Eclampsia: Case Studies; Laparoscopic Reconstructive Pelvic Surgery

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Table of Contents

Preface ................................................................. .ii
Chapter 1—Eclampsia: Case Studies  ....................... 1
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Chapter 2—Laparoscopic Reconstructive Pelvic Surgery ............................. 18
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The Hospital Physician Obstetrics and Gynecology Board Review Manual is a quarterly publication intended to supplement study material and to provide a review for board certification candidates. Each board review manual covers anatomy, pathophysiology, principles and theories of disease, as well as clinical aspects of the diagnosis and treatment of pertinent obstetrical, gynecologic, and medical processes. The content of the board review manual is guided by the content specified by the Council on Resident Education in Obstetrics and Gynecology (CREOG) in the Educational Objectives: Core Curriculum in Obstetrics and Gynecology, 6th edition, 2000.

Topics covered include:

- Infertility
- Intrapartum care
- Management of the climacteric period
- Management of nongynecological conditions
- Medical complications of pregnancy
- Menstrual and endocrine disorders
- Obstetric complications
- Office procedures
- Oncology therapies
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- Pediatric and adolescent gynecology
- Postpartum care
- Practice management
- Primary care
- Professional growth and development
- Vulvar and vaginal malignancies

The format of the Hospital Physician Obstetrics and Gynecology Board Review Manual is case-based clinical vignettes that are commonly encountered in the practice of obstetrics and gynecology. This format presents essential information in an easy-to-read, concise manner. The board review questions are intended for reader self-assessment and to direct the reader to pertinent information concerning the topics. The tables and figures are selected for their clarity, educational value, and ability to illustrate, highlight, and/or summarize essential facts and concepts that the candidate must know to successfully complete the Board examination. Coverage is not intended to be complete. Recommended reading and references are listed.

This manual has been developed without the involvement of the ABOG. The manual is based on the Series Editors’ and contributing authors’ clinical experiences, awareness of new developments, experiences as resident educators, and knowledge of the certification examinations in obstetrics and gynecology. The editors wish all the candidates success with their training and Board examinations and rewarding careers in obstetrics and gynecology.

**Preface**

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I. INTRODUCTION

This manual describes the diagnosis, treatment, prevention, and management of seizures in women with eclampsia or who are at risk for eclampsia. Seizure prophylaxis and treatment are discussed for various situations ranging from prophylaxis for at-risk women who have never had eclampsia to the management of serious complications in women with complex eclampsia. Three case patients are presented to highlight and illustrate major concepts as well as to provide a mechanism for self-assessment. Questions provided throughout the text mimic those that might be asked on the oral board examinations; the answers are provided somewhere in the text.

DEFINITION

- What is eclampsia?
- How is eclampsia unique from other causes of seizure activity?

Eclampsia is the development of seizure activity during pregnancy that can be attributed to pregnancy-induced hypertension (PIH) and cannot be attributed to other causes. PIH encompasses the range of hypertensive disorders that are specifically associated with pregnancy. PIH exhibits a broad spectrum of clinical disease from mild involvement to life-threatening complications. When categorizing patients with PIH, those with eclampsia are considered to have severe disease, but clinical evidence of progressive mild signs and symptoms of PIH does not reliably precede the onset of seizure activity.

The goals of managing patients with eclampsia are the same as the goals for managing any woman with severe PIH. These goals include assessment and stabilization of the patient’s airway, breathing, circulation, and higher neurologic function; evaluation for the presence of other potentially life-threatening conditions and initiation of corrective action; and implementation of a plan for delivery. Delivery has been identified as the only definitive treatment for any manifestation of PIH, including eclampsia.1

EPIDEMIOLOGY

- How common is eclampsia?
- Can patients who are at increased risk for developing eclampsia be identified?

Hypertensive disease has been identified as a major cause of maternal mortality in the United States, accounting for 10.4% of the deaths reviewed in a 1988 report.2 The incidence of eclampsia ranges from 1 in 337 to 1 in 3448 deliveries, with the higher incidence rates reported at large, metropolitan, tertiary referral centers.3 Eclampsia is described as primarily a disease of primigravid younger women (eg, women
Chapter 1—Eclampsia: Case Studies

Younger than 20 years). It is reasonable to assume that any condition that increases the risk for PIH should also increase the risk for developing eclampsia; these conditions include nulliparity, being older than 40 years, having a family history of PIH, and other risk factors such as chronic hypertension, chronic renal disease, antiphospholipid syndrome, diabetes mellitus, twin gestation, and presence of angiotensinogen gene T235. Although low socioeconomic status remains a controversial risk factor for PIH, it has been shown to be a risk factor for eclampsia. Because eclampsia is partially preventable by careful observation and timely delivery in patients with PIH and because only a few of the women who develop eclampsia have prenatal care, a reasonable conclusion is that a major component of low socioeconomic status is access to prenatal care. The risk for eclampsia increases in the face of worsening HELLP (hemolysis, elevated liver enzyme levels, and low platelet count) syndrome, another severe variant of PIH. A short duration of sexual cohabitation before conception has also been associated with an increased risk of eclampsia, possibly suggesting an immunologic factor in the pathophysiology of PIH.

II. PATHOPHYSIOLOGY

- What are the general pathologic features of PIH that are characteristic of eclampsia?
- What physiologic features unique to the brain and its vascular structure may further contribute to eclampsia and other neurologic complications of PIH?

The sequence of changes that leads to eclampsia begins with the formation of the placenta itself. Immunologic factors are proposed to interfere with trophoblastic prostacyclin function, leading to incomplete trophoblastic tissue invasion into the arteriole blood supply of the placenta and increased blood clotting in the placental intervillous space. An imbalance between various prostaglandins that regulate vascular tone and platelet aggregation, principally prostacyclin and thromboxane, has been proposed as an important difference between pregnant women who are at risk for developing PIH and women who are not at risk. Normal pregnancy also produces a state of decreased responsiveness to angiotensin II. Among pregnant women who later developed clinical PIH, their response to angiotensin II was pronounced from very early in these gestations and did not return to a normal response until after delivery.

Another potentially important early-onset difference among pregnant women who are later diagnosed with PIH includes an excessive increase in cardiac output. This increase has been suggested to lead to increased intracapillary pressure, in turn leading to endothelial cell damage in the capillary beds of many end-organ systems but not necessarily raising systemic pressure in early stages of the disease. Endothelial cell damage leads to exposure of the endothelial basement membrane and release of thrombogenic and vasoactive mediators that lead to deposition of fibrin and platelets. Endothelial cell damage also disrupts the barrier function of endothelium, leading to interstitial edema and deposition of plasma proteins. Endothelial cell damage and the local release of vasoactive mediators within a vascular system that is already predisposed to increased tone leads to further overall increase in vascular tone and intermittent vasoconstriction. The combination of microvascular obstruction resulting from plasma protein, fibrin, and platelet deposition and aggregation; interstitial edema; and vasoconstriction can lead to decreased oxygen delivery to end-organ tissues, tissue hypoxia, end-organ damage, and end-organ failure. When this process becomes pronounced within the vasculature and tissue of the brain, the clinical result may be eclampsia.

A great deal has been learned regarding the pathogenesis of clinical disease involving the brain in patients with PIH. Autopsy studies initially provided information; more recent studies used imaging techniques such as angiography, computed tomography (CT), magnetic resonance imaging (MRI), and Doppler ultrasound. Various clinical complications are possible with vascular and tissue changes in the brain, including eclampsia, cortical blindness or amaurosis, hypertensive encephalopathy, cerebral edema, intracranial bleeding, and cortical infarction. Acute hypertension can induce a loss of cerebrovascular autoregulation, resulting in passive dilation of arterioles. With the decrease in transmural pressure comes an increase in intravascular hydrostatic pressure, producing further extravasation of fluid and plasma proteins into the interstitial space. Focal, intermittent cerebral vasospasm that accompanies severe hypertension produces tissue ischemia that leads to cell damage and a cytotoxic-derived form of cerebral edema. Mechanisms of vascular autoregulation associated with hypertension can exacerbate the pathologic process that occurs with PIH in the microcirculation of the brain, leading to further interstitial and cytotoxic edema as well as tissue hypoxia. The combination contributes to the occurrence of eclampsia and other PIH-related neurologic complications.
III. CLINICAL PRESENTATION

- Do any reliable clinical signs or symptoms precede eclampsia?
- What are the general characteristics of an eclamptic seizure?

A fundamental problem with the term “preeclampsia” is that up to 38% of women who develop eclampsia do not have clinically significant hypertension or proteinuria before the onset of seizure activity. Apprehension, excitability, or hyperreflexia can precede seizure activity in eclampsia, although a preseizure aura is uncommon.20 Eclampsia can occur in the antepartum, intrapartum, or postpartum periods, but it is more common before delivery. Approximately 55% to 75% of those affected are antepartum and intrapartum patients, versus 25% to 45% of postpartum patients.20 A review of the more common signs and symptoms that precede the onset of eclampsia can be found in Table 1. Headache, hyperreflexia, proteinuria, and edema are commonly associated with the onset of eclampsia; however, these symptoms are not universal. Although clonus and visual changes would be accepted as a warning of a high degree of neurologic irritability and impending eclampsia, they are not as commonly associated with the onset of eclampsia as might be expected. The severity of hyperreflexia, proteinuria, and hypertension does not directly correlate with the severity of the overall disease or the risk for neurologic complications such as eclampsia.1,6

Serum chemistry studies have been of limited value, although serum uric acid levels greater than 5.0 mg/dL have been associated with poor perinatal outcome. In a comparison between 2 groups of women with eclampsia, those with a serum uric acid greater than 10.0 mg/dL demonstrated no significant difference in degree of blood pressure elevation, perinatal outcome, or incidence of recurrent PIH in subsequent pregnancies compared with those who had serum uric acid levels less than 6.0 mg/dL.22 A significantly lower serum colloid oncotic pressure was seen in patients with eclampsia compared with those who had severe preeclampsia, although the clinical utility of this observation has yet to be established.23

A gradual process of development, beginning with weight gain, usually precedes eclampsia. Gaining 2 pounds or more per week in the third trimester with or without clinical evidence of edema can be a marker for a potential problem. About 20% of cases of eclampsia are preceded by relative hypertension, with systolic blood pressures of 130 to 140 mm Hg or diastolic blood pressures of 80 to 90 mm Hg. The first sign of seizure activity usually involves facial distortion, eye protrusion, and a congested facial expression. The patient may also have foaming at the mouth, tongue biting, and absence of respiratory movements. The first phase of the seizure lasts about 20 seconds and is characterized by facial twitching, then generalized rigidity, and then generalized muscle contractions. The second phase lasts about 60 seconds and involves rapid muscular tonic-clonic contractions in succession, starting with the muscles of the jaw or eyelids, progressing to other facial muscles, and then becoming generalized to other muscles in the body. The seizure can last about 60 to 75 seconds and is usually followed by a slow return to normal muscular, neurologic, and respiratory function. A period of amnesia for recent events, including the seizure, follows the seizure. A deceleration of the fetal heart rate with recovery to the baseline rate and reactivity is common.24

IV. MANAGEMENT

PREVENTION

- Who should receive prophylaxis for eclamptic seizures?
- Why is intravenous magnesium sulfate used for eclamptic seizure prophylaxis?
- How is magnesium sulfate administered and monitored?

