

## **Critical Care of the Obstetric Patient**

Janet M. Shapiro

*J Intensive Care Med* 2006 21: 278

DOI: 10.1177/0885066606290390

The online version of this article can be found at:

<http://jic.sagepub.com/content/21/5/278>

---

Published by:



<http://www.sagepublications.com>

**Additional services and information for *Journal of Intensive Care Medicine* can be found at:**

**Email Alerts:** <http://jic.sagepub.com/cgi/alerts>

**Subscriptions:** <http://jic.sagepub.com/subscriptions>

**Reprints:** <http://www.sagepub.com/journalsReprints.nav>

**Permissions:** <http://www.sagepub.com/journalsPermissions.nav>

**Citations:** <http://jic.sagepub.com/content/21/5/278.refs.html>

>> [Version of Record](#) - Aug 31, 2006

[What is This?](#)

---

# Critical Care of the Obstetric Patient

---

Janet M. Shapiro, MD, FCCP

**The obstetric patient poses exceptional challenges in the intensive care unit. Knowledge of the physiologic changes of pregnancy and specific pregnancy-related disorders is necessary for optimal management. Intensive care unit diagnoses may include preeclampsia, including the HELLP syndrome, pulmonary embolic disease, amniotic fluid embolism, status asthmaticus, respiratory infection, the acute respiratory distress syndrome, and sepsis. The management of mechanical ventilation is based on principles of avoiding lung injury, and hypercapnia may be tolerated even during the pregnancy. When the clinician is faced with the extraordinary instance of cardiopulmonary arrest, perimortem cesarean delivery must be considered to improve the potential for maternal and fetal survival.**

Key words: *pregnancy, obstetric, intensive care unit, perimortem cesarean delivery, mechanical ventilation, cardiopulmonary resuscitation, preeclampsia, HELLP syndrome, amniotic fluid embolism, status asthmaticus, pulmonary embolism*

---

The intensive care management of the obstetric patient presents particular challenges. Critical illness complicates fewer than 0.3% of deliveries [1,2]. Obstetric admissions to the intensive care unit (ICU) are uncommon, comprising less than 1% of ICU admissions [1,2]. Obstetric disorders account for the majority of admission diagnoses, and most patients are admitted to ICU postpartum. The most common ICU diagnoses in obstetric patients are obstetric hemorrhage in 26% to 33%; hypertensive disorders, especially preeclampsia, in 21% to 42%; respiratory failure in 10%; and infection in 10% of obstetric admissions [1-4]. Disorders specific to pregnancy such as preeclampsia and amniotic fluid

From Medical Intensive Care Unit, St. Luke's Hospital; Columbia University; and Division of Pulmonary and Critical Care Medicine, St. Luke's and Roosevelt Hospital Center, New York, New York.

Received November 21, 2005, and in revised form February 2, 2006. Accepted for publication February 5, 2006.

Address correspondence to Janet M. Shapiro, MD, FCCP, Division of Pulmonary and Critical Care Medicine, St. Luke's Hospital MU 316, 1111 Amsterdam Avenue, New York, NY 10025, or e-mail: Jshapiro@chpnet.org.

Shapiro JM. Critical care of the obstetric patient. *J Intensive Care Med.* 2006;21:278-286.

DOI: 10.1177/0885066606290390

embolism may lead to respiratory failure in pregnancy. Diseases such as status asthmaticus, severe respiratory infection and sepsis, the acute respiratory distress syndrome (ARDS), and venous thromboembolic disease may develop during pregnancy or postpartum. Intensive care unit mortality rate of obstetric patients is generally 2% to 3% [1,2,4].

This article focuses on several important disorders of critically ill obstetric patients and reviews issues of respiratory failure in pregnancy. The rare situation of cardiopulmonary arrest and perimortem delivery is addressed.

---

## Changes in Pregnancy

---

Pulmonary physiology changes in normal pregnancy [5]. Upper airway mucosal edema, mucus secretion, nasal congestion, and rhinitis can create difficulties with mask ventilation and endotracheal intubation. The respiratory stimulant effect of progesterone leads to an increase in minute ventilation through an increase in tidal volume and generally unchanged respiratory rate. Consequently, arterial blood gas analysis typically shows respiratory alkalosis with  $Paco_2$  in the range of 28 to 32 mm Hg [5,6]. Elevation of the hemidiaphragm is attributable to the enlarging uterus, and functional residual capacity is reduced. There is no change in lung compliance, but chest wall compliance is reduced.

The pregnant patient has reduced respiratory system reserve in the face of elevated maternal and uteroplacental oxygen demand. In a critical illness, rapid and severe respiratory decompensation may occur. The pregnant patient is also at greater risk of gastric aspiration related to reductions in lower esophageal sphincter pressure and gastric motility [5].

Circulatory changes include an increased cardiac output attributable to the expanded circulation and a decrease in systemic vascular resistance attributable to the uteroplacental circulation [5,6]. Aortocaval compression by the gravid uterus is extremely important; in fact, in late pregnancy, the inferior vena cava may be completely obstructed in the supine position, and venous return occurs through azygous, lumbar, and paraspinal veins. The uterus receives up to 30%

of the cardiac output. The uteroplacental circulation lacks autoregulation. When the maternal circulation is compromised and compensatory vasoconstriction occurs, uteroplacental perfusion will be reduced, leading to rapid fetal hypoxia and acidosis. For this reason, evidence of fetal distress such as fetal bradycardia may be a sign of maternal deterioration [7]. For the team of intensivist and obstetrician, fetal heart monitoring provides crucial information about both the maternal and fetal conditions.

