Critical Care of the Obstetric Patient

Series Editor and Author: Eric H. Gluck, MD, FCCP, FCCM
Chief, Pulmonary and Critical Care Medicine, North Chicago Veterans Affairs Medical Center, North Chicago, IL

Consulting Editor: Cory M. Franklin, MD
Professor of Medicine, Finch University of Health Sciences, The Chicago Medical School, North Chicago, IL; Director, Medical Intensive Care Unit, Cook County Hospital, Chicago, IL

Contributing Author: Domenick J. Sorresso, MD
Assistant Professor of Medicine, Finch University of Health Sciences, The Chicago Medical School; Associate Director of Critical Care Medicine, North Chicago Veterans Affairs Medical Center,

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Cover Illustration by Mary K. Bryson
Preface

Over the past two volumes, the Critical Care Medicine Board Review Manual has covered the basics of critical care medicine, including mechanical ventilation, hemodynamic monitoring, endocrine emergencies, and electrolyte disturbances. In Volume 3, we will focus on more specific issues and will use a case-based format for presenting the material. Brief introductory remarks will be followed by a case report. Broad-based questions following the case presentation will be answered in the subsequent discussion. Most articles will conclude with 7 to 10 board review questions. This format will present the information in a relevant, concise manner and will serve as a review tool for board examinations as well as for in-service examinations.

Volume 3 will address the following topics:

Part 1—Critical Care of the Obstetric Patient
Part 2—Environmental Emergencies
Part 3—Nosocomial Infections in the Intensive Care Unit
Part 4—Upper Airway Diseases in the Intensive Care Unit

The intensive care of the pregnant patient is often very anxiety-provoking. Two lives are at stake, and the infant is usually perceived as being quite fragile. As physicians, we are well aware that until this century, women got pregnant and delivered healthy babies without any medical support at all, so we are not comfortable with the concept that the pregnancy state can result in a life-threatening condition. This manual addresses the most common causes of life-threatening medical complications that occur, as ascertained from the literature and from obstetrical specialists. The physiologic changes that occur during pregnancy, and their potential for interacting with the signs and symptoms of a patient’s presentation, are briefly described. Conditions presented in the case reports herein include: amniotic fluid embolism, tocolytic-induced pulmonary edema, preeclampsia, and peripartum cardiomyopathy. Not only are these among the most common causes of medical emergencies among obstetric patients, they also lend themselves to the development of differential diagnoses of these signs and symptoms, allowing a broad discussion of these patients.

This peer-reviewed manual has been developed without the involvement of the American Board of Internal Medicine or the Society of Critical Care Medicine. It is based on the Series Editors’ and Contributing Authors’ clinical experience, awareness of recent documents and trends in critical care medicine, and a review of the literature. These topics have served as the basis of the education of our medical residents and critical care fellows.

Eric H. Gluck, MD, FCCP
Chief, Pulmonary and Critical Care Medicine
North Chicago Veterans Affairs Medical Center
North Chicago, IL
I. CARDIOPULMONARY CONSIDERATIONS

Many anatomic and physiologic changes occur in pregnant women. An understanding of some of the cardiopulmonary alterations that occur during pregnancy is important for the critical care physician because these changes can greatly impact management strategies.

Anatomic changes occur in the airway itself, in both the upper and lower respiratory tracts. Most notable are airway mucosal congestion and edema. Mucosal edema peaks by the middle of the third trimester and is primarily the result of increased estrogen production, which causes capillary congestion, hyperplasia, and hypersecretion by mucus glands. Many pregnant women have sinus pain, headaches, sneezing, and a nagging cough. Polypsis of the upper respiratory tract may recur in patients who have a history of this condition. Nasal obstruction may cause a variety of practical problems in caring for the critically ill patient. Administration of anesthesia may be hampered, and the patient may be intolerant of face masks. Nasal appliances such as nasogastric tubes, nasal trumpets, and nasal endotracheal tubes may be difficult to use. These appliances should be well lubricated because nasal membranes are quite friable.