The prevention of eclampsia requires identifying those patients with the greatest risk: those who have

| Table 1. Frequency of Signs and Symptoms Preceding Eclampsia |
|------------------|-----------------|
| Sign/Symptom     | Patients, %     |
| Headache         | 83              |
| Hyperreflexia    | 80              |
| Proteinuria      | 80              |
| Edema            | 60              |
| Clonus           | 46              |
| Visual changes   | 45              |
| Epigastric pain  | 20              |

Table 2. Protocols for Eclampsia Prophylaxis Using Intravenous Magnesium Sulfate

<table>
<thead>
<tr>
<th>Author</th>
<th>Prophylaxis for Eclampsia</th>
<th>Treatment of Eclamptic Seizures</th>
</tr>
</thead>
</table>
| Roberts             | 2 to 4 g bolus then 1.0 g/hr GTT | 4 g bolus then 1.0 g/hr GTT  
Recurrent seizure:  
Phenobarbital 125 mg IV OR  
Diazepam 5 mg IV |
| Cotton and Dildy    | 4 to 6 g bolus over 20 min then 2 to 3 g/hr GTT | 4 to 6 g bolus over 20 min then 2 to 3 g/hr GTT  
Recurrent seizure:  
Second 2 g bolus |
| Sibai               | 6 g bolus over 15 to 20 min then 2 g/hr GTT | 6 g bolus over 15 to 20 min then 2 g/hr GTT  
Recurrent seizure:  
Second 2 g bolus over 3 to 5 min |

GTT = drops; IV = intravenous.

discontinued and the patient should be thoroughly assessed. Table 3 shows possible toxic side effects after treatment with magnesium sulfate. If respiratory depression is observed, an ampule of calcium gluconate (1.0 g in 10 mL solution) is given by intravenous push over 3 minutes. Properly labeled ampules of calcium gluconate should be readily available for this purpose. If respiratory arrest were to occur, intubation and mechanical ventilation should be undertaken without delay. The use of intravenous magnesium sulfate for the purpose of eclamptic seizure prophylaxis has not been associated with delays in the progress of labor when compared with patients placed on phenytoin for the same purpose. Eclamptic seizure prophylaxis is continued until 24 hours after delivery.

INITIAL STABILIZATION AND EVALUATION

• What are the initial steps in managing a pregnant woman who is having an eclamptic seizure?
• Why is magnesium sulfate the agent of choice for eclampsia?
• What other conditions must be considered and how are they identified?

When a seizure occurs in an antepartum, intrapartum, or postpartum woman, several steps should be undertaken to restore and maintain maternal as well as fetal well-being:

1. Protect the maternal airway during the seizure: place a padded tongue blade between the patient’s back teeth to reduce tongue biting while being careful to avoid stimulating a gag reflex that could precipitate vomiting and aspiration; move patient to her side (preferably the left); using a proper oral suction device, carefully remove foam and secretions from the mouth while taking care not to stimulate a gag reflex.

2. Protect the patient from injury during the initial seizure by removing or covering any surfaces or objects that the patient’s head, arms, or legs might strike. Health care professionals should protect themselves from injury as well.

3. Expect the initial seizure to last approximately 60 to 75 seconds.

4. Do not attempt to shorten or abort the initial seizure.

5. After the initial seizure has resolved, the patient will usually begin to breathe on her own with deep, rapid respirations and may initially be disoriented, combative, and difficult to control. At this time, the following actions should be taken: ensure restoration of respiratory function; begin continuous pulse oxymetry monitoring; supplement oxygen as indicated; auscultate bilateral lung fields for evidence of aspiration; obtain a radiograph of the chest if aspiration is suspected or vomiting has occurred; measure arterial blood gas levels to determine blood oxygen content and to evaluate the degree and type of acidosis, if present.

6. If a long-acting antiepileptic medication with sedation activity (eg, diazepam) has been given during the initial or subsequent seizures, monitor the patient closely for apnea and cardiac arrest. In addition, a health care professional skilled in the management of airway and ventilation in pregnancy (eg, an anesthesiologist or intensivist) should be available if necessary.

7. Assure that large-bore intravenous access has been obtained or maintained after the initial seizure has resolved.

8. Initiate seizure control management with magnesium sulfate intravenous infusion. To review protocols for dosage and administration of magnesium sulfate for eclamptic seizures, refer to Table 2. To review recommendations for monitoring and management of patients on magnesium sulfate intravenous infusion, refer to the previous section on “Prevention.”

<p>| Table 3. Side Effects Associated with Increasing Serum Magnesium Concentrations |</p>
<table>
<thead>
<tr>
<th>Effect</th>
<th>Magnesium Serum Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seizure prophylaxis</td>
<td>4 to 6</td>
</tr>
<tr>
<td>ECG changes</td>
<td>5 to 10</td>
</tr>
<tr>
<td>Loss of deep tendon reflexes</td>
<td>10</td>
</tr>
<tr>
<td>Respiratory arrest</td>
<td>15</td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td>&gt; 25</td>
</tr>
</tbody>
</table>

ECG = electrocardiogram.


9. After stabilization of maternal airway, breathing, circulation, and mental status has been accomplished and seizure control management has been initiated, attention can be directed to fetal concerns if the patient has not yet delivered the fetus. Most fetal heart rate patterns return to a normal baseline level, and fetal/neonatal outcome is generally good after an eclamptic seizure. Transient abnormal fetal heart rate patterns are probably caused by a combination of PIH-induced uterine vasospasm, uterine hypertonia, and the absence of maternal respiration during the acute phase of a seizure. Fetal heart rate patterns that may be associated with eclampsia include: deceleration or bradycardia lasting 30 seconds to 9 minutes, decrease in fetal heart rate up to 5 minutes after the onset of the seizure, a period of transient decreased beat-to-beat variability with late decelerations after deceleration or bradycardia, and transient tachycardia that may occur after deceleration or bradycardia.

10. Tocodynamometry usually demonstrates uterine hyperactivity and increased uterine tone, with contractions ranging from 2 to 14 minutes in duration. Uterine patterns and fetal heart rate should be carefully monitored. Persistent abnormal patterns are more common in preterm and growth-restricted fetuses and may be associated with placental abruption.

11. Expect an eclamptic seizure to recur in 10% to 15% of patients. If a seizure recurs, protect and ensure airway, breathing, circulation as described previously; repeat magnesium sulfate bolus as described in Table 2; and do not exceed a total of 8 g of magnesium sulfate within 1 hour for the management of eclamptic seizures.

DEFINITIVE TREATMENT

• What is the definitive treatment for eclampsia regardless of gestational age?
• How is the route of fetal delivery selected for a given patient with eclampsia?
• How is atypical eclampsia defined?

A plan for definitive treatment of eclampsia is formulated after seizure activity has been controlled, the patient has been restored to a near-normal state of mental function, and initial evaluation of maternal and fetal status has been accomplished. The definitive treatment for eclampsia is delivery of the fetus. Women with eclampsia require delivery regardless of gestational age. The monitoring protocols as outlined previously for evidence of magnesium toxicity are continued. Maternal condition is monitored closely and frequently, and fetal heart rate and uterine patterns (assessed using tocodynamometry) are continuously and carefully monitored. The delivery plan should consider the gestational age of...
the fetus, fetal presentation, status of fetal well-being, status of the labor, and maternal well-being. Magnesium sulfate is continued until 24 hours after delivery. The delivery plan can be initiated when the patient is stable. Induction of labor can be initiated regardless of cervical dilation for patients who are at 30 weeks of gestation or later and whose fetuses have normal presentation. Induction or augmentation of labor can be initiated for the patient who is at less than 30 weeks of gestation, has a favorable cervix or established labor, and whose fetus has a normal presentation. An elective cesarean section can be done for patients who are at less than 30 weeks of gestation and have an unfavorable cervix or for those with a fetal malpresentation at any gestational age. For patients in labor with eclampsia who are at less than 30 weeks of gestation, 30% will have fetal growth restriction, 23% will have placental abruption, and 65% will have an abnormal fetal heart rate pattern.

No recommendation is given in the biomedical literature regarding delaying delivery after corticosteroid administration for treatment of fetal lung maturity. However, this author believes that corticosteroid administration for this purpose is reasonable because the process of stabilization and evaluation before delivery may take some time (approximately an hour) and the induction process may also take up to several hours. At least some benefit for the fetus/neonate can be expected without delaying the delivery process.

Cesarean delivery should be reserved for deteriorating maternal condition or obstetric indications. Vaginal delivery can be achieved in 50% to 82% of patients with eclampsia who undergo induction of labor. Regional anesthesia is possible for either labor analgesia or operative anesthesia. Epidural is preferred over spinal anesthesia because the agents can be slowly and carefully titrated against the patient’s potential high degree of peripheral vasoconstriction, intravascular volume constriction, and hypertension.