---

## Preeclampsia

---

Hypertension is one of the most common disorders complicating pregnancy. Preeclampsia is characterized by hypertension and proteinuria occurring after the 20th week of gestation [6]. This disorder complicates 5% to 8% of pregnancies. Patients present with edema, visual disturbances and headache, and epigastric pain. Preeclampsia is a procoagulant and proinflammatory state caused by placental hypoperfusion. Impaired trophoblast invasion of the uterine spiral arteries prevents the normal increase in blood flow to the placenta [8,9]. The placenta secretes antagonists to vascular endothelial growth factor and placental growth factor. Locally, there is endothelial release of the vasoconstrictor thromboxane, activation of platelets, reduction in nitric oxide, and vascular sensitivity to angiotensin. In addition, inflammatory cytokines such as interleukin-6 and tumor necrosis factor are released [8]. Placental hypoperfusion is the result.

The local vascular dysfunction becomes generalized in severe preeclampsia, leading to dysfunction of hepatic, renal, cerebral, and hematologic systems. Clinical findings of severe preeclampsia are severe blood pressure elevation >160/110 mm Hg, proteinuria, oliguria, blurred vision, headache, cerebrovascular accident, elevation of transaminases, liver capsule distention leading to pain, nausea and vomiting, platelet count below 100 000/mm<sup>3</sup>, and pulmonary edema [9]. Placental pathology reveals fibrinoid necrosis, thrombosis, and infarction. Preeclampsia can progress rapidly to eclampsia, characterized by development of seizures [8].

The HELLP syndrome—hemolysis, elevated liver enzymes, and low platelets—complicates 10% to 20% of cases of severe preeclampsia [8]. Although this syndrome is believed to be in the spectrum of severe preeclampsia, 15% of patients do not have hypertension or proteinuria. Patients present with abdominal pain, nausea, and malaise. The key features are thrombocytopenia <100 000/mm<sup>3</sup>, microangiopathic hemolytic anemia with schistocytes and

elevated lactate dehydrogenase >600 IU/L, and elevated transaminases [10,11]. Complications may include hemorrhage, but more prominent are hepatic failure or infarction and rupture. Pathology of the liver shows similar changes of preeclampsia in the placenta: fibrin microthrombi, periportal hemorrhage, and necrosis [8]. The differential diagnosis of HELLP includes acute fatty liver of pregnancy [11]. Only 50% of these patients have a diagnosis of preeclampsia. Signs of acute fatty liver of pregnancy are more prominent findings of hepatic failure with hypoglycemia, coagulopathy, and encephalopathy. In contrast to the vasculopathy of preeclampsia, liver pathology demonstrates microvesicular steatosis [6].

Pulmonary edema complicates 3% of cases of preeclampsia and usually occurs postpartum [8]. Respiratory failure occurs in the setting of fluid overload, hypoalbuminemia and decreased colloid oncotic pressure, and increased pulmonary capillary hydrostatic pressure. Pulmonary edema may develop in severe preeclampsia complicated by cardiopulmonary arrest, hypertensive crisis, disseminated intravascular coagulation, acute renal failure, and cerebral edema.

Delivery is indicated for severe maternal organ failure, placental abruption, or gestation over 34 weeks [6,8]. Supportive treatments with oxygen, mechanical ventilation, and antihypertensive therapy are administered. Fluid management may be challenging because intravascular volume is often low. The goal is perfusion of vital organs, including placental perfusion, guided by clinical examination and, at times, by hemodynamic monitoring [6]. Magnesium sulfate is effective for the prevention of seizures in severe preeclampsia as well as treatment of eclamptic seizures [8]. Management of the HELLP syndrome includes corticosteroid therapy for fetal lung maturation antepartum; corticosteroids have been shown to increase platelet count [12]. Plasma exchange has been advocated in patients with severe HELLP syndrome (platelet count below 50 000/mm<sup>3</sup>) that does not resolve postpartum. In 1 case series of daily plasma exchange in severe HELLP, there were no maternal deaths [13]. Maternal mortality rate in preeclampsia is reported up to 11% [6].

---

## Pulmonary Embolism

---

Venous thromboembolism (VTE) is an important cause of maternal morbidity and mortality [14,15]. It is estimated that VTE complicates 0.06% of pregnancies and is a leading cause of maternal mortality [14]. VTE may occur throughout the pregnancy: the highest incidence of pulmonary embolism is in

the immediate postpartum period. Risk factors for venous thromboembolism in pregnancy include prior VTE, recent surgery, older age, obesity, and thrombophilia.

Specific changes related to pregnancy favor the development of venous thrombosis: venous stasis, endothelial injury, and an increase in coagulation [14]. Venous stasis is attributable to a progesterone-induced increase in venous capacitance and vena caval obstruction from the uterus. Vascular injury at the uteroplacental surface occurs at delivery. Favoring coagulation are the increase in coagulation factors I, II, VII, VIII, X; a decrease in the coagulation inhibitor protein S; an increase in resistance to activated protein C; impaired fibrinolysis attributable to an increase in plasminogen activator inhibitors; and activation of platelets. Pregnant women with thrombophilic disorders may present with the initial VTE during pregnancy. These thrombophilic disorders include activated protein C resistance attributable to factor V Leiden mutation, prothrombin gene mutation, and deficiencies of protein C, protein S, and antithrombin III [14].