The major anatomic changes that occur in the thoracic cavity are secondary to the gravid uterus’ pushing upward on the diaphragm. This effect, along with a widening of the lower rib cage, increases the thoracic anteroposterior diameter. In addition, abdominal musculature is more relaxed during pregnancy, which allows increased inspiratory capacity.
The major pulmonary functional change is a decrease in functional residual capacity. Expiratory reserve volume may decrease by 10% to 40% and residual volume may decrease by 20%. Total lung capacity is reduced by approximately 5%.

Minute ventilation increases, primarily as a result of increased tidal volume, which may rise by as much as 30%. Respiratory rate does not change. The increase in the minute ventilation, the result of higher serum levels of progesterone, causes a chronic respiratory alkalosis.

Pregnant women are hypersensitive to changes in arterial carbon dioxide tension. The diffusing capacity for carbon monoxide increases early in the course of pregnancy but drops back down to normal, or slightly below normal, levels by parturition.

Cardiac output may increase by 30% to 50% and peaks at approximately 20 weeks’ gestation. This increase is primarily caused by a 20% increase in heart rate. The compression of the inferior vena cava by the gravid uterus is responsible for the lower extremity edema typical in normal pregnancies as well as the marked postural hypotension present in many pregnancies.

Many of these physiologic effects are secondary to hormonal changes. An increased level of progesterone is the primary factor responsible for the augmented hypercapnic ventilatory drive. The resulting chronic respiratory alkalosis leads to an increase in bicarbonate excretion. Serum bicarbonate levels among pregnant women are usually between 18 and 21 mEq/L; however, the acute or chronic respiratory alkalosis seen during labor is not readily compensated, and pH may increase to up to 7.6. Patients are frequently aware of their imposed dyspnea. Increased levels of both estrogen and progesterone result in greater mouth occlusion pressure.

Prostaglandin F₂α (PGF₂α), a uterine smooth muscle relaxant, is also a bronchoconstrictor. Prostaglandin E maintains a bronchodilatory effect. There is also a 2- to 3-fold increase in free cortisol levels during pregnancy. The effect of these hormones on a pregnant patient with asthma may be positive, negative, or neutral. Of pregnant patients with asthma, symptoms in one third improve during pregnancy, symptoms in another third worsen, and symptoms in the remaining third remain the same.

II. CASE PATIENT 1

PRESENTATION

A 30-year-old woman, gravida 2, para 1, presents to the hospital at 38 weeks’ gestation after her membranes rupture. Labor does not advance, and she is started on an intravenous infusion of oxytocin to advance labor. In the fourteenth hour of the second stage of labor, the patient becomes agitated but delivers a healthy 3.6-kg (8-lb) male infant. There is a mild drop in the patient’s blood pressure; pulse oximetry is 93%. Respiratory rate is 20 breaths/ min and mildly labored. A chest radiograph shows clear lung fields with mild basilar atelectasis. The patient is given fluids intravenously and supplemental oxygen. Several hours later, she appears confused and markedly dyspneic with laboring breathing. Her hypotension has not responded to fluids, and her pulse oximetry has fallen despite oxygenation. A second chest radiograph reveals bilateral hilar infiltrates. Persistent oozing occurs at the intravenous site. She begins to seize and is intubated.

• What is the differential diagnosis of dyspnea in a peripartum or immediately postpartum woman?
• What is the most likely diagnosis in this patient?
• What diagnostic test would be most helpful in establishing the diagnosis?
• What is the goal of management?

DISCUSSION

Differential Diagnosis

Tocolytic-induced pulmonary edema. Initially, the patient’s chest radiograph was clear, but after several hours of fluid administration, it showed bilateral infiltrates. If this patient had presented earlier in her gestation, had premature labor, and had received tocolytic
agents, tocolytic-induced pulmonary edema would have been a possibility. This patient is near full term, has ruptured membranes, and received a uterine stimulant. Tocolytic-induced pulmonary edema usually reverses quite rapidly with treatment.

**Pneumonitis and adult respiratory distress syndrome.** Another important cause of dyspnea in a pregnant patient is aspiration of gastric contents leading to a pneumonitis or even adult respiratory distress syndrome (ARDS). The patient's recent clinical history would include vomiting during labor. Patients become grossly hypoxemic and febrile, with crackles auscultated on chest examination. The chest radiograph frequently reveals a localized infiltrate that appears as a pulmonary edema pattern unless ARDS is present.