For patients with eclampsia who require airway management by intubation (ie, those who have general anesthesia for delivery, airway protection with coma, or hyperventilation for cerebral edema), care must be taken to reduce laryngeal stimulation, which may precipitate transient severe hypertension, pulmonary edema secondary to a sudden increase in cardiac afterload, tachycardia, or a precipitous rise in intracranial pressure. The greatest risk is at times of intubation and extubation, and laryngeal stimulation can be reduced with intravenous labetalol, nitroprusside, or nitroglycerin.

After a woman with eclampsia has been evaluated and stabilized on magnesium sulfate, a careful and honest evaluation of the medical personnel, including available consultants, gestational age, state of other issues related to PIH, facilities for emergency delivery, and intensive care for both the patient and her neonate should be undertaken. Consideration should then be given to transferring the stable patient to a tertiary medical center as appropriate for the optimal care of both the patient and neonate.

A small subset of women with eclampsia will be at least less than 20 weeks of gestational age or will have onset of seizure activity more than 48 hours after delivery. Such cases have been termed “atypical eclampsia,” and these patients require special attention. For pregnancies at less than 20 weeks of gestation, consideration must be given to gestational trophoblastic neoplasm and serious underlying maternal disease. Although rare, eclampsia can occur in the first half of an apparently normal pregnancy, confusing the diagnostic evaluation. Late postpartum eclampsia occurs 48 hours or more after delivery. A full evaluation for other causes of seizure activity is recommended with this condition. The approach for initial stabilization, control of seizures with magnesium sulfate, and evaluation is the same for patients with gestational eclampsia as it is for other antepartum patients with eclampsia; definitive therapy requires termination of the pregnancy. Late postpartum eclampsia, the approach for initial stabilization, control of seizures with magnesium sulfate, and evaluation is the same except that a more thorough evaluation of other potential causes of seizure activity is necessary. Magnesium sulfate therapy can be continued for 24 to 72 hours after initiation until a brisk diuresis of 500 to 1000 mL/hr is established.

V. DIAGNOSIS AND MANAGEMENT OF COMPLICATIONS

- When does a simple case of eclampsia become complex?
- What other disease entities must be considered when a patient does not improve with straightforward management?
- What modalities are available to detect other disease processes?

Further evaluation and management are imperative when a patient presents with a constellation of findings initially consistent with a diagnosis of eclampsia but does not improve with antiseizure management using magnesium sulfate, develops additional neurologic deficits, or is at increased risk for other neurologic
Conditions that may mimic or accompany eclampsia include cortical blindness or amaurosis, hypertensive encephalopathy, cerebral edema, intracranial bleeding, and cortical infarction.

When seizure activity persists despite adequate serum magnesium levels, further management should include obtaining or repeating arterial blood gas levels. If acidemia (pH < 7.10) is present, consider giving sodium bicarbonate intravenously; if hypoxemia is present, provide oxygen supplementation and evaluate for aspiration pneumonitis. Additional management for seizure control includes administration of a short-acting barbiturate (eg, sodium amobarbital or sodium pentobarbital) and consideration of additional diagnostic techniques (eg, CT scan, MRI, cerebral angiography, transcranial Doppler ultrasonography, or an EEG) to rule out complications.

COMPUTERIZED AXIAL TOMOGRAPHIC SCANNING

CT scanning can be useful in identifying a wide range of complications in the obstetric patient with seizures. The technique is considered safe for the second and third trimesters. Possible findings in patients with eclampsia and other neurologic complications include cerebral edema (characterized by diffuse or patchy areas of low density in white matter, particularly in the occipital region; flattening or loss of cortical sulci; decreased intracranial ventricle dimensions; and acute hydrocephalus), cerebral hemorrhage (characterized by intraventricular or high-density parenchymal bleeding), and cerebral infarction (characterized by low attenuation areas or basal ganglia). CT scanning can be useful in identifying a wide range of complications in the obstetric patient with seizures.

MAGNETIC RESONANCE IMAGING

MRI offers a number of advantages over CT. MRI provides better definition of intracranial anatomy and pathologic processes. Because no ionizing radiation is used, MRI can theoretically be used at any gestational age. Contraindications to the use of this technique include the presence of certain metallic aneurysm clips and some cardiac pacemakers.

Almost all patients with eclampsia have multifocal areas of increased signal at the gray-white matter junction on T2-weighted images, consistent with focal edema. T1- and T2-weighted images are generated in a field with different magnetic field strengths as follows. For T1, resonance of hydrogen in fat produces a brighter image. For T2, hydrogen in water produces a brighter image because myelin in brain tissue contains a great deal of lipid and because edema is the result of accumulation of excessive water in tissue. Cerebral edema and intracranial bleeding are seen with a great deal of specificity on T2-weighted images. In a comparison of women with eclampsia who were evaluated by both CT and MRI, abnormal findings were identified in 46% of the MRI studies and in 33% of the CT studies. Of patients with severe PIH but without eclampsia, 50% have nonspecific foci of increased signal in the deep cerebral white matter on T2-weighted images that may be associated with focal vascular changes. Imaging studies may not be necessary for women with uncomplicated eclampsia that responds to initial treatment with adequate magnesium sulfate.

MRI with diffusion-weighted imaging can detect hydrogen in water that has moved from the intravascular to the extravascular space. Therefore, MRI can differentiate diffusion-dependent vasogenic edema seen in hypertensive ischemic encephalopathy from non–diffusion-dependent cytotoxic edema seen with infarction. Conventional MRI cannot make this distinction. The difference is important because hypertensive encephalopathy is treated by controlling blood pressure and infarction is treated with anticoagulation.

OTHER IMAGING STUDIES

Transcranial Doppler Ultrasonography

Significantly higher middle cerebral artery velocities are seen in patients with eclampsia compared with normal pregnant controls and patients with preeclampsia alone. The degree and extent of cerebral vasospasm can be correlated in the face of hypertension and eclampsia.

Electroencephalography

The most common finding on an EEG in patients with eclampsia is change consistent with recent seizure activity, which helps to confirm the diagnosis of seizure but adds no other information. Evidence of seizure activity was persistent in patients with therapeutic serum magnesium levels, suggesting that the mechanism of action of magnesium sulfate does not involve suppression of the type of neurologic activity that produces EEG patterns. No correlation between the degree of hypertension and the degree of EEG abnormalities has been established.

Cerebral Angiography

The role of cerebral angiography in eclampsia has become very limited with the current availability of detailed studies with CT, MRI, and Doppler ultrasound.

ADDITIONAL INTRACRANIAL PATHOLOGY

• What other pathologic processes are possible in the patient with complex eclampsia?
A significant portion of women who die from complications of eclampsia are found to have 1 or more of the following processes: cerebral edema, temporary blindness, intracranial bleeding, cerebral infarction, or hypertensive encephalopathy. With each individual case, it is difficult to determine the specific degree that eclampsia and the pathophysiologic changes inherent to PIH have contributed to the overall disease process compared with these other entities. A great deal of overlap exists in the clinical definition as well as the pathophysiology of eclampsia and these other entities. Occasionally, 1 of these entities presents in the absence of eclampsia and PIH. A high index of suspicion and aggressive management is warranted for these unusual disease states in the patient with atypical or complex eclampsia because each can precipitate significant morbidity and mortality. Because these disease processes can present suddenly and severely, intensive monitoring of the patient with eclampsia is justified.

Cerebral Edema

Cerebral edema occurs with the movement of fluid into the extravascular, interstitial space, resulting in a net increase in intracranial fluid. In general, cerebral edema can result from breakdown at the level of vascular endothelium and the blood-brain barrier, increased intravascular hydrostatic pressures, cellular damage within the parenchyma, plasma osmotic imbalance, or outflow obstruction of cerebrospinal fluid. When cerebral edema accompanies eclampsia, the most likely cause is disruption of the barrier function of the vascular endothelium. Increased intravascular hydrostatic pressure is also an important cause. Cerebral edema can be classified according to the pathophysiologic process thought to produce the given clinical situation. Particularly among patients with eclampsia that is difficult to manage, complex combinations of certain mechanisms are certainly possible. These mechanisms include vasogenic edema with breakdown of the blood-brain barrier, hydrostatic edema secondary to increased intravascular hydrostatic pressure, cytotoxic edema caused by cell membrane instability and intracellular water loss, hypo-osmotic edema caused by increased intravascular free water and decreased plasma osmolality, and interstitial edema related to acute obstructive hydrocephalus.44

Among patients with eclampsia who have recurrent or persistent seizures or other neurologic deficits, vasogenic edema results from the primary pathophysiologic mechanism of PIH, which is the breakdown of vascular endothelial cell barrier function that allows the diffusion of fluid and plasma proteins into the interstitial space. Hydrostatic edema occurs if the endothelial cell damage and release of vasoactive mediators result in severe systemic vasoconstriction and hypertension. With continued damage to the endothelium, the local release of vasoactive mediators as well as microvascular occlusion associated with fibrin and platelet deposition reduces tissue oxygen delivery and causes ischemia, producing hypoxic damage to brain tissue cell membranes and cytotoxic edema. Decreased serum osmolality noted in patients with eclampsia may also contribute to cerebral edema.

The best approach in management of a woman with eclampsia and suspected cerebral edema includes establishing the diagnosis (using cerebral imaging with CT or MRI), correcting hypoxemia and hypercarbia, controlling body temperature, controlling blood pressure, and avoiding use of inhalation anesthesia.45 A patient with severe edema and intracranial hypertension can be managed by providing assisted hyperventilation to produce hypocarbia and correct hypoxemia and by administering intravenous hyperosmotic solutions (ie, mannitol) to draw interstitial fluid into the intravascular space.28

Temporary Blindness

The most common causes identified in patients with temporary blindness during pregnancy include: PIH, eclampsia, cavernous sinus thrombosis, and hypertensive encephalopathy. For patients with PIH, blindness can result from severe retinal vascular involvement or occipital lobe edema. The vascular array that is most sensitive to the described fluctuations of vascular tone in patients with vasoconstriction and severe hypertension is the posterior cerebral arterial distribution, serving the occipital lobes. MRI in such cases has revealed focal occipital cortical edema and focal occipital cortical petechial hemorrhage. Temporary blindness associated with PIH and eclampsia has been reported to last from 4 hours to 9 days.46 The condition resolves with delivery.28

Intracranial Bleeding or Infarction

Among women with eclampsia who develop either intracranial bleeding or infarction, 47% have been noted to have nonhemorrhagic infarction and 44% have intraparenchymal hemorrhage.47 Related conditions include cerebral venous thrombosis, cardiac-derived emboli, nontraumatic hemorrhagic stroke, and hypertensive encephalopathy.

