

# Acute Pulmonary Edema in Pregnancy

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Pulmonary edema is a secondary disease process characterized by an excess accumulation of fluid in the pulmonary interstitial and alveolar spaces, preventing adequate diffusion of both oxygen and carbon dioxide. The excess fluid accumulation interferes with maternal oxygenation and, if not identified and corrected, can lead to maternal and fetal hypoxemia. The purpose of this article is to review principles of oxygen transport and the pathophysiology, diagnosis, management, and nursing implications of acute pulmonary edema in pregnancy. **Key words:** acute respiratory dysfunction, HELLP syndrome, maternal complications, multiorgan dysfunction syndrome, oxygenation, preeclampsia, pregnancy, pulmonary edema, tocolytic-induced complications

**P**ULMONARY EDEMA is a secondary disease process characterized by an excess accumulation of fluid in the pulmonary interstitial and alveolar spaces, preventing adequate diffusion of both oxygen and carbon dioxide.<sup>1,2</sup> The development of pulmonary edema quickly leads to pulmonary dysfunction and is potentially life-threatening for the woman and her fetus.<sup>3</sup>

During pregnancy, pulmonary edema is commonly associated with preeclampsia, pre-existing cardiac disease, tocolytic therapy, infection, and fluid and blood replacement therapy.<sup>1-12</sup> Discontinuation of the inciting medication usually resolves tocolytic-induced pulmonary edema. However, when acute pulmonary dysfunction presents as a comorbid

process in the patient with underlying disease or preeclampsia, pulmonary edema is associated with substantial perinatal mortality.<sup>5</sup>

Acute pulmonary dysfunction can quickly produce a state of maternal hypoxemia. Maternal hypoxemia, in turn, may lead to fetal hypoxemia. Clinical situations where maternal hypoxemia presents a challenge to care providers for fetal management include, but are not limited to, acute respiratory distress, pulmonary edema with or without preeclampsia, and pulmonary edema from underlying cardiac decompensation (eg, valvular disease). Fetal and neonatal outcomes are dependent on the underlying maternal disease process that precipitated the development of pulmonary edema and on maternal status. Recent mortality data indicate a significant increase in maternal mortality when medical conditions, including pulmonary edema, complicate the pregnancy.<sup>13</sup> Of all maternal deaths from medical conditions, 34% were from cardiovascular problems and 11% were related to pulmonary complications.<sup>13</sup> The purpose of this article is to review principles of oxygen transport and the pathophysiology, diagnosis, management, and nursing implications of pulmonary edema in pregnancy.

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## PHYSIOLOGIC ADAPTATION OF THE MATERNAL PULMONARY SYSTEM

### Cardiopulmonary adaptations

Pregnancy is a dynamic physiologic state resulting in dramatic mechanical and biochemical changes in maternal cardiovascular and pulmonary systems affecting respiratory function and gas exchange. These physiologic adaptations and increased metabolic demands of pregnancy may increase the risk of acute pulmonary edema.

Maternal blood volume increases by approximately 45% to 50% by 32 weeks of gestation, with plasma volume expansion accounting for a greater proportion of the increase.<sup>14-16</sup> This increased plasma volume produces a hypervolemic, dilutional effect on serum albumin, fibrinogen, and globulins, thereby decreasing serum colloid osmotic pressure (COP).<sup>15,17</sup> COP is the pressure resulting from the effect of plasma proteins (ie, albumin, fibrinogen, and globulins) to hold water in the intravascular space.<sup>17,18</sup> This decrease in intravascular COP during pregnancy may predispose a pregnant woman to the development of pulmonary edema. See Table 1 for a summary of the cardiopulmonary adaptations of pregnancy. See Figure 1 for an illustration of normal Starling's forces within the pulmonary bed.

### Oxygenation

The physiologic adaptations of the cardiopulmonary system during pregnancy enable a woman to optimize oxygen delivery to maternal tissues, the uteroplacental bed, and the fetus. Although plasma volume expansion results in a dilutional reduction in hemoglobin concentration and arterial oxygen content, maternal oxygen delivery remains at or above normal levels because of a 50% increase in cardiac output.<sup>14,16,21</sup> This adaptation makes the woman more dependent on cardiac output for maintenance of oxygen delivery during pregnancy as compared to the nonpregnant woman.<sup>21,22</sup>

Oxygen consumption increases steadily throughout the pregnancy and increases further during labor.<sup>23</sup> Normally oxygen delivery to the tissues exceeds consumption, so a woman during pregnancy is able to meet oxygen needs for herself and her fetus during pregnancy and labor.<sup>16</sup> However, when oxygen delivery is insufficient to meet metabolic demands, the fraction of delivered oxygen actually consumed, or the oxygen extraction ratio, increases. A normal oxygen extraction ratio is approximately 25%; the extraction ratio increases as a compensatory mechanism to meet increasing oxygen needs.<sup>16,21</sup> If the maternal cardiopulmonary system becomes compromised, thereby reducing oxygen supply, oxygen extraction from the blood increases to maintain oxygen consumption. As oxygen delivery decreases, oxygen extraction ratios continue to rise until the limits of oxygen extraction are reached; tissues are unable to maintain aerobic energy production, and oxygen consumption decreases.<sup>16,21</sup> At this point, the individual is in an oxygen-dependent state, having reached critical oxygen delivery.<sup>15,16,24</sup> If a pregnant woman's oxygen delivery decreases, she will quickly reach critical oxygen delivery, compromising both herself and her fetus.<sup>15</sup> Since fetal oxygenation is dependent on maternal oxygenation, it is critical to carefully assess and monitor maternal cardiopulmonary function for signs of decreased oxygen delivery.