**Heart failure and peripartum cardiomyopathy.** Heart failure and peripartum cardiomyopathy are always possibilities when pulmonary edema is present, especially if the patient received fluid resuscitation. However, these conditions are usually gradual in onset and typically occur 1 month postpartum.

**Asthma.** Asthma is the most common cause of respiratory problems in pregnancy, occurring in about 1% of pregnant women. If this patient had had a history of asthma or was noted to have wheezing during the course of her labor, status asthmaticus would be a consideration. This patient does not present with signs and symptoms of a patient with status asthmaticus, which include diaphoresis, altered speech, and a silent chest. A chest radiograph in a patient with status asthmaticus is typically consistent with hyperinflation.

**Pneumomediastinum or pneumothorax.** These are both rare complications. Pneumomediastinum occurs in approximately 1/2000 to 1/100,000 deliveries, usually in the second stage of labor, and is heralded by chest or shoulder pain with radiation to the extremities and neck. Pneumomediastinum is associated with prolonged labor. The chest radiograph of a patient with pneumomediastinum is consistent with the presence of pneumomediastinal or subcutaneous air. Pneumothorax also is rare and can occur with or without a pneumomediastinum.

**Postpartum cardiopulmonary collapse.** Causes of postpartum cardiopulmonary collapse should also be included in the differential diagnosis. These include hemorrhagic shock, usually resulting from uterine rupture. Air embolism may also present with features of cardiopulmonary collapse. Left ventricular failure, aspiration pneumonia, and anaphylaxis to medications given during labor also may cause cardiopulmonary collapse. When present, thromboembolic disease is usually not evident until several days postpartum.

**Pulmonary embolism.** Pulmonary embolism is one of the most common complications of pregnancy. Pregnant patients are at an increased risk for thromboembolic disease, especially if they have a previous history of thromboembolic episodes. Patients with pulmonary embolism usually have pleuritic chest pain and shortness of breath. Patients may also have evidence of deep venous thrombosis. Patients with pulmonary emboli may have a clear chest radiograph, with atelectasis as the only sign. Pulmonary embolism typically occurs 2 to 3 days postpartum.

**Amniotic fluid embolism.** Amniotic fluid embolism is the result of amniotic fluid entering the maternal circulation, disrupting or occluding maternal pulmonary circulation. Amniotic fluid embolism is the most likely diagnosis for this patient. Amniotic fluid embolism should be suspected when cardiopulmonary collapse occurs peripartum or immediately postpartum.

**AMNIOTIC FLUID EMBOLISM**

**Epidemiology**

The incidence of amniotic fluid embolism is between 1/8000 and 1/80,000 births. The mortality rate is high (80% to 90%). Of mortalities, 28% are associated with a tumultuous labor and 10% to 15% are associated with grand mal seizures and disseminated intravascular coagulation.

Risk factors for amniotic fluid embolism include premature rupture of the membranes, meconium-stained amniotic fluid, and the use of uterine stimulants.
Pathophysiology

Amniotic fluid does not normally enter the maternal circulation. As long as the fetal membranes remain intact, amniotic fluid cannot enter the maternal circulation. In addition to the rupture of the fetal membranes, there must be a disruption of a uterine vein with a pressure gradient favoring entry of amniotic fluid from the uterus to the maternal circulation. There are two possible sites where such a disruption can occur: at the uterine veins at the site of placental separation or at a small tear in the lower uterus and endocervix.

A variety of mechanisms may be responsible for the respiratory failure encountered with an amniotic fluid embolism. Mechanical obstruction of the pulmonary vasculature may occur as a result of the accumulation of large amounts of fetal cellular debris, mucinous material, and other debris within the pulmonary vasculature. Presence of this debris may be confirmed by aspirating blood from the distal port of a pulmonary artery catheter, a process known as pulmonary microvascular cytology.

Another possible mechanism is the presence of an alveolar capillary leak. Characteristically, the bronchoalveolar lavage specimens obtained from these patients tend to have higher protein content than specimens found in patients with ARDS not associated with amniotic fluid embolism and in patients with congestive heart failure.

