal femur. With the other hand, apply anterior force to the proximal tibia. If an end-point is not appreciated or significant movement of the anterior tibia occurs, the test is considered positive.

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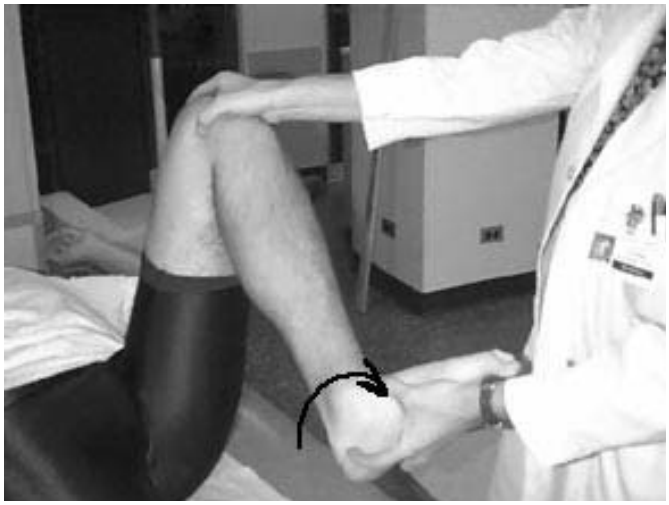
Figure 4. The McMurray Test



The examiner flexes the knee beyond 90° with the foot externally rotated in the examiner's hand. The other hand palpates the medial joint line as the knee is extended gradually beyond 90°. A medial meniscus injury is indicated by pain, a popping sensation, or crepitus.

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hours of injury is highly suggestive of cruciate ligament rupture or an osteochondral fracture. A full popliteal fossa may be seen with popliteal artery disruption, suggesting that the knee may have been dislocated, that a bicruciate ligament rupture may be present, or that both injuries have occurred simultaneously.

It is extremely important to remember that a reliable examination may not be possible within the first few hours of injury, and the patient may need to have a repeat exam in 24-48 hours when the pain and swelling have subsided. In the multiply injured patient, other priorities may limit the initial examination of the knee and associated structures. Following stabilization, a complete and directed physical examination will reveal likely injury patterns.[16]

Valgus and varus stress should be applied to the knee in 20-30°of flexion and in full extension. This action will help to identify MCL and LCL injuries. Examination of the knee in slight flexion allows for relaxation of the major muscle groups that provide support to the knee and isolates the supporting ligamentous structures of the knee joint.

Anterior and posterior drawer tests should be performed with the hip flexed at 45°, the knee flexed at 90°, and the foot secured in a neutral position on the exam table. Anterior force should be applied to the proximal tibia to assess for ACL integrity and posterior force applied to assess for PCL integrity. The same examinations also should be repeated with the foot secured in internal and external rotation.[2] The tibia should be limited by a discrete end-point in the anterior and posterior directions. Any sliding forward or backward suggests an ACL or PCL injury.

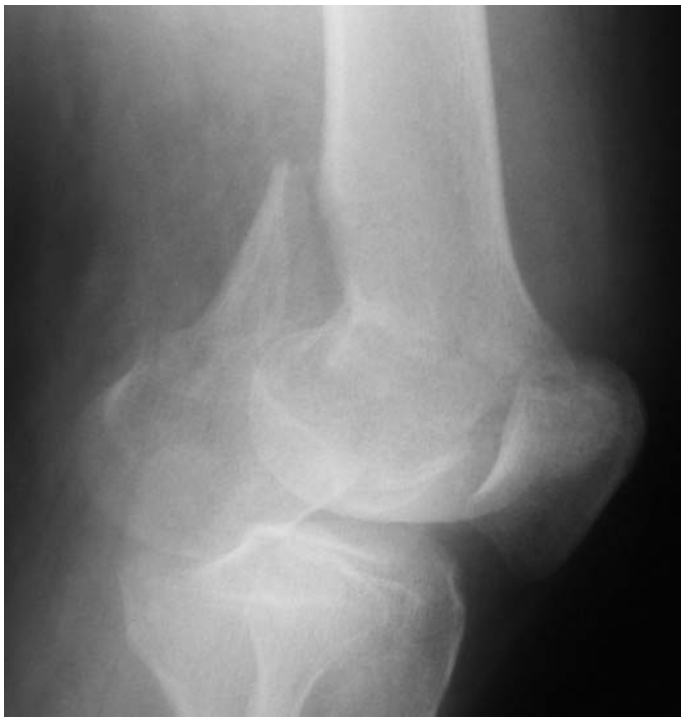
Lachman's test is more sensitive for ACL rupture (up to 99% sensitivity vs 77% for the anterior drawer test).[17] Hold the distal femur with one hand, and apply anterior force to the proximal tibia with the other hand while the knee is flexed 20-30° (*See Figure 3.*). Here again, a discrete end-point should be appreciated. A lack of a discrete end-point or significant anterior movement of the tibia is indicative of a positive test.

The Lateral Pivot Shift test is performed with the knee in full extension. The leg is lifted by the distal tibia, which will produce subluxation of the proximal tibia with ACL disruption. The knee is flexed carefully to 20-40°, while a mild valgus force is applied to the knee, and the foot is moved laterally. A thud can be appreciated as the tibia subluxes against the femur with this motion.

Meniscal injuries are detected using the McMurray's test, Apley compression test, or medial-lateral grind test.[18] The McMurray test involves flexion of the knee beyond 90° with the foot externally rotated in the examiner's hand. (*See Figure 4.*) The other hand palpates the medial joint line as the knee is extended gradually beyond 90°. Pain, a popping sensation, or crepitus indicates a medial meniscus injury. Repeat the test with the foot internally rotated while the lateral joint line is palpated. Pain and crepitus with flexion indicate lateral meniscus injury. The McMurray's test alone has been shown to have a low sensitivity and specificity for detecting medial meniscus injuries. [19,20]

Apley's compression test is performed with the patient in a prone position, and the knee flexed to 90°. Pain with rotation of

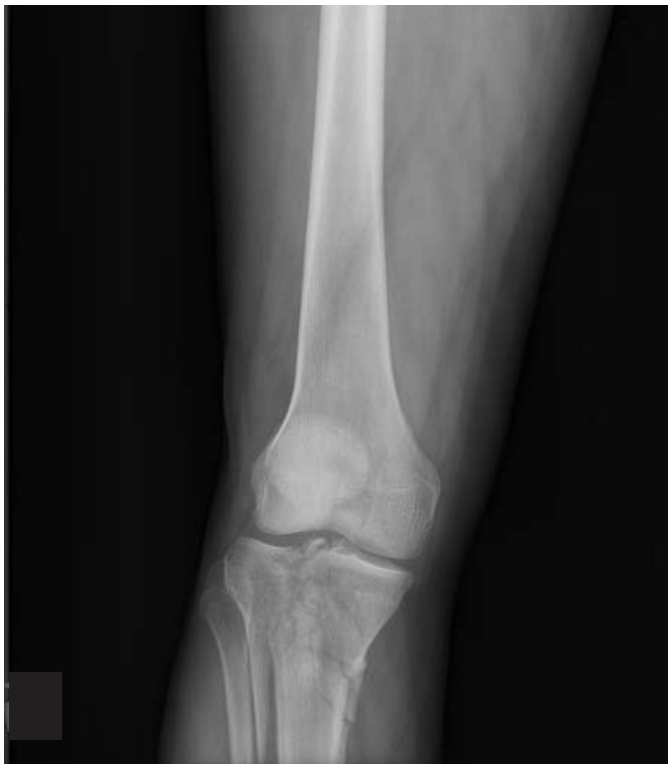
Figure 5. Intercondylar Femoral Fracture



This patient sustained an intercondylar femur fracture. Because a large amount of force is necessary to produce this injury, the patient should be assessed carefully for other injuries. In addition, the close proximity of the popiteal artery and vein to the distal femur should alert the clinicial to the possibility of vascular injury.

Image courtesy of Howard Werman, MD.

Figure 6. Tibial Plateau Fracture



This patient sustained a tibial plateau fracture. Meniscal and ligamentous injuries commonly accompany these fractures.

Image courtesy of Howard Werman, MD.

the foot while upward traction is applied suggests damage to the capsule and/or ligaments. Pain with rotation of the foot while downward traction is applied indicates meniscal injury.

The medial-lateral grind test is performed in the supine patient by cradling the calf in one hand and palpating the medial and lateral joint lines with the other. Valgus and varus stress is applied to the joint. Any crepitus felt along the joint line indicates possible meniscal injury.

Bragard's sign is tenderness along the anterior medial joint line upon internal rotation and extension of the tibia and indicates a likely medial meniscus injury.

Payr's sign is pain with pressure applied to the medial aspect of the knee with the patient sitting cross-legged. The knees should be flexed at 90°. This finding is also consistent with medial meniscus injury.

First Steinmann's sign is either pain in the anterolateral joint space with internal rotation and flexion of the knee, which is consistent with a lateral meniscus tear, or pain in the anteromedial joint space with external rotation and flexion of the knee, which suggests a medial meniscus tear.

Secondary Steinmann's sign helps to distinguish a meniscal injury from an LCL or MCL disruption. It involves a shifting location of tenderness from the region of the collateral ligament when the knee is flexed to the anterior aspect of the knee when fully extended.

In addition, a thorough evaluation of the neurovascular status of the knee should be done, focusing on the presence or absence of pulses in the posterior tibial, dorsalis pedis, and popliteal arteries, as well as the sensory function of the various nerves supplying the area below the knee.

Injury to the Bony Structures of the Knee

Bony injuries to the knee can involve the distal femur, proximal tibia and fibula, patella, or dislocation of the knee. The management of these various injuries differs based on the mechanism and location of injury.

Femoral Fractures. Fractures of the distal femur are rare injuries [1,21,22] and are grouped into four different categories: Class A, supracondylar fractures; Class B, intercondylar fractures; Class C, condylar fractures; and Class D, epiphyseal fractures *(See Figure 5.*)

All femoral fractures present with knee pain or hip pain, inability to bear weight, and tenderness over the distal femur. These fractures usually occur as the result of a direct blow to the distal femur, such as from a dashboard or steering wheel. A large amount of force is required to produce a femoral fracture and

consequently, these fractures often are associated with other injuries. Additionally, because of the muscular attachments of the quadriceps group, the hamstring muscles and both the gastrocnemius and soleus muscles, displacement of the fracture fragments is common. Remember that the popliteal artery and vein are in close proximity to the distal femur and are prone to injury with such fractures.

