Focus on: Obstetrics

Critical care of the obstetric patient

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Pregnancy is often regarded as a simple process that proceeds without complication, but a significant proportion of women will develop critical illness requiring intensive care. Once admitted to an intensive care unit (ICU) the age-adjusted mortality rate for obstetric patients may also be high, despite a fall in the overall maternal mortality rate.

Critical illness may develop as a direct result of pregnancy or from a coincidental disease, aggravated by the physiological demands of pregnancy. Its management in the face of altered maternal physiology, diseases specific to pregnancy and the presence of a fetus is a unique challenge.

We review the normal physiological changes of pregnancy, the general principles of intensive care in the pregnant patient, including a review of respiratory support, haemodynamic monitoring, drug therapy, nutrition, radiological considerations and specific management of pre-eclampsia, trauma, septic shock, and acute lung injury in pregnancy. © 2000 Harcourt Publishers Ltd

Key words: intensive care, pregnancy.

Introduction

Although pregnancy is usually a natural process that proceeds without complication, approximately 0.1–0.9% of pregnant women will develop critical illness requiring intensive care. Once admitted to an intensive care unit (ICU), the age-adjusted mortality rate for obstetric patients is high at 4.5–20%, despite a fall in the overall maternal mortality rate.4–6

Critical illness may develop as a direct result of pregnancy or from a coincidental disease, aggravated by the physiological demands of pregnancy. Its management in the face of altered maternal physiology, diseases specific to pregnancy and the presence of a fetus is a unique challenge. Hypertensive disorders of pregnancy, acute respiratory failure and sepsis remain the major indications for admission.4 Obstetric haemorrhage is covered fully in another article.

Failure to provide intensive care is associated with increased mortality and morbidity in obstetric patients and early admission may minimize progression to multiple organ failure.7

In this review we consider the normal physiological changes of pregnancy, the general principles of intensive care in the pregnant patient and specific management of a number of conditions.

Physiological changes in pregnancy

Cardiovascular system

Maternal blood volume increases to 40% above normal values by 30 weeks’ gestation. Increased levels of prostacyclin and arteriovenous shunting to the placental bed
cause a decrease in systemic vascular resistance (SVR). Cardiac output increases by 40% through an increase in stroke volume and heart rate, maintaining blood pressure. A dilutional anaemia occurs because plasma volume increases by 40%, while red cell mass only increases by 20%. This provides some protection against haemorrhage at delivery. Cardiac output increases a further 10–15% during labour with an increase in venous return from the contracting uterus coupled with maternal pain and effort.

All this represents a significant stress to the cardiovascular system, greatest in the third trimester, during labour and immediately after delivery, when circulating blood volume transiently increases in preparation for a postpartum diuresis.

In the supine patient, caval compression by the gravid uterus causes a decrease in venous return and cardiac output, with a decrease in placental perfusion. In extreme cases, compression of the aorta may also occur, impairing uterine blood flow and causing fetal distress. Aortocaval compression may be minimized by placing the patient in the full left lateral position.

Respiratory system

Changes in respiratory drive, thoracic cage mechanics and airway aperture affect the respiratory system. Increased tidal volume and a smaller increase in respiratory rate raise minute ventilation by about 50% at term. Elevated serum progesterone levels are thought to augment ventilation by increasing the sensitivity of the medullary respiratory centre to carbon dioxide (CO₂), so producing a respiratory alkalosis. Renal compensation maintains blood pH in the normal range.

Cephalad displacement of the diaphragm by the gravid uterus causes a progressive decrease in functional residual capacity (FRC) of 10–20% by term. Oxygen consumption increases by 20–30% by the third trimester and during labour there is a further increase to 40–75% above baseline, making the pregnant patient particularly susceptible to the rapid development of hypoxia, secondary to hypoventilation or apnoea.

Vital capacity remains unchanged owing to a widening in the diameters of the thoracic cage. Although lung compliance is unchanged, chest wall and total respiratory compliance are reduced in late pregnancy. Besides these changes in respiratory parameters, there are structural changes to the airways during pregnancy rendering them oedematous and friable.

Renal system

Principally as a result of increased renal blood flow, glomerular filtration rate is increased during pregnancy by approximately 50%. Consequently, plasma urea and creatinine levels decrease by 40–50%, so that values within the ‘normal’ (non-pregnant) range in parturients may suggest evidence of renal dysfunction. Dilatation of the renal pelvis and ureters during pregnancy may increase the incidence of urinary tract infections. Physiological diuresis occurs between the second and fifth days postpartum.