Cerebral venous thrombosis is associated with hypercoagulable states, infection, and hyperviscosity states (ie, sickle cell disease or severe dehydration). Symptoms
are similar to what would be expected for ischemic or hemorrhagic infarction and include increased intracranial pressure caused by poor venous outflow, seizure (occurring in 80% of patients), severe headache, stupor or coma, and limb paralysis.

In patients with cardiac-derived emboli, thrombi usually originate on valves, although mural thrombi occasionally occur. The anterior cerebral arterial circulation is most commonly affected in nonpregnant patients, the middle cerebral arterial circulation is more often affected in pregnant patients, and the internal carotid arterial circulation is more often affected in postpartum women. In addition, these patients commonly have hypertension; however, coma, seizure, and eclampsia are all infrequent.

Nontraumatic hemorrhagic stroke is usually associated with a vascular abnormality such as an aneurysm or arteriovenous malformation. Intracranial bleeding leads to an increase in overall intracranial pressure that precipitates compensatory cerebral vascular hypertension. Clinical features include an association with systemic hypertension and sudden onset of severe headache followed by seizure and coma. Approximately 14% of all patients have albuminuria. The onset of these clinical features is not associated with the hemodynamic changes normal to the process of labor and delivery. The mode of delivery does not affect outcome; cesarean section is reserved for obstetric indications only.

Hypertensive encephalopathy is marked cerebral vasospasm with severe systemic hypertension. This produces cerebral ischemia and cytotoxic edema followed by marked cerebral vasodilatation secondary to loss of cerebral vascular autoregulation. Clinical features include severe systemic hypertension, severe headache, nausea and vomiting, visual disturbances, altered mental status, focal neurologic deficits, and seizures.

**PREGNANCY PLANNING AFTER ECLAMPSIA**

- For a patient with a history of eclampsia during a previous pregnancy, what is the prognosis for a subsequent pregnancy?
- Are close relatives of patients with eclampsia at increased risk for eclampsia?
- Does a woman with a history of eclampsia have an increased risk for chronic hypertension?

The overall prognosis for subsequent pregnancy for women with a history of eclampsia is favorable. An increased risk for PIH and eclampsia in subsequent pregnancies is noted when compared with women without a history of eclampsia, although this risk is not excessive. Subsequent pregnancy for women with a history of eclampsia should be considered high risk for the development of PIH and related complications and requires careful management. Table 4 reviews the findings of 4 large studies regarding subsequent pregnancy among women with eclampsia. The incidence of PIH and related complications in subsequent pregnancy is much higher among women with eclampsia after delivery (>48 hours after delivery or remote from term) and who therefore have the highest risk in subsequent pregnancy. The sisters and daughters of women who have a history of eclampsia are at increased risk for PIH and eclampsia. Women who have had eclampsia do not have an increased risk for the development of chronic hypertension compared with the general population.

### Table 4. Pregnancy After Eclampsia

<table>
<thead>
<tr>
<th>Complication</th>
<th>Women, N</th>
<th>Pregnancies, N</th>
<th>Complications, n</th>
<th>Complications (range), %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preeclampsia</td>
<td>527</td>
<td>938</td>
<td>228</td>
<td>24.3 (21.9–35.4)</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>417</td>
<td>828</td>
<td>21</td>
<td>2.5 (1.0–15.6)</td>
</tr>
<tr>
<td>Placental abruption</td>
<td>353</td>
<td>764</td>
<td>17</td>
<td>2.2 (2.0–2.5)</td>
</tr>
<tr>
<td>Preterm delivery</td>
<td>417</td>
<td>828</td>
<td>98</td>
<td>11.8 (6.5–34.0)</td>
</tr>
<tr>
<td>Intrauterine growth restriction</td>
<td>356</td>
<td>540</td>
<td>54</td>
<td>10.0 (6.0–22.0)</td>
</tr>
<tr>
<td>Perinatal death</td>
<td>527</td>
<td>938</td>
<td>37</td>
<td>3.9 (2.7–6.25)</td>
</tr>
</tbody>
</table>

VI. CASE PRESENTATIONS

CASE PATIENT 1
Presentation
Patient 1 is a 17-year-old woman who is gravida 1, at 38 weeks of gestation. She presents to the general obstetric service with regular uterine contractions. Cervical examination shows dilation of 2 cm, 90% effacement, and station 0. Patient 1 reports mild nausea and mild headache since the onset of contractions about 8 hours before admission. Her blood pressure is 130/70 mm Hg, and urine shows a trace of protein. Vital signs and physical examination are otherwise unremarkable. Fetal heart tones are in the normal range. When she reports increased discomfort with contractions 2 hours later, cervical examination shows dilation of 4 cm, 100% effacement, and station +1. Vital signs remain within normal limits, and fetal heart tones continue to be in the normal range. Her pregnancy is complicated by the fact that she did not receive prenatal care until 32 weeks, when the fact of her pregnancy could no longer be hidden from her family or school personnel.

• What risk factors does patient 1 have for eclampsia?
• Does patient 1 have PIH at this time?
• Should patient 1 have seizure prophylaxis?

Further Presentation
Shortly after the second examination, patient 1 exhibits generalized tonic-clonic activity that lasts for 60 seconds. A tongue blade is placed and padding is placed over the bedside rails. External fetal heart rate monitoring demonstrates a deceleration of the baseline heart rate to 70 bpm. The baseline fetal heart rate recovers to normal within 3 minutes. During fetal monitoring, intravenous access is obtained and secured. An intravenous bolus of magnesium sulfate is given followed by a maintenance drip. Patient 1 is groggy, but she appropriately responds to stimulation and questioning. She is able to protect her airway. Pulse oximetry is 98% on room air. Repeat physical examination demonstrates no evidence of injury, irregular heart rate, or abnormal heart sounds. Her lungs are clear, as was noted on admission. Neurologic examination is nonfocal, and mental status remains stable. Fetal heart tones demonstrate a normal heart rate with diminished short-term variability present, but no further decelerations are noted. Contractions continue in a regular pattern.

Diagnostic Evaluation
A diagnostic evaluation is undertaken, including laboratory evaluation of complete blood count and platelet count, serum electrolyte, creatinine, and uric acid levels, blood urea nitrogen, lactate dehydrogenase and transaminase activity, coagulation studies with fibrinogen, fibrin split products, urinalysis, and toxicology screen as well as a CT scan of the head. The fetal heart tones resume a pattern in the normal range within 25 minutes of the seizure. Patient 1 remains oriented and tolerates intravenous magnesium sulfate with minimal side effects. She and her fetus are carefully monitored for vital signs, symptoms of progression of PIH, fetal heart tones, and contraction pattern. Systolic blood pressure ranges between 130 and 150 mm Hg, whereas diastolic blood pressure ranges between 80 and 90 mm Hg. About 30 minutes after the seizure, artificial rupture of membranes and placement of an intrauterine pressure catheter and a fetal scalp electrode are undertaken.

• What other diagnostic tests might have been done for patient 1?
• Was the CT scan of the head justified at this point? Why or why not?
• Why should the fetal heart tones and uterine contraction pattern be closely monitored?

Discussion
Laboratory evaluation shows a serum uric acid level of 5.8 mg/dL, with all other values within normal limits. Toxicology screening is negative for common substances of abuse. CT scan of the head shows no abnormalities. Labor is augmented with oxytocin. An uncomplicated vaginal delivery of a female infant occurs 6.5 hours after the seizure. Apgar scores are 7 at 1 minute and 8 at 5 minutes; the cord pH is 7.26. Patient 1 remains on an intravenous magnesium sulfate drip until 24 hours after delivery. She experiences adequate diuresis. Her blood pressure decreases to the 120/70 mm Hg range. No further seizure activity is noted. The mother and her infant are discharged to home on the third postpartum day in good condition.

• What is the preferred method of delivery for patient 1’s fetus?
• Under what conditions would a cesarean section have been recommended?
• What information should patient 1 and her family understand for subsequent pregnancy planning?

CASE PATIENT 2

Presentation

Patient 2 is a 38-year-old woman, para 2012, who is admitted to the high-risk obstetric service at 32 weeks of gestation. She has a dichorionic, diamniotic twin gestation and chronic hypertension controlled by alphamethyldopa. She has increased weight gain, nondependent edema, and increased blood pressure despite taking the antihypertensive as prescribed. Her obstetric and surgical history is pertinent for a previous cesarean section at term because of failure to progress in active labor. On initial evaluation, blood pressure is 150/98 mm Hg, and +2 proteinuria is noted. Fetal heart tones are in the normal range for both fetuses, and no contractions are present. Neurologic examination is nonfocal with brisk bilateral lower extremity reflexes. Cervical examination shows no evidence of labor.

Initial laboratory evaluation reveals normal findings for complete blood count, platelet count, serum electrolytes, blood urea nitrogen, creatinine, lactate dehydrogenase, and serum transaminase activities. Serum uric acid level is 6.7 mg/dL. Repeat blood pressure is 148/94 mm Hg. Patient 2 is admitted for further evaluation and monitoring for a diagnosis of chronic hypertension with superimposed mild pre eclampsia. A corticosteroid series is administered to address fetal lung immaturity. An obstetric ultrasound shows a twin pregnancy with breech/breech presentation; fetal biometrics and estimated weights are concordant with the gestational age. A 24-hour urine collection reveals creatinine clearance of 105 mm³/min and urinary protein of 3.2 g. Close fetal and maternal monitoring indicate values are in the normal range throughout the evaluation period of approximately 48 hours.