Other possible mechanisms are pulmonary edema secondary to left ventricular dysfunction and anaphylaxis due to an antigenic response to fetal debris.

Management

The main goal of therapy is supportive. There is no role for prophylactic antibiotics or anticoagulation with heparin. Patients with amniotic fluid emboli most likely will require endotracheal intubation, mechanical ventilation, and hemodynamic stabilization.

III. CASE PATIENT 2

PRESENTATION

A 25-year-old woman, gravida 2, para 1, presents to the hospital at 31 weeks’ gestation. She is having contractions regularly, 8 to 10 minutes apart. On physical examination, she is neither effaced nor dilated. She is administered terbutaline (as an intravenous infusion) to stop her labor and methylprednisolone (intravenously) to help hasten fetal lung development. The patient’s contractions become less frequent and stop approximately 10 hours later, and she is continued on both medications. The next morning, the patient is alert but appears cyanotic and is tachypneic, tachycardic, and markedly short of breath. Auscultation of her lungs reveals bilateral moist crackles to her mid-lung fields. Pulse oximetry is 82%, and a chest radiograph is consistent with bilateral infiltrates.

- What is the most likely diagnosis?
- What is the pathophysiology of this disorder?
- What are some clinical risk factors?
- What is the treatment?

DISCUSSION

Diagnosis

This patient most likely has pulmonary edema secondary to β-adrenergic tocolytic therapy. Pulmonary edema is a unique complication of this treatment. It occurs in approximately 4% of pregnant women who are administered tocolytic therapy for premature labor. Although signs and symptoms of pulmonary edema secondary to β-adrenergic tocolytic therapy are nonspecific, the following suggest the diagnosis: 1) recent use (currently or less than 24 hours previously) of β-adrenergic tocolytic therapy; 2) dyspnea before delivery or shortness of breath less than 12 hours postpartum; and 3) chest radiograph demonstrating either unilateral or bilateral acinar infiltrates.

Patients with this condition have hemodilution that responds rapidly to diuretics and supplemental oxygen. Some of the more common tocolytics (eg, terbutaline, ritodrine) enhance secretion of antidiuretic hormone, resulting in increased water retention and hypotonicity.

Pathophysiology

The pathophysiology of pulmonary edema secondary to tocolytic therapy remains obscure. Neither
left ventricular failure nor myocardial toxicity seems to be a factor. Cardiac enzymes, echocardiography, and pulmonary capillary wedge measurements are usually normal, unless an underlying cardiac condition is present. Although the presence of a capillary leak syndrome has been suggested, the mechanism of action of β-adrenergic medications theoretically should prevent or decrease lung water and capillary leakage. Also, the rapid reversibility of this condition with therapy makes capillary leakage unlikely.

Many patients with pulmonary edema resulting from β-agonist tocolytic therapy have evidence of generalized fluid overload. They probably would have received crystalloid infusions to help combat some of the postural hypotension as well as having received corticosteroids to hasten fetal lung maturity in the event that tocolysis was not successful and delivery was inevitable. The mineralocorticoid effects would cause both salt and water retention.

Risk Factors

Patients at increased risk for pulmonary edema induced by β-adrenergic tocolytics include those who have had intravenous infusions of β-adrenergic tocolytics for more than 24 to 48 hours, those who have had large-volume crystalloid infusions, those who are carrying multiple fetuses, those who have sepsis concurrent with labor, and possibly those who have preeclampsia.

Treatment

The mainstay of treatment is to terminate β-adrenergic tocolytic therapy. Many patients require diuresis and oxygen supplementation. A pulmonary artery catheter is generally not needed unless there is difficulty in assessing fluid status or there is evidence of cardiopulmonary dysfunction, renal disease, or concomitant sepsis. With treatment, the pulmonary edema usually reverses rapidly and patients recover fully.