### Acid-base status

Physiologic adaptations of the maternal cardiopulmonary and renal systems induce a state of chronic compensated respiratory alkalemia.<sup>14,25</sup> Alveolar and arterial  $PCO_2$  levels decrease because of an increased minute ventilation. Bicarbonate excretion by the renal system increases during pregnancy to compensate for the lowered  $PCO_2$ .<sup>14,15</sup> Since fetal excretion of carbon dioxide is dependent on the maternal respiratory system, the decrease in maternal  $PCO_2$  allows the fetus to offload carbon dioxide at the uteroplacental level when maternal acid-base status is

**Table 1.** Maternal physiologic adaptations\*

System	Increased	Decreased	Clinical significance
Cardiovascular	Blood volume (plasma > RBC) Cardiac output Stroke volume Heart rate Uterine blood flow Myocardial hypertrophy	Systemic vascular resistance Pulmonary vascular resistance Blood pressure Mean arterial pressure Colloid osmotic pressure	Increased risk for pulmonary edema Dilutional anemia is common with decreases seen in hemoglobin and hematocrit concentration May unmask underlying cardiovascular disease Changes may exacerbate decompensation from chronic underlying disease process
Pulmonary	State of compensatory respiratory alkalemia with increased pH, PO <sub>2</sub> Oxygen consumption Respiratory rate, minute ventilation Tidal volume, inspiratory reserve volume, inspiratory capacity Airway conductance (dilatation of larger airways below larynx) Elevation of diaphragm Thoracic cage circumference; anteroposterior and transverse diameters Capillary engorgement of nasal and oropharyngeal mucosae and larynx	Functional residual capacity, expiratory reserve volume, residual volume Internal diameter of trachea reduced Total lung capacity (vital capacity unchanged) PCO <sub>2</sub> , HCO <sub>3</sub> Oxygen reserve	Hypermetabolic Diminished buffering capacity results in rapid development of hypoxemia and acidemia during periods of hypoventilation and apnea Airway edematous and tissue more friable, which predisposes to intubation difficulties; need smaller endotracheal tube Surgical airway equipment readily available; avoid nasotracheal intubation Hypoventilation possible secondary to parenteral opioid analgesia Second-stage labor, especially with closed glottis, results in increased oxygen consumption and lactate concentration Changes may exacerbate decompensation from chronic underlying disease process

\* From Blackburn,<sup>14</sup> Norwitz et al,<sup>15</sup> and Whitty and Dombrowski.<sup>16</sup>

normal.<sup>14,15</sup> See Table 2 for arterial blood gas values in pregnancy.

#### Fetal oxygenation

Delivery of oxygen to the fetus is dependent on the maternal cardiopulmonary

system's ability to meet the metabolic and oxygenation needs of both herself and her fetus. Fetal oxygen delivery depends on multiple factors, including, but not limited to, uteroplacental blood flow, differences in maternal and fetal partial pressures of oxygen,

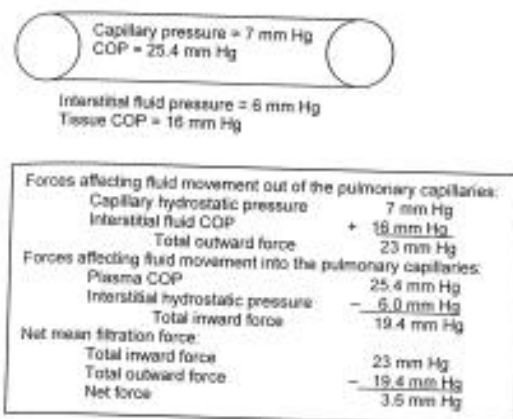


Figure 1. Colloid oncotic pressures and fluid movement: Starling forces in normal lung. From Darovic,<sup>18</sup> Clark et al,<sup>19</sup> and Guyton.<sup>20</sup> COP indicates colloid osmotic pressure.

maternal oxygen-carrying capacity, and placental surface area.<sup>14,27</sup>

To facilitate fetal oxygen delivery/carbon dioxide excretion, the maternal oxyhemoglobin dissociation curve shifts to the right, increasing release of oxygen from maternal hemoglobin at the uteroplacental bed. Fetal hemoglobin releases carbon dioxide to bind

with oxygen. As the fetus releases carbon dioxide, fetal pH rises, shifting the fetal oxyhemoglobin curve to the left. This shifting of the oxyhemoglobin dissociation curve increases the affinity of fetal hemoglobin for oxygen. Within the placental vascular bed, as the distance between the maternal and fetal oxyhemoglobin dissociation curves increases, oxygen transfers rapidly from maternal circulation to fetal circulation.<sup>14,27</sup>

Maternal hypoxemia, especially when  $PO_2$  is below 60 mm Hg, shifts the maternal oxyhemoglobin curve to the left toward the fetal curve, decreasing oxygen available for the fetus.<sup>14,28</sup> Maternal hyperventilation also shifts the maternal curve to the left, decreasing release of oxygen from maternal hemoglobin, which in turn decreases fetal oxygenation.<sup>14</sup>

#### PATHOPHYSIOLOGY OF PULMONARY EDEMA

The most simplistic description of the pathophysiologic mechanism for pulmonary edema defines cardiogenic or noncardiogenic factors. However, the underlying disease process is more complex. A contemporary classification scheme defines the type of pulmonary edema by the mechanisms that produce increased fluid movement across the pulmonary capillary membrane.<sup>18</sup> This movement of fluid results from an imbalance of hydrostatic and protein osmotic pressures, increased endothelial permeability, lymphatic insufficiency, and unknown or poorly understood mechanisms<sup>17,18</sup> (see Figures 1 and 2). The underlying mechanisms, while discussed as isolated events, may overlap or be multicausal.

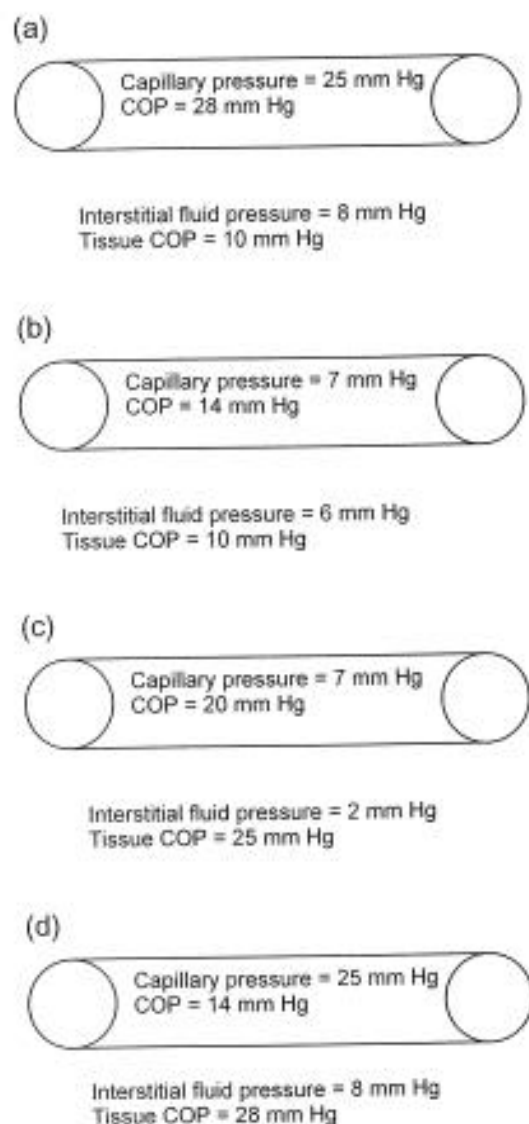
#### Hydrostatic pulmonary edema

Hydrostatic, or high-pressure, pulmonary edema results from an imbalance of intravascular volume and intravascular and interstitial pressures. The underlying mechanisms that lead to the development of

Table 2. Arterial blood gas values in pregnancy\*

Parameter	Reference range
pH	7.40-7.46
$PCO_2$	26-32 mm Hg
$PO_2$	106-108 mm Hg (sea level) 101-104 mm Hg (third trimester) -13 mm Hg (supine position in third trimester)
$HCO_3^-$	18-22 mm Hg
Base deficit	-3 to -4 mEq/L
$P(A-a)O_2$	20 mm Hg +6 mm Hg (supine position in third trimester)

\*From Blackburn,<sup>14</sup> Norwitz et al,<sup>15</sup> and Gei and Suarez.<sup>20</sup>



**Figure 2.** Underlying mechanisms for pulmonary edema: (a) increased capillary hydrostatic pressure; (b) low plasma colloid osmotic pressure; (c) increased capillary permeability; (d) obstruction of pulmonary lymphatics. From Darovic,<sup>18</sup> Clark et al,<sup>19</sup> and Guyton.<sup>20</sup> COP indicates colloid osmotic pressure.

hydrostatic pulmonary edema include cardiogenic causes, decreased COP, and increased negative interstitial pressure.<sup>17</sup> Cardiogenic causes and decreased COP are the more common mechanisms for hydrostatic pulmonary edema in pregnancy and are discussed in this

article.<sup>17,18</sup> Negative interstitial pressure from such causes as acute airway obstruction or rapid reexpansion of a pneumothorax may occur during pregnancy, but these are the least common mechanisms involved in the development of pulmonary edema.

### **Cardiogenic pulmonary edema**

Cardiogenic pulmonary edema occurs when the cardiac muscle is in a state of dysfunction. This dysfunction may be systolic dysfunction (decreased myocardial squeeze, ejection fraction <45%), diastolic dysfunction (impaired ventricular muscle relaxation resulting in high filling pressures), or valvular disease.<sup>17</sup> One of the major causes of pulmonary edema in pregnancy and congestive heart failure is systolic dysfunction.<sup>29,30</sup>

Systolic dysfunction occurs with failure of either the left heart (pulmonary edema) or right heart (congestive heart failure). Left ventricular failure, or backward failure, occurs when there is an accumulation of excess fluid behind the failing left ventricle secondary to inadequate ventricular emptying. As excess fluid accumulates, left ventricular end-diastolic pressure, pulmonary artery wedge pressure, and pulmonary artery pressures increase, causing a redistribution of intravascular volume from systemic circulation to the pulmonary circulatory circuit.<sup>17,18</sup> Simply put, hydrostatic pressure increases within the pulmonary capillary bed, pushing fluid out of the capillaries into lung tissue.

If the systolic dysfunction results from a forward failure, there is an inadequate discharge of blood into the arterial system. Whereas backward failure results in pulmonary congestion, forward failure decreases systemic perfusion.<sup>31</sup> With left ventricular forward failure, cardiac output decreases and blood pressure falls. This decrease in cardiac output causes a reduction in effective blood flow through the renal vascular system, with a compensatory increased renin, angiotensin, and aldosterone production. Renal secretion of salt and water decreases and systemic vascular resistance increases. Right ventricular forward failure results in an accumulation of

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excess blood in the right ventricle and a left interventricular septal shift compromising left ventricular capacity, with a resulting decrease in stroke volume. Ultimately, right ventricular forward failure causes an increase in left ventricular filling pressure. This in turn results in a decreased left preload, leading to a decreased left ventricular output and systemic blood pressure.<sup>17,18,31,32</sup>

While diagnostic procedures can identify an underlying mechanism for cardiogenic pulmonary edema, the ventricular muscles are interdependent. Once one ventricle fails, the other will also fail.

The third mechanism leading to cardiogenic pulmonary edema is valvular heart disease. In pregnancy, rheumatic mitral stenosis is the most common valvular lesion resulting in pulmonary edema. Hypervolemia of pregnancy coupled with an increase in maternal heart rate leads to decreasing ventricular filling time and allows for an accumulation of excess blood volume, or left preload.<sup>17</sup> Maternal tachycardia, intravascular fluid overload, decreased cardiac output states, or conditions leading to increased pulmonary vascular resistance will further impede left ventricular filling, leading to pulmonary congestion and edema. During active labor, the nurse must remember that cardiac output will increase 20% to 30% above normal pregnancy resting values, further increasing the risk of pulmonary edema.<sup>10,14,17,33</sup>

Women with valvular lesions are at an increased risk for postpartum pulmonary edema. Following childbirth, the blood that perfused the uteroplacental bed now returns to central circulation as an autotransfusion. Maternal cardiac output increases further by an additional 10% to 20% in the immediate postpartum period.<sup>10,14,17,33</sup> This autotransfusion increases the pulmonary artery wedge pressure, causing redistribution of the blood volume into the pulmonary circulation, leading to the development of pulmonary congestion. Other hemodynamic changes that may exacerbate normal postpartum pulmonary congestion and contribute to the development of pulmonary edema in-

clude the presence of a relative bradycardia and a decrease in myocardial contractility. These changes may persist for up to 2 weeks postpartum.<sup>10,14,17,33</sup>

#### **Decreased colloid osmotic pressure**

As previously mentioned, COP is the pressure resulting from the ability of plasma proteins (eg, albumin, fibrinogen, and globulins) to hold water in the intravascular space.<sup>17,18</sup> Intravascular COP opposes hydrostatic pressure and interstitial COP, which tend to pull water from the intravascular bed. The decrease in intravascular COP during pregnancy may predispose the woman to the development of pulmonary edema. Blood loss and crystalloid fluid replacement will further reduce the woman's intravascular COP. Women diagnosed with preeclampsia also demonstrate a lower intravascular COP, secondary to endothelial permeability and proteinuria. Decreased COP is rarely an independent cause of pulmonary edema; however, it can exacerbate the edema that occurs from another precipitating event.<sup>17,34</sup> See Table 3 for COP values in pregnancy.

