Assessment and management of the critically ill pregnant patient may be affected by the physiologic changes induced by pregnancy, by physicians’ lack of familiarity with pregnancy-specific conditions, and by the presence of a fetus. This review describes the cardiopulmonary complications that may result in intensive care unit admission and the principles of management of these patients.

MATERNAL CARDIORESPIRATORY PHYSIOLOGY

Changes in lung volumes occur during pregnancy as the uterus enlarges (Table 1). Functional residual capacity decreases by 10–25% (1, 2), whereas total lung capacity decreases only minimally as the thoracic cage widens to compensate (2). Forced expiratory volume in 1 sec is not altered by pregnancy (3), although asthmatic patients respond variably with improvement, no change, or deterioration (4). Lung compliance is unchanged in pregnancy, but chest wall and total respiratory compliances are reduced (5). An increase in tidal volume and minute ventilation occurs beginning in the first trimester, reaching 20–40% above baseline by term (6). The increased ventilation is mediated by the elevated serum progesterone level (7) and a mild respiratory alkalosis results, with compensatory renal excretion of bicarbonate. By term, the arterial carbon dioxide pressure falls to about 28–32 torr (3.7–4.3 kPa), with plasma bicarbonate decreased to 18–21 mEq/L (8). Oxygen consumption is increased due to increasing demands of the fetus and maternal metabolic processes, to a level 20–33% above baseline by the third trimester (6, 9). The reduced functional residual capacity and increased oxygen consumption can result in the rapid development of hypoxemia in response to hypoventilation or apnea (10).

Maternal blood volume and cardiac output increase progressively through pregnancy, beginning from as early as 5 wks gestation and reaching 30–50% above baseline levels by about 28 wks (11–13). Systemic blood pressure decreases, reaches a nadir at about 28 wks, and then slowly increases toward term (14). Hemodynamic measurements by pulmonary artery catheter in the near-term patient demonstrate the increased cardiac output, with systemic vascular resistance and pulmonary vascular resistance decreased by 20–30% (Table 1) (13). In the supine position, the enlarged uterus may compress the inferior vena cava, producing a drop in cardiac output, sometimes associated with reflex vasovagal effects (15).
During labor, cardiac output increases by a further 10–15%, augmented during contractions by the return of 300–500 mL of blood to the central circulation (16, 17). Immediately after delivery, there is an increase in preload, producing an increased cardiac output. Cardiac output remains elevated at the levels seen during pregnancy for about 48 hrs after delivery (18).

**DETERMINANTS OF FETAL OXYGENATION**

Oxygen delivery to the fetus is determined by the maternal arterial oxygen content, the hemoglobin concentration, and the uterine blood flow. As the uterine vasculature is normally maximally dilated, maternal illness tends to adversely affect fetal oxygenation (19). Maternal hypotension, alkalosis, and endogenous or exogenous catecholamines can vasoconstrict the uterine artery (20, 21). Umbilical venous blood returning to the fetus has a low oxygen tension, but an adequate oxygen content is maintained by the left shift of the oxygen dissociation curve of fetal hemoglobin (22). Uteroplacental oxygen delivery can be improved by increasing oxygen-carrying capacity by blood transfusion, improving maternal cardiac output, or optimizing maternal oxygenation. Tilting the patient to the left lateral position to increase cardiac output should always be considered (23).

**PREGNANCY-SPECIFIC CONDITIONS**

**Preeclampsia.** Preeclampsia is a pregnancy-induced condition characterized by hypertension and proteinuria occurring after 20 wks gestation (24). The cause of this condition is unclear, but it may be initiated by a placental abnormality that produces a diffuse maternal endothelial effect. This leads to vasospasm, reduced organ perfusion, and multiple organ dysfunction (24). Preeclampsia and its complications account for 20–50% of obstetrical admissions to the intensive care unit (25–29) and 12–17% of all maternal deaths in the United States (24, 30).

Given the multiple-organ nature of the disease, the differential diagnosis would often include systemic lupus erythematosus, thrombotic thrombocytopenic purpura, idiopathic thrombocytopenic purpura, and hemolytic-uremic syndrome. The complications of preeclampsia include pulmonary edema, cerebral edema, renal failure, hypertensive crisis, seizures ( eclampsia), and the HELLP syndrome (microangiopathic hemolytic anemia, thrombocytopenia, and hepatic involvement) (24, 31). Pulmonary edema occurs most commonly in the early postpartum period, often associated with intrapartum fluid administration and return of blood to the central circulation as the uterus contracts (32).