Gastrointestinal system

Gastric motility and food absorption are reduced in pregnancy, although basal gastric pH and acid output are unchanged. There is a decrease in lower oesophageal sphincter tone caused by an increase in plasma progesterone levels. Gastric emptying is further delayed by narcotic analgesia, increasing the risk of aspiration of stomach contents into the early postpartum period.

Fetal considerations

Oxygen delivery to the feto-placental unit is the product of maternal arterial oxygen content and uterine blood flow. However, arterial oxygen content is reduced by 20–25%, due to the anaemia of pregnancy. Therefore, maintenance of maternal cardiac output is critical to fetal oxygen delivery, since the uterine vasculature is normally maximally dilated and can not adapt to a fall in maternal cardiac output.

In any critically ill obstetric patient, the effects of intensive care management on fetal well being should be considered. Delivery of the baby may be the most appropriate solution, depending on gestational age. If this is not possible, uteroplacental oxygen delivery must be maximized. Increasing maternal oxygen carrying capacity by transfusion, and enhancing cardiac output and oxygenation can often improve fetal oxygen delivery, but simple manoeuvres such as left lateral tilting should not be overlooked.

General principles of ICU management

Respiratory support

The management of the pregnant patient with respiratory failure is similar to that of other patients but has certain additional considerations.

Tracheal intubation and controlled ventilation is indicated for airway protection, pulmonary toilet, or management of severe hypoxia and ventilatory failure, as manifest by significant uncompensated respiratory acidosis. Mucosal hyperaemia of the upper respiratory tract results in narrowed upper airways which bleed easily, and tracheal intubation may be difficult.

There is little information concerning prolonged ventilation in the pregnant patient, and data are derived principally from experience with anaesthetic ventilation for operative delivery. It is appropriate to aim for an arterial CO₂ tension (PₐCO₂) of 4 kPa to mimic the mild respiratory alkalosis of pregnancy, but significant hypocapnia and alkalosis may decrease placental blood flow and fetal oxygenation. The current trends of controlled hypoventilation and permissive hypercapnia, avoiding high ventilatory pressures (and hence alveolar overdistention), have not been evaluated extensively in pregnancy. Early information suggests that provided there is adequate oxygena-
tion, maternal $P_{\text{CO}_2}$ may be allowed to rise to 8 kPa without affecting the fetus. In fact, fetal oxygenation may actually be improved.  

Non-invasive ventilation techniques in acute respiratory failure are rarely used during pregnancy because of the risks of oesophageal reflux and aspiration. Consequently, few data exist concerning their use.

The use of a variety of unconventional modes of respiratory support in pregnant patients including extracorporeal membrane oxygenation, extracorporeal $CO_2$ removal, high-frequency jet ventilation and the use of an intracaval oxygenator have been described in case reports, but clinical experience is limited.

Haemodynamic monitoring

The indications for haemodynamic monitoring during pregnancy are similar to those in other patients. As stressed already, interpretation of haemodynamic data must take into account the normal physiological changes in cardiac output and systemic vascular resistance during pregnancy. Central venous and pulmonary capillary wedge pressures are normally unchanged in pregnancy. Left ventricular function is usually well preserved even in the presence of significant pre-eclampsia. Since it is difficult to demonstrate a significant outcome benefit of pulmonary artery catheterization in pregnant patients, recommendations for its use must remain qualified. In addition, there are significant risks of internal jugular venous cannulation which may be worse in the pregnant patient at term. The use of ‘long line’ cephalic vein catheterization may be safer.

In addition, monitoring of the fetal heart rate and wellbeing may give valuable clues to maternal haemodynamic status.

Drug therapy

Fetal concerns often limit prescription of drugs during pregnancy. The major risk period for teratogenesis is the first ten weeks of gestation. During the second and third trimesters, drugs may affect the growth and functional development of the fetus or have toxic effects on fetal tissues. Drugs given shortly before term may have adverse effects on labour or on the neonate after delivery.

The British National Formulary contains a list of drugs to be avoided or used with caution in pregnancy, although absence of a drug from the list does not imply safety.