The clinical findings of pulmonary embolism include dyspnea and chest pain. Syncope is an uncommon presentation. As in nonpregnant individuals, a massive pulmonary embolism may present with shock attributable to embolic obstruction to right ventricular cardiac output. The hypotension with elevated right ventricular wall tension may lead to coronary ischemia and further impair myocardial function.

The diagnosis of pulmonary embolism during pregnancy raises concerns about diagnostic yield and safety of the radiographic studies for the fetus. Although measurement of serum D-dimer, a breakdown product of fibrin, has been applied to exclude pulmonary embolism, levels are generally increased in the postpartum period and so this test is not as useful in pregnancy [14]. A chest radiograph should always be performed to assess for an alternative diagnosis, particularly pneumonia. The ventilation-perfusion (V/Q) lung scan has been the traditional initial test for pulmonary embolism during pregnancy. In the largest series of 121 V/Q scans in pregnant women with suspected pulmonary embolism, there was a higher proportion of diagnostic scans (ie, 1.8% high probability and 73.5% normal scans) than in nonpregnant individuals [16]. The yield of V/Q scanning may be higher in pregnant patients because of less comorbid pulmonary disease and the lower prevalence of pulmonary embolism in this younger age population.

With a nondiagnostic V/Q scan, lower extremity compression ultrasound is the next test, because

demonstration of lower extremity deep venous thrombosis will render a diagnosis of venous thromboembolic disease, which requires anticoagulation. If compression ultrasound is negative and the pretest probability of pulmonary embolism remains moderate or high, additional testing is mandatory.

Pulmonary angiography has been considered the gold standard for the diagnosis of pulmonary embolism for patients with suspected pulmonary embolism and nondiagnostic noninvasive tests. In practice, helical chest computed tomography (CT) scanning has become a common initial test for pulmonary embolism. The diagnostic yield is excellent for major pulmonary embolism involving the main and lobar pulmonary arteries. The studies of helical CT, however, have excluded pregnant patients. A calculation of fetal radiation exposure with helical CT for pulmonary embolism supported its use, because the fetal exposure was less than that in V/Q lung scanning [17]. A survey of radiologists revealed that most perform CT angiogram in pregnant women [18]. A recently published decision-tree analysis supported CT angiogram as the most cost-effective initial test in diagnosis of pulmonary embolism in pregnant women [19].

It is recommended that fetal radiation exposure be limited to less than 5 rad. Estimates of fetal radiation exposure are [14,17] as follows:

Chest radiograph: <0.001 rad  
 Ventilation-perfusion scan: <0.011 rad  
 Pulmonary angiogram (via brachial artery): <0.05 rad  
 Chest CT scan: <0.016 rad

These values should assure clinicians that diagnostic evaluation for pulmonary embolism can be safely pursued in the pregnant patient with suspected pulmonary embolism. Among 120 pregnant women who underwent V/Q scanning for suspected pulmonary embolism, there was no increase in spontaneous fetal loss, congenital abnormalities, or malignancies compared with general pregnant women [16].

Echocardiography may be useful in supporting the diagnosis and in guiding therapy of massive pulmonary embolism. Right ventricular dilation and hypokinesis, paradoxical septal motion, and compression of the left ventricle may be observed in major pulmonary embolism. The echocardiographic findings alone are generally not sufficient to establish the diagnosis of pulmonary embolism [20].

For treatment of pulmonary embolism, heparin is administered during the pregnancy because it does not cross the placenta, in contrast to warfarin, which can cause fetal hemorrhage and malformations [21]. Low-molecular weight heparin or

intravenous unfractionated heparin may be used. Low-molecular weight heparin is advantageous in terms of fixed dosing and lack of monitoring, potentially lower incidence of heparin-induced thrombocytopenia, and decreased risk of osteoporosis in women who must continue heparin throughout the pregnancy [21]. Low-molecular weight heparin may be adjusted during the pregnancy based on weight gain or targeted to anti-Xa level [21]. Warfarin is administered for postpartum anticoagulation. Anticoagulation for pulmonary embolism is generally administered for 6 months and should include 6 weeks postpartum.

Massive pulmonary embolism may complicate pregnancy or the postpartum state. Intravenous unfractionated heparin is administered. Right ventricular failure may occur attributable to the embolic obstruction and may require hemodynamic support. Hypotension is first addressed by positioning the patient in the left lateral decubitus position, so that compression of the inferior vena cava may be relieved and venous return improved. Intravenous fluids should be administered. Vasopressors should be administered to achieve adequate maternal perfusion; although uterine artery vasoconstriction may occur, improving maternal circulation will enhance vital organ perfusion. Dopamine has been used as an effective vasopressor during pregnancy [6] and may be required in the setting of massive pulmonary embolism.