#### **Permeability pulmonary edema**

Permeability pulmonary edema is a severe form of acute respiratory failure that occurs when the tight junctions between the endothelial cells open, allowing passage of water, proteins, and cells into the interstitial and alveolar spaces.<sup>17,18</sup> The degree of pulmonary insult ranges from acute lung injury to the development of acute respiratory distress syndrome.<sup>17,18,35</sup> "Acute lung injury (ALI) is clinically characterized by an intense inflammatory response and fibrosis of lung tissue to infectious or noninfectious insults. Pathophysiologic and clinical characteristics include diffuse alveolar damage, increased permeability of the pulmonary membrane that allows a leak of protein-rich fluid into the interstitial and alveolar spaces, hypoxemia that is poorly responsive to oxygen supplementation, increasing dyspnea and tachypnea, and a progressive decrease in lung compliance."<sup>18(p457)</sup>

**Table 3.** Colloid osmotic pressure in pregnancy\*

Variable		Colloid osmotic pressure (mean $\pm$ SD), mm Hg
Normal pregnancy	Antepartum (at term)	22.4 $\pm$ 2.3
	Postpartum (first 24 h)	15.4 $\pm$ 2.1
Preeclampsia	Antepartum	17.9 $\pm$ 0.7
	Postpartum	13.7 $\pm$ 0.5
Betamimetic therapy	12 h post-intravenous therapy (ritodrine)	14.3 $\pm$ 1.7

\*From Clark et al.<sup>19</sup>

Acute respiratory distress syndrome is the end stage of acute lung injury.

Clinical presentations for hydrostatic and permeability pulmonary edema are similar. However, management and clinical courses differ. Hydrostatic pulmonary edema responds quickly to aggressive diuresis. Permeability pulmonary edema takes days to weeks to clear.

#### Other mechanisms

The last category used to describe mechanisms of pulmonary edema includes disease processes in which the development of pulmonary edema is either unknown or poorly understood. Unfortunately, this category includes many pregnancy-related cases of pulmonary edema, including tocolytic-induced and preeclampsia.

Tocolytic-induced pulmonary edema is associated with multifetal gestations, maternal anemia, low maternal weight, use of intravenous betamimetic therapy for longer than 24 hours, simultaneous use of 2 or more tocolytic agents, and corticosteroid therapy to accelerate fetal lung maturity. Proposed underlying mechanisms for the development of tocolytic-induced pulmonary edema include maternal heart disease, fluid overload, occult chorioamnionitis, hypokalemia, myocardial ischemia, mineralocorticoid effect of corticosteroids, catecholamine injury to the myocardium, and permeability edema.<sup>2,3,5-7,10-12,26,36-40</sup>

Pulmonary edema that occurs in women diagnosed with preeclampsia also falls into this category. This patient population frequently has multiple abnormalities present simultaneously, including increased capillary permeability secondary to endothelial dysfunction, hypoalbuminemia, and afterload-induced left ventricular dysfunction.<sup>1-3,7,9,10,13,14,17,26,37,41-43</sup>

#### DIAGNOSIS

Diagnosis of pulmonary edema is based on patient history, physical examination findings, laboratory data, and chest radiograph. Information obtained in the history includes, but is not limited to, the onset and duration of symptoms, precipitating factors, comorbidities (eg, anemia, underlying disease processes), current medications, and a review of risk factors (Table 4). Symptoms suggestive of evolving pulmonary edema include dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and decreased exercise tolerance. Physical examination findings include tachypnea, upright posture, air hunger, sweating, rales, use of accessory muscles of respiration, resting tachycardia, displaced point of maximal impulse, and presence of third heart sound, neck/jugular vein distention, hepatjugular reflux, hepatomegaly, jaundice, and peripheral edema.

Laboratory studies, including complete blood count, serum electrolyte values, and

**Table 4.** Risk factors for pulmonary edema in pregnancy

- Multiple gestation
- Preeclampsia
- HELLP syndrome
- Eclampsia
- Tocolytic therapy
- Cardiac dysfunction
- Blood replacement therapy
- Fluid overload
- Pneumonia
- Septic shock
- Aspiration

renal function studies (blood urea nitrogen, serum creatinine) should be immediately assessed for any woman experiencing acute pulmonary edema. Since pregnancy is also a hypercoagulable state, evaluation for a pulmonary embolus may also be indicated.

Chest radiography reveals bilateral air space disease, with prominence in the bases; perihilar infiltrates in a "bat-wing" distribution, redistribution of blood flow with prominent upper-lobe veins, or pleural effusions may be present. Interstitial edema is also present. Unfortunately, chest radiography does not differentiate between hydrostatic and permeability pulmonary edema.

Oxygenation status is monitored through arterial blood gas analysis or noninvasive pulse oximetry. Arterial blood gases are indicated to rule out acidosis and evaluate for carbon dioxide retention.

Cardiomegaly may be present on chest radiography. A 12-lead electrocardiogram will help determine the presence of chamber hypertrophy, ischemia, conduction defects, or dysrhythmias. Echocardiogram allows evaluation of cardiac structure and function and can be a useful diagnostic tool.

For the woman failing to respond to traditional therapy, a pulmonary artery (Swan-Ganz) catheter may be indicated to establish a differential diagnosis for the underlying mechanism causing the pulmonary edema.

Indications for a pulmonary artery catheter include tight mitral stenosis (valve area  $<1$  cm<sup>2</sup>), severe systolic dysfunction (ejection fraction  $<30\%$ ), a "white out" on chest X-ray, and for those patients who do not respond after 2 hours of aggressive diuresis with furosemide.<sup>17,44</sup> While a pulmonary artery catheter can provide information about specific hemodynamic parameters—including right preload (central venous pressure), left preload (pulmonary artery wedge pressure), cardiac output, mixed venous oxygen saturation (SvO<sub>2</sub>), and right ventricular ejection fraction—the use of this technology is not without risk and controversy. While the use of a pulmonary artery catheter has not demonstrated improved outcomes, the data generated may challenge traditional management plans, allowing for better understanding of the underlying causative mechanisms and improvement of patient care.<sup>17,45</sup>

## MANAGEMENT

The management of pulmonary edema focuses on removal of the underlying cause. Management options include pharmacologic and nonpharmacologic measures. Table 5 summarizes management options. Critical care management of pulmonary edema will not be discussed in this article.