Supracondylar Fractures. By definition, these fractures occur above the femoral condyles and do not involve the knee joint. As a result, effusions are not associated with this type of injury. Five percent to 10% of these fractures are open fractures.[23] All of the other fractures mentioned above have an intra-articular component. Epiphyseal fractures usually occur in children older than 10 years. Because bone growth around the knee joint accounts for almost two-thirds of adult leg length, these injuries commonly are associated with leg shortening, and all Salter-Harris fractures of the knee joint should have urgent orthopedic evaluation.[10,21,22]

Distal femur fractures all require urgent orthopedic consultation and can be treated with immobilization, traction, or operative fixation at the discretion of the consulting orthopedic specialist. Skeletal traction often is the preferred method by orthopedic surgeons once the patient is admitted; however, a hare traction splint can be used to immobilize the fracture in the ED until the skeletal pins can be placed. Significant pain control often is required; traction on the femur is extremely painful.[2] Commonly associated injuries include ipsilateral hip fractures, popliteal vascular injuries, peroneal nerve injury, and damage to the quadriceps or hamstring groups.

Tibial Fractures. Tibial fractures are divided into four classes: Class A, condylar fractures; Class B, spine fractures; Class C, tuberosity fractures; and Class D, subcondylar fractures.

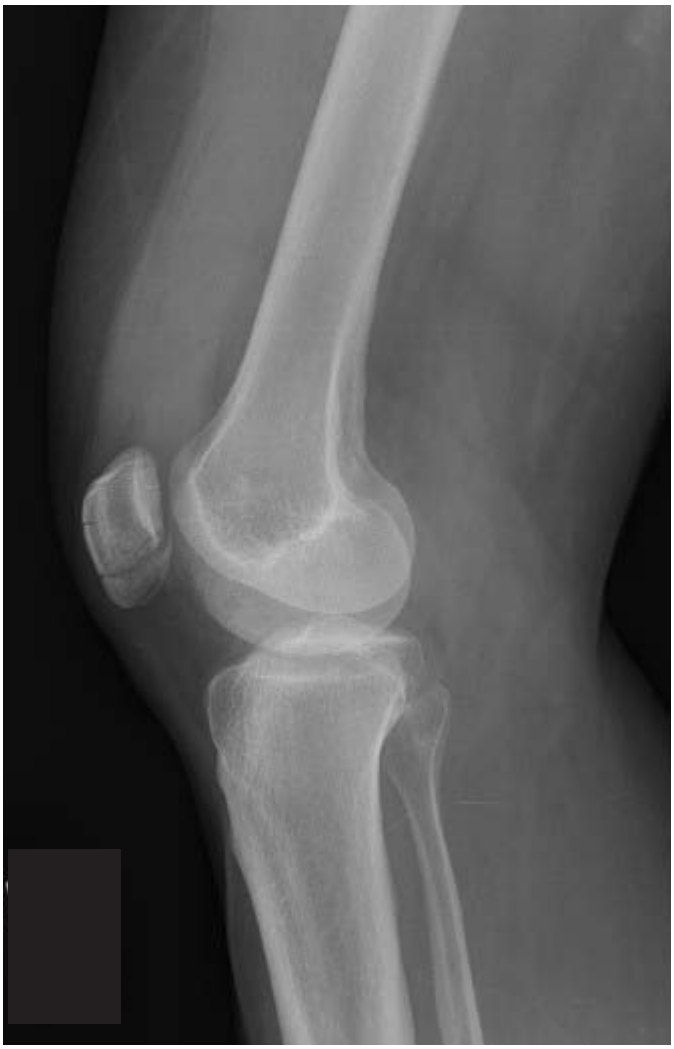
Tibial fractures typically occur in one of two ways: 1) a direct blow to the tibia (bumper injury); or 2) axial compression of the tibia, which occurs when the knee is forcibly extended in anticipation of a frontal impact.

Class A injuries (condylar and plateau fractures) usually are produced by axial loading (e.g., a fall from a height). (*See Figure 6.*) The rotational stress on the knee will determine whether the medial or lateral tibial plateau is affected.[24] The lateral plateau is the site of fracture in the majority of cases.[1] Meniscal and ligamentous injuries commonly accompany these fractures; the menisci and medial and lateral collateral ligaments are related closely to and attached to the tibial plateaus.[25] A plateau fracture without depression of the articular surface can be treated by a long leg cast. Any depression or irregularity of the articular surfaces requires immediate orthopedic consultation and surgical repair. ACL and MCL tears are associated with lateral plateau fractures; PCL and LCL tears are associated with medial plateau fractures. Lateral plateau injuries are more common and usually are due to involvement of the tibial spine.[25]

Class B injuries (spine fractures) are rare but present similar to ACL and PCL tears, depending upon which spine is fractured. The mechanism of injury typically involves an anterior or posterior force to the proximal tibia with the knee in flexion. Anterior spine fractures are 10 times more common than posterior spine fractures and are associated with ACL injuries.[25] Complete spine avulsion requires urgent orthopedic consultation and surgical repair, while incomplete fractures can be treated by a knee immobilizer and orthopedic follow-up.

Class C injuries (tuberosity fractures) involve the attachment of the quadriceps tendon, and present with pain over the anterior tibia with signs similar to either partial or complete quadriceps tendon rupture (difficulty or inability to perform a straight leg raise). These fractures can occur as the result of a shearing force or more commonly, due to forced flexion of the knee, creating stress on the quadriceps tendon. Partial avulsions can be treated by immobilization and orthopedic follow-up; complete avulsions require operative fixation. Clinically, this type of injury is indistinguishable from quadriceps tendon rupture, but plain x-rays

Figure 7. Patellar Fracture



This radiograph demonstrates a patellar fracture. Patellar fractures typically occur secondary to direct knee trauma. Nondisplaced patellar fractures may be managed with a knee immobilizer and pain control.

Image courtesy of Howard Werman, MD.

Figure 8. Angiogram of Popliteal Artery Showing Intimal Tear

Injury to the popiliteal artery is common in patients with knee dislocations. These injuries require early recognition and, optimally, a repair within 6-8 hours of the injury. Intact pulses in the foot do not exclude vascular injury.

Image courtesy of Howard Werman, MD.

will show the avulsed segment of the tibial spine attached to the end of the quadriceps tendon.

Class D injuries (subcondylar fractures) can be treated with immobilization and do not require urgent orthopedic consultation unless there is comminution of the fragments or associated injuries. Like supracondylar patellar fractures, these injuries do not involve the joint space.

Proximal Fibular Fractures. These fractures are typically noted as the result of a direct blow to the area or a valgus stress on the knee joint. The latter mechanism typically produces lateral collateral ligament injury and peroneal nerve injury.[1] Isolated proximal fibular fractures can be treated symptomatically with a knee immobilizer and analgesics. A variant of the proximal fibular fracture, the Maisonneuve's fracture, involves a distal tibial fracture or ligamentous disruption, resulting in instability of the ankle. Thus, the ankle joint should be assessed carefully if a proximal fibular fracture is present, and x-rays should be obtained if the ankle is tender.

Patellar Fractures. Patellar fractures typically are caused by direct knee trauma (e.g., a knee striking a dashboard), and are divided into several classes: non-displaced, transverse, pole fractures, comminuted (non-displaced or displaced), vertical, or osteochondral. High impact injuries are associated with hip fractures and dislocations. A secondary mechanism may involve an avulsion caused by the pull of the quadriceps mechanism. Patellar fractures are more common in males.

A non-displaced fracture can be treated with a knee immobilizer and pain control. If the quadriceps mechanism is intact (See Figure 7), the patient can bear weight as well. Fractures associated with disruption of the extensor mechanism or displacement more than 3 mm requires operative repair in an urgent manner.

Remember that anatomical variants of the patella can include a bipartite and tripartite patella. With a bipartite patella, a secondary ossification center typically is noted in the upper outer portion of the patella, which may be confused with a fracture. Because these variants tend to be bilateral, a comparison view of the uninjured patella should be obtained.

Patellar Dislocation

Patellar dislocations typically occur when the patient twists on an extended knee. Rarely, dislocation can occur as the result of a lateral or medial blow to the patella. The condition is more common in women.[26] Many patients (10–50%) will have recurrent episodes during their lifetimes. The patella almost always dislocates laterally (80%).[26] Sometimes this injury is associated with medial joint capsule injury.

Patellar dislocation is very painful, and the patient often presents with a fixed, partially flexed knee with obvious displacement of the kneecap. The reduction can be performed in one of two manners.

Passive Hyperextension of the Knee. This is a painful procedure and often requires pain control and sedation. The lower leg is grasped in one hand, the upper leg in the other, and the knee is straightened passively. The patella then is moved back into place by hand, forcing the patella medially.

Active Hyperextension of the Knee. The patient can be asked to straighten the knee (e.g., to touch the bed with the back of his knee while standing or lying). A cooperative patient can perform this maneuver, and reduction causes immediate cessation of pain.

Whichever method is chosen, plain knee radiographs should be obtained following successful reduction to identify any associated patellar fractures or osteochondral injuries, which are seen in up to 5% of cases.[1,26] Additionally, a thorough knee examination should be performed once the patella is in anatomic alignment; up to 12% of patients with a patellar dislocation have an associated ligamentous or meniscal injury.[1,26] Knee immobiliza-

tion should be used to prevent recurrent dislocation, and follow-up within one to two weeks is indicated. If recurrent dislocation occurs, operative fixation may be needed.

Knee Dislocation

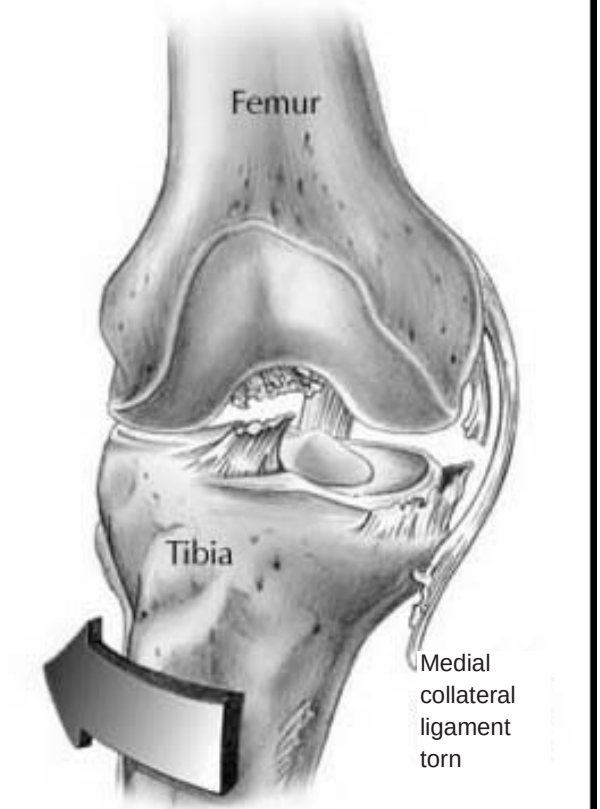
Knee dislocation is the result of a significant force delivered to the knee and occurs as the result of hyperextension, posterior forces to the tibia, or significant rotary forces.[27] One example is a bumper impact by a motor vehicle against a planted lower extremity. Any knee dislocation is associated with significant ligamentous disruption.[5] There are three types of dislocation of the knee: anterior, posterior, and lateral. In anterior dislocations (the most common type), the tibia is displaced anteriorly to the femur and typically is the result of hyperextension of the knee joint. In posterior dislocations, the tibia is forced posteriorly by a direct force applied to the proximal tibia. Lateral dislocations can be medial or lateral, and occur with equal frequency. Lateral dislocations can be complicated further with rotary deformity of the knee as well. Any type of knee dislocation carries a 20% incidence of open joint injury[5] and classically presents with a deformed knee and severe pain. Pulses in the foot must be assessed immediately and may be present or absent. Reduction of all three types of dislocations requires longitudinal traction. Sedation and pain relief commonly are required.[1,5] Post-reduction radiographs should be obtained to assess for concomitant fractures, which can include tibial and patellar fractures, as well as fibular head fractures. A thorough neurovascular examination must be conducted following successful reduction to determine if there is injury to the peroneal and tibial nerves. One additional injury pattern is worthy of mention—proximal tibiofibular dislocations or subluxations—rare injuries that are seen after a significant fall. Peroneal nerve injuries commonly are associated with this type of dislocation.