Thrombolytic therapy with tissue plasminogen activator may be administered for massive pulmonary embolism with hemodynamic compromise [22]. Case reports and series describe the use of thrombolytic therapy during pregnancy and the immediate postpartum period, times considered to be relative contraindications to this therapy [23,24]. In the largest series of 172 uses of thrombolytic therapy, maternal mortality rate was 1%, maternal hemorrhage 8%, and pregnancy loss 6% [23].

---

## Amniotic Fluid Embolism

---

Amniotic fluid embolism syndrome is characterized by abrupt cardiorespiratory collapse and coagulopathy during labor or immediately postpartum [25,26]. Amniotic fluid includes particulates such as squames, lanugo hairs, mucin, and bile from meconium; prostaglandins E<sub>2</sub> and F<sub>2a</sub>; leukotriene B<sub>4</sub>; and a thrombokinase-like element. Amniotic fluid gains entry into the maternal circulation by disruption of the uterine wall and rupture of the placental membranes. Induction medications and difficult labor with forceful contractions have been disproved as

causing amniotic fluid embolism. These amniotic fluid elements may produce pulmonary artery obstruction, activation of inflammation, and activation of coagulation. For this reason, the term "anaphylactoid syndrome of pregnancy" has been applied [25]. Components of amniotic fluid such as fetal squames have been discovered in the circulation of patients without the clinical syndrome, so the mere presence of the particulates is not sufficient to cause the syndrome.

In animal models, injection of amniotic fluid causes pulmonary vascular occlusion and vasospasm leading to cor pulmonale [26]. The human syndrome is characterized by left ventricular failure, as documented in patients who survived to have a pulmonary artery catheter placed. It is postulated that the human response may be biphasic, with initial pulmonary hypertension [27], followed by myocardial depression from inflammatory mediators and hypoxemia. The left heart failure then manifests as pulmonary edema and shock or cardiopulmonary arrest. Neurologic dysfunction with seizures and coma and coagulopathy complete the clinical picture.

Lung pathology may reveal gross findings of edema or hemorrhage, but the lung may have a normal appearance. Histologic examination demonstrates foreign material in the pulmonary capillaries, arterioles, and arteries [25]. Special stains such as TKH-2, a monoclonal antibody to fetal glycoprotein sialyl Tn antigen, have been applied to pathologic specimens and also evaluated in maternal serum [28]. Testing maternal serum has not been validated and is not currently recommended for diagnosing the syndrome.

The results of a national registry of 46 clinically diagnosed cases provide information on the syndrome [26]. The majority of cases occurred during labor or within 8 minutes of delivery. Hypotension and fetal distress were universal; almost all patients (93%) were in respiratory failure, described as pulmonary edema or ARDS, and 87% suffered cardiopulmonary arrest. Coagulopathy was present in 83% of cases. Supportive management includes advanced cardiac life support protocol for cardiopulmonary arrest, mechanical ventilation, hemodynamic support, and blood product administration. Maternal outcome was poor: maternal mortality rate was 61%, and 85% died or survived with permanent neurologic damage [26]. Bleeding from the delivery site caused by the coagulopathy may be difficult to control, and cases are reported of treatment with uterine artery embolization [29] and recombinant factor VIIa administration [30].

---

## Respiratory Infection

---

Pneumonia can lead to maternal death, preterm delivery, and low-birth weight infants. Pathogens in community-acquired pneumonia are similar in pregnant and nonpregnant individuals and include *Streptococcus pneumoniae*, *Hemophilus influenzae*, *Mycoplasma*, *Chlamydia*, and *Legionella* [31].

Cell-mediated immunity is impaired in pregnancy; findings include a decrease in T-helper cells, reduced natural killer cell activity, and reduced lymphocyte proliferation. Viral pathogens, such as influenza and varicella, may thus cause severe infection in pregnancy. Influenza caused excess deaths among pregnant women during influenza epidemics, and women in the second and third trimesters are at increased risk for influenza-related morbidity and mortality [32]. Influenza may cause primary pneumonia or be complicated by secondary bacterial infection. Patients may present with acute hypoxemic respiratory failure and bilateral opacities on chest radiograph. Early diagnosis with a rapid enzyme-linked immunosorbent assay for influenza A can provide a diagnosis in 30 minutes, and viral culture provides influenza subtype and strain. Antiviral agents such as amantadine and oseltamivir may reduce the duration of illness; however, these agents have not been shown to affect the course of influenza complicated by respiratory failure. Amantadine has been administered to pregnant women with respiratory failure, despite concern about teratogenicity. The neuraminidase inhibitor oseltamivir is preferred in pregnant patients [32].

Pregnant patients infected with the human immunodeficiency virus (HIV) present additional infectious possibilities. Respiratory failure attributable to *Pneumocystis carinii* pneumonia may be rarely diagnosed and successfully treated with antibiotics and corticosteroids during pregnancy [33]. In recent large series of obstetric patients requiring ICU, HIV-related disorders have not been reported [3,4].

---

## Status Asthmaticus

---

Asthma is a common respiratory disorder in pregnancy. Poor control of asthma is associated with adverse maternal and fetal outcomes: preeclampsia, uterine hemorrhage, preterm delivery, and low birth weight [34]. Mechanical ventilation for status asthmaticus is uncommon in the general population, and the incidence of intubation for status asthmaticus in pregnancy is unknown. In our series of

87 patients with status asthmaticus, 3 pregnant women required intubation [35].