• Does patient 2 have preeclampsia?
• What other diagnosis might be considered?

Further Presentation

On the third hospital day, patient 2’s blood pressure increases to a systolic pressure of 190 to 210 mm Hg and a diastolic pressure of 90 to 110 mm Hg. She reports some visual disturbances and a moderate headache. Funduscopic examination is unremarkable. With the onset of signs and symptoms consistent with severe preeclampsia, patient 2 is started on intravenous magnesium sulfate for seizure prophylaxis, blood pressure is brought under control with intravenous labetalol, and an elective repeat cesarean section is scheduled. The infants are delivered, and the procedure concludes without complication. Patient 2 is maintained on intravenous magnesium sulfate and transferred to the high-risk obstetric unit for careful postoperative monitoring and management.

• Are patients with suspected severe preeclampsia more likely to develop eclampsia than those with mild preeclampsia?
• What are the signs and symptoms of magnesium toxicity? How is it managed?

Management

Approximately 2 hours after delivery, patient 2 experiences generalized tonic-clonic activity lasting 90 seconds consistent with a grand mal seizure. A repeat intravenous magnesium sulfate bolus is given. Patient 2’s mental status returns to normal, and her neurologic examination after the seizure is nonfocal. Within 24 hours after the seizure, her systolic blood pressure is 130 to 140 mm Hg and her diastolic pressure is 80 to 90 mm Hg; she experiences a large diuresis. The intravenous magnesium sulfate is discontinued 24 hours after the seizure occurred. Patient 2 is then transferred to the postpartum unit.

On the second postoperative day, patient 2 notes a constant sense of numbness, tingling, and mild weakness of the left hand over the thumb, index, and middle fingers extending up to the elbow; she notes that she is right-handed. Neurologic examination is otherwise unremarkable. No further seizure activity is noted. An EEG demonstrates diffuse changes, although no focal abnormality is suggested. A CT scan and MRI (Figure 1) of the head demonstrate a right parietal lobe infarction. Typically, MRI provides better definition of an infarction. Long-term thromboembolic prophylaxis is started with low-dose oral warfarin sodium. Six weeks after the delivery, patient 2 reports near-complete recovery of strength and sensation in the affected hand and arm.

CASE PATIENT 3

Presentation

Patient 3 is a 26-year-old, para 1021, woman at 29 weeks of gestation is admitted to the high-risk obstetric unit in an obtunded state after having 3 seizures at home (which were witnessed) and being transported by
the emergency medical system. Patient 3 has a history of idiopathic seizure disorder since she was 14 years old. She had been taking valproic acid but discontinued the medication without discussing it with her neurologist when she learned of her pregnancy at about 6 weeks of gestation. She experienced the first generalized tonic-clonic seizure event during early afternoon before being brought for admission on the same day.

She had reported to the emergency department of another hospital where her neurologic examination was reported as nonfocal and fetal heart tones appeared to be normal. No contractions were present. Her blood pressure was 140/80 mm Hg, and trace proteinuria was present. She reported a moderate headache that seemed a little better with acetaminophen. Her mental status was blunted but oriented; she responded appropriately to questions. Because the seizure activity was consistent with her history of seizures, she was given oral valproic acid and received a prescription for her usual maintenance dose. She was discharged home. Within a few minutes of arriving at home that evening, a family member saw her have 2 generalized tonic-clonic seizures, at which time patient 3 was transported to the second emergency department and then to the high-risk obstetrical unit.

**What evidence indicated that patient 3 might have had PIH on presentation to the emergency department at the first hospital?**

**Is a diagnosis of a seizure disorder a risk factor for PIH or eclampsia?**

**Further Presentation**

On admission to the high-risk obstetric unit, patient 3 is noted to be markedly obtunded and minimally responsive to noxious stimulation. Blood pressure is 170/98 mm Hg; otherwise, her general examination and neurologic evaluation are grossly normal. She is able to maintain airway protection. External fetal monitoring demonstrates a normal fetal heart rate with diminished beat-to-beat variability. Intravenous access is obtained, and patient 3 receives a magnesium sulfate bolus followed by a maintenance drip. Blood pressure is noted to increase to 210/110 mm Hg, which is controlled with intravenous labetalol. Within 40 minutes of the intravenous magnesium sulfate bolus, a recurrent generalized tonic-clonic seizure occurs that lasts approximately 45 seconds. A smaller repeat intravenous bolus of magnesium sulfate is given.

On assessing patient 3’s mental status after the latest seizure, she can no longer protect her airway. Her continuous peripheral oxymetry drops to 88% during the seizure then increases to 90% afterwards. Patient 3 receives additional intravenous labetalol; she is intubated and mechanically ventilated. A prolonged deceleration of the fetal heart rate is noted with the seizure, although the fetal heart rate recovers to the previously normal baseline within 6 minutes of onset of the seizure. Within 20 minutes of onset of intubation and mechanical ventilation, continuous pulse oxymetry recovers to 98% and some variability returns to the fetal heart rate baseline.

Blood work obtained on admission to the intensive care unit is consistent with HELLP syndrome, with a platelet count of 70,000/mm^3^, aspartate aminotransferase of 780 mg/dL, and lactate dehydrogenase (LDH) of 1100 mg/dL. The rest of her serum coagulation studies are normal except for elevated fibrin degradation products. Serum electrolyte levels and renal function are normal. Corticosteroids are started to manage the HELLP syndrome and to promote fetal maturity. CT and MRI (Figures 2 and 3) of the head are consistent with diffuse generalized cerebral edema. Patient 3 is started on intravenous mannitol, and normal oxygenation is maintained by mechanical ventilation. Invasive hemodynamic monitoring is undertaken to better monitor cardiac output and oxygen delivery because intravascular volume shifts are expected given the need for mechanical ventilation, acute hypertension management, and control of
cerebral edema. An obstetric sonogram reveals a fetus with asymmetric intrauterine growth restriction and oligohydramnios in a cephalic presentation. On examination, the cervix is dilated 2 cm with 75% effacement, station –2, and irregular contractions are noted.

• Did patient 3 have eclampsia or hypertensive encephalopathy?
• Is HELLP syndrome a risk factor for eclampsia?
• Would the combination of intravenous magnesium sulfate and mannitol require additional careful monitoring of the serum magnesium level and clinical examination of patient 3?
• What are the dangers of intubation for patient 3? How are they controlled?

Management

Patient 3 is started on an oxytocin drip to augment her preterm labor. Six hours into the augmentation, the fetal heart rate tracing demonstrates absence of variability and recurrent late decelerations. Continuous pulse oxymetry is in the normal range. Laboratory monitoring demonstrates only slight progression of the HELLP syndrome. Repeat cervical examination shows little progress in labor. Because of concerns about the fetal heart rate, a primary cesarean section is undertaken. A viable preterm male infant is delivered. His Apgar scores are 4 at 1 minute and 6 at 5 minutes; the cord pH is 7.12. Postoperatively, the intravenous magnesium sulfate drip is continued, and a therapeutic serum valproic acid level is achieved.

Patient 3's mental status improves to the point where she can respond to commands, and extubation is successful. Her blood count, platelet count, serum LDH, and transaminase activities begin to return to normal levels; an adequate diuresis is underway 48 hours after delivery. The intravenous magnesium sulfate is discontinued, and valproic acid is continued orally. After extubation, patient 3 is able to maintain oxygenation and protect her airway. Her neurologic examination is nonfocal, but she has marked compromise of short-term and long-term memory. The HELLP syndrome resolves over 1 week. Her blood pressure returns to the normal range. Serum valproic acid levels remain in the therapeutic range. Her memory recovers completely by 2 weeks after delivery with the exception of little recall surrounding the events of her seizures and subsequent delivery. A complete evaluation is repeated at 6 weeks postpartum, and results are in the normal range. The baby does well in the neonatal intensive care unit and special care unit for premature infants.

• Was a trial of labor appropriate management for patient 3?
• What additional evaluation might be considered for patient 3 postpartum?
• What issues should be addressed before patient 3's next pregnancy?
VII. SUMMARY POINTS

GENERAL PRINCIPLES

- Eclampsia is the presence of seizure activity during pregnancy that can be attributed to the existence of pregnancy-induced hypertension (PIH) and cannot be attributed to other causes.
- The incidence of eclampsia ranges from 1 in 337 deliveries to 1 in 3448 deliveries, with the higher incidence rates reported at large, metropolitan, tertiary referral centers.
- Focal, intermittent cerebral vasospasm that accompanies severe hypertension produces tissue ischemia that leads to cell damage and a cytotoxic derived form of cerebral edema.
- Hypertension triggers vascular autoregulation that can then exacerbate the pathologic process involving the microcirculation of the brain that occurs with PIH, leading to further interstitial and cytotoxic edema and tissue hypoxia.
- A fundamental problem with the term “preeclampsia” is that up to 38% of women who develop eclampsia do not have clinically significant hypertension or proteinuria before the onset of seizure activity.

MANAGEMENT

- The only definitive treatment for any manifestation of PIH, including eclampsia, is delivery of the fetus.
- The severity of hyperreflexia, proteinuria, and hypertension does not directly correlate with the severity of the overall disease or the risk for neurologic complications such as eclampsia.
- A substantial portion (20% to 38%) of women who develop eclampsia have only minimal elevations of blood pressure, no significant proteinuria, and no edema. Therefore, the use of seizure prophylaxis has been advocated for patients who meet the blood pressure criteria for PIH regardless of a lack of clinically significant proteinuria or edema.
- Magnesium sulfate given as an intravenous infusion is the agent of choice for seizure prophylaxis in the United States.
- Phenytoin is not as effective as magnesium sulfate for preventing eclamptic seizures. Diazepam has been associated with a high incidence of excessive maternal and neonatal sedation.
- Women with eclampsia require delivery regardless of gestational age. Vaginal delivery can be achieved in 50% to 82% of patients with eclampsia undergoing induction of labor, whereas cesarean delivery should be reserved for deteriorating maternal condition or obstetric indications.

