Management Principles of the Critically Ill Obstetric Patient

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KEYWORDS

- Critical illness • Pregnancy • Peripartum cardiomyopathy
- Cardiopulmonary resuscitation • Hypertensive crisis
- Massive transfusion protocol

The goals in management of critically ill obstetric patients involve intensive monitoring and physiologic support for patients with life-threatening but potentially reversible conditions. Management principles of the mother should also take the fetus and gestational age into consideration. The most common reasons for intensive care admissions (ICU) in the United States and United Kingdom are hypertensive disorders, sepsis, and hemorrhage. The critically ill obstetric patient poses several challenges to the clinicians involved in her care, because of the anatomic and physiologic changes that take place during pregnancy.

CRITICAL ILLNESS IN PREGNANCY

Prevalence

The estimated prevalence of obstetric patients requiring ICU admission is 0.9% both in the United States and the United Kingdom. Mortality of critically ill obstetric patients ranges from 12% to 20%.[1] The most common cause of maternal death in the ICU is acute respiratory distress syndrome (ARDS).[2]

Prognosis

Patients with primary obstetric disorders tend to have a better overall prognosis as delivery of the fetus usually reverses the illness and resuscitation is more effective. Preterm babies also have a chance of survival in hospitals with established neonatal intensive care units. Several retrospective studies have analyzed the racial differences with regard to ICU admissions and the outcome of parturients. Ethnic minorities, recent immigrants, and low socioeconomic status have been associated with poor outcome.[3] Obstetric patients admitted to the ICU have a better prognosis and mortality is lower than for general medical ICU patients. Nonobstetric critical illness in pregnant women significantly affects fetal and neonatal outcomes. Maternal shock, blood product transfusion, and lower gestational age are associated with an increased risk of fetal loss.[4]

The Confidential Enquiry into Maternal and Child Health in the United Kingdom made a few recommendations aimed at improving child health and reducing maternal mortality.[5] These recommendations highlighted the importance of early recognition and management of severely ill pregnant women, and routine use of early warning scoring systems to be used for obstetric patients.[6] Early recognition of critical illness is essential for a favorable outcome for mother and baby. Prognostic criteria such as APACHE scoring may not predict mortality as accurately in pregnancy as they do

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outside of pregnancy. One of the reasons for this difference is the physiologic changes of pregnancy such as an increase in heart rate, change in white cell count, or even a drop in normal values for creatinine that can affect the score. In many cases, delivery results in a drastic improvement in the disease course and a lower mortality, even when initial indicators suggest a high mortality.

Obstetric Versus Nonobstetric Disorders

Primary obstetric disorders account for 50% to 80% of ICU admissions during pregnancy and the puerperium in all parts of the world.[3,7] More than 80% of these admissions are because of preeclampsia and its complications, hemorrhage, and sepsis. Nonobstetric disorders in pregnancy show large geographic variations. In developed countries, asthma, pneumonia, drug abuse, complicated urinary infections, preexisting autoimmune disorders, chronic pulmonary disease, endocrine disorders, trauma, and pulmonary thromboembolism are common.[8,9] Medical disorders commonly seen in developing countries include severe malaria, viral hepatitis, cerebral venous sinus thrombosis, tetanus, tuberculosis, rheumatic valvular heart disease, and anemia.[7] Some of the common obstetric and nonobstetric causes are listed in **Box 1**.

ICUs in developed countries are increasingly challenged with a unique subgroup of pregnant women. Advances in health care have resulted in survival to child-bearing age of women with disorders such as surgically corrected complex congenital heart disease and organ transplant, and chronic disorders such as cystic fibrosis. Pregnant women with these conditions have increased morbidity and tend to require intensive medical care.[10,11]

Box 1
Causes for critical illness in pregnancy

Obstetric causes
- Obstetric hemorrhage
- Placental abruption
- Preeclamsia/eclampsia
- HELLP syndrome (HELLP is an abbreviation of the main findings: hemolytic anemia; elevated liver enzymes and low platelet count)
- Acute fatty liver of pregnancy
- Chorioamnionitis
- Amniotic fluid embolism
- Puerperal sepsis
- Pelvic septic thrombophlebitis
- Peripartum cardiomyopathy

Nonobstetric causes
- Respiratory failure
- ARDS
- Acute renal failure
- Urinary tract infection
- Diabetic ketoacidosis
- Drug abuse

MANAGEMENT PRINCIPLES OF A CRITICALLY ILL PARTURIENT

Some of the common indications for transfer of patients to the ICU are listed in **Table 1**. As in a nonpregnant critically ill patient, the initial assessment of a parturient is focused on airway, breathing, and circulation.