IV. CASE PATIENT 3

PRESENTATION

Case patient 3 is an 18-year-old black woman, gravi-da 1, para 0, who presents to the emergency department at 32 weeks’ gestation with an unrelenting headache. She has had two episodes of blurred vision in the past 3 weeks. She was last seen at a family planning clinic 24 weeks ago. She has gained 45 pounds during the course of her pregnancy. On physical examination, the patient’s temperature is 37.2°C (98.9°F), pulse rate is 84 bpm and regular, respiratory rate is 12 breaths/min and unlabored, and blood pressure is 180/110 mm Hg. Cardiac examination shows a 1–2/6 flow murmur auscultated at the aortic area. Lungs are clear, and abdomen is consistent with that of a gravid woman, with the uterus measuring approximately 34 cm. The patient’s lower extremities reveal 2+ pitting edema. A urine dipstick test is consistent with 3+ proteinuria.

• What is the correct diagnosis for case patient 3?
• What are the risk factors for this condition?
• What major complications should be considered?
• How should this patient be treated?

DISCUSSION

Diagnosis

Case patient 3 has severe preeclampsia. Preeclampsia is characterized by hypertension, edema, and proteinuria or hyperuricemia after 20 weeks' gestation in a previously normotensive woman. Preeclampsia occurs in approximately 8% of pregnant women and is generally considered either mild or severe. The characteristics of severe preeclampsia are shown in Table 1. If untreated, preeclampsia may progress to eclampsia, which is characterized by convulsions in addition to the signs of preeclampsia.

Risk Factors

Risk factors for preeclampsia include nulliparity, multiple gestation, diabetes mellitus, history of chronic hypertension, underlying renal disease, and polyhydramnios. Preeclampsia is most prevalent among black women between 20 and 35 years of age and from a lower socioeconomic class.

Table 1. Signs of Severe Preeclampsia

| High blood pressure (systolic blood pressure > 160 mm Hg or diastolic blood pressure > 110 mm Hg) |
| Proteinuria (> 5 g in 24 hours or 3+ to 4+ protein on dipstick test) |
| Oliguria (< 500 mL of urine in 24 hours) |
| Evidence of cerebral or visual disturbances |
| Pulmonary edema |
| Epigastric pain |
| Thrombocytopenia (≤ 70,000 cells/mm³) |

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Complications
In patients with preeclampsia, cardiac output is reduced, plasma volume is decreased, and systemic vascular resistance is increased. These changes are the opposite of what occurs in a normal pregnancy. Preeclampsia leads to endothelial injury, which may result in vasospasm, altered vascular permeability, and activation of the coagulation cascade. Perfusion to the placenta, kidneys, liver, and brain is decreased.

The major complications of preeclampsia are convulsions (ie, eclampsia), cerebral hemorrhaging, placental abruption, disseminated intravascular coagulation, pulmonary edema, liver failure, renal failure, and death.

Treatment
Early delivery. Prompt delivery is indicated if eclampsia seems inevitable, if there is evidence of multiorgan dysfunction or fetal distress, or if severe preeclampsia occurs after 34 weeks' gestation. Early delivery is the only cure for either preeclampsia or eclampsia, but it increases the rates of neonatal morbidity and mortality. An earlier gestational age may necessitate a more prolonged pregnancy in order to improve the chances of neonatal survival.

Reduction in blood pressure. The main objective of therapy for patients with preeclampsia is to prevent cerebral complications (eg, convulsions, encephalopathy, hemorrhaging). A sustained diastolic pressure greater than 110 mm Hg requires treatment. Blood pressure should be reduced such that mean arterial pressure is between 105 and 126 mm Hg or diastolic blood pressure is between 90 and 105 mm Hg. Treatment is usually initiated with hydralazine (5 mg intravenously every 20 minutes for a total dose of 20 mg). Other treatment regimens include labetalol (20 mg intravenously) and nifedipine (10 mg orally).

Anticonvulsants. Prophylactic anticonvulsant therapy in women with preeclampsia is controversial. Progression to eclampsia occurs in approximately 0.2% of preeclampsia patients. Some researchers advocate use of anticonvulsants, whereas others claim that lowering blood pressure to a more acceptable level is sufficient. If the decision to use anticonvulsants is made, the agent of choice is magnesium sulfate (a 4-mg intravenous loading dose over 10 minutes followed by a 1- to 2-g intravenous maintenance dose over 1 hour). Magnesium sulfate is superior to either phenytoin or diazepam. For therapeutic use in the face of eclampsia, 1 g/min magnesium sulfate is given until the seizure is controlled, up to a total of 6 grams.

V. CASE PATIENT 4

PRESENTATION
Case patient 4 is a 24-year-old woman who presents to the emergency department with a 3-day history of progressively increasing shortness of breath and dyspnea. She has noted a gradual onset of difficulty breathing associated with orthopnea, postnasal drip, and a dry cough. She has noted calf tenderness over the past 2 weeks. She does not note fever, chills, sweats, or chest pain; she does not have a history of heart disease. The patient had an uncomplicated vaginal delivery of a healthy, 3-kg (7-lb) boy 6 weeks ago. She was in the hospital for 2 days and was subsequently discharged home.

Physical Examination
On physical examination, case patient 4 is in mild respiratory distress. Her temperature is 37°C (98.6°F), pulse rate is 108 bpm, respiratory rate is 40 breaths/ min, and blood pressure is 150/90 mm Hg. The patient has jugular venous distension, bilateral crackles over the lower half of both lung fields, diffuse rhonchi, and 2+ pitting edema of the ankles. There are no murmurs, rubs, or thrills. An S4 gallop is audible. Her calves are asymmetric with the left calf larger than the right, and Homans' sign is positive.

Laboratory Studies
Laboratory studies show hemoglobin level of 11.2 mg/dL; leukocyte count of 11,500/µL with a differential of 80 polymorphonuclear leukocytes, 15 lymphocytes, and 5 band forms; and platelet count of 350,000/µL. Electrolytes are normal except for a minimally elevated blood urea nitrogen level of 29 mg/dL. Serum creatinine level is normal.
• What is the most likely diagnosis for case patient 4?
• What further diagnostic tests are required at this point?
• What therapies should be started immediately?

DISCUSSION
Diagnosis
The most likely diagnosis for case patient 4 is peripartum cardiomyopathy resulting in congestive heart failure. Peripartum cardiomyopathy is rare, occurring in approximately 1 in 4000 deliveries. It usually occurs during the last trimester but may occur up to 5 months postpartum. Symptoms of peripartum cardiomyopathy usually include shortness of breath, exertional dyspnea, edema, palpitations, tachycardia, cough (sometimes with hemoptysis), and, occasionally, pleuritic chest pain. These symptoms often make differentiating between peripartum cardiomyopathy and other causes of heart failure or pulmonary emboli difficult.

Etiology
The etiology of peripartum cardiomyopathy is unknown. Postulates include infectious agents such as viruses, parasites, and bacteria; metabolic disturbances associated with pregnancy; nutritional abnormalities; hormonal changes; maternal-fetal immunologic disorders; stress; toxic insult; and preexisting subclinical heart disease. Peripartum cardiomyopathy is initially confused most often with pulmonary embolism, pregnancy-induced hypertension, congestive heart failure, myocardial infarction, or asthma. The incidence increases with multiparity, pregnancy-induced hypertension, and postpartum hypertension, and is higher in African American women.

Pathophysiology
Because many physiologic changes occur during pregnancy, determining at what point the changes become pathologic is sometimes difficult. During the postpartum period, mild depression of left ventricular function is normally present, which leads to a prolongation of ejection fraction.

Reduction of the ejection time and ventricular contractility are the main pathologic features of peripartum cardiomyopathy. These changes result in elevated pulmonary capillary wedge pressure and in tachycardia. Left heart failure eventually proceeds to biventricular failure and, ultimately, to pulmonary edema.

The histologic changes that occur during peripartum cardiomyopathy are indistinguishable from those of idiopathic dilated cardiomyopathy. These changes include disintegration of heart muscle cells, a pale flabby heart, four-chamber dilation, endomyocardial thickening, mural thrombi, and myocardial fibrosis. Heart valves remain normal.

Diagnostic Tests
Typically, the diagnostic work-up centers around a chest radiograph, electrocardiogram (ECG), and echocardiogram. A chest radiograph of a patient with peripartum cardiomyopathy shows signs of pulmonary congestion or edema and an enlarged heart. The echocardiogram typically shows four-chamber enlargement and reduced systolic function. Sometimes, mural thrombi also are visible. The ECG is nonspecific and may show ST-T segment wave changes, arrhythmias, QRS abnormalities, and left bundle branch block.