In status asthmaticus, airway inflammation and edema and smooth muscle bronchoconstriction lead to severe airflow obstruction. Incomplete exhalation leads to lung hyperinflation, which compromises diaphragm function. Hypoxemia attributable to V/Q mismatching and increased muscle metabolic demands may eventuate in respiratory muscle failure. Inhaled  $\beta$ -agonist and anticholinergic therapy is administered, and systemic corticosteroids are given. Although parenteral  $\beta$ -agonist therapy has not been proven more effective than inhaled treatment, if subcutaneous  $\beta$ -agonist therapy is considered, terbutaline is the preferred agent in pregnancy [36].

Ventilator management in the pregnant patient with status asthmaticus is identical to that of the nonpregnant patient. Dynamic hyperinflation with an increase in intrathoracic pressure can lead to hypotension or barotrauma. Thus, controlled ventilation with slow respiratory rate to allow sufficient expiratory time and prevent dynamic hyperinflation remains the goal. Decreasing ventilation may lead to hypercapnia; permissive hypercapnia is well-accepted for management of ventilatory failure attributable to obstructive airways disease. Studies have not specifically addressed pregnancy; however, in one small series, hypercapnia (mean  $Paco_2$  57 mm Hg) was induced during labor without maternal or fetal complication [37].

We have cared for 1 patient in her first trimester of a twin pregnancy who required mechanical ventilation, sedation and neuromuscular blockade, and aggressive inhaled and systemic therapy. She experienced marked hypercapnia with  $Paco_2$  greater than 80 mm Hg (maximum  $Paco_2$  150 mm Hg) during 3 days of mechanical ventilation; she was extubated on the fourth day, continued outpatient asthma therapy, and delivered normal twins at term.

In status asthmaticus, ventilation must be controlled to minimize dynamic hyperinflation and its consequences. To control ventilation, sedatives, and rarely neuromuscular blockade, are used. Morphine, fentanyl, and propofol have all been used in pregnant patients [36].

---

## Acute Respiratory Distress Syndrome (ARDS)

---

The ARDS may occur in the obstetric patient as a complication of pneumonia, aspiration, sepsis, and amniotic fluid embolism. Cytokines and inflammatory cells and their products injure the alveolar epithelium and capillary endothelium. Physiologic consequences

are hypoxemia, decreased compliance, and pulmonary hypertension [38]. Clinical evaluation reveals a rapid decline in oxygenation and bilateral opacities on chest radiograph. Respiratory system compliance may be reduced in pregnant patients near term because of upward displacement of the diaphragm [5], and so measurements of end-inspiratory (plateau) pressure may be elevated without severe ARDS.

The essential support in patients with ARDS is mechanical ventilation. Recent studies have refined the ventilatory approach. It is postulated that ventilator-induced lung injury may perpetuate and add to the lung injury and increase the risk of mortality. The ARDS Network trial found that the use of lower tidal volumes (6 mL/kg compared with 12 mL/kg ideal body weight) was associated with a decrease in mortality rate [39]. Additional strategies have been used in ventilatory management. Prone positioning to attempt to improve oxygenation by enhanced V/Q matching, augment secretion drainage, and relieve lower lobe compression by the heart has been applied but has shown no mortality benefit in large trials. In pregnancy, prone positioning is not feasible.

The approach in pregnant patients is similar to the nonpregnant population with ARDS. Ensuring adequate oxygenation and allowing hypercapnia to prevent ventilator-induced lung injury are goals. Pregnant women have been excluded from trials of ARDS. Few obstetric patients are described with ARDS [1,3]. In two series addressing outcome of ARDS, mortality was 25% and 39% [40,41]; these values reflect the range of mortality in nonpregnant patients.

---

## Sepsis

---

Sepsis is an uncommon complication during pregnancy or the postpartum period. The most common site of infection among critically ill obstetric patients is the urinary tract. Obstetric causes include chorioamnionitis, endometritis following cesarean delivery, and infection of the episiotomy site [6]. Necrotizing fasciitis can complicate the obstetric wound. Specific organisms that must be covered include the *Enterobacteriaceae* (specifically *Klebsiella* and *Escherichia coli*), *Streptococcus* (including toxin-producing strains), and anaerobes.

The management of sepsis is the same as in the nonpregnant individual and depends on appropriate antibiotics, source control, volume resuscitation, and hemodynamic support. Activated protein C, a circulating anticoagulant with anti-inflammatory properties, was shown to reduce mortality rate in patients with severe sepsis with high Acute

Physiology and Chronic Health Evaluation II score and high risk of death [42]. This has not been studied specifically in pregnancy, but 2 case reports describe the administration of activated protein C during pregnancy [43,44].

---

## Patients With Diabetes Mellitus

---

Diabetes mellitus may complicate pregnancy. Among pregnancies in women with diabetes, diabetic ketoacidosis is reported to occur in 1% to 3% and most often in patients without a prior diagnosis of diabetes [45]. It is rarely reported as a reason for ICU admission. In 1 series of 174 obstetric admissions, diabetes mellitus was the medical disorder in 2.3% [4]. Among 74 critically ill obstetric patients, diabetic ketoacidosis was the admission diagnosis in 3 patients (4%) [3]. Management with intravenous fluids and insulin is analogous to that of nonpregnant patients.

Current critical care practice has included strict glycemic control in critically ill patients, particularly patients with sepsis [46]. The goals of tighter control may be especially prudent in obstetric patients.

---

## Does Delivery Improve Maternal Condition in Respiratory Failure?

---

Several issues must be considered in the management of respiratory failure in the pregnant patient. The first priority is oxygenation and ventilatory support of the mother. The critically ill obstetric patient may develop spontaneous labor in the ICU. Evidence of fetal distress on fetal heart monitoring may include bradycardia, lack of heart rate variability, or late decelerations with contractions that indicate fetal hypoxia or acidosis [47]. The fetus is exposed to many medications; however, medications necessary for life-saving treatment of the mother should be administered. In managing the ventilated patient, sedatives such as opiates and propofol, and rarely neuromuscular blocking agents, are administered. These drugs cross the placenta, and it must be anticipated if delivery occurs, the neonate will be sedated (and possibly paralyzed) and will require respiratory support.

In managing the pregnant patient with respiratory failure, intensivists and obstetricians confront the question of whether delivery—when the patient is not in labor—will improve the maternal condition. In specific disorders such as preeclampsia, amniotic fluid embolism, and placental abruption, delivery is indicated.

In patients with respiratory failure attributable to pneumonia or ARDS, for example, the answer is not established. One would not expect delivery to lead to immediate recovery of respiratory failure attributable to ARDS. The obstetric literature addresses this question in case series of intubated pregnant patients. Tomlinson et al<sup>48</sup> described 10 intubated pregnant patients; 7 were delivered vaginally and 3 underwent cesarean delivery. Postdelivery, there was a decrease in FiO<sub>2</sub> but no other changes in ventilator parameters. The average duration of ventilation was 7 days, 2.6 days after delivery. Three of the 10 patients died. In another series of mechanically ventilated obstetric patients, 37 intubated patients who delivered during the course of respiratory failure were identified [49]. Eleven of 24 cesarean deliveries were for "maternal condition." Of the 7 maternal deaths, 5 had cesarean deliveries and 4 were performed for expected improvement in maternal condition [49]. In addition, in a series that included 5 intubated pregnant patients, the maternal condition was believed to have improved in only 1 patient who was extubated 2 days postdelivery [50]. Authors express caution in proceeding with elective delivery in the hope of improving the maternal condition [49].

---

## Cardiopulmonary Arrest

---

Cardiopulmonary arrest is an uncommon event during pregnancy [51]. Causes of cardiopulmonary arrest include venous thromboembolism, pregnancy-induced hypertension, sepsis, amniotic fluid embolism, hemorrhage, trauma, and cardiac disease. Pregnant patients may have preexisting cardiac disorders, myocardial infarction, or peripartum cardiomyopathy as causes of cardiopulmonary arrest [52].

During cardiopulmonary resuscitation (CPR), blood flow is less than 30% of normal blood flow. Circulation during CPR requires achieving adequate cardiac compression with elevation in intrathoracic pressure and adequate venous return from the systemic veins and pulmonary circulation. Physiologic and anatomic changes in pregnancy affect the circulation during CPR [53]. In late pregnancy, the gravid uterus may completely obstruct the inferior vena cava. Hypotension is thus initially treated with positioning in the left lateral decubitus position. Respiratory compromise may be precipitous because of the increase in oxygen consumption and the decrease in reserve. The upward displacement of the diaphragm alters chest wall compliance and position of intrathoracic organs.

When CPR is performed in pregnancy, the airway must be achieved immediately. Inferior vena caval compression must be relieved. Chest compressions are not effective in the lateral position; a 30° elevation of the right hip is the optimal position [53]. No changes in hand position or defibrillation energy have been recommended for pregnant patients [52]. Medications are given in the standard fashion; although epinephrine causes uteroplacental vasoconstriction, it is given according to the Advanced Cardiac Life Support (ACLS) protocol. Concerns have been raised that bicarbonate leads to fetal acidosis; however, bicarbonate is no longer standard in the ACLS protocol because of experimental findings of worsening intracellular acidosis.

The most important intervention during CPR in a patient with late pregnancy is cesarean delivery [52,53]. The term "4-minute rule" has been coined to indicate that a perimortem cesarean delivery should be initiated within 4 minutes of cardiopulmonary arrest. Delivery will immediately relieve the uterine compression and improve venous return and allow more effective chest compressions. A review of 61 cases of perimortem cesarean delivery by Katz et al<sup>54</sup> revealed that fetal survival was 70% if delivery occurred within 5 minutes of arrest. Initiating the delivery within 4 minutes and accomplishing delivery within 5 minutes can provide the possibility of maternal code survival and opportunity for fetal survival. Cases are reported of maternal survival after 25 to 30 minutes of CPR with return of circulation immediately after delivery [55-57]. CPR must be continued during and following the delivery. There is uncertain benefit with a smaller fetus, and perimortem cesarean delivery is not recommended in a pregnancy of less than 24 weeks gestation.

---

## Conclusion

---

The management of the obstetric patient in the ICU is especially difficult and stressful for the patient, family, and clinicians. The presence in the ICU of a young patient with a viable pregnancy or postpartum with a neonate, who suffered a drastic, unanticipated illness, demands many skills. Obstetric nurses can offer valuable guidance and support to the patient and family. Communication is vital to prepare and inform the patient and family about the course of ICU. Anticipation of a poor outcome for mother or fetus can prompt consultations for additional support systems and possibly bereavement services.