Airway

Airway evaluation and management remains the first priority as in a nonpregnant patient. Supplemental oxygen may be required in some patients depending on their oxygen saturation. Tracheal intubation is needed in the setting of persistent hypoxemia, airway obstruction, impaired laryngeal reflexes, or altered consciousness. Because pregnant women are at a high risk for aspiration of gastric contents, endotracheal intubation should be performed sooner rather than later to protect the airway. If the airway examination indicates that tracheal intubation is likely to be difficult, awake intubation should be performed with

Table 1
Causes of shock in obstetric patients

Hypovolemic shock	Hyperemesis gravidarum, ruptured ectopic pregnancy, placental abruption, placenta previa, postpartum hemorrhage, uterine rupture, trauma
Septic shock	Chorioamnionitis, puerperal sepsis, septic abortion, pneumonia, pyelonephritis
Cardiogenic shock	Valvular heart disease, peripartum cardiomyopathy, acute myocardial infarction, myocarditis

good topical anesthesia. Rapid sequence induction with cricoid pressure and orotracheal intubation is recommended in the obtunded or unconscious parturient without a potentially difficult airway. Difficult airway equipment for airway management must always be available in the ICU and intensivists should be familiar with use of at least a few alternative airway devices.

Breathing

Adequacy of respiration must be established rapidly. Supplemental oxygen and bag/mask ventilation may be required initially. If respiratory effort is inadequate, tracheal intubation is performed and mechanical ventilation initiated without any further delay.

Circulation

Hypotension and shock should be treated promptly to maintain uteroplacental perfusion. After 20 weeks of gestation, pressure of the gravid uterus on the inferior vena cava and abdominal aorta in the supine position can cause supine hypotension syndrome and decrease cardiac output by up to 30%. For chest compressions to be more effective during the second half of pregnancy, studies have confirmed that applying a partial left lateral tilt to the patient relieves the aortocaval compression.[12] Rees and Willis[13] concluded that the best compromise for cardiopulmonary resuscitation is achieved by wedging the patient at 27°.

Two large-bore intravenous cannulae (14G or 16G) should be placed to administer fluids and a Foley catheter should be placed to monitor urine output. Central venous access may be needed for volume resuscitation, bolus drug administration, infusion of vasopressors and central venous oxygen saturation monitoring. Femoral vein catheterization should be avoided if possible because of the risk of thromboembolism and infection. The jugular route is preferred over the subclavian route in patients with coagulopathy, as the subclavian site cannot be compressed in case of excessive bleeding or accidental arterial puncture. Hypotension is treated by aggressive volume resuscitation. If hemorrhage is life threatening, blood group O Rh-negative packed red blood cells are transfused until type specific or cross-matched blood is available. It is preferable to place an arterial line at the earliest to measure the blood pressure continuously. Severe maternal hypotension may require treatment with vasopressors.

If the parturient has pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF), defibrillation is the treatment of choice and is not contraindicated in pregnancy. However, if defibrillation is required, it is important to remember to remove any internal fetal monitoring equipment that might conduct the electricity to the fetus.[14]

The next step during resuscitation of a patient with pulseless VT or VF is to administer appropriate medications that improve cardiac response to defibrillation. Vasopressin has been added to the Advanced Cardiac Life Support (ACLS) guidelines as an alternative to epinephrine. However, in middle to late pregnancy, there is a 4-fold increase in vasopressinase, a cystine aminopeptidase produced by placental trophoblasts, that enhances the clearance of vasopressin. The effect of vasopressors on uteroplacental perfusion in a cardiac arrest situation is unknown; however, the ACLS guidelines must be followed and the use of these vasopressors must not be withheld.[15] The use of epinephrine can enhance placental blood flow and improve fetal outcome.[16] Circulate the medication with 60 to 90 seconds of cardiopulmonary resuscitation (CPR), then defibrillate at 360 J.