OUTCOME

- Between 10% and 15% of patients are expected to have a recurrent eclamptic seizure.
- A significant portion of women who die from complications of eclampsia are found to have 1 or more of the following: cerebral edema, temporary blindness, intracranial bleeding, cerebral infarction, or hypertensive encephalopathy.
- The overall prognosis for subsequent pregnancy for women with a history of eclampsia is favorable. However, the incidence of PIH and related complications in subsequent pregnancy is much higher among women who had eclampsia remote from term and therefore bear the highest risk in subsequent pregnancy.
- The sisters and daughters of women who have a history of eclampsia are at increased risk for PIH and eclampsia.
- Compared with the general population, women who have had eclampsia are at no increased long-term risk for the development of chronic hypertension.

REFERENCES

Chapter 1 — Eclampsia: Case Studies


I. INTRODUCTION

Several structures are thought to support the pelvic organ system, vagina, and neighboring structures; however, the endopelvic fascia and pelvic floor muscles provide most of the support in this area. Laparoscopic reconstructive pelvic surgery requires a thorough knowledge of pelvic floor anatomy and its supportive components before repair of defective anatomy is attempted. This review describes pelvic supportive anatomy and the different surgical techniques used to repair specific pelvic defects. Two case patients are presented to illustrate specific steps involved in diagnosing and surgically treating different conditions, including problematic stress incontinence and enterocele. The techniques for accomplishing laparoscopic pelvic surgery to repair the defects are described in detail.

II. ANATOMY OF PELVIC SUPPORT

- What is the nature and structural arrangement of the supportive pelvic cellular connective tissues?

ENDOPELVIC FASCIA

To understand the pelvic support system of the female pelvic organs, it is useful to subdivide the support system into 3 axes: (1) the upper vertical axis, (2) the midhorizontal axis, and (3) the lower vertical axis. The endopelvic fascia—a network of connective tissue and smooth muscle—constitutes the physical matrix for the integrity of the axes and maintains the bladder, urethra, uterus, vagina, and rectum in their respective anatomic relationships.

DeLancey further describes the 3 levels of support axes as follows: level 1—superior suspension of the vagina to the cardinal-uterosacral complex; level 2—lateral attachment of the upper two thirds of the vagina; and level 3—distal fusion of the vagina into the urogenital diaphragm. In this support system, the endopelvic fascial system is thought to be continuous, extending from the origin of the cardinal-uterosacral complex to the urogenital diaphragm and providing structural support to the vagina and adjacent organs (Figure 4).

Level 1—Superior Suspension

The cardinal-uterosacral complex provides apical support by suspending the uterus and upper one third of the vagina. This complex can be discussed as 2 separate entities: the cardinal ligament and the uterosacral ligament. The cardinal ligament is the fascial sheath of collagen that envelops the internal iliac vessels then continues along the uterine artery, merging into the visceral capsule of the cervix, lower uterine segment, and upper vagina. The uterosacral ligament is denser and more prominent than the cardinal ligament. Collagen fibers of the uterosacral ligament fuse with the visceral fascia over the cervix, lower uterine segment, and upper vagina. The uterosacral ligament is denser and more prominent than the cardinal ligament. Disruption of the cardinal-uterosacral complex may result in uterine descensus. Likewise, partial vaginal vault prolapse after hysterectomy may also occur from such level 1 detachment. The endopelvic fascial defects associated with several specific clinical findings are shown in Table 5.

Level 2—Lateral Attachment

Level 2 provides horizontal support to the bladder,
upper two thirds of the vagina, and rectum. Additionally, the vaginal wall supports itself to some degree by its fibromuscular tissue, which is often referred to as fascia. Anterior support of the vaginal wall is provided by the pubocervical fascia, whereas posterior support is provided by the rectovaginal fascia. The pubocervical fascia, found between the bladder and the vaginal epithelium, attaches laterally to the tendinous arch of pelvic fascia, often referred to as the white line. Posteriorly, the rectovaginal septum, found between the vaginal epithelium and the rectum, attaches laterally to the fascia over the levator ani muscles. The white line is a linear thickening of the parietal fascia overlying the levator ani muscles and can be traced along its course starting at its origin at the ischial spine, along the pelvic sidewall (internal obturator muscle) to its insertion into the pubic bone.

The rectovaginal septum lies between the vaginal epithelium and rectum, suspended superiorly by the cardinal-uterosacral complex and laterally attached to the fascia of iliococcygeal muscles and white line. This intact rectovaginal septum is the support system of the posterior vaginal wall and helps maintain the rectum in its posterior position. A breach in the integrity of the rectovaginal septum often results in development of a rectocele.

### Level 3—Distal Fusion

The vagina and its support structures of pubocervical and rectovaginal septum traverse the urogenital hiatus to distally fuse into the parietal fascia of the pubococcygeal and puborectal muscles and the perineal membrane. The rectovaginal septum fuses to the perineal body and the pubocervical fascia fuses to the perineal membrane of the urogenital triangle, which subsequently fuses to the perineal body.

### III. Diagnosis of Paravaginal Defects

- **What types of site-specific defects may result in cystocele formation?**
- **How is a paravaginal defect determined on pelvic examination?**

A basic knowledge of the endopelvic fascia support system and the 3 support axes is essential in understanding defects in vaginal wall support. All forms of vaginal prolapse—whether anterior, apical, or posterior—represent a breach of integrity in the continuity of the endopelvic fascia system. Defects in the anterior wall...
result primarily in cystoceles, urethroceles, or cystourethroceles; defects in the posterior wall result primarily in rectoceles; and apical defects usually yield enteroceles.

**CYSTOCELE**

As discussed previously, the pubocervical fascia of the anterior vaginal wall primarily supports the bladder and urethra. This fascia is superiorly suspended to the pericervical ring at the cervix, which is ultimately attached to the cardinal-uterosacral complex, being joined laterally to the white line to give horizontal support and being fused distally to the urogenital diaphragm. A breach or break in the integrity of this support may result in a cystocele, including specific tears such as a transverse break from the pericervical ring, a lateral break at the fascial white line, or a midline/longitudinal tear along the anterior vaginal wall.

The surgical correction of the cystocele depends on the type of defect found in the pubocervical fascia. Clinical assessment in the office is important in determining the correct surgical approach. On examination of the anterior vagina, anterolateral support should be confirmed. If one or both anterolateral sulci are absent and vaginal epithelium rugation is present, then a detachment of the pubocervical fascia from the fascial white line—termed a paravaginal defect—should be suspected. If the anterolateral sulci are present, vaginal rugation is absent, and a cystocele is noted, then a midline tear or defect in the pubocervical fascia should be suspected. A transverse defect also has preservation of the anterolateral sulci with absence of rugae; however, this defect tends to be limited to the upper 33% of the anterior wall.

In patients with these defects, the appropriate surgical repair is selected based on the site-specific defect; a paravaginal defect requires only paravaginal repair, whereas a midline or transverse defect requires an anterior repair or transverse defect repair, respectively.

**IV. LAPAROSCOPIC BURCH TYPE URETHROPEXY**

- **What are the operative indications and critical steps in performing a laparoscopic Burch type urethropexy?**

Since the introduction of the retropubic urethral suspension in 1910, more than 100 different surgical techniques for the treatment of stress urinary incontinence (SUI) have been described. Many of these techniques have been modifications of original procedures that attempted to improve clinical outcome, shorten operative time, or reduce surgical morbidity. Despite the number of surgical procedures developed each year, the Burch urethropexy and pubovaginal sling operations continue to yield the highest rate of success.

Emphasizing the principles of minimally invasive surgery, laparoscopy has evolved as an alternative to many operations that rely on an abdominal or transvaginal approach. Since first described in 1991, the laparoscopic retropubic colposuspension (urethropexy) has rapidly gained popularity. Reported advantages include improved visualization, shorter hospital stay, more rapid recovery, and decreased blood loss.

**OPERATIVE INDICATIONS**

Laparoscopy should be thought of as merely a mode of access, not as a different operative technique. Ideally, the indications for a laparoscopic approach to retropubic urethropexy should be the same as for an open (laparotomy) approach. Patients who are candidates for laparoscopy include those with genuine SUI and urethral hypermobility. Traditionally, an open Burch urethropexy would be performed on these patients, but the laparoscopic Burch urethropexy appears to be an acceptable substitute for most patients. Factors that might influence this decision include history of previous pelvic or incontinence surgery, age and weight of the patient, the need for concomitant surgery, contraindications to general anesthesia, and experience of the surgeon. The decision to proceed with a laparoscopic approach should be based on an objective clinical assessment of the patient and on the surgeon’s skills.

**SURGICAL TECHNIQUE**

We recommend that all patients undergo a modified bowel preparation consisting of a full liquid diet 48 hours before scheduled surgery, then take 1 bottle (10 oz) of magnesium citrate and switch to a clear liquid diet 24 hours before surgery. This regimen results in bowel decompression, which appears to improve visualization of the operative field and to reduce the risk for contamination in case of accidental bowel injury. A single dose of prophylactic intravenous antibiotics (cefazolin, 1 g) is administered 30 minutes before surgery. Antiembolic compression stockings are routinely used. The patient is intubated, given general anesthesia, and placed in a dorsal lithotomy position with both arms placed against her sides. A 16F 3-way Foley catheter with a 5-mL balloon tip is inserted into the bladder and attached to continuous drainage.

The following laparoscopic technique parallels our open technique and has previously been described. We routinely perform open laparoscopy at the inferior margin of the umbilicus. A 10-mm access port is used at this
Chapter 2—Laparoscopic Reconstructive Pelvic Surgery

The abdomen is insufflated with 15 mm Hg of CO₂. Three additional ports are placed under direct vision (Figure 5). The choice of individual port size depends on whether concomitant surgery is planned for the patient.