Injury to the popliteal artery is common in knee dislocations (up to 40% of all dislocations).[3,28] (*See Figure 8.)* It is critical that such injuries be identified early and repaired within 6-8 hours of injury.[28] Intact pulses in the foot do not exclude the possibility of arterial injury. An arteriogram should be performed on these patients according to most experts, although an alternative strategy of admission and repeated clinical examination after normal ankle brachial indices (ABI) in the ED has been suggested.[5] An ABI is performed by obtaining a blood pressure in the arm, by standard technique, then taking a blood pressure at the ankle. Dividing the ankle pressure by the arm pressure determines the ABI. Ankle pressure should be greater than brachial pressure. Thus, a ratio of one or greater is considered normal. An ABI of 0.9 or less is concern for possible arterial injury. Patients who have peripheral arterial disease also will have an ABI of less than 0.9, possibly leading to a false positive result. Whichever strategy is used, knee immobilization and urgent orthopedic consult is required.

Treatment of Bony Knee Injuries

Many bony knee injuries can be treated by casting; immobilization of the joint above and below the injury is critical for optimal healing. For tibial and fibular fractures, it can be accomplished either with a long leg cast or through the combined use of a short leg cast and a knee immobilizer. When properly applied, the knee immobilizer will combine effectively with the cast to immobilize both the knee and ankle. This technique can be used for a variety of knee fractures. Some fractures, however, require surgical repair and should have urgent orthopedic consultation.

Figure 9. Knee Ligamentous Injury



Sprains involve injury to the ligamentous structures of the knee. They vary in severity from a Grade 1 sprain (a small incomplete with no instabiity of the knee) to a Grade 3 sprain (a complete ligamentous disruption with mal complete joint instabilty).

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Injury of the Supporting Structures of the Knee

Sprains involve injury to the ligaments of the knee. A Grade 1 sprain corresponds with a small incomplete tear and involves local tenderness with minimal swelling, no instability of the knee, and little increased pain with stress applied to the knee. A Grade 2 sprain corresponds with a moderate incomplete tear and involves local tenderness with moderate swelling, and some joint instability with a firm end-point on stress. A Grade 3 sprain cor-

Table 1. Ottawa Knee Rules

Plain radiographs are indicated if the patient has any one of the following conditions:

- Age 55 years or older
- Tenderness at head of fibula
- Isolated tenderness of the patella (no bone tenderness of knee other than patella)
- Inability to flex to 90°
- Inability to bear weight for four steps both in the ED and immediately after injury

Adapted from: Bachman LM, et al. The accuracy of the Ottawa knee rule to rule out knee fractures: A systematic review. *Ann Intern Med* 2004;140:121-124.

responds with a complete ligamentous disruption and involves local tenderness, swelling that can be minimal or marked, and significant joint instability with no clear end point.[29] (*See Figure 9.*)

Partial ligament tears typically are more painful and can produce significantly more swelling than complete disruptions. The MCL is the most commonly injured knee ligament. (*See Figure 9.)* The mechanism usually involves a strong lateral force applied to a slightly flexed knee. This type of injury also can produce ACL disruption and tearing of the medical meniscus. Isolated Grade 1 or Grade 2 sprains can be treated with immobilization, compression, rest, ice, elevation, and non-steroidal anti-inflammatory medications with orthopedic or primary care follow-up within a few days. Grade 3 injuries can be treated similarly with prompt orthopedic referral (within 24 hours).

The medial and lateral menisci also are commonly injured structures within the knee. They are not innervated, but injury may produce pain because of irritation from injury to adjacent ligaments. These structures are relatively avascular with their blood supply coming from peripheral capillaries. This poor blood supply results in slower accumulation of joint effusions associated with meniscal injury. The medial meniscus is fixed more securely and is more commonly injured. Acute meniscal tears are usually longitudinal and located peripherally.[14] If it extends along the entire length of the meniscus, it is considered a bucket-handle tear. Traumatic ruptures are rare before age 10 years. They typically produce a triad of symptoms including joint line pain, swelling, and locking of the knee. The patient also may complain of the knee "giving out." Treatment is the same as for a grade 1 or 2 sprain.