Maternal and Fetal Evaluation

After stability of the airway, breathing, and circulation, a thorough evaluation with a detailed history and physical examination is performed. Routine ICU monitoring includes electrocardiogram, pulse oximetry, and noninvasive blood pressure monitoring. As well as blood grouping and cross-matching, blood should be sent for analysis of arterial pH and blood gases, hemoglobin concentration, electrolytes, glucose, renal and liver function. Platelet count, prothrombin time (PT), partial thromboplastin time (PTT), and serum fibrinogen and fibrin degradation product levels are obtained if disseminated intravascular coagulopathy is suspected. Thromboelastography is an alternative test that measures the viscoelastic properties of clot formation, to diagnose thrombocytopenia, platelet dysfunction, and coagulation factor abnormalities. The Kleihauer-Betke analysis should be obtained to detect the presence and percentage of fetal red blood cells in the maternal circulation. Commercially available anti-D immunoglobulin should be administered in the situation of an Rh-positive fetus carried by an Rh-negative mother. Ultrasonography is performed to evaluate the fetus and uteroplacenta.

Fetal well-being is closely monitored in the critically ill parturient. The biophysical profile has gained popularity as a test of fetal well-being. This includes fetal breathing, tone, movement, amniotic fluid volume, and the results of a nonstress test. Each parameter is assigned a score of 2 when present and 0 when absent. Scores of

8 and 10 imply fetal well-being, 6 is equivocal and 4 or less indicates the need to deliver, provided that delivery does not pose a serious risk to the mother. If preterm delivery is anticipated and there is no medical contraindication, betamethasone (2 intramuscular doses of 12 mg, 24 hours apart) may be given to enhance fetal lung maturity. The fetus should be monitored either intermittently or continuously by a trained obstetric nurse in the ICU according to the fetal status.

CARDIOVASCULAR DYSFUNCTION

Shock

Causes of shock in obstetric patients are listed in **Table 1**. Shock presents as tachycardia, tachypnea, hypotension, oliguria, altered mental status, and lactic acidosis.[17–19] Orthostatic hypotension can be the only manifestation of early hemorrhagic shock; the diagnosis may be missed in the supine patient. Signs of external or internal hemorrhage may be present. Rales on auscultation are found in left ventricular failure or ARDS, a third heart sound in peripartum cardiomyopathy and pulmonary thromboembolism, and cardiac murmurs in valvular heart disease.

Patients with shock require invasive monitoring of arterial and central venous pressures. Pulmonary artery catheterization (PAC) is used to monitor pulmonary artery wedge pressure (left ventricular filling pressure), pulmonary arterial pressure, and cardiac output. The role of PAC is controversial. Recent studies in nonpregnant patients show that it does not increase mortality, but failed to show any benefit.[20–22] Echocardiography may therefore be preferred in coagulopathic patients or those requiring only a single hemodynamic evaluation to classify their disease, but PAC remains the mainstay of management of complex problems in critically ill obstetric patients similar to nonobstetric patients.

The parturient should be placed in the left lateral position to relieve aortocaval compression. Cardiac filling should be optimized with rapid intravenous infusion of crystalloids such as normal saline or lactated Ringer's solution. Approximately 3 L of crystalloids are required to replenish 1 L of lost blood (3:1 ratio). Colloids such as hetastarch or albumin remain in the circulation longer than crystalloids. However, in a large multicenter, randomized, double-blind study, Finfer and colleagues[23] found that fluid replacement with normal saline was as effective as human albumin and a lot cheaper. Low central venous pressure (CVP) or pulmonary arterial wedge pressure (PAWP) indicates decreased cardiac preload requiring fluid replacement. However, a normal CVP or PAWP does not rule out hypovolemia and should be treated by repeated fluid challenges with 200 to 500 mL of crystalloids infused over 10 to 15 minutes until the CVP or PAWP increases by 3 mm Hg or more and stays persistently increased.

If the mean arterial pressure (MAP) remains less than 60 mm Hg after fluid replacement, vasopressor therapy is started with infusion of dopamine (2–20 μg/kg/min) or norepinephrine (0.5–20 μg/min) through the central line. Vasopressin (0.01–0.04 units/min) infusion may work if hypotension does not respond to norepinephrine or dopamine. In cardiogenic shock, inotropic agents such as dobutamine (intravenous infusion at 2–20 μg/kg/min) can be started when the MAP is less than 60 mm Hg once intravascular volume status is optimized. Most vasopressors are known to negatively affect the uteroplacental circulation. Phenylephrine and ephedrine are the most commonly used drugs and possibly the safest in the treatment of hypotension associated with regional anesthesia. Maternal heart rate can be used as a guide to therapy when using phenylephrine or ephedrine. However, in situations such as shock, if one vasopressor is believed to have a clear benefit compared with another, the clinician should not hesitate to use the most beneficial drug while monitoring fetal well-being. Many patients with septic shock have relative adrenal insufficiency. Some investigators recommend intravenous hydrocortisone (200–300 mg/d in 3–4 doses) only if the basal serum cortisol level is less than 150 μg/L or if the cortisol level fails to increase by 90 μg/L after adrenocorticotropic hormone (ACTH) stimulation, whereas others treat all patients with corticosteroids.[18] In a multicenter randomized controlled trial, hydrocortisone did not improve survival, although hydrocortisone hastened reversal of shock in patients in whom shock was reversed.[24]