Inotropes. Although inotropic support is often vital for the critically ill mother, it may cause fetal compromise through uterine vasoconstriction.

Ephedrine has been shown to increase maternal blood pressure while preserving uterine blood flow and is associated with an improvement in fetal hypoxia and acidosis associated with hypotension. Recently, both ephedrine and phenylephrine have been shown to be safe and effective in treating maternal hypotension following spinal anaesthesia, without adversely affecting neonatal outcome. Unfortunately, tachyphalaxis limits the effectiveness of ephedrine, and it is unsuitable for use as a continuous infusion in the ICU.

Dobutamine, noradrenaline and adrenaline, despite favourable maternal haemodynamic responses, have adverse effects on uterine blood flow in animal studies. Studies of dopamine, yield conflicting results. If these inotropes are necessary they should not be withheld, but careful monitoring, and if necessary, delivery of the fetus will help avoid fetal compromise, unless it is very premature.

Antihypertensive drugs. A number of vasodilator agents are used in the treatment of pregnancy-induced hypertension. Hydralazine is the most widely used agent and has a record of safety and efficacy in pregnancy. Labetalol, a valuable alternative, combines $\alpha$- and $\beta$-adrenoceptor blocking activity and has been shown to control maternal blood pressure effectively, without adversely affecting uterine blood flow. Methyldopa has a long history of effective use for the treatment of essential hypertension in pregnancy and a good record with respect to fetal and neonatal safety.

Nifedipine, a calcium antagonist, has been used but it may cause severe hypotension in combination with magnesium sulphate in pre-eclampsia.

Angiotensin-converting enzyme inhibitors carry a risk of serious fetal renal dysfunction and should be avoided if possible. There are more than 30 case reports of perinatal renal failure, many of which have been fatal, following in utero exposure to captopril or enalapril.

Antiarrhythmic agents. Supraventricular and ventricular arrhythmias are more common during pregnancy, and may cause serious haemodynamic disturbance in the critically ill.

Supraventricular arrhythmias may be diagnosed and treated with intravenous adenosine. Heart block or transient asystole are possible complications, since adenosine slows conduction through the atrioventricular node. Although it probably crosses the placenta, no adverse fetal effects have been identified. Verapamil, a calcium channel-blocker, may also be used to terminate paroxysmal supraventricular tachycardia in pregnancy, and has been used to treat fetal arrhythmias as it crosses the placenta.

Ventricular tachyarrhythmias in pregnancy are often responsive to $\beta$-adrenoceptor blockade or to type I antiarrhythmic drugs. However, cardioversion or synchronous defibrillation should not be withheld during pregnancy if clinically indicated. Lignocaine has been found to be safe and effective in the pregnant patient. However, it does cross the placenta and may affect the fetus if maternal plasma levels are high. Amiodarone can be used during pregnancy to treat maternal and fetal arrhythmias but the
major metabolite, desethylamiodarone, crosses the placenta and may cause neonatal hypothyroidism.

**Drugs for analgesia, sedation and neuromuscular blockade.** Benzodiazepines such as midazolam and diazepam cross the placenta and may increase the risk of cleft palate when used early in pregnancy, but opiate analgesics such as morphine, pethidine and fentanyl do not appear to cause congenital malformation. Short term use of non-depolarizing neuromuscular blockers also appears to be safe.

**Nutrition**

Nutrition is vital in the critically ill pregnant patient. If intake is low, maternal energy stores are maintained despite the needs of the fetus. If starvation occurs before 26 weeks’ gestation, intrauterine growth retardation and neurological impairment may result. Pregnancy increases calorific requirements by approximately 300 kcal/day. In critical illness, optimal nutritional support may be provided by supplying approximately 80% of estimated energy requirements with 20% of the calories administered as lipids. Protein intake should be augmented by 20–50% to 1.5 g/kg/day, in order to meet the demand for maternal and fetal protein synthesis; all protein or amino acid preparations should include glutamine. There is an increased need for vitamins and minerals including iron, zinc and magnesium. Serum calcium, phosphate and magnesium levels also tend to fall in critical illness.

Enteral nutrition is the method of choice, preferably by nasoduodenal feeding in view of reduced lower oesophageal sphincter tone. Total parenteral nutrition has been successfully administered to pregnant patients for up to several months in a variety of disorders, including inflammatory bowel disease, oesophageal stricture and malignancy; however its use in critically ill obstetric patients is less well described. There are concerns over the use of lipid emulsions leading to fatty infiltration of the placenta and premature labour, but these have not been supported by clinical studies.

**Radiological considerations**

Despite the risk of exposing the fetus to ionizing radiation, radiological procedures (particularly chest X-rays) are imperative for investigation and should not be withheld. A chest radiograph exposes the maternal lungs to approximately 0.5 cGy and fetal exposure may be reduced to about 0.001 cGy with lead shielding. Significantly more fetal exposure occurs with abdominal and pelvic investigations, such as computed tomography which can deliver between 5 and 10 cGy to the fetus. The potential adverse effects of radiation exposure to the fetus include teratogenicity and oncogenicity; low dose (under 5 cGy) exposure is associated with a slightly increased risk of childhood cancer but does not predispose to congenital developmental abnormalities. It is likely that the risk to the fetus increases with the dose of radiation and therefore every effort should be made to limit this, particularly in the first trimester.

**Management of specific conditions in intensive care**

**Pre-eclampsia**

Pre-eclampsia is responsible for 20–60% of obstetric admissions to the ICU; the main admission indications for ICU in pre-eclampsia are listed in Table 1. The overall management of pre-eclampsia and eclampsia are covered in the article on obstetric emergencies. Here we will review some of the most serious complications of pre-eclampsia needing ICU admission.

**Pulmonary oedema.** Pulmonary oedema occurs in approximately 3% of severe pre-eclampsics, and usually presents in the early postpartum period. It may be associated with aggressive fluid administration at a time when plasma colloid osmotic pressure is low and fluid leaks out into the alveoli. Fluid restriction and diuretic therapy may be guided by invasive haemodynamic monitoring. Pulmonary oedema associated with pre-eclampsia is a particular danger in a subgroup of chronically hypertensive, obese women who exhibit evidence of left ventricular dysfunction with elevated filling pressures. 21

**Renal dysfunction.** Oliguria commonly occurs in pre-eclampsia as a result of generalized arterial vasospasm, but only rarely proceeds to renal failure. Vascular permeability is increased with a fall in plasma colloid osmotic pressure, which leads to generalized oedema and expansion of the interstitial space in the face of a constricted intravascular compartment. This makes fluid balance difficult to assess, but in the face of the vascular changes described, the worst mistake is to give too much fluid, resulting in pulmonary oedema, rather than too little.

**HELLP syndrome.** HELLP syndrome characterized by microangiopathic Haemolytic anaemia, Elevated Liver enzymes and Low Platelets may develop in 4–20% of

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<td><strong>Mechanical ventilation</strong></td>
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<td><strong>Disseminated intravascular coagulation</strong></td>
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patients with severe pre-eclampsia. Maternal and perinatal mortality rates of 24 and 33% respectively have been reported. It is important to remember that HELLP syndrome may present after delivery of the fetus in 30% of cases. Even with supportive ICU management, HELLP syndrome is associated with a poor outcome, despite a number of novel strategies including, antiplatelet agents, thromboxane synthetase inhibition, steroids and plasmapheresis. The maternal complications of HELLP syndrome include, acute renal failure, acute respiratory distress syndrome and haemorrhage. Haemorrhage occurs in 2% of cases and may progress to hepatic rupture. It may be diagnosed clinically or by ultrasound.

Trauma

Death from road traffic accidents is a leading cause of non-obstetric maternal mortality. The pregnant woman is susceptible to unique injuries including, amniotic membrane rupture, uterine rupture and direct fetal trauma. With increasing gestation, the uterus becomes increasingly vulnerable to trauma and particularly haemorrhage as blood flow to the pelvis is increased. Placental abruption and uterine rupture are the most serious sequelae of blunt abdominal trauma in late pregnancy. Cephalad displacement of abdominal contents by the uterus increases the risk of visceral injury following penetrating trauma of the abdomen. The bladder assumes an intra-abdominal position and becomes a target for injury after the 12th week of gestation.

Once again, maternal haemodynamics are affected by the physiological changes of pregnancy. Increased maternal blood volume after 20 weeks' gestation tends to delay signs of hypovolaemia until blood loss is very severe. Lesser degrees of hypovolaemia may induce vasoconstriction that maintains perfusion to maternal vital organs at the expense of uteroplacental blood flow; so signs of fetal distress are a sinister marker of significant maternal haemodynamic compromise. Severity of maternal injury and haemodynamic compromise, as well as direct fetoplacental injury, are the factors which determine fetal death.