**Approaches**

The bladder is filled in a retrograde fashion with 200 to 300 mL of normal saline, allowing identification of the superior border of the bladder edge. Entrance into the space of Retzius can be accomplished by using either a transperitoneal or an extraperitoneal approach. Typically, the decision is the surgeon’s preference, although previous abdominal surgery may make a transperitoneal approach more difficult. The transperitoneal approach uses a harmonic scalpel or endoshears. The incision is made approximately 3 cm above the bladder reflection, beginning along the medial border of the right obliterated umbilical ligament. Immediate identification of loose areolar tissue at the point of incision confirms a proper plane of dissection. At this time, instilling 50 mL of indigo carmine or methylene blue into the bladder is recommended by some for easier detection of inadvertent bladder injury during initial dissection.5

At this time, an assistant uses index and middle fingers to elevate the anterior vaginal wall toward Cooper’s ligament while the surgeon uses both hands to tie the suture with a series of extracorporeal knots using an endoscopic knot pusher. An additional double-purchase suture is then placed in a similar fashion at the level of the urethrovaginal junction, approximately 2 cm lateral to the viscera, on the same side. The procedure is repeated on the opposite side. Excessive tension on the vaginal wall should be avoided when tying down the sutures; we routinely leave a suture bridge of approximately 2 to 3 cm (Figure 6).

**Suturing**

The laparoscopic urethropexy is performed using nonabsorbable number 0 sutures; we prefer to use polytetrafluoroethylene. With the nondominant hand, the surgeon uses the index finger to elevate the vagina. The first suture is placed 2 cm lateral to the urethra at the level of the midurethra. A second needle pass or “purchase” is taken, incorporating the entire thickness of the anterior vaginal wall excluding the epithelium; then the suture is passed through the ipsilateral Cooper’s ligament.

When the Burch urethropexy is completed, the intra-abdominal pressure is reduced to approximately 10 to 12 mm Hg of CO₂ and the retropubic space is inspected for hemostasis. Cystoscopy is performed to

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**Figure 5.** Location of abdominal surgical ports. The numbers refer to the diameter of the access ports (mm).
determine whether urinary tract injury has occurred. The patient is given 5 mL of indigo carmine and 10 mL of furosemide intravenously. A 70-degree cystoscope is then used to visualize the bladder lumen, exclude unintentional stitch penetration, and confirm bilateral patency of the ureters. After cystoscopy, attention is returned to laparoscopy. Peritoneal defect closure is encouraged; we routinely use a multifire hernia stapler for closure. All ancillary trocar sheaths are removed under direct vision to ensure hemostasis and exclude iatrogenic bowel herniation. Excess gas is expelled, and fascial defects of 10 mm or more are closed using delayed absorbable sutures. Postoperative bladder drainage and voiding trials are accomplished using either a transurethral or suprapubic catheter.

V. PARAVAGINAL REPAIR COMBINED WITH BURCH URETHROPEXY

- For patients with SUI secondary to urethral hypermobility and a lateral paravaginal defect cystocele, what is the most appropriate laparoscopic approach?

DISCUSSION

If the physician encounters paravaginal defects during the dissection of the retropubic anatomy, we (and most surgeons) believe these defects should be surgically corrected before the laparoscopic urethropexy is done. By repairing the paravaginal defect first, normal anatomical support is established, which minimizes the risk for over-elevating the paraurethral Burch sutures, theoretically reducing voiding dysfunction. Our approach combines the paravaginal repair with Burch urethropexy for treatment of anterior vaginal prolapse and SUI associated with urethral hypermobility (Figure 7).

After dissection of the space of Retzius, the anterior vaginal wall and its point of lateral attachment should be visualized throughout its course from its origin at the pubic symphysis to insertion at the ischial spine. If paravaginal wall defects are present, the lateral margins of the pubocervical fascia will not be approximated to the pelvic sidewall at the tendinous arch of pelvic fascia (white line). The lateral margins of the detached pubocervical fascia and the broken edge of the white line can be visualized to confirm the paravaginal defect. Unilateral or bilateral defects may be present.

CORRECTION OF PARAVAGINAL DEFECTS

The paravaginal repair is performed using a 2-0 non-absorbable suture with intracorporeal needle placement and extracorporeal knot tying. With the nondominant hand, the surgeon uses the index finger to elevate the anterior vaginal wall and the pubocervical fascia to their normal site of attachment along the tendinous arch of pelvic fascia. The needle and suture are introduced through the 12-mm port, and the needle is grasped and driven using a laparoscopic needle driver.
The first suture is placed near the apex of the vagina through the paravesical portion of the pubocervical fascia. The needle is then passed through the ipsilateral internal obturator muscle and overlying fascia around the tendinous arch of fascia at its origin 1 to 2 cm distal to the ischial spine. The suture is secured using an extracorporeal knot-tying technique. Good tissue approximation is accomplished with an intervening suture bridge. Sutures are placed sequentially along the paravaginal defects from the ischial spine toward the urethrovesical junction. In most cases, a series of 2 to 4 sutures is placed between the ischial spine at a point 1 to 2 cm proximal to the urethrovesical junction, allowing space for completion of the Burch urethropexy (Figure 7). The surgical procedure is repeated on the patient’s opposite side. When the bilateral paravaginal repair is completed, the Burch urethropexy is performed.

VI. OUTCOME OF LAPAROSCOPIC BURCH COLPOSUSPENSIONS

- According to the literature, what is the current understanding regarding the clinical results of laparoscopic Burch colposuspensions?

DISCUSSION

Since Vancaillie and Schuessler published the first laparoscopic colposuspension case series in 1991, many other investigators have reported their experience. Review of the literature reveals a lack of uniformity in surgical technique and surgical materials used for colposuspension. This lack of uniformity is seen not only with laparoscopic colposuspension but also with the conventional open (laparotomy) technique. We believe that the laparoscopic approach should be identical to the open technique to allow for comparative studies. Because of the lack of standardization and the steep learning curve associated with laparoscopic suturing, surgeons have attempted to develop faster, easier, and often substantially different ways of performing a laparoscopic Burch colposuspension. These modifications have included the use of stapling devices, bone anchors, mesh, and fibrin glue. The long-term outcomes for these modifications may prove to be substantially different than the original technique.

Many laparoscopic Burch colposuspension case series have been reported, all of which have used conventional surgical techniques and suture material. The published cure rates range from 69% to 100%, with most studies indicating cure rates of greater than 80% (Table 6). Most authors have reported decreased blood loss, decreased duration of hospital stay, decreased postoperative pain, and shorter recovery time. A recent series of 107 laparoscopic Burch procedures has reported an overall complication rate of 10%. Traditional colposuspension has a complication rate of approximately 17%. Complications associated with both techniques include injury to the lower urinary tract, hemorrhage, urinary tract infections, de novo detrusor instability, voiding dysfunction, or even urinary retention. In addition, trocar-related injuries can occur with laparoscopy. For a laparoscopic Burch procedure, intraoperative bladder injury is the most common complication, occurring in 3% to 4% of patients; this injury usually occurs during initial dissection into the retropubic space. Although some surgeons perform subsequent laparotomy to repair the cystotomy, most minor bladder injuries can be repaired laparoscopically. Early recognition of bladder injury and proficiency in laparoscopic suturing techniques are critical elements in this approach. Reports suggest that bladder injury occurs much less frequently in procedures performed by experienced surgeons.

Postoperative voiding dysfunction, such as urinary retention and detrusor instability, appears to occur much less frequently when the laparoscopic approach is used. Recent studies report that detrusor instability is 3% to 5% after a laparoscopic approach compared with 10% to 18% after an open retropubic urethropexy. Despite its recent introduction and the lack of long-term data, the laparoscopic Burch colposuspension has become popular for treatment of SUI. Although initial data suggest that this technique is a safe and effective alternative to traditional laparotomy, surgeons should approach it with caution. Laparoscopic suturing and thorough knowledge of urogynecologic anatomy are essential to yield long-term outcome data equivalent to the traditional open technique. Future prospective randomized clinical trials may establish the laparoscopic approach as a minimally invasive method for successful long-term treatment of genuine anatomic SUI.

VII. CASE PATIENT 4

PRESENTATION

Patient 4 is a 47-year-old women, gravida 4 para 3, who was referred by her primary care physician for evaluation and treatment of SUI. For 1 year, she has been regularly performing biofeedback-directed pelvic floor
muscle exercises under the supervision of a physical therapist. Although these exercises have resulted in mild improvement, patient 4 now seeks surgical treatment because she continues to experience problematic stress incontinence. Her history shows no evidence of urgency, urge-related incontinence, or any voiding dysfunction. Patient 4 only has urine loss associated with activities such as coughing, sneezing, or moderate exercise. In addition, she has vague pelvic pressure or the sensation of vaginal fullness associated with prolonged standing or vigorous activity. She recalls having been told by her previous gynecologist several years ago that her "bladder was beginning to drop."

**DIAGNOSIS AND TREATMENT**

On physical examination, patient 4 is found to have a positive standing stress test. She also has urethral hypermobility (Q-tip strain angle = 70 degrees) and a grade 2 cystocele of a lateral defect type, which would be classified as a stage 2–Aa on the ICS Pelvic Organ Prolapse ordinal staging system. Preoperative urodynamic studies indicate she has an abdominal stress leak point pressure of 120 cm of water and a maximum urethral closing pressure of 65 cm of water. These measurements suggest that patient 4 does not have intrinsic sphincter deficiency. Because she does not have any risk factors such as chronic pulmonary disease, a retropubic urethropexy is recommended over a sling-type procedure. If certain risk factors are present, including chronic coughing or straining, then a sling type of procedure may be preferred. The corrective reconstructive laparoscopic pelvic surgery recommended for patient 4 consists of a laparoscopically performed retropubic Burch type colposuspension combined with a bilateral paravaginal defect repair to repair her cystocele. A suprapubic catheter will also be placed to facilitate postoperative voiding trials. The anticipated length of stay is 1.5 days. Patient 4 is counseled that she can expect to return to normal daily activities, except for strenuous exercise or lifting, within 2 weeks. Strenuous activity (e.g., high-impact aerobics) should be avoided until 12 weeks after surgery.