Knee trauma also can produce disruption or injury to the many tendinous insertions about the knee. Disruption of the quadriceps mechanism can occur in a number of possible locations. Such injuries commonly occur as the result of forced flexion of the knee with the quadriceps muscle contracted.[30] Rupture of the quadriceps tendon superior to the patella is more frequent in the elderly population, while patellar tendon ruptures inferior to the patella are more common in young athletes.[30] The extensor tendon mechanism also can be disrupted with a complete transverse fracture of the patella and with avulsion of the tibial tubercle. Split and referral for orthopedic repair of the injury is recommended. Repeated, low-grade stress to the patella tendon may produce tendonitis or jumper's knee.[31,32]

Radiography

Plain radiography can be very helpful in the diagnosis of fracture, dislocation, and effusion about the knee after trauma; however, it reveals little useful information about ligamentous, meniscal, or tendon injuries.[9,33-35] Standard views include a true anterior-posterior and lateral view of the knee along with one or two oblique views of the knee. Additional views may include a sunrise view of the knee, which is specifically used to identify patellar injuries.[1] In this projection, the knee is flexed, and the beam is directed tangentially to the longitudinal axis of the patella.

Historically, x-rays of the knee have been overused, and several clinical decision rules have been proposed to assist the examiner in determining which patients do not require imaging. The most widely accepted are the Ottawa Knee Rules,[36] which state that plain films of the knee are indicated if the patient is: 1) 55 years of age or older; 2) has tenderness at the head of the fibula; 3) has isolated tenderness of the patella; 4) is unable to flex the knee to 90°; or 5) is unable to bear weight for four steps both in the ED and immediately after the injury. (*See Table 1.*) These rules have helped decrease radiography rates, decrease cost to the patient, and decrease the length of stay in the ED without decreasing the rate of identifying clinically significant fractures of the knee.[15,36]

Computerized tomography (CT) scanning of the knee occasionally is used by the orthopedic consultants to delineate further the anatomy of a fracture, but it has a limited role in the ED evaluation of the acutely injured knee.[37] Magnetic resonance imaging (MRI) is useful in the outpatient setting for evaluating the soft-tissue structures about the knee when the specific injury is in question, but usually is not necessary as part of the emergency assessment.[9,33] When the type of injury is readily apparent from history and physical examination, the patient often will undergo arthroscopy without having an MRI.

Other Diagnostic Studies

Saline Arthrogram. When concerned about the possibility of a penetrating joint injury (e.g., a laceration that overlies the joint space), a saline arthrogram can be used safely to exclude communication with the joint. Proper performance of a saline arthrogram can help identify otherwise occult, open joint injuries.[38]

To perform a saline arthrogram, the knee is prepped and draped in a sterile fashion, with the knee in a 30° flexion. Lidocaine 1% with or without epinephrine can be used as local anesthesia. A superior medial or lateral approach should be used similar to an arthrocentesis. The choice of approach is based upon avoiding the suspected area of penetration. Using the margin of the patella as a landmark, the clinician inserts an 18-gauge needle attached to a syringe containing sterile saline into the knee joint under negative pressure. Once placement is confirmed by aspiration of synovial fluid, 20-30 mL of saline is instilled into

the joint space, while the laceration or other skin injury is observed. Leakage of saline from the laceration confirms that there is communication with the joint space. Open joint injuries require emergent orthopedic consultation, and usually operative debridement and irrigation.

After finishing the procedure, be sure to remove all injected saline from the joint; the extra fluid may be quite uncomfortable for the patient.

Angiography. Angiography is a critical imaging modality used with suspected anterior dislocations, posterior dislocations, or bicruciate ruptures. Popliteal artery injury occurs in association with these injuries in 30-40% of cases.[3,28] The prognosis is good when arterial injury is identified and repaired within 8 hours of injury. When the diagnosis is delayed, up to 86% of patients with popliteal artery injury require amputation, and 66% of those remaining have permanent ischemic changes of the leg and foot.[16,21] If distal pulses are absent with this type of injury post-reduction, immediate surgical exploration is necessary.

Pitfalls

Several potential pitfalls exist in the evaluation of patients with knee injuries, including: 1) not getting a complete history regarding the mechanism of injury; 2) failure to have the knee re-examined in 24-48 hours when pain and swelling have decreased; 3) failure to examine the knee in flexion and extension; and 4) failure to consider popliteal artery injury in the face of significant knee injury, especially in the case of dislocations, and address this condition with either angiography or immediate surgical exploration.

Summary

Patients frequently present in the ED with trauma to the knee. It is critical for the ED physician to be familiar with the anatomy of the knee, trauma associated with certain mechanisms of injury, and appropriate diagnostic testing that is indicated based on the history and a thorough physical examination. Limitations of the initial examination should be recognized, as well as the more subtle findings of potential vascular compromise. Appropriate diagnostic testing should be performed and orthopedic consultation and follow-up obtained when indicated.