### Positioning

Upright positioning is critical to management of pulmonary edema to help displace blood volume to the dependent parts of the body. When positioning the woman who is still pregnant, uterine displacement will further facilitate respiratory effort. Since central hemodynamic values are equivalent when comparing the left lateral position to sitting with uterine displacement, maternal cardiac output is maintained and respiratory function is optimized.<sup>46</sup>

### Oxygenation

Supplemental oxygen is administered by nasal cannula or a tight-fitting facemask to maintain a PaO<sub>2</sub> higher than 60 mm Hg and

**Table 5.** Summary of management for pulmonary edema in pregnancy

Pharmacologic interventions
Supplemental oxygen using a nonrebreather mask
Fluid and sodium restriction
Discontinuation of any tocolytics
Diuretic therapy will be initiated intravenously with furosemide (Lasix) to promote diuresis.
Electrolytes (especially potassium) should be supplemented as needed <sup>1</sup>
Reduction of preload and afterload by the use of vasodilators such as nitrates <sup>10</sup>
Intravenous morphine can relieve pain and anxiety, decrease the work of breathing, and decrease venous and arteriolar constriction <sup>62</sup>
Nonpharmacologic interventions
Continuous fetal heart tracing
Monitoring the patient's vital signs (including continuous monitoring of oxygen saturation using a pulse oximeter) and arterial blood gas monitoring
Foley catheter placement for accurate measurement of urinary output as well as preventing unnecessary physical exertion, and facilitating continuous EFM
Reposition the patient. Elevate the head of the bed at least 30° to prevent hypoventilation and facilitate deep breathing. This position improves ventilation by reducing pulmonary capillary wedge pressure. <sup>1</sup> Some patients may breathe easier in a sitting or semi-sitting position
Care should be taken to continue to enhance uteroplacental perfusion by wedging 1 hip to relieve any aortal-vena caval compression. This positioning may increase the difficulty of continuous electronic fetal monitoring. Before repositioning the mother to "keep the baby on the monitor," remember that increasing the mother's respiratory difficulty will further compromise fetal oxygenation, resulting in a nonreassuring tracing

If the patient does not respond rapidly to the above interventions, prepare to proceed with hemodynamic monitoring. Intubation and mechanical ventilation with positive pressure may be required in patients with hypoxia who are unresponsive to initial diuretics and supplemental oxygen. Noncardiogenic pulmonary edema that does not respond to the above measures may represent acute respiratory distress syndrome.<sup>10</sup>

an oxygen saturation ( $\text{SaO}_2$ ) of more than 90%.<sup>1,10,11,14,17,18,41</sup> Administration of oxygen by nasal cannula does not increase the flow rate to more than 4 L/min; higher rates do not improve the inhaled oxygen fraction and cause nasal irritation. If higher flow rates are required to maintain oxygenation, facemasks are required. Flow rates up to 15 mL/min can be used to administer oxygen by non-rebreathing facemasks. Continuous positive airway pressure may be maintained noninvasively by using a tight-fitting facemask or nasal mask. Continuous positive airway pressure is often used as a temporizing measure to maintain oxygenation until furosemide-induced diuresis reduces preload to clear the lungs. Advantages of continuous positive airway pressure include increased intra-alveolar pressure,

reduced transudation of fluid from alveolar capillaries, and impeded venous return to the thorax. If continuous positive airway pressure fails to maintain adequate oxygenation, mechanical ventilation may be indicated.

#### Diuretic therapy

Iatrogenic fluid overload from inadvertent intravenous therapy or aggressive fluid/blood replacement usually resolves quickly. The priorities for management include administration of diuretic therapy, fluid restriction, and supplemental oxygen therapy. For patients with preexisting cardiac disease, a sudden onset of pulmonary edema may indicate left ventricular failure, necessitating aggressive management.

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Diuretic therapy or preload reduction with furosemide (Lasix) helps relieve intravascular volume overload. Furosemide is a high-potency diuretic that inhibits reabsorption of sodium, water, and chloride from the ascending limb of the Loop of Henle; it also decreases reabsorption of sodium and water and increases potassium excretion in the distal renal tubule.<sup>47</sup> This drug causes a vasodilatory effect on the renal vascular system, increasing renal blood flow. Effective renal blood flow is increased during pregnancy, and coupled with the further increased flow secondary to the effects of furosemide; any drug received by the woman that is eliminated by renal excretion may have a decreased serum level and suboptimal therapeutic effect.<sup>47</sup>

Furosemide acts as an immediate venodilator, increasing venous capacitance, which further decreases preload. Dosing is titrated to patient response. Typical dosing is 20 to 80 mg, as a slow intravenous bolus administered over 2 to 3 minutes. In the absence of a diuretic response in 30 to 60 minutes, an additional dose may be administered. Additional dosing may be incremental, increased by 20 to 40 mg. The goal is to obtain approximately 2000 mL diuresis over several hours. The effectiveness of furosemide is generally evident by radiological clearing of the pulmonary edema. If the underlying mechanism for pulmonary edema is cardiogenic, preload reduction produces clinical improvement within minutes, before diuresis begins.<sup>17,18,47</sup>

#### Venodilator therapy

Morphine sulfate, in doses of 2 to 5 mg intravenously to a possible total of 15 mg, helps relieve anxiety and promote a sense of well-being, reduces the sympathetically induced arteriolar/venous constriction, and increases venous capacitance. This action reduces the preload, afterload, and workload of the heart, while increasing the stroke volume and cardiac output. Morphine therapy, furosemide, and oxygen supplementation may be the only interventions required in the obstetric patient.

While the goal is to reduce intravascular volume, diuretic and venodilator therapy is not without risks. Diuretic and venodilator therapy both decrease left ventricular filling pressure and stroke volume. Care should be taken to avoid hypotension and further worsening the imbalance of systemic and myocardial oxygen supply and demand.

#### Pulmonary edema with comorbidities

In the obstetric patient with severe hypertension or preeclampsia who develops pulmonary edema, management will include antihypertensive therapy. Traditional antihypertensive therapy with hydralazine, labetalol, or nicardipine will reduce afterload, improving cardiac performance. Additional antihypertensive agents such as sodium nitroprusside or nitroglycerine may be used in the critical care setting. Sodium nitroprusside, a potent balanced arterial and venous vasodilator, can provide minute-to-minute titration to control afterload. However, this drug is rarely used in obstetrics because of the risk for fetal cyanide and thiocyanate toxicity. In comparison, although nitroglycerine, which is primarily a venular vasodilator, crosses the placenta it appears safe for the fetus.