From the obstetrical perspective, management requires a well-timed delivery; delivery is always in the mother's best interest (24). No specific treatment is effective other than removal of the fetus and placenta. Antihypertensive therapy is used only to prevent maternal hypertensive complications and does not alter the natural history of preeclampsia or benefit the fetus (24, 33). Commonly used antihypertensive regimens include intravenous hydralazine or labetolol or oral calcium antagonists (24). Rapid decreases in blood pressure must be avoided due to the risk of reducing uteroplacental perfusion. Magnesium sulfate is the treatment of choice for seizure prophylaxis and treatment (34, 35). An initial intravenous bolus is followed by an infusion of 2–3 g/hr, usually for 24 hrs. Magnesium levels are maintained at 2–3.5 mmol/L; toxic levels may occur in patients with renal dysfunction and can cause respiratory muscle weakness and cardiac conduction defects. Hypocalcemia commonly occurs and should not always be treated—intravenous calcium will reverse the toxic and therapeutic effects of magnesium. Fluid management in preeclampsia usually requires careful volume expansion, but excessive fluid administration can cause pulmonary or cerebral edema (36–38). Pulmonary artery catheterization may be indicated in the presence of severe hypertension requiring intravenous therapy, pulmonary edema, and persistent oliguria despite careful fluid challenge (24).

**Amniotic Fluid Embolism.** Amniotic fluid embolism is an uncommon but catastrophic complication of pregnancy, with a mortality rate of 10–86% (39, 40). Usually associated with labor and delivery, it may also occur with uterine manipulations or uterine trauma or in the early postpartum period (39). Amniotic fluid is believed to enter the vascular circulation through endocervical veins or uterine tears, and particulate cellular contents or humoral factors produce acute pulmonary hypertension and acute left ventricular dysfunction (39–42). The clinical presentation is with sudden onset of dyspnea, hypoxemia, and cardiovascular collapse, often followed by seizures (39, 40). Less commonly, fetal distress or hemorrhage caused by disseminated intravascular coagulopathy may be the initial problem. A significant proportion of patients with amniotic fluid embolism die within the first hour. The diagnosis of amniotic fluid embolism is usually based on a typical clinical picture. Although the finding of fetal squames in an occluded pulmonary capillary aspirate has been suggested to confirm the diagnosis, this is not a specific finding (39). Differential diagnoses to consider include septic shock, pulmonary thromboembolism, abruptio placenta, tension pneumothorax, or a myocardial ischemic event.

Initial treatment involves routine resuscitative and supportive measures, with attention to oxygenation, mechanical...
ventilation, and inotropic support (39). There is no specific therapy, but a role for corticosteroids has been suggested (40). Survivors of the initial cardiorespiratory collapse are likely to develop disseminated intravascular coagulopathy and/or acute respiratory distress syndrome. Neurologic damage caused by hypotension and hypoxemia is common.

**Tocolytic Pulmonary Edema.** Beta-adrenergic agonists have been used to inhibit uterine contractions in preterm labor, although this practice is less frequent following studies demonstrating a lack of benefit from tocolysis (43). A complication of this β-agonist use in pregnancy is the development of pulmonary edema (44). The mechanism of this complication is unclear but may relate to myocardial effects or changes in permeability. The clinical presentation is of respiratory distress with clinical features of pulmonary edema, during or immediately following intravenous infusion of tocolytic drugs. Treatment involves discontinuing the β-agonist and supportive treatment including diuresis (44, 45). Early recognition and management reduce the need for mechanical ventilation. Failure of the pulmonary edema to resolve in 12–24 hrs should prompt a search for an alternative diagnosis (45). Pulmonary edema also occurs following intrauterine fetal surgery, possibly related to the use of tocolytic agents (46).