References

1. Marx J, Hockberger R, Walls R, eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. New York: Mosby; 2002:674-705.
2. Tintinalli JE, Kelen GD, Stapczynski JS, et al, eds. *Emergency Medicine: A Comprehensive Study Guide.* New York: McGraw Hill; 2000:1814-1822.
3. Seybold EA, Busconi BD. Traumatic popliteal artery thrombosis and compartment syndrome of the leg following blunt trauma to the knee: A discussion of treatment and complications. *J Orthop Trauma* 1996;10:138-141.
4. Fadale PD, Noerdlinger MA. Sports injuries of the knee. *Curr Opin Rheumatol* 1999;11:144-150.
5. Schenck RC Jr, Hunter RE, Ostrum RF, et al. Knee dislocations. *Instr Course Lect* 1999;48:515-522.
6. Harner CD, Hoher J. Evaluation and treatment of posterior cruciate ligament injuries. *Am J Sports Med* 1998;26:471-482.
7. Duri ZA, Aichroth P M, Zorrilla P. The posterior cruciate ligament: A review. *Am J Knee Surg* 1997;10:149-165.
8. Mallory N, Kelsberg G, Ketchell D. Does a knee brace decrease recurrent ACL injuries? *J Fam Pract* 2003;52: 803-804.
9. Ho CP, Marks PH, Steadman JR. MR imaging of knee anterior cruciate ligament and associated injuries in skiers. *Magn Reson Imaging Clin N Am* 1999;7:117-130.
10. Smith BW, Green GA. Acute knee injuries: Part II. Diagnosis and management. *Am Fam Physician* 1995;51:799-806.
11. Oeppen RS, Connolly SA, Bencardino JT, et al. Acute injury of the articular cartilage and subchondral bone: A common but unrecognized lesion in the immature knee. *AJR Am J Roentgenol* 2004; 182:111-117.
12. Fritz RC. MR imaging of meniscal and cruciate ligament injuries. *Magn Reson Imaging Clin N Am* 2003;11: 283-293.
13. Miller MD, Bergfeld JA, Fowler PJ, et al. The posterior cruciate ligament injured knee: Principles of evaluation and treatment. *Instr Course Lect* 1999;48:199-207.
14. Swenson TM, Harner CD. Knee ligament and meniscal injuries. Current concepts. *Orthop Clin North Am* 1995;26:529-546.
15. Shearman CM, el-Khoury GY. Pitfalls in the radiologic evaluation of extremity trauma: Part II. The lower extremity. *Am Fam Physician* 1998;57:1314-1322.
16. Solomon DR, Simel DL, Bates DW, et al. Does this patient have a torn meniscus or ligament of the knee. *JAMA* 2003;286:1610-1620.
17. Honari DB, Young CC, Boynton MD, et al. Diagnosis and treatment of common knee injuries in athletes. *Wis Med J* 1996;95:361-366.
18. Muellner T, Nikolic A, Vecsei V. Recommendations for the diagnosis of traumatic meniscal injuries in athletes. *Sports Med* 1999;27: 337-345.
19. Anderson AF, Lipscomb AB. Clinical diagnosis of meniscal tears. Description of a new manipulative test. *Am J Sports Med* 1986; 14:291-293.
20. Noble J, Erat K. In defense of the meniscus: A prospective study of 200 meniscectomy patients. *J Bone Joint Surg Br* 1980;62:7-11.
21. Zionts LE. Fractures around the knee in children. *J Am Acad Orthop Surg* 2002;10:345-355.
22. Stover M. Distal femoral fractures: Current treatment, results and problems. *Injury* 2001;32: SC3-13.
23. Iobst CA, Stanitski CL. Acute knee injuries. *Clin Sports Med* 2000;19:621-635.
24. Dirschl DR, Dahners LE. Current treatment of tibial plateau fractures. *J South Orthop Assoc* 1997;6:54-61.
25. Ruth JT. Fractures of the tibial plateau. *Am J Knee Surg* 2001;14: 125-128.
26. Garner JP, Pike JM, George CD. Intra-articular dislocation of the patella: Two cases and literature review. *J Trauma* 1999;47:780-783.
27. Ghalambor N, Vangsness CT Jr. Traumatic dislocation of the knee: A review of the literature. *Bull Hosp Jt Dis* 1995;54:19-24.
28. Tominaga GT, Connolly JE, Wilson SE. Bilateral popliteal artery injury from bumper crush injury. *J Trauma* 1996;40:311-313.
29. Perryman JR, Hershman EB. The acute management of soft tissue injuries of the knee. *Orthop Clin North Am* 2002;33:575-585.

30. Ilan DI, Tejwani N, Keschner M, et al. Quadriceps tendon rupture. *J Am Acad Orthop Surg* 2003;11:192-200.
31. Warden SJ, Brukner P. Patellar tendinopathy. *Clin Sports Med* 2003; 22:743-759.
32. Fredberg U, Bolvig L. Jumper's knee. Review of the literature. *Scand J Med Sci Sports*1999;9:66-73.
33. Laughlin WJ Jr. Clinically appropriate, cost-effective MR scan of the acute knee injury. *J La State Med Soc* 1998;150: 308-311.
34. Tandeter HB, Shvartzman P. Acute knee injuries: Use of decision rules for selective radiograph ordering. *Am Fam Physician* 1999; 60:2599-2608.
35. Gray SD, Kaplan PA, Dussault R G. Imaging of the knee. Current status. *Orthop Clin North Am* 1997;28:643-658.
36. Bachmann LM, Haberzeth S, Steurer J, et al. The accuracy of the Ottawa knee rule to rule out knee fractures: A systematic review. *Ann Intern Med* 2004;140:121-124.
37. Bharam S, Vrahas MS, Fu FH. Knee fractures in the athlete. *Orthop Clin North Am* 2002;33:565-574.
38. Coumas JM, Palmer WE. Knee arthrography. Evolution and current status. *Radiol Clin North Am* 1998;36:703-728.