The critical care clinician must be knowledgeable about the physiologic changes and distinctive



illnesses and complications in the critically ill obstetric patient. Optimal management requires the close collaboration of intensivists and obstetricians to review changes in maternal condition and modify care plans throughout each ICU day. For the patient near term, the ICU must be prepared for spontaneous delivery or perimortem delivery. Maternal cardiac arrest necessitates immediate perimortem cesarean delivery to improve the possibility of maternal and fetal survival.

## References

- Mahutte NG, Murphy-Kaulbeck L, Le Q, et al. Obstetric admissions to the intensive care unit. *Obstet Gynecol.* 1999; 94: 263-266.
- Hazelgrove JF, Price C, Pappachan VJ, et al. Multicenter study of obstetric admissions to 14 intensive care units in southern England. *Crit Care Med.* 2001;29:770-775.
- Afessa B, Green B, Delke I, et al. Systemic inflammatory response syndrome, organ failure, and outcome in critically ill obstetric patients treated in an ICU. *Chest.* 2001;120: 1271-1277.
- Munnur U, Karnad DR, Bandi VDP, et al. Critically ill obstetric patients in an American and an Indian public hospital: comparison of case-mix, organ dysfunction, intensive care requirements, and outcomes. *Intensive Care Med.* 2005;31: 1087-1094.
- Chesnett AN. Physiology of normal pregnancy. *Crit Care Clin.* 2004;20:609-615.
- Lapinsky SE, Kruczynski K, Slutsky AS. Critical care in the pregnant patient. *Am J Respir Crit Care Med.* 1995;152: 427-455.
- Clark SL, Haney AF, eds. *Danforth's Obstetrics and Gynecology.* 9th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2003:461-475.
- Henry CS, Biedermann SA, Campbell MF, et al. Spectrum of hypertensive emergencies in pregnancy. *Crit Care Clin.* 2004;20:697-712.
- Lain KY, Roberts JM. Contemporary concepts of the pathogenesis and management of preeclampsia. *JAMA.* 2002;287: 3183-3186.
- Stone JH. HELLP syndrome: hemolysis, elevated liver enzymes, and low platelets. *JAMA.* 1998;280:559-562.
- Sibai BM. Diagnosis, controversies, and management of the syndrome of hemolysis, elevated liver enzymes, and low platelet count. *Obstet Gynecol.* 2004;103:981-991.
- Martin JN, Thigpen BD, Rose CH, et al. Maternal benefit of high-dose corticosteroid therapy for HELLP syndrome. *Am J Obstet Gynecol.* 2003;189:830-834.
- Eser B, Guven M, Unal A, et al. The role of plasma exchange in HELLP syndrome. *Clin Appl Thromb Hemost.* 2005;11:211-217.
- Shapiro JM. Venous thromboembolism in pregnancy. *J Intensive Care Med.* 2001;16:22-28.
- Stein PD, Kayali F, Olson RE, et al. Venous thromboembolism in pregnancy: 21-year trends. *Am J Med.* 2004;117:121-125.
- Chan WS, Ray JG, Murray S, et al. Suspected pulmonary embolism in pregnancy. *Arch Intern Med.* 2002;162: 1170-1175.
- Winer-Muram HT, Boone JM, Brown HL, et al. Pulmonary embolism in pregnant patients: fetal radiation dose with helical CT. *Radiology.* 2002;224:487-492.
- Schuster ME, Fishman JE, Copeland JF, et al. Pulmonary embolism in pregnant patients: a survey of practices and policies for CT pulmonary angiography. *AJR Am J Roentgenol.* 2003;181:1495-1498.
- Doyle NM, Ramirez MM, Mastrobattista JM. Diagnosis of pulmonary embolism: a cost-effectiveness analysis. *Am J Obstet Gynecol.* 2004;191:1019-1023.
- Wood KE. Major pulmonary embolism. Review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. *Chest.* 2002;121: 877-905.
- Bates SM, Greer IA, Hirsh J, et al. Use of antithrombotic agents during pregnancy. The seventh ACCP conference on antithrombotic and thrombolytic therapy. *Chest.* 2004;126: 627S-644S.
- Buller HR, Agnelli G, Hull RD, et al. Antithrombotic therapy for venous thromboembolic disease. The seventh ACCP conference on antithrombotic and thrombolytic therapy. *Chest.* 2004;126:401S-428S.
- Turrentine MA, Braems G, Ramirez MM. Use of thrombolytics for the treatment of thromboembolic disease during pregnancy. *Obstet Gynecol Surv.* 1995;50:534-541.
- Shapiro JM. Thrombolytic therapy of massive pulmonary embolism following cesarean delivery. *Clin Intensive Care.* 2001;12:1-4.
- Aurangzeb I, George L, Raoof S. Amniotic fluid embolism. *Crit Care Clin.* 2004;20:643-650.
- Clark SL, Hankins GDV, Dudley DA, et al. Amniotic fluid embolism: analysis of the national registry. *Am J Obstet Gynecol.* 1995;172:1158-1169.
- Stanten RD, Iverson LIG, Daugharty TM, et al. Amniotic fluid embolism causing catastrophic pulmonary vasoconstriction: diagnosis by transesophageal echocardiogram and treatment by cardiopulmonary bypass. *Obstet Gynecol.* 2003;102: 496-498.
- Oi H, Kobayashi H, Hirashima Y, et al. Serological and immunohistochemical diagnosis of amniotic fluid embolism. *Sem Thromb Hemost.* 1998;24:479-484.
- Goldszmidt E, Davies S. Two cases of hemorrhage secondary to amniotic fluid embolus managed with uterine artery embolization. *Can J Anesth.* 2003;50:917-921.
- Lim Y, Loo CC, Chia V, et al. Recombinant factor VIIa after amniotic fluid embolism and disseminated intravascular coagulopathy. *Int J Gynecol Obstet.* 2004;87:178-179.
- Rigby FB, Pastorek JG. Pneumonia during pregnancy. *Clin Obstet Gynecol.* 1996;39:107-119.
- Bridges CB, Harper SA, Fukuda K, et al. Prevention and control of influenza. *MMWR Morbid Mortal Wkly Rep.* 2003;52:1-36.
- Albino JA, Shapiro JM. Respiratory failure in pregnancy due to *Pneumocystis carinii*: report of a successful outcome. *Obstet Gynecol.* 1994;83:823-824.
- Guy ES, Kirumaki A, Hanania NA. Acute asthma in pregnancy. *Crit Care Clin.* 2004;20:731-745.
- Shapiro JM, McLeroth P. Status asthmaticus: a large MICU experience. *Clin Intensive Care.* 2002;13:89-93.
- Shapiro JM. Management of respiratory failure in status asthmaticus. *Am J Respir Med.* 2002;1:409-416.
- Ivanovic AD, Elam JO, Huffman J. Effect of maternal hypercarbia on the newborn infant. *Am J Obstet Gynecol.* 1970;107: 939-946.
- Ware LB, Matthay MA. The acute respiratory distress syndrome. *N Engl J Med.* 2000;342:1334-1349.
- The Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med.* 2000;342: 1301-1308.
- Perry KG, Martin RW, Blake PG, et al. Maternal mortality associated with adult respiratory distress syndrome. *South Med J.* 1998;91:441-444.
- Catanzarite V, Willms D, Wong D, et al. Acute respiratory distress syndrome in pregnancy and the puerperium: causes, courses, and outcomes. *Obstet Gynecol.* 2001; 97:760-764.