**Gestational Trophoblastic Disease.** Pulmonary hypertension and pulmonary edema may rarely occur in the setting of benign hydatidiform mole, as a result of trophoblastic pulmonary embolism. This most commonly occurs during evacuation of the uterus, with a higher incidence of pulmonary complications occurring in woman later in pregnancy (47). Treatment is supportive, with resolution occurring within 48–72 hrs (48). Molar pregnancy may be associated with chorioangioma, which can produce multiple, discrete pulmonary metastases.

**Peripartum Cardiomyopathy.** Cardiac failure may occur in the absence of preexisting heart disease as a result of peripartum cardiomyopathy, an idopathic condition presenting in the last month of pregnancy or within 5 months of delivery. The incidence is about 1:3500 live births, and the etiology remains uncertain (49). The clinical presentation is usually after 36 wks gestation (in contrast to women with preexisting cardiac disease) and involves the gradual onset of symptoms of heart failure, late in pregnancy or in the postpartum period. The diagnosis is made by the demonstration of left ventricular systolic dysfunction and exclusion of other causes of cardiomyopathy (49). During labor and the early postpartum period, tachycardia and increased cardiac output may precipitate acute pulmonary edema. Treatment is not different than for other patients with heart failure, but angiotensin-converting enzyme inhibitors should be avoided before delivery. Anticoagulation is essential due to the hypercoagulable state of pregnancy. A subset of patients may have an inflammatory myocarditis, and immunosuppressive therapy may be considered in those who do not improve after 2 wks of standard treatment (49). A high mortality rate has been described (10–50%), although about half of patients recover normal ventricular function (50, 51).

**Obstetrical Hemorrhage.** Due to the high uterine blood flow near term (600 ml/min), the pregnant patient is at risk of devastating blood loss (52). Obstetrical assessment of major hemorrhage may require ultrasound of the uterus and examination under anesthesia (53). Supportive management involves rapid volume replacement, supplemental oxygen administration, and blood product support for associated dilutional coagulopathy. In the presence of uterine atony, the intramuscular administration of methylergonovine may be of value but is contraindicated in the presence of hypertension (53). Oxytocin is infused at a higher dose than for augmentation of labor, at an infusion rate up to 100 mU/min (53). This dose has significant antiuretic effect and may cause hypotension. An analogue of prostaglandin F2α, (carboprost tromethamine—Hemabate) given intramuscularly (0.25 mg, repeated every 15–90 mins to maximum 2 mg) or intramyometrially has a high success rate in controlling hemorrhage (53, 54). Reported side effects include vomiting, hypertension, bronchoconstriction, and increased intrapulmonary shunt. Recombinant factor VIIa has also been successfully used in the management of severe obstetrical hemorrhage (55). If pharmacologic methods are unsuccessful, radiologic transcatheter embolization of the internal iliac or uterine artery may be required (56). If these measures fail, surgical exploration will be necessary to repair lacerations, reduce blood flow by arterial ligation, or remove the uterus (53).

The pregnant patient is at risk of hyperperfusion injury to the kidney particularly when renal function is already compromised by preeclampsia (57). A high incidence of myocardial ischemia has been noted in pregnant women with hemorrhagic shock, and this should be considered in the management of these patients (58).

**Septic Shock.** Obstetrical sepsis is an important cause of hemodynamic instability requiring intensive care unit support. A spectrum of microorganisms may be implicated, originating from the vagina or intestine, caused by sexual transmission, or blood-borne (59). Pregnancy appears to be associated with a decreased cell-mediated immune response, which increases susceptibility to microorganisms such as *Listeria monocytogenes*, disseminated herpesvirus, varicella, and coccidioidomycosis (60).

Antepartum infection produces choioamnionitis, but the bulk of obstetrical sepsis occurs in the postpartum period. The usual principles of management should be followed, with prompt resuscitation with volume expansion. If inotropic therapy is necessary in the antepartum situation, the effects of such drugs on uteroplacental perfusion must be considered (61). Initial empirical antibiotic therapy aims to provide broad Gram-negative, Gram-positive, and anaerobic cover, using drugs such as ampicillin, gentamicin and clindamycin; ampicillin/sulbactam; ticarcillin-clavulinate; piperacillin and gentamicin; or carbapenems (59, 62). Surgical intervention may be required and can be life-saving. Activated protein C should be considered in the postpartum patient (63).