The cause of shock should also be aggressively managed. Septic shock is treated with antibiotics and control of the source of sepsis. Blunt curettage of the infected uterus or aspiration or surgical drainage of pelvic abscesses may be required.

Postpartum hemorrhage remains one of the leading causes of preventable maternal mortality, both in the developed and the developing world. Initial assessment of a bleeding patient requires monitoring blood pressure, pulse, capillary refill, mental status, and urinary output.[19] Severe hemorrhage is associated with peripheral vascular constriction, depression of mental status, and severe hypotension leading to multiorgan failure.[25] In postpartum hemorrhage, correction of coagulopathy and thrombocytopenia is vital. When medical therapy is unsuccessful, surgical approaches to postpartum hemorrhage are often considered. These may include uterine curettage,

laceration repair, balloon tamponade, embolization, compressive suture techniques, uterine or hypogastric artery ligation, and ultimately hysterectomy as a last resort. Blood and blood products should be transfused based on coagulation profile and massive transfusion protocol (MTP) activated. Most trauma centers have an MTP in place. Burtelow and colleagues[26] have successfully used 6:4:1 (red blood cells/fresh-frozen plasma/platelets) for immediate resuscitation in massive obstetric hemorrhage in their obstetric unit. Recombinant activated Factor VII has recently become available. Initial reports of its use in women with exsanguinating obstetric hemorrhage are encouraging. It is extremely expensive and should only be considered when conventional therapy fails.[19]

Hypertensive Crisis

Arterial pressure greater than 160/110 mm Hg in preeclampsia can result in pulmonary edema, seizures, intracerebral hemorrhage, and requires rapid blood pressure control. Intravenous labetalol 20 mg can be given initially followed by a 40-mg dose and 2 80-mg doses at 10-minute intervals until blood pressure is controlled or a cumulative dose of 300 mg is reached. Once initial blood pressure is controlled, a continuous infusion at a rate of 0.5 to 2 mg/min of labetalol can be used instead of intermittent dosing. As delivery is the ultimate treatment of resistant gestational hypertension, it may obviate the need to use continuous infusions in many cases. Another effective intravenous agent, hydralazine, is administered at a dose of 5 to 10 mg every 20 minutes (maximum of 40 mg) until blood pressure is controlled. Reduction of pressure to normal levels (<140/90 mm Hg) should be avoided as it may compromise placental perfusion. Hypertension refractory to these drugs is an indication for intravenous nitroglycerin (10−100 μg/min) or sodium nitroprusside (2−8 μg/min). Prolonged use of nitroglycerin may lead to methemoglobinemia. Cyanide toxicity in the mother and fetus may occur with sodium nitroprusside, limiting its use to less than 4 hours and only as a last resort.

Peripartum Cardiomyopathy

Even in the absence of preexisting heart disease, cardiac failure may occur as a result of peripartum cardiomyopathy (PCCM). PCCM is defined as cardiomyopathy that develops in the last month of gestation or in the first 5 months in the postpartum period without any identifiable cause.[27,28] The incidence is 1:3000 to 1:15,000 live births in the United States. Pathogenesis is poorly understood. However, infections and immunologic and nutritional causes have been implicated. Clinical presentation includes the usual signs and symptoms of heart failure. Diagnosis is based on clinical presentation of congestive heart failure and objective evidence of left ventricular systolic dysfunction. Early diagnosis and initiation of treatment are essential to optimize the outcome of the parturient. Medical management comprises sodium restriction, loop diuretics, afterload reducing agents (hydralazine, nitrates), and digoxin. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers should be avoided during pregnancy because of severe adverse neonatal effects and can be substituted for by hydralazine and nitrates during pregnancy. Some ACE inhibitors can be used in the postpartum period even in women who are breast feeding. Patients with persistent left ventricular abnormalities have a poor prognosis.[29]

If medical therapy fails, patients may then be treated with mechanical circulatory support devices and (or) cardiac transplantation. Mechanical assist devices can be used as a bridge to recovery or a bridge to transplantation.[29−31]

Cardiopulmonary Resuscitation in Pregnancy

During cardiopulmonary resuscitation in pregnancy, the team needs to follow the revised 2005 American Heart Association guidelines with modifications to compensate for the altered anatomy and physiology of pregnancy as outlined later. The major modifications include (1) prompt airway management, (2) special attention to lateral displacement of uterus and avoidance of aortocaval compression, (3) optimal performance of chest compressions in the lateral decubitus position, (4) caution in the use of sodium bicarbonate as it may not cross the placenta quickly enough to reverse fetal acidosis, and (5) early consideration of perimortem cesarean delivery to optimize CPR and survival of mother and baby.[32,33]

Amiodarone has a large iodine load and may have an adverse effect on fetal thyroid function; however, advantages should be weighed against this risk of fetal thyroid dysfunction and the drug used if it is considered to be the best option. Vasopressin can be cleared more quickly starting mid pregnancy and may need to be substituted with epinephrine.

Perimortem Cesarean Delivery

Perimortem cesarean deliveries were recommended in 1986.[34] Katz and colleagues[34] recommended a 4-minute rule from the maternal arrest to the initiation of the cesarean delivery, with the fetus being delivered within 5 minutes. This approach was promoted principally on fetal grounds to allow

the potential salvage of a viable fetus. The timing of delivery was based on theoretic considerations such as oxygen consumption, and prevention of neurologic injury. Since the initial description by Katz and colleagues,[35] numerous case reports have described often dramatic reversal of the maternal hemodynamic collapse, even in refractory situations. If initial resuscitation is not effective during cardiac arrest in pregnancy, delivering the fetus within 5 minutes may facilitate maternal and fetal survival. The 5-minute rule from arrest to delivery is now recommended by the American Heart Association when the intrauterine gestation is greater than 24 weeks (the cut-off for fetal viability).

RESPIRATORY FAILURE

Numerous insults can lead to acute lung injury and ARDS. ARDS is a form of respiratory failure characterized by acute hypoxemia and increased alveolar-capillary permeability resulting from diffuse pulmonary inflammation. Risk factors for ARDS can be classified into 4 main categories: (1) sepsis from pulmonary or nonpulmonary sources, (2) major trauma, (3) transfusion with multiple-unit blood products, and (4) aspiration of gastric contents. Eighty-five percent of ARDS cases result from 1 of these risk factors and sepsis accounts for up to 50% of all cases.[36] Risk factors for ARDS in pregnant patients can be divided into causes that are unique to pregnancy and those that are not unique to pregnancy as outlined in **Box 2**.

Box 2
Causes of ARDS

- Unique to pregnancy
  - Preeclampsia/eclampsia
  - Tocolytic-induced pulmonary edema
  - Aspiration of gastric contents
  - Chorioamnionitis
  - Amniotic fluid embolism
  - Placental abruption
  - Obstetric hemorrhage-related cause
  - Endometritis
  - Retained placental products
  - Septic abortion
- Not unique to pregnancy
  - Sepsis
  - Pneumonia
  - Severe trauma
  - Multiple transfusions
  - Aspiration of gastric contents
  - Acute pancreatitis
  - Fat emboli
  - Near drowning

Data from Katz VL, Dotters DJ, Droegemueller W. Perimortem cesarean delivery. Obstet Gynecol 1986;68:571–76.

In cases of ARDS and obstructive lung disease, achieving normocapnea leads to more volutrauma, biotrauma, and atelectrauma. The current goals of ventilation are permissive hypercapnea, and maintaining the pH between 7.25 and 7.35. This is accomplished predominantly by the use of small tidal volumes. It is not unusual to allow a $Paco_2$ of 60 mm Hg or higher. In pregnancy there are no clear-cut data on the role of permissive hypercapnea. There are a few short-term animal experiments evaluating the effects of hypercapnia on uteroplacental blood flow but no long-term studies. The hypercapnia was induced rapidly without providing time for changes in the compensatory mechanisms to bring the pH to more normal levels. On the contrary, in an anaesthetized sheep model, Walker and colleagues[37] found no significant changes in uterine blood flow even when the maternal $Paco_2$ reached 60 mm Hg. At greater than 60 mm Hg $Paco_2$ they noted increased uterine vascular resistance, resulting in decreased uterine blood flow. There are no human data regarding the effect of permissive hypoventilation on uteroplacental and umbilical blood flow. Human extrapolation of animal data may not be valid for multiple reasons. The permissive hypoventilation as applied to patients with ARDS is not a sudden change in minute ventilation, but a gradual change while monitoring the patient. The ARDS net protocol provides guidance with regard to the rapidity of CO_2 accumulation and the pH change.[38] Sodium bicarbonate infusions can be given to compensate for severe acidosis without causing maternal alkalosis. There are no published studies investigating the use of low tidal volumes in the treatment of pregnant patients with acute lung injury and ARDS. However, a fetal-maternal gradient of $Paco_2$ is around 10 to 13 mm Hg, resulting in a higher $Paco_2$ in the fetus and a potential for fetal acidosis, an increase in intracranial pressure, and a right shift in the hemoglobin dissociation curve. In addition, hypercapnia in the first 72 hours of life may lead to retinopathy of prematurity. Although bicarbonate infusion can reverse maternal acidosis, the rate of transfer of these ions across the placenta is not well studied and varies between species. It is possible that the