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**Table 6. Review of Laparoscopic Burch Urethropexy Using the Conventional Suturing Technique**

<table>
<thead>
<tr>
<th>Author (yr)</th>
<th>Patients, N</th>
<th>Follow-up, mo</th>
<th>Objective Data</th>
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*Some or all urethropexies were performed using only 1 suture on each side.
VIII. CASE PATIENT 5

PRESENTATION

Patient 5 is a 69-year-old woman referred by her primary care physician for evaluation and treatment of suspected vaginal vault inversion and possible enterocele formation. She had a standard vaginal hysterectomy approximately 15 years ago because of uterine prolapse. She says she has had the sensation of vague pressure that causes discomfort, but not pain, in the vaginal and perineal region for the past 2 years. During the past several months, she has noticed vaginal tissue protruding through the introital opening if she spends much of the day on her feet. She reports no changes in lower urinary tract or rectal function. She is sexually active and has been receiving hormonal replacement therapy.

A detailed, site-specific pelvic examination reveals incomplete vaginal vault inversion with a small enterocele (POPQ stage III-C). The appropriate reconstructive laparoscopic pelvic surgery for patient 5 would consist of a bilateral uterosacral vaginal vault suspension in combination with repair of the enterocele. The anticipated length of stay is 1 day. Patient 5 is expected to return to normal daily living in 7 to 10 days. Strenuous activities should again be avoided until 10 to 12 weeks after surgery.

• Which level of endopelvic fascia support is defective in the development of vaginal vault inversion?

ENTEROCELE

As previously mentioned, level 1 support involves the long paracolpial fibers that serve to suspend the proximal vagina and cervical vaginal junction. The cardinal and uterosacral ligaments previously described merge with these fibers and attach to the pericervical ring. This network of connective tissue fibers and smooth muscle serves to prevent vaginal eversion. A disruption of the integrity of these fibers, as opposed to stretching, results in apical vaginal vault eversion. The most common cause of this condition is previous hysterectomy with failure to adequately reattach the cardinal-uterosacral complex to the pubocervical fascia and rectovaginal fascia at the vaginal cuff.

• How is an enterocele defined, and what is the anatomic defect associated with development of an enterocele?

An enterocele exists when the parietal peritoneum comes into direct contact with the vaginal epithelium with no intervening fascia. In normal pelvic supportive anatomy, the anterior pubocervical fascia, posterior rectovaginal fascia, cardinal-uterosacral ligaments, and paracolpial fibers all converge, or fuse, to form the pericervical ring. The integrity and continuity of these supportive tissues can be compromised in patients who have had a complete hysterectomy. We believe (as do other surgeons) that development of an enterocele is likely to be directly related to a disruption of the fusion of the cephalad margin of the pubocervical fascia and the corresponding cephalad margin of the posterior rectovaginal fascia. Although vaginal mucosa may cover this defect, it is not supportive, which greatly increases the likelihood that an enterocele will eventually develop within the vaginal cavity. Although the depth and overall anatomic configuration of the cul-de-sac have been implicated in the development of an enterocele, it is the authors’ belief that it is not the primary etiology.

LAPAROSCOPIC TECHNIQUE

• What are the operative steps involved in a laparoscopically performed bilateral uterosacral-vaginal vault suspension and enterocele repair?

The technique of laparoscopic uterosacral-vaginal vault suspension begins with identification of the vaginal apex, the proximal uterosacral ligament, and the course of the pelvic ureter. The identification of the vaginal vault and delineation of the rectum are facilitated by the use of a vaginal probe, end-to-end anastomosis sizer, or similar instrument. Using the vaginal probe, traction is placed cephalad and ventrally, causing the uterosacral ligaments to stretch so they can be identified and traced backward to their most proximal point of origin, lateral to the sacrum. At this level, the uterosacral ligament is usually about 4 cm below the pelvic ureter. The peritoneum overlying the vault apex is incised to expose the pubocervical fascia anteriorly and the rectovaginal fascia posteriorly.

A full-thickness purchase of the uterosacral ligament at its proximal portion is secured with a 0 nonabsorbable suture. Two or 3 helical stitches are placed every few millimeters along the uterosacral ligament. These sutures usually end at the level of the ischial spine. The suture is then placed full thickness, sparing vaginal mucosa, through the ipsilateral rectovaginal fascia and pubocervical fascia in the region of the lateral vaginal fornix. The suture is temporarily held while the opposite uterosacral ligament is reattached in a similar fashion to the ipsilateral vaginal fornix. The sutures are then tied down using the extracorporeal knot-tying
Chapter 2—Laparoscopic Reconstructive Pelvic Surgery

Two or 3 additional reinforcing sutures are then placed on each side. Any openings lateral to the suture attachment are closed because they increase the risk for future small bowel entrapment and subsequent obstruction. The authors (and other surgeons) do not feel it is necessary to plicate the uterosacral ligaments to each other across the midline for this procedure to be successful.

For patients with a coexisting enterocele, the enterocele sac is dissected laparoscopically, which allows for the endopelvic fascial margins to be identified. The delineation of the cephalad margin of the pubocervical fascia anteriorly and the rectovaginal fascia posteriorly may be enhanced by the placement of marking sutures (eg, prolene) placed vaginally, which can be felt on examination during the surgery. The complete loss of rugae with shining, taut vaginal epithelium often demarcates the edge of the enterocele. Depending on the size, the enterocele sac may or may not be excised because redundant tissue will resorb over time. The pubocervical and rectovaginal fascia are then re-approximated with a series of interrupted #0 nonabsorbable sutures. Placing a grasping forceps beneath the suture for downward traction while plicating the fascial margins is often helpful in completing this step (Figure 8).

Figure 8. Placement of interrupted sutures to reapproximate pubocervical fascia (PCF) and rectovaginal fascia (RVF) cephalad margins at vault apex. (A) Placement of curved grasper within suture loop between pubocervical and rectovaginal edge. (B) Early phase in surgical step. Closed-loop knot pusher securing suture while counter traction is applied by curved grasper imbricating the vaginal mucosa allowing approximation of PCF and RVF. (C) Final phase in surgical step. Closed-loop knot pusher securing another suture while counter traction is applied by curved grasper imbricating the vaginal mucosa allowing approximation of PCF and RVF. (D) After the first suture is placed, subsequent interrupted sutures may not require counter traction with grasping forceps.
IX. SUMMARY POINTS

ANATOMY

• Laparoscopic reconstructive pelvic surgery requires a thorough knowledge of pelvic floor anatomy and its supportive components.

• The support system of the pelvic organs can be divided into 3 support axes, or levels: level 1—superior suspension of the vagina to the cardinal-uterosacral complex; level 2—lateral attachment of the upper two thirds of the vagina; and level 3—distal fusion of the vaginal into the urogenital diaphragm.

• The endopelvic fascia, a network of connective tissue and smooth muscle, is a continuous system that constitutes the physical matrix for the integrity of the axes, providing structural support and maintaining the bladder, urethra, uterus, vagina, and rectum in their respective anatomic relationships.

DEFECTS

• All forms of vaginal prolapse, whether anterior, apical, or posterior, represent a breech of integrity in the continuity of the endopelvic fascia system. Defects in the anterior wall result primarily in cystoceles, urethroceles, or cystourethroceles, whereas defects in the posterior wall result primarily in rectoceles and apical defects usually yield enteroceles.

• A breech or break in the integrity of the pubocervical fascia of the anterior vaginal wall may result in a cystocele, including specific tears such as a transverse break from the pericervical ring, a lateral break at the fascial white line, or a midline/longitudinal tear along the anterior vaginal wall. The surgical correction of the cystocele depends on the type of defect found in the pubocervical fascia.

• An enterocele exists when the parietal peritoneum comes into direct contact with the vaginal epithelium with no intervening fascia. The complete loss of rugae with shining, taut vaginal epithelium often demarcates the edge of the enterocele. Development of an enterocele is likely to be directly related to a disruption of the fusion of the cephalad margin of the pubocervical fascia and the corresponding cephalad margin of the posterior rectovaginal fascia, which can occur in patients who have had a complete hysterectomy. Although vaginal mucosa may cover this defect, it is not supportive, which greatly increases the likelihood that an enterocele may develop.

SURGERY

• Despite the number of surgical procedures developed each year, the Burch urethropexy and pubovaginal sling operations continue to produce the highest rate of success for the treatment of stress urinary incontinence (SUI).

• Laparoscopy has evolved as an alternative to many operations that rely on an abdominal or transvaginal approach. Reported advantages of laparoscopic retropubic colposuspension include visualization, shorter hospital stay, more rapid recovery, and decreased blood loss.

• Laparoscopy should be considered as a mode of access, not as a different operative technique, with the indications for a laparoscopic approach to retropubic urethropexy ideally being the same as for an open (laparotomy) approach. Factors that influence whether an open procedure or laparoscopic approach would be used include history of previous pelvic or incontinence surgery, age and weight of the patient, the need for concomitant surgery, contraindications to general anesthesia, and experience of the surgeon.

• If the surgeon encounters paravaginal defects during the dissection of the retropubic anatomy, the authors believe that surgical correction should take place before the laparoscopic urethropexy. This approach combines the paravaginal repair with Burch urethropexy for treatment of anterior vaginal prolapse and SUI associated with urethral hypermobility.

• The published cure rates after laparoscopic Burch colposuspension range from 69% to 100%, with most studies indicating cure rates of higher than 80%. In addition, most authors have reported decreased blood loss, decreased length of stay in the hospital, decreased postoperative pain, and a shorter recovery time.

• Complications of laparoscopic Burch procedures include intraoperative bladder injury as the most common, occurring in 3% to 4% of patients. Bladder injury occurs much less frequently when procedures are performed by experienced surgeons.

• Although initial data suggest that the laparoscopic Burch colposuspension is as safe and effective as traditional laparotomy for treatment of SUI, surgeons should exercise caution when performing this procedure. Laparoscopic suturing and thorough knowledge of urogynecologic anatomy are essential to yield results equivalent to the traditional open technique.
REFERENCES