In the obstetric patient with systolic dysfunction complicated by pulmonary edema, management focuses on supportive therapy. Such management focuses first on afterload reduction and then inotropic therapy.

When first-line therapies fail to obtain the desired response, critical care management is required. The scope of critical care management will not be discussed in this article.

#### NURSING IMPLICATIONS

Nursing vigilance is a key component to possible prevention and early diagnosis of pulmonary edema. The development of pulmonary edema quickly leads to pulmonary dysfunction and becomes an obstetrical emergency that potentially threatens the life of the mother and the fetus.<sup>3</sup> Early diagnosis and intervention improve perinatal outcomes and may prevent more severe complications.

### Pulmonary function and gas exchange

Influencing care of the pregnant woman diagnosed with pulmonary edema are the principles of pulmonary function, oxygen transport, and gas exchange. Pregnancy is characterized by a state of chronic compensated respiratory alkalemia, which places the woman at greater risk for alterations in oxygen transport and gas exchange when pulmonary dysfunction occurs. Pulmonary edema results in reduced lung distensibility and alveolar edema. Alveolar edema prevents ventilation of the alveoli, which in turn prevents perfusion of the unventilated alveoli; this results in shunting and hypoxemia. The resulting hypoxemia causes pulmonary vasoconstriction, which decreases shunting but increases pulmonary vascular resistance. The increased pulmonary vascular resistance increases right afterload, increasing right ventricular workload. As right afterload and right ventricular workload increase, airway resistance is increased, causing more fluid to fill the airways.

Pulmonary function is more than just a respiratory rate. The respiratory function includes ventilation and oxygen-carbon dioxide exchange. Ventilation involves the process of inspiration and expiration. During inspiration, oxygen is obtained from an extracorporeal source (either the atmosphere or a ventilator) and transferred to the pulmonary bed, where oxygen transfers across the alveolar-endothelial barrier, where it binds with hemoglobin and is carried to the tissues. At the tissue level, hemoglobin releases oxygen and picks up carbon dioxide prior to returning to the alveolar-endothelial barrier in the pulmonary bed, with removal of carbon dioxide through expiration. Any process that interferes with inhalation or exhalation sets up a potential imbalance that can quickly lead to pulmonary dysfunction, decreased oxygen transport, and inefficient gas exchange.

### Maternal assessment

Maternal assessment will include assessment for risk factors as well as clinical find-

ings. See Table 4 for risk factors. These are to be assessed by review of prenatal and intrapartum records as well as patient interview. The most common etiologies include hypertension, multiple gestation, use of tocolytic agents, cardiac disease, fluid overload (colloid and/or blood products), infection, and preeclampsia.<sup>1,2,11</sup> Tocolytic-induced pulmonary edema is associated with current or recent use of  $\beta$ -adrenergic drugs, primarily  $\beta_2$ -agonists like terbutaline or ritodrine. The onset of symptoms can occur within as little as 24 hours of the initiation of tocolytic therapy; symptoms can occur within the first 12 hours postpartum.<sup>3</sup> Concurrent use of multiple tocolytics has been associated with the development of pulmonary edema. The most common combination of tocolytic agents leading to the development of pulmonary edema were subcutaneous terbutaline and intravenous magnesium sulfate.<sup>2</sup> Accurate intake and output records will alert the nurse to the possibility of fluid overload.

In a sample of women with preeclampsia, pulmonary edema was found to have developed in 62%; onset of symptoms can occur more than 72 hours postpartum.<sup>3</sup> For most women, this may well be after hospital discharge. It is important to note that the likelihood for pulmonary edema increases with the addition of each risk factor (eg, tocolytic usage *and* multiple gestations). The absence of these risk factors is not blind reassurance, however. In a group of 51 women diagnosed with pulmonary edema during pregnancy or in the postpartum period, 6 of the 13 with cardiac disease had previously undiagnosed structural heart disease.<sup>2</sup>

### Cardiopulmonary assessment

Early clinical features of pulmonary edema include tachypnea, tachycardia, and dyspnea. The nurse will frequently be the healthcare provider who must first assess a woman's report of dyspnea during pregnancy, labor, or childbirth. This is a common and generally innocuous consequence of normal physiologic changes during pregnancy. Proposed explanations for dyspnea of pregnancy include

progesterone-induced respiratory center stimulation, with resultant hyperventilation, and pressure exerted by the enlarging uterus on the diaphragm, which may compromise residual capacity.<sup>3</sup> Whether the nurse is at the bedside in a women's center, or triaging phone calls for a midwife or physician's office, it is essential that she assess all complaints of dyspnea against risk factors and symptoms for pulmonary edema. The nurse should ask the woman to describe the dyspnea. A woman with pulmonary edema might feel as if suffocating; one with a reactive airways disorder may feel breathing to be tight.<sup>42</sup>

The woman should be assessed for tachypnea (respiratory rate  $>26$ - $28$ ) and tachycardia (heart rate  $>110$ ). In the antepartum and postpartum setting, these are significant clinical findings. These findings can present a challenge in the laboring woman, however, where tachypnea and tachycardia can also be symptomatic of ineffective pain management. As edema worsens, the woman may exhibit anxiety and diaphoresis<sup>2</sup>; although these are not normal findings for the antepartum or postpartum woman, they are symptoms that are more common in active labor. Therefore, thorough lung auscultation is needed immediately for any dyspneic complaint, especially those associated with tachypnea and tachycardia. Baseline assessments should occur at a minimum of every 8 to 12 hours throughout the intrapartum period. In the presence of pulmonary edema, initially bibasilar crackles will be heard. As the condition worsens, the nurse may hear crackles throughout the lungs.<sup>42</sup> Frothy and blood-tinged secretions may appear. Wheezing and carbon dioxide retention can occur because of peribronchial edema and increased airway resistance.<sup>3</sup> One of the complications of pulmonary edema is poor oxygen saturation, so one of the best methods of monitoring oxygen status is continuous measurement of oxygen saturation with a pulse oximeter.<sup>43</sup> Values should be 95% or higher during pregnancy to prevent fetal compromise.<sup>14,21,27,28</sup> Postpartum values should be maintained within a range consistent with institutional guidelines for inten-

sive care unit or postanesthesia care unit discharge.