Septic shock

Ten per cent of direct maternal deaths in the UK between 1994 and 1996 were associated with infection, and septic shock remains a major cause of maternal death. Septic shock occurs most commonly in the peripartum period, often following chorioamnionitis, postpartum endometritis, septic abortion and urinary tract infection. There is an association with operative delivery, prolonged rupture of membranes, retained products of conception and prior instrumentation of the genito-urinary tract. Although Gram-negative coliform are the most frequent causative organisms, aerobic and anaerobic streptococci, bacteroides and clostridia have been implicated. Empirical antibiotic regimens should provide broad Gram-negative, Gram-positive and anaerobic cover for what is typically a polymicrobial infection.

Prompt volume expansion, to optimize cardiac output and tissue oxygen delivery, in conjunction with invasive haemodynamic monitoring to guide inotropic therapy is indicated. A microbiological diagnosis should be sought to guide therapy, and surgical drainage of infected collections is indicated.

Respiratory disorders of pregnancy

Acute lung injury

The pregnant patient is at risk of acute lung injury from obstetric causes and coincidental pulmonary insults. It frequently precedes the development of multiorgan failure. Common obstetric causes include, pre-eclampsia, obstetric haemorrhage, amnionitis or endometritis and amniotic fluid embolism.

The diagnosis is established on clinical and radiological grounds. Maternal stabilization and the general principles of respiratory support have been discussed above. Management strategies must consider the cardiorespiratory alterations of pregnancy and the well-being of the fetus. Both diuresis and positive end expiratory pressure may diminish maternal cardiac output, and inotropic agents may reduce uteroplacental perfusion.

If the maternal condition continues to deteriorate, delivery of the fetus should be considered as a therapeutic option. This is likely to be advantageous for three reasons. First, the fetus is unlikely to prosper in an increasingly hostile environment, and abrupt fetal deterioration may accompany progression of acute lung injury in the mother. Second, delivery per se, may improve the maternal condition, and third, delivery of the baby increases the range of treatment options available for the mother in the postpartum period.

Although it is difficult to predict survival for individual patients with acute lung injury during pregnancy, prolonged ventilation is not necessarily associated with excessive maternal mortality. In fact, survival of acute lung injury during pregnancy is marginally better than in the general population, reflecting the young age of the group, and the transient nature of many of the predisposing conditions.

Pulmonary oedema

There are a number of conditions that may cause pulmonary oedema during pregnancy, including pre-eclampsia (vide supra), tocolytic therapy and cardiac dysfunction.

Tocolytic induced pulmonary oedema. Acute pulmonary oedema is a recognized complication of β-adrenergic agonists such as ritodrine and terbutaline, which are used to inhibit the onset or progress of labour. The pathophysiological mechanisms remain unclear, but may involve a combination of catecholamine induced myocardial dysfunction, fluid overload, increased capillary permeability and reduced colloid osmotic pressure in pregnancy. The
Concomitant use of steroids to enhance fetal lung maturation may contribute by promoting fluid retention. Typically it presents with acute respiratory distress, subternal chest pain in 25% of cases and signs of pulmonary oedema during or within 12 h of tocolytic therapy. Discontinuation of tocolytic agents and treatment with oxygen and diuretic is often sufficient.

**Cardiogenic pulmonary oedema.** Cardiogenic pulmonary oedema is an important differential diagnosis of acute respiratory failure in pregnancy. The haemodynamic stress of labour and delivery make this an especially hazardous time for women with cardiac dysfunction. Invasive monitoring may be helpful in following fluid shifts and optimizing haemodynamic parameters. The vasodilatory effects of epidural anaesthesia may be of benefit in these patients; however, caution needs to be exercised as a precipitous fall in afterload may lead to acute decompensation in patients with aortic stenosis, hypertrophic cardiomyopathy or pulmonary hypertension. 26

**Conclusion**

Successful management of the critically ill obstetric patient requires an awareness of the normal cardiorespiratory changes of pregnancy, familiarity with pregnancy-specific diseases and a consideration for the well being of both mother and fetus. Recognition of the severity of the patient’s condition, followed by rapid referral to an ICU is vital to reduce maternal mortality and morbidity.

**References**