rate of transfer of bicarbonate to the human fetus may not occur fast enough to correct fetal acidosis. The proven efficacy of a low tidal volume strategy in nonpregnant patients with ARDS provides strong support for its universal use.[39] In managing the ARDS patients, the authors routinely use small tidal volumes, permissive hypoventilation (up to $Paco_2$ of 60 mm Hg) while closely monitoring the fetal status with the biophysical profile.

Data on fetal oxygenation are derived from sheep models and are discussed by Tomimatsu and colleagues.[40] Oxygen levels should be closely monitored in pregnancy and kept higher than in nonpregnant women, especially in patients with acute changes in oxygenation who may not have a normal placental oxygen transport. Oxygen tension levels should be monitored rather than oxygen saturations because it is the difference in Pao_2 at the placental level that determines oxygen transfer.

Another caveat to keep in mind is that positive pressure ventilation and the application of positive end expiratory pressure increase intrathoracic pressure and decrease venous return. In a pregnant patient, these effects remain true and may be worsened by compression of the inferior vena cava by the gravid uterus, which may reduce venous return further and lead to a decreased urine output.

Management of respiratory failure in pregnancy is similar to management in nonpregnant women, although being mindful of the normal physiologic changes that occur in the parturient. Protective mechanical ventilation using smaller tidal volumes, elevation of the head of the bed to prevent ventilator-associated pneumonia, and routine use of spontaneous awakening trials and spontaneous breathing trials minimize complications caused by mechanical ventilation.

ACUTE RENAL FAILURE

Creatinine clearance is increased in pregnancy to 120 to 160 mL/min and serum creatinine level decreases to 0.4–0.7 mg/dL. Acute renal failure comprises oliguria, azotemia, and metabolic acidosis. It has become rare in the developed world with an incidence of approximately 1:15,000, but continues to be associated with significant mortality and long-term morbidity. The most common cause of renal failure in pregnancy is preeclampsia. Other pregnancy-specific causes include acute fatty liver of pregnancy, thrombotic thrombocytopenic purpura, amniotic fluid embolism, infection, sepsis, intravascular volume depletion, obstruction, or idiopathic causes.[6]

The initial management of renal failure is similar to nonpregnant patients. Nephrotoxins (aminoglycosides, radiocontrast dye) should be avoided if possible. Drugs should be dosed based on renal function with particular attention to magnesium. Dopamine has been administered for both prevention and treatment of acute renal failure in critically ill patients. However, clinical studies have not demonstrated the efficacy of this approach and it is not recommended for routine use for either prophylaxis or treatment of acute renal failure.[41] Initiation of dialysis seems to be safe when indicated. Indications for dialysis include intravascular volume overload, hyperkalemia refractory to medical management, metabolic acidosis, or symptomatic uremia.

SUMMARY

Critical illness may complicate any pregnancy. Obstetricians must be familiar with the issues pertaining to care of pregnant women with multiple organ failure. Many obstetric disorders may mimic medical disorders. Once the correct diagnosis is made, the obstetrician and the intensivists must decide whether delivery will alter the natural history of the disease process and improve maternal survival. If the maternal condition is expected to improve after delivery, then the decision to deliver vaginally or by cesarean section must be made. Fetal viability should obviously be taken into consideration. Hypovolemia, hypotension, and respiratory failure are treated while preparations are made to deliver the fetus. Timely delivery improves not only maternal outcome but also fetal outcome. No efforts should be spared in the management of critically ill obstetric patients because their outcomes are often dramatically better than expected from the initial severity of illness.

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