Treatment
First-line treatment is hospitalization and bed rest. Prophylaxis for venous stasis is important to prevent phlebitis and, ultimately, pulmonary emboli. Standard treatment regimens for congestive heart failure are effective and include diuretics, digitalis, afterload reduction, anticoagulation, salt restriction, and vasodilating agents. The use of corticosteroids is controversial. Vasodilators must be used cautiously in a pregnant patient to prevent inadequate uteroplacental blood flow. Heparin, which does not cross the placenta, is the anticoagulant agent of choice during pregnancy.

Heart transplantation may be considered in patients with unresponsive progressive heart failure.

Prognosis
The prognosis of peripartum cardiomyopathy depends on when and if cardiomegaly resolves. Most patients who recover within 6 months have a very favorable prognosis. The mortality rate for those who do not
show improvement in heart size within 6 months is 20% to 60% over the ensuing year. Death is usually attributable to progressive heart failure, pulmonary emboli, cerebral emboli, or fatal arrhythmias.

VI. SUMMARY POINTS

- The many anatomic and physiologic changes that occur in pregnant women have a tremendous impact on critical care management strategies.
- Cardiopulmonary alterations include increased airway mucosal edema, increased inspiratory capacity, decreased functional residual capacity, increased minute ventilation, increased cardiac output, increased total blood volume, increased extracellular water, and reduced systemic vascular resistance.
- An important cause of dyspnea in the pregnant patient is aspiration of gastric contents, which may lead to pneumonitis or adult respiratory distress syndrome.
- Asthma is the most common cause of respiratory problems in pregnancy, occurring in about 1% of pregnant women.
- Dyspnea may be a sign of cardiopulmonary collapse. Amniotic fluid embolism should be suspected when cardiopulmonary collapse occurs peripartum or immediately postpartum.
- If dyspnea occurs following administration of β-adrenergic tocolytic therapy with coadministration of glucocorticoids, pulmonary edema should be suspected.
- Preeclampsia is characterized by hypertension, edema, and proteinuria or hyperuricemia after 20 weeks' gestation in a previously normotensive woman. It occurs in approximately 8% of pregnant women.
- Early delivery is the only cure for preeclampsia but carries with it increased neonatal morbidity and mortality rates. If antihypertensive therapy is effective, delivery may be delayed to improve the chances of neonatal survival.
- Peripartum cardiomyopathy resulting in congestive heart failure is a rare disease occurring in approximately 1 in 4000 deliveries. It usually occurs during the last trimester but may occur up to 5 months postpartum.
- Clinical signs of peripartum cardiomyopathy include pulmonary congestion or edema, an enlarged heart, four-chamber enlargement, reduced systolic function, mural thrombi, and nonspecific electrocardiographic changes.
- First-line treatment is hospitalization and bed rest. Prophylaxis for venous stasis should be initiated. Standard treatment regimens for congestive heart failure are effective and include diuretics, digitalis, afterload reduction, anticoagulation, salt restriction, and vasodilating agents.

SUGGESTED READINGS


TOPICS COVERED IN THE CRITICAL CARE MEDICINE BOARD REVIEW MANUALS

Volume 2

Part 1  Hypothermia
Part 2  Disorders of Sodium Metabolism
Part 3  Acute Poisonings I: General Management
Part 4  Acute Poisonings II: Specific Agents

Volume 3

Part 1  Critical Care of the Obstetric Patient
Part 2  Environmental Emergencies (to be published)
Part 3  Nosocomial Infections in the Intensive Care Unit (to be published)
Part 4  Upper Airway Diseases in the Intensive Care Unit (to be published)

TIME TO PREPARE

Candidates who meet the requirements of the American Board of Internal Medicine should apply for examination.

The certification examination in critical care medicine is on November 4, 1998. Late registration is April 2 to July 1, 1998.

For information and applications:
Registration Section, American Board of Internal Medicine
510 Walnut Street, Suite 1700, Philadelphia, PA 19106
(215) 446-3500 • (800)441-ABIM
Fax (215) 446-3590 • Web Site http://www.abim.org