In the postpartum patient who deteriorates despite antibiotic therapy, a resistant organism (e.g., *Enterococcus*), localized abscess, myometrial microabscesses, or septic pelvic thrombophlebitis should be considered. Gas in the subcutaneous tissues or uterine walls on radiographic studies suggests clostridial gas gangrene (64). Early surgical evacuation of the uterus and high-dose penicillin are essential, but hysterectomy and wide excision of gangrenous areas may be required (65). Necrotizing fasciitis and streptococcal toxic shock syndrome, due to group A streptococci, may occur unexpectedly following an uncomplicated pregnancy and delivery (66). Management includes antibiotic therapy with penicillin and clindamycin and early surgical intervention. Intravenous immunoglobulin (2 g/kg) is thought to be beneficial (67, 68). Other organisms producing a toxic shock syndrome include *Staphylococcus aureus*.
and *Clostridium sordelli*, usually associated with instrumentation and septic abortion (69).

**CONDITIONS NOT SPECIFIC TO PREGNANCY**

**Pulmonary Thromboembolic Disease.** Pulmonary thromboembolism occurs in up to 0.13% of pregnancies, more frequently in the early postpartum period than during pregnancy (70). The high incidence results from hypercoagulability, from hormonally mediated venous stasis, and from local pressure effects of the uterus on the inferior vena cava. Investigation of suspected pulmonary embolism is similar to that in the patient who is not pregnant, with duplex ultrasound as the initial investigation of choice. Ventilation-perfusion scanning can be performed with <1.0 mGy (<100 mrad) radiation exposure to the fetus, and a computed tomography pulmonary angiogram may be carried out with similarly low fetal exposure (Table 2) (71, 72).

Warfarin is usually avoided in pregnancy due to the risk of an embryopathy with first trimester use and central nervous system abnormalities with second and third trimester exposure (73). Heparin has the advantage that it does not cross the placenta and can be readily reversed. Low molecular weight heparins are both safe and effective in pregnancy but are less easy to reverse acutely (74). When administered with adequate precautions, thrombolysis has been used successfully in pregnancy but should be limited to life-threatening situations (75). Transvenous placement of an inferior vena cava filter can be performed, although there is some risk of dislodgement because of the dilated venous system and pressure effects during labor (76).

**Acute Respiratory Distress Syndrome in Pregnancy.** The pregnant patient is at risk of developing pulmonary edema and acute lung injury from pregnancy-associated complications or other conditions (Table 3) (45, 77) and may be predisposed to pulmonary edema by the cardiovascular changes and the reduced albumin level occurring in pregnancy (45).

There are few differences in the management of pregnant patients who have acute respiratory distress syndrome compared with those who are not pregnant. Adequate maternal oxygen saturation is essential for fetal well-being, and fetal delivery may benefit both the mother and the fetus (78). Survival appears to be as good as or better than that in the general population, possibly because of their young age, lack of comorbidity, and the reversibility of many of the predisposing conditions (79).

**Asthma.** The hormonal changes of pregnancy can affect asthma control variably, with worsening, improvement, or no substantial change being noted (4, 80, 81). As asthma is a common condition, acute asthmatic attacks represent an important cause of respiratory compromise in pregnancy. The reduced PaO₂ occurring in late pregnancy should be considered in the assessment of arterial blood gases. Treatment is not different from the nonpregnant patient, and pregnancy is not an absolute contraindication to corticosteroid therapy (81). Although there is obviously a reluctance to prescribe drug therapy in pregnancy, uncontrolled asthma is more dangerous for the fetus than the recommended medications.

**Cardiac Disease.** Due to the circulatory changes of pregnancy, women with preexisting heart disease may develop acute cardiac decompensation. Those with prior cardiac events, cyanosis, left heart obstructive lesions, or systolic dysfunction are at most risk (82).

Mild to moderate regurgitant valvular disease is generally well tolerated in pregnancy, but patients with mitral and aortic stenosis may develop hemodynamic deterioration as the physiologic changes peak at about 28 wks (83). New-onset atrial fibrillation or severe hypertension can precipitate sudden hemodynamic deterioration even in women with less severe cardiac disease. Patients with moderate to severe mitral stenosis are likely to experience hemodynamic deterioration during the third trimester or during labor. Treatment is similar to the nonpregnant patient; digoxin and β-blockers can reduce heart rate and diuretics reduce left atrial pressure (83, 84), but angiotensin-converting enzyme inhibitors should be avoided. Electrical cardioversion can be performed safely if indicated. Most patients with mitral stenosis can undergo vaginal delivery; invasive hemodynamic monitoring may be beneficial during labor and the early postpartum period. Epidural anesthesia is usually better tolerated hemodynamically than general anesthesia during labor and delivery.