CE/CME Questions

1. Which of the following statements regarding the popliteal fossa is *not* true?
   A. The popliteal fossa is a depression found behind the knee joint.
   B. The popliteal artery represents the continuation of the femoral artery into the knee joint and is located in this recess.
   C. The artery is not fixed within the fossa, which protects it from injury when the knee is stressed.
   D. The peroneal nerve is located within this recess.

2. Which of the following statements is true regarding meniscal injuries?
   A. Meniscal injuries are detected using the McMurray's test, Apley compression test or medial-lateral grind test.
   B. The McMurray test involves flexion of the knee beyond 90° with the foot externally rotated in the examiner's hand. The other hand palpates the medial joint line as the knee is extended gradually beyond 90°. Pain, a "popping" sensation, or crepitus indicates a medial meniscus injury.
   C. The McMurray test alone has been shown to have a low sensitivity and specificity for detecting medial meniscus injuries.
   D. Apley's compression test is performed with the patient in a prone position and the knee flexed to 90°. Pain with rotation of the foot while upward traction is applied suggests damage to the capsule and/or ligaments. Pain with rotation of the foot while downward traction is applied indicates meniscal injury.

3. Which of the following statements regarding tibial fractures is *not* true?
   A. Tibial fractures typically occur in one of two ways: 1) a direct blow to the tibia (bumper injury); or 2) axial compression of the tibia, which occurs when the knee is forcibly extended in anticipation of a frontal impact.
   B. Class A injuries (condylar and plateau fractures) usually are produced by axial loading (e.g., a fall from a height).
   C. The rotational stress on the knee will determine whether the medial or lateral tibial plateau is affected.
   D. The medial plateau is the site of fracture in the majority of cases.

4. Which of the following statements regarding disruption of the quadriceps mechanism is true?
   A. Such injuries commonly occur as the result of forced extension of the knee with the quadriceps muscle contracted.
   B. Rupture of the quadriceps tendon superior to the patella is more frequent in the elderly population, while patellar tendon ruptures inferior to the patella are more common in young athletes.
   C. The abduction tendon mechanism also can be disrupted with a complete transverse fracture of the patella and with avulsion of the tibial tubercle.
   D. Immediate arteriography is indicated.

5. Based upon the Ottawa rules, in which of the following patients should plain radiographs *not* be obtained?
   A. A 65-year-old male who has fallen down the steps and complains of knee pain.
   B. A 42-year-old female who presents after falling at a football game with tenderness at the head of the fibula.
   C. A 37-year-old skiier who presents after falling and has isolated tenderness of the patella.
   D. A 52-year-old male who presents with knee pain and can flex his knee to 90°.

CME Objectives

Upon completing this program, the participants will be able to:

a.) Quickly recognize or increase index of suspicion for occult knee injury;
b.) Be educated about how to correctly and quickly stabilize, and then to manage patients with knee trauma;
c.) Understand various diagnostic modalities for arterial injuries; and
d.) Understand both likely and rare complications that may occur.

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

6. Which of the following statements regarding knee dislocations is *not* true?
   A. Knee dislocation is the result of a significant force delivered to the knee and occurs as the result of hyperextension, posterior forces to the tibia, or significant rotary forces.
   B. Any knee dislocation is associated with significant ligamentous disruption.
   C. There are three types of dislocation of the knee: anterior, posterior, and lateral.
   D. In anterior dislocations (the most common type), the tibia is displaced anteriorly to the femur and typically is the result of hyperextension of the knee joint.
   E. In a lateral dislocation, the tibia is forced posteriorly by a direct force applied to the proximal tibia.

7. Which of the following statements regarding lateral dislocations is *not* true?
   A Lateral dislocations can be medial or lateral and occur with equal frequency
   B. Lateral dislocations can be complicated further with rotary deformity of the knee.
   C. Any type of knee dislocation carries a 75% incidence of open joint injury.
   D. Reduction of the dislocation requires longitudinal traction.
   E. Sedation and pain relief commonly are required.

8. Which of the following statements regarding vascular injuries is true?
   A. Injury to the popliteal artery is common in knee dislocations (up to 40% of all dislocations).
   B. It is critical that such injuries be identified early and repaired within 6 to 8 hours of injury.
   C. Intact pulses in the foot do not exclude arterial injury.
   D. An arteriogram should be performed on these patients according to most experts, although an alternative strategy of admission and repeated clinical examination after normal ankle brachial indices (ABI) in the ED has been suggested.
   E. All of the above statements are true.

9. Which of the following statements regarding an ABI is *not* true?
   A. An ABI is performed by obtaining a blood pressure in the arm by standard technique, then taking a blood pressure at the ankle. Dividing the ankle pressure by the arm pressure determines the ABI.
   B. Ankle pressure should be greater than brachial pressure.
   C. A ratio of one or greater is considered normal.
   D. An ABI of 0.9 or less always means that the patient has sustained an arterial injury.
   E. Patients who have peripheral arterial disease also may have an ABI of less than 0.9.

10. A 35-year-old male presents with a patellar fracture. Which of the following statements is true?
   A. A patellar fractures typically is caused by direct knee trauma.
   B. A secondary mechanism may involve an avulsion caused by the pull of the quadriceps mechanism.
   C. Patellar fractures are more common in males.
   D. A non-displaced fracture can be treated with a knee immobilizer and pain control.
   E. All of the above statements are true.

Answer Key:
1. C; 2. B; 3. D; 4. B; 5. D; 6. E; 7. C; 8. E; 9. D; 10. E

In Future Issues:

Ocular Trauma