In addition to the physical assessments, the nurse caring for the woman with pulmonary edema must also provide emotional support during this hospitalization. Care providers can have excellent clinical skills, but for the woman experiencing pulmonary dysfunction, care and compassion are equally important. A vicious cycle begins when the woman experiences shortness of breath or complains that she is "having difficulty breathing." A feeling of suffocation will cause an increase in the woman's pulse rate, blood pressure, and oxygen consumption—all further increasing the work of breathing. The increased work of breathing further increases the workload of the heart to meet the woman's oxygenation and perfusion needs. Coupled with the increased cardiac workload there is pulmonary congestion and increased anxiety and difficulty breathing for the woman.

Women enter pregnancy with preset assumptions and plans. Emotional support for the woman and her family is important. If she is on an antepartum unit, the woman may be concerned for her own well-being and the well-being of her fetus; postpartum she may feel stresses from impaired parent-infant interactions. This stress increases if the woman gives birth to a preterm, low-birth-weight, or critically ill infant admitted to an intensive care nursery. The nurse should explain all procedures, and the woman should have support persons present. The nurse should encourage the woman to express her concerns and feelings. If the woman desires pastoral support, the nurse can notify the woman's pastoral care (eg, priest, rabbi, pastor, minister), or available hospital pastoral personnel. The nurse can also provide information about hospital or community supportive social services.

#### Fetal assessments

Fetal well-being is dependent on the effectiveness of the maternal cardiopulmonary system's ability to deliver oxygen to the fetus while compensating to meet her own oxygenation/perfusion needs. A guiding

principle for management of pregnancy complicated by underlying maternal conditions, maternal well-being is the first priority. Moreover, for the woman diagnosed with pulmonary edema, therapies directed to improve maternal oxygenation will also improve fetal oxygen delivery and overall fetal status. Depending on the gestational age of the fetus, maternal stabilization is preferable to immediate delivery.

Continuous electronic fetal monitoring allows the assessment of fetal status. Fetal assessment may show the effects of maternal hypoxemia even before the mother voices a complaint of clinical symptoms. Even small decreases in maternal  $PO_2$  can lead to a significant drop in fetal oxygenation.<sup>3</sup> Early mild fetal hypoxia may produce the compensatory mechanism of increased variability. As the hypoxia increases, the fetus will become tachycardic secondary to activation of the sympathetic nervous system because of inadequate oxygenation. Variability decreases and flattens. If the hypoxia becomes profound, bradycardia and decelerations appear. If the mother has received medications with similar fetal side effects, this fetal early warning system may not be given adequate credence. As always, it is important to know the baseline fetal heart rate and to thoroughly investigate deviations.

#### **Monitoring response to therapy**

Assessment of maternal fetal status is part of the clinical picture evaluated by the perinatal care provider. In addition to the general assessments for maternal/fetal well-being, the nurse must also evaluate and monitor the maternal and fetal responses to therapies initiated to improve maternal cardiopulmonary function and improve oxygenation and perfusion.

#### **Positioning**

As mentioned previously, an upright maternal position is critical to the management of woman diagnosed with pulmonary edema. In an upright position, with uterine displacement maintained, approximately 75% of the

blood displaced to the dependent parts of the body comes from pulmonary circulation. This displacement of blood to the dependent body parts may reduce pulmonary blood volume by approximately 25%.<sup>18</sup> By reducing pulmonary blood volume, pulmonary capillary hydrostatic pressure and fluid movement from the interstitial areas into the lung decrease. Upright positioning is an easy, non-invasive measure to significantly reduce right preload and decrease pulmonary congestion.

While an upright position is beneficial, the nurse must be aware of the woman's true body position. Placing a bed in a semi-Fowler's or high-Fowler's position does not ensure that the woman will maintain an upright position. With the head of the bed raised and the foot of the bed remaining flat, the woman's legs are in a horizontal position. If the legs remain horizontal, the force of gravity is not maximized, and the displacement of blood is minimized. Furthermore, in an unsupported upright position, the woman will typically slip down in bed after approximately 5 to 10 minutes and assume a flatter or more supine position, further compromising maternal cardiopulmonary function.

To assist the woman maintain a true upright position, the foot of the bed must be lowered or, if possible, adjusted into a chair position. The side rails are in the up position to assist the woman to "pull up" in the bed, if needed. Pillows can be placed to support the woman in the upright position and to maintain uterine displacement; the over-bed table may be useful as well. A pillow placed vertically between the woman's scapulae brings the shoulders back and facilitates effective ventilatory movements.

#### **Oxygenation status**

Supplemental oxygen is administered by either facemask or nasal cannula to maintain  $SpO_2$  at or above 95% during pregnancy to prevent fetal compromise.<sup>14,21,27,28</sup> However, oxygen saturation, as measured by continuous or intermittent pulse oximetry, is only part of the oxygenation/perfusion equation. To maintain adequate maternal/fetal oxygenation and

perfusion, the woman must have adequate hemoglobin to transport the oxygen to the tissues and adequate cardiac output to perfuse the tissues. The underlying assumption for pulse oximetry monitoring is that a normal  $\text{SaO}_2$  indicates that oxygen is being delivered to the lungs, that the lungs are diffusing oxygen into the pulmonary arterial blood, and that hemoglobin binds with oxygen, and is delivered to the tissues. However, even when pulse oximetry readings are within the reference range, there is no guarantee that oxygen is being delivered to or used by end-organ cells or body tissues.

Assessment for adequate oxygenation involves a systematic review of multiple factors, including maternal and fetal parameters. Maternal assessments consistent with adequate tissue oxygenation include stable vital signs; appropriate level of consciousness, mentation, verbal responses, and behaviors; skin color and temperature appropriate for race; and urinary output of at least 25 to 30 mL/h. Fetal assessments consistent with adequate uteroplacental perfusion include a stable baseline heart rate and variability appropriate for gestational age, presence of accelerations, and absence of decelerations. For the woman on an antepartum or intrapartum unit, fetal status is an appropriate indicator of maternal oxygenation and perfusion.

#### **Furosemide**

Furosemide is a high-potency diuretic used in the management of acute pulmonary edema to decrease right preload and pulmonary congestion. When administered intravenously, onset of diuresis is 5 to 15 minutes, peak action within 20 to 60 minutes, with duration of approximately 2 hours.

Furosemide and supplemental oxygen therapy are generally the only therapies required when women develop pulmonary edema during pregnancy. However, since the drug is eliminated by hepatic metabolism and renal excretion, and preeclampsia is a common precipitating event for pulmonary edema in pregnancy, careful monitoring of lab data is indicated. Maternal pulse rate and blood pressure,

urinary output, serum electrolytes (including potassium, sodium, chloride, and magnesium levels), and acid-base status should be monitored during diuretic therapy.