Severe aortic stenosis is associated with significant risk during pregnancy (84). Symptoms such as dyspnea, angina pectoris, or syncope may appear from late in the second trimester. Percutaneous aortic balloon valvuloplasty is possible during pregnancy. Spinal and epidural anesthesia are both safe and effective in pregnancy but should be limited to life-threatening situations. Low molecular weight heparins cross the placenta and can be readily reversed. Low molecular weight heparins have the advantage that it does not occlude the placenta and can be readily reversed. Low molecular weight heparins are both safe and effective in pregnancy but should be limited to life-threatening situations (75). Transvenous placement of an inferior vena cava filter can be performed, although there is some risk of dislodgement because of the dilated venous system and pressure effects during labor (76).

Table 2. Fetal radiation exposure and the risk of radiological investigations in the critically ill pregnant patient

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Fetal Radiation Exposure, mGy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest radiograph (abdomen shielded)</td>
<td>0.01</td>
</tr>
<tr>
<td>Ventilation-perfusion scan</td>
<td></td>
</tr>
<tr>
<td>Perfusion</td>
<td>0.1–1.0</td>
</tr>
<tr>
<td>Ventilation</td>
<td>0.1–0.4</td>
</tr>
<tr>
<td>CT pulmonary angiogram</td>
<td>0.1–0.9</td>
</tr>
<tr>
<td>CT pelvis and abdomen</td>
<td>30–50</td>
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</tbody>
</table>

Radiation effect

- Teratogenicity: 50–100
- Increased risk of childhood leukemia: 20–50

CT, computed tomography.

Table 3. Causes of pulmonary edema and acute respiratory distress syndrome in the pregnant patient

<table>
<thead>
<tr>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnancy-specific conditions</td>
</tr>
<tr>
<td>Amniotic fluid embolism</td>
</tr>
<tr>
<td>Tocolytic-induced pulmonary edema</td>
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<tr>
<td>Preeclampsia</td>
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<tr>
<td>Placental abruption</td>
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<tr>
<td>Peripartum cardiomyopathy</td>
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<tr>
<td>Trophoblastic embolism</td>
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<tr>
<td>Fetal surgery</td>
</tr>
<tr>
<td>Related to pregnancy</td>
</tr>
<tr>
<td>Obstetric sepsis</td>
</tr>
<tr>
<td>Secondary to obstetric hemorrhage</td>
</tr>
<tr>
<td>Gastric aspiration</td>
</tr>
<tr>
<td>Pulmonary edema secondary to preexisting</td>
</tr>
<tr>
<td>heart disease</td>
</tr>
<tr>
<td>Increased infection risk</td>
</tr>
<tr>
<td>Listeria</td>
</tr>
<tr>
<td>Pyelonephritis</td>
</tr>
<tr>
<td>Varicella</td>
</tr>
<tr>
<td>Blastomycosis</td>
</tr>
<tr>
<td>Not specific to pregnancy</td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Drug overdose</td>
</tr>
<tr>
<td>Other infections</td>
</tr>
</tbody>
</table>
A
dmission of the pregnant or post-
partum woman to the intensive care unit is un-
common but may require specialized knowledge for successful management.

anesthesia during labor may adversely af-
flect hemodynamics due to the vasodila-
tory effect. Although antibiotic prophylaxis is not indicated for routine deliveries, many centers use prophylaxis as uncomplicated delivery cannot always be anticipated (84).