42. Bernard GR, Vincent JL, Lateve PF, et al. Efficacy and safety of recombinant human activated protein C for severe sepsis. *N Engl J Med*. 2001;344:699-709.
43. Medve L, Csitari IK, Molnar Z, et al. Recombinant human activated protein C treatment of septic shock syndrome in a patient at 18<sup>th</sup> week of gestation: a case report. *Am J Obstet Gynecol*. 2005;193:864-865.
44. Mikaszewska-Sokolewicz M, Mayzner-Zawadzka E. Use of recombinant human activated protein C in treatment of severe sepsis in a pregnant patient with fully symptomatic ovarian hyperstimulation syndrome. *Med Sci Monit*. 2005; 11: CS27-CS32.
45. Ramin KD. Diabetic ketoacidosis in pregnancy. *Obstet Gynecol Clin North Am*. 1999;26:481-488.
46. Dellinger RP, Carlet JM, Masur H, et al. Surviving sepsis campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med*. 2004;32:858-871.
47. Cunningham FG, Gant NF, Leveno KJ, et al. Intrapartum assessment. In: Cunningham FG, Gant NF, Leveno KJ, et al, eds. *William's Obstetrics*. 21st ed. New York, NY: McGraw-Hill; 2001:331-360.
48. Tomlinson MW, Caruthers TJ, Whitty JE, et al. Does delivery improve maternal condition in the respiratory-compromised gravida? *Obstet Gynecol*. 1998;91:108-111.
49. Jenkins TM, Troiano NH, Graves CR, et al. Mechanical ventilation in an obstetric population: characteristics and delivery rates. *Am J Obstet Gynecol*. 2003;188:549-552.
50. Collop NA, Sahn SA. Critical illness in pregnancy. *Chest*. 1993;103:1548-1552.
51. Morris S, Stacey M. Resuscitation in pregnancy. *BMJ*. 2003; 327:1277-1279.
52. Whitty JE. Maternal cardiac arrest in pregnancy. *Clin Obstet Gynecol*. 2002;45:377-392.
53. Dildy GA, Clark SL. Cardiac arrest during pregnancy. *Obstet Gynecol Clin North Am*. 1995;2:303-314.
54. Katz VL, Dotters DJ, Droegemueller W. Perimortem cesarean delivery. *Obstet Gynecol*. 1986;68:571-576.
55. O'Connor RL, Sevarino FB. Cardiopulmonary arrest in the pregnant patient: a report of a successful resuscitation. *J Clin Anesth*. 1994;6:66-68.
56. Lanoix R, Akkapeddi V, Goldfeder B. Perimortem cesarean section: case reports and recommendations. *Acad Emerg Med*. 1995;2:1063-1067.
57. Lurie S, Mamet Y. Cesarean delivery during maternal cardiopulmonary resuscitation for status asthmaticus. *Emerg Med*. 2003;20:296-297.