Strict intake and output records must be maintained. Administration of furosemide can result in profound diuresis, with resulting hypovolemia and electrolyte losses. Attempts to force diuresis in a potentially hypovolemic or volume-contracted state such as preeclampsia may lead to hypovolemic shock and possibly vascular thrombosis. Loop diuretics, like furosemide, may cause significant losses of potassium, sodium, chloride, and magnesium. Electrolyte levels must be monitored, with results evaluated within the context of the underlying disease process and the use of furosemide. Tinnitus, hearing impairment, and reversible deafness have been reported to occur with rapid intravenous administration of furosemide; doses must be administered slowly. Lastly, prior to administering furosemide the woman's medication allergies should be verified; if a sulfonamide sensitivity is present, furosemide may cause an allergic reaction.

#### **Morphine sulfate**

Morphine sulfate is a widely administered narcotic analgesic that also allays anxiety and creates a sense of well-being. For the woman diagnosed with pulmonary edema, these actions reduce levels of circulating catecholamines and decreases cardiac workload. An additional benefit of morphine sulfate administration is the venodilatory effect of the drug: increased venous capacitance, decreased pulmonary artery blood flow and pressure (right preload/afterload), decreased left ventricular end-diastolic volume and pressure (left preload), and improved stroke volume. For the woman diagnosed with pulmonary edema, these cardiopulmonary effects are useful in improving myocardial oxygenation/perfusion and decreasing cardiac workload, myocardial oxygen consumption, and pulmonary vascular congestion. With intravenous administration, the effect of

morphine is immediate and plasma levels decrease rapidly over the first hour following administration.

Because morphine is a central nervous system depressant, maternal vital signs, including pulse rate, respiratory rate, level of consciousness, and oxygen saturation, are monitored; this is important especially if the woman is receiving magnesium sulfate. Respiratory depression and hypotension are potentially serious side effects of morphine administration, so naloxone and resuscitative equipment must be immediately available.

### CONCLUSIONS

Pulmonary edema complicating pregnancy can occur as an acute condition, threatening both maternal and fetal well-being. The peri-

natal caregiver must be able to quickly assess and recognize deviations from normal to identify the woman at greatest risk for developing pulmonary edema. Once risk factors or predisposing conditions are identified, the goal of care is to prevent further compromise as much as possible. Vigilant assessments of maternal and fetal well-being and assessment of adequate oxygenation and perfusion will identify problems early, so appropriate therapies can be initiated in a timely manner. Positioning of the woman, supplemental oxygen therapy, diuresis and venodilator therapy, and systematic assessments are key elements of optimal nursing care for the woman diagnosed with pulmonary edema. Through an interdisciplinary plan of care perinatal outcomes in the pregnancy complicated by acute onset of pulmonary edema can be positive.

### REFERENCES

- Norwitz E, Hsu D, Repke J. Acute complications of preeclampsia. *Clin Obstet Gynaecol.* 2002;45:308-329.
- Sciscione A, Ivester T, Largoza M, Manley J, Shlossman P, Colmoegen G. Acute pulmonary edema in pregnancy. *Obstet Gynaecol.* 2003;101:511-515.
- Jiva T. Critical care of pregnant women, I: pulmonary edema, ARDS, thromboembolism. How to manage life-threatening cardiopulmonary complications of pregnancy. *J Crit Illn.* 2000;15:316-324.
- Bal L, Thierry S, Brocas E, Adam M, Van de Louw A, Tenaillon A. Pulmonary edema induced by calcium-channel blockade for tocolysis. *Anesth Analg.* 2004;99:910-911.
- Caughy A, Paret J. Tocolysis with beta-adrenergic receptor agonists. *Semin Perinatol.* 2001;25:248-255.
- de La Chapelle A, Benoit S, Bouregba M, Durand-Reville M. The treatment of severe pulmonary edema induced by beta adrenergic agonist tocolytic therapy with continuous positive airway pressure delivered by face mask. *Anesth Analg.* 2002;94:1593-1594.
- Deblieux P, Summer W. Acute respiratory failure in pregnancy. *Clin Obstet Gynaecol.* 1996;39:143-152.
- Elliott J, Istwan N, Rhea D, Stanziano G. The occurrence of adverse events in women receiving continuous subcutaneous terbutaline therapy. *Am J Obstet Gynaecol.* 2004;191:1277-1282.
- Gilson G, Kramer R, Barada C, Izquierdo L, Curet L. Does labetalol predispose to pulmonary edema in severe pregnancy-induced hypertensive disease? *J Matern Fetal Med.* 1998;7:142-147.
- Graves C. Acute pulmonary complications during pregnancy. *Clin Obstet Gynaecol.* 2002;45:369-372.
- Huang W, Chen C. Pulmonary edema in pregnancy. *Int J Obstet Gynaecol.* 2002;78:241-243.
- Pryde P, Besinger R, Gianopoulos J, Mittendorf R. Adverse and beneficial effects of tocolytic therapy. *Semin Perinatol.* 2001;25:316-340.
- Chang J, Elam-Evans L, Berg C, et al. Pregnancy-related mortality surveillance—United States, 1991-1999. *MMWR Surveill Summ.* 2003;52:1-8.
- Blackburn S. *Maternal, Fetal & Neonatal Physiology: A Clinical Perspective.* 2nd ed. St Louis: WB Saunders; 2003.
- Norwitz E, Robinson J, Malone F. Pregnancy physiology. In: Dildy GA III, Belfort M, Saade G, Phelan J, Hankins G, Clark S, eds. *Critical Care Obstetrics.* 4th ed. Malden, Mass: Blackwell Science; 2004:19-42.
- Whitty J, Dombrowski M. Respiratory diseases in pregnancy. In: Creasy R, Resnik R, Iams J, eds. *Maternal-fetal Medicine: Principles and Practice.* 5th ed. Philadelphia: WB Saunders; 2004:953-974.
- Mabie W. Pulmonary edema. In: Dildy GA III, Belfort M, Saade G, Phelan J, Hankins G, Clark S, eds. *Critical Care Obstetrics.* 4th ed. Malden, Mass: Blackwell Science; 2004:346-353.
- Darovic G. Monitoring patients with acute pulmonary disease. In: Darovic G, ed. *Hemodynamic Monitoring: Invasive and Noninvasive Clinical Application.* 3rd ed. Philadelphia: WB Saunders; 2002:421-470.