Woman with cyanotic congenital heart disease and those with associated pulmonary hypertension are at significant risk during pregnancy (84). Pulmo-
nary hypertension, either primary or sec-
ondary, is associated with a significant mortality rate in pregnancy, due to the inability of the right ventricle to tolerate the increases in cardiac output (85). Treatment with vasodilators such as pros-
tacyclin and nitric oxide has been de-
scribed, with or without invasive hemo-
dynamic monitoring (83, 86). These patients should be managed in a center with expertise and experience. Ischemic heart disease is uncommon in pregnancy but may be missed due to masking of symptoms, signs, and cardiac enzyme levels by pregnancy. Coronary artery dissec-
tion may be responsible, particularly in the immediate postpartum period. Aortic dissection may occur related to hyperten-
sion, aortic coarctation, or Marfan’s syn-
drome and is associated with significant maternal and fetal mortality (87).

**MANAGEMENT ISSUES**

A detailed review of management of the critically ill pregnant patient is be-

doing the scope of this review but is ad-
dressed elsewhere (61, 88–90). Manage-
ment issues relevant to the patient with cardiopulmonary complications include the use of vasopressor drugs, cardiopul-
monary resuscitation, and mechanical ventilation.

**Vasopressor Therapy**

Although vasopressor or inotropic therapy may be essential for maternal (and fetal) survival, all commonly used drugs (e.g., dobutamine, dopamine, nor-

epinephrine, epinephrine) may reduce uterine blood flow (61). Animal studies and limited human data suggest that ephedrine has an advantage in increasing uterine blood flow as well as maternal blood pressure (91). The rapidity of cor-

rection of blood pressure may be more important than the specific inotropic agent used (92). Nonpharmacologic in-
terventions such as volume replacement therapy and left lateral positioning must be stressed. If inotropic therapy is needed to support maternal hemodynamics, it should not be withheld due to concerns for potential adverse effects on the fetus.

**Cardiopulmonary Resuscitation**

The management of cardiac arrest in the pregnant patient uses standard pro-

tocols, with some modifications (93). Electrical cardioversion and defibrillation may be performed in pregnancy, but fetal monitoring leads must be removed to prevent arcing (94). Pharmacologic ther-

apy required for maternal resuscitation should not be withheld when clinically indicated. Cardiac massage in the supine position may cause aorto-caval compres-
sion, impairing venous return and car-
diac output. A wedge under the right hip or manual displacement of the uterus to the left is recommended (93). Perimor-
tem cesarean section may be indicated when initial attempts at resuscitation have failed in a woman with a viable fetus (95).

**Ventilatory Support**

**Noninvasive Ventilation.** Intubation in pregnancy can be associated with in-
creased risk, making noninvasive support an attractive consideration. This modality is also ideally suited to short-term ventilatory support, which may be the case in many obstetrical complications that re-
verse rapidly. The major concern of mask ventilation in pregnancy is the risk of vomit-
ing and aspiration, aggravated by the increased intra-abdominal pressure, delayed gastric emptying, and reduced lower esophageal sphincter pressure in pregnancy.

**Airway Management.** Failed intuba-
tion in the obstetrical population occurs at a significantly higher rate than in other anesthetic intubations (96). The diminished functional residual capacity and increased oxygen consumption produce rapid desaturation in response to apnea or hypoventilation (10). In view of the delayed gastric emptying and elevated intra-abdominal pressure, the pregnant pa-
tient should always be considered to have a full stomach and appropriate precau-
tions taken (97). Upper airway hyperemia and edema may reduce visualization and increase bleeding.

**Mechanical Ventilation.** Data on me-
chanical ventilation in pregnancy are limited. Hyperventilation should be avoided as this adversely affects uterine blood flow, due to the resulting alkalemia and the effect of positive pressure venti-
lation in reducing cardiac output (98). Although pressure–limited ventilatory strategies suggest limiting plateau pressure to 30 cm H2O (99), chest wall compli-
ance is reduced in late pregnancy (5) and the transpulmonary pressures may not be elevated at a plateau pressure of 35 cm H2O. Higher ventilatory pressure may be acceptable in pregnant patients near term. In terms of the level of acceptable PaCO2, limited data suggest that maternal hypercapnia up to 60 torr (8 kPa), in the presence of adequate oxygenation, may not be detrimental to the fetus (100, 101), although fetal heart rate changes associated with fetal acedia may be noted. If marked respiratory acidosis results from permissive hypercapnia, treatment with bicarbonate may improve maternal and fetal academia (102). Delivery of the fetus may improve the mother’s condition, but this is not always effective (103).

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