Chapter 2
Management Principles of the Critically Ill Obstetric Patient

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Pregnant women constitute a small percentage of admissions to the intensive care units in most institutions. However, given the profound physiologic changes of pregnancy, these women present a challenge for most practitioners. Pregnancy physiology is discussed in the context of the various interventions described below. This chapter will review basic interventions in the ICU to help the clinician manage this unique population.

Pregnancy Physiology

Many anatomical and physiological changes occur during pregnancy that impact the management of critically ill gravidas.

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Respiratory Changes

Both the anteroposterior and the transverse diameters of the chest increase in pregnancy, and the diaphragm moves upward by about 4 cm [1]. Enlargement of the breasts may affect chest compliance and the ease of intubation [2]. The increase in blood and interstitial fluid volume is responsible for the development of mucosal edema especially in the oropharyngeal and laryngeal structures reducing the airway diameter. Hyperemia, which is possibly related to hormonal changes and the increase in plasma volume, makes the mucosa friable and prone to bleeding with minimal trauma. This increased friability may then compromise the visualization of the upper airway landmarks. Longitudinal upper airway assessment with Mallampati grading system suggests that airways become less patent with pregnancy progression (Pilkington). In addition, Mallampati grades I–II may progress to III–IV during labor and do not return to pre-labor grades for an additional 12 h [2].

During pregnancy, oxygen consumption is increased by 30–60 % [2]. There is a 20 % decline in functional residual capacity by term. An additional drop of nearly 25 % may be observed in the supine position [2]. The increase in oxygen consumption and the reduction in functional residual capacity in the supine position contribute to decreased oxygen reserve and lead to faster desaturations in response to apnea. A study by Choi et al. has shown that compared to nonpregnant controls and following sedation and the use of paralytics, pregnant women have a significantly faster decline in oxygenation and increase in PaCO2 in response to induced apneas [3]. Thus, intubation protocols should be modified in pregnant women to account for these changes.

Cardiovascular Changes

Pregnancy is associated with profound hemodynamic changes that prepare gravidas for the blood loss that is anticipated during delivery. Blood volume increases by 40–52 % by the end of gestation [4, 5]. This increase begins as early as 6 weeks gestation, peaks in the late second trimester, and then plateaus. Blood volume in pregnancy varies but is generally 1,200–1,600 ml greater than in a nonpregnant individual [6]. The rise in blood volume results from an expansion of erythrocytes as well as significant rise in plasma volume. The latter is due to an increase in sodium and water retention related to enhanced renin-angiotensin-aldosterone system activity, and an increase in the hepatic production of renin related to the hyperestrogenic state of pregnancy [1]. The disproportionate increase in red blood cells to plasma volume results in a physiologic anemia of pregnancy [7].

Heart rate increases by 10–20 % during gestation, but this increase usually occurs in the latter part of pregnancy. Cardiac output (CO) starts rising in the first trimester, peaking by the end of the second trimester [8, 9], and may be as high as 50 % above prepregnancy values. This rise is more pronounced in twin, compared to singleton

pregnancies. The rise in CO is caused by a significant increase in preload and stroke volume in the early part of gestation. CO is then maintained in the second half of pregnancy primarily by an increase in heart rate. Nearly 17 % of cardiac output is directed to the uterine arteries to supply a flow of 500 ml/min to the placenta and the growing fetus. Both central venous and pulmonary capillary wedge pressure are unchanged in pregnancy.

Systemic vascular resistance falls early in pregnancy as a result of vasodilation in response to hormonal stimulation [10]. Despite an increase in cardiac output, the end result is a fall in blood pressure (BP) in the first trimester. Diastolic blood pressure falls are more pronounced than systolic blood pressure changes and result in a widened pulse pressure. Mean BP decreases by 10–15 mm Hg starting in the first trimester with a nadir in BP occurring at approximately 24–28 weeks [11]. Similar changes occur in the pulmonary circulation, leading to unchanged pulmonary pressures despite the increase in cardiac output.

Plasma colloid osmotic pressure (COP) falls during normal pregnancy from 23.2 mm Hg in the first trimester to 21.1 mmHg at term [12] to 16 mm after delivery and that fall is even more pronounced in PEC [13]. This drop in colloid osmotic pressure not only contributes to the development of pulmonary edema even in the absence of elevated hydrostatic pressures but also contributes to its higher incidence in the postpartum, compared to the antenatal period.

Aortocaval compression caused by the gravid uterus in the supine position may result in decreased venous return, reduced filling of the right heart cavities resulting in maternal hypotension and potential for uteroplacental insufficiency in women with tenuous intravascular status [2]. Combining these changes with the increased oxygen consumption observed in pregnancy and labor and delivery, the decrease in FRC in the supine position and limited oxygen reserve and the reduction in cardiac output in the supine position may all compromise placental perfusion in susceptible individuals [1].

Gastrointestinal Changes

Gastrointestinal physiologic changes occurring in pregnancy and around labor and delivery complicate airway intubation further. A generalized slowing of the gastrointestinal tract function occurs in pregnancy due to the effect of progesterone on smooth muscles. Lower esophageal sphincter tone is reduced with a subsequent increase of acid reflux frequency [14]. During labor, gastric emptying is slower [2]. The gravid uterus compresses the duodenum and the stomach increasing the chances of regurgitation. When opioids are administered during labor or added to the epidural analgesia, gastric emptying is further delayed, but the use of local anesthetics in epidural analgesia alone has no effect [2].

Airway Management

Airway management in pregnancy remains a true challenging task, and pregnant women are eight times more at risk for tracheal intubation failure than the general surgical population. In addition to the usual indications for airway intubation that are relevant for the nonpregnant population, airway intubation in pregnancy may need to be performed when regional anesthesia around delivery fails or is contraindicated in cases such as sepsis, local infection at the site, coagulopathy, or spine abnormalities [2].

As in the nonpregnant population, predicting a difficult intubation or anesthesia complications requires the evaluation of several criteria. These criteria include Mallampati or modified Mallampati score, receding mandible, protruding upper incisors, and a short neck. Part of this assessment may be affected by pregnancy physiology. The physiological and anatomical changes related to pregnancy contribute to complications with sedation or rapid sequence induction procedures, reduction in upper airway size and gastrointestinal changes that may predispose to a higher risk of aspiration. The risk is further augmented by the urgency of certain obstetric interventions. The presence of comorbid conditions such as obesity or preeclampsia may also increase the risk of difficult airway management. In addition, the huge reduction in general anesthesia practice and the shift toward the safer locoregional methods in obstetric populations implicate a major reduction in teaching this technique to the younger generations of obstetric anesthesiologists, possibly impacting the level of comfort and competency in performing this intervention [1].

Thus, to limit the complications of difficult airway management, it would be reasonable to use regional anesthesia when appropriate but to also adopt a thorough and repeated evaluation of the upper airway in pregnant women and access the most experience provider for intubation.

Pathological Conditions Associated with Increased Risk of Difficult Airway Management in Pregnancy

Obesity

Obesity is a major issue in the modern world and has reached heights of an epidemic in certain developed countries. Despite efforts to reduce obesity, women of reproductive age and pregnant women are no exception [1]. Obesity is associated with numerous pregnancy-related complications, including anesthesia-related complications. Obesity is a risk factor for difficult intubation in the general as well as in obstetric population: a BMI > 26 kg/m^2 is by itself a risk factor for difficult mask ventilation [1]. Morbidly obese parturients have a higher incidence of cesarean delivery necessitating anesthetic intervention [2].

Preeclampsia and Eclampsia

Preeclamptic parturients have narrowed upper airways secondary to soft tissue swelling. This is well observed in sitting and supine position [15, 16]. Neck, facial, and tongue edema may be cues for possible difficult airway. Moreover, thrombocytopenia in the settings of preeclampsia constitutes a major risk since it shifts the choice from regional to general anesthesia in the case of an operative delivery. In addition, the risk of bleeding during airway intubation is higher with thrombocytopenia which may significantly impact visualization of the upper airway landmarks and complicate airway intubation further.

Preoperative Evaluation of Airways in Obstetrics

Identification of patients who may require airway intubation is a joint responsibility of the anesthesiologist and the obstetrician. In these situations, early planning of anesthetic management can be initiated. Many findings on physical exam may help in predicting difficult airway management including a Mallampati grade above II, a mouth opening less than 3.5 cm, a thyroid-chin distance less than 6.5 cm, and a sternum-chin distance less than 13.5 cm [1]. In addition, cervical spine and mandibular mobility need to be assessed.

Patients considered at an intermediate to high risk should have a comprehensive discussion of their analgesia options as well as alternate plans after failed intubation, if general anesthesia is required.

Management Steps

In summary, key points in airway management of pregnant women include airway assessment prior to intubation even in urgent situations; aspiration precautions such as elevation of the head of the bed, cricoid pressure, and possibly administration of citric acid/sodium citrate; smaller endotracheal tube size may be necessary; and lower doses of sedatives and anesthetics may be needed.

Although preoxygenation is a necessary step, care should be taken with manual ventilation to avoid the risk of aspiration. In patients who are hemodynamically compromised, the gravida may need to be placed in a supine position with a left-sided tilt to relieve inferior vena cava obstruction and improve cardiac output and organ perfusion. The tilt may be achieved by using the Cardiff wedge or the human wedge [17] or simply by using a manual uterine displacement.

Given the potential difficulties in airway management in pregnant women, the provider most experienced in airway management should be performing airway intubation. A difficult airway cart should be available. Laryngeal mask airway (LMA) is the first option in the difficult to intubate patient.

Hemodynamic Monitoring

Not surprisingly, pregnant women have been excluded from many critical care trials. Hence, many of the data are based on either pregnancy-specific disorders or on expert opinion and logical decisions informed by pregnancy physiology. Indications for hemodynamic monitoring in obstetrics are similar to the non-obstetric population but also include some pregnancy-specific disorders such as severe preeclampsia with refractory hypertension or severe cardiovascular collapse such as in the setting of an acute amniotic fluid embolism. Other indications in obstetrics include structural heart disease with a potential for decompensation during labor and delivery. Indications for hemodynamic monitoring in adult respiratory distress syndrome (ARDS) or septic shock are unchanged in pregnancy compared to the nonpregnant population. In general, conditions requiring hemodynamic monitoring in pregnancy are uncommon. However, when they do occur, interpretation of hemodynamic data requires a good understanding of the physiologic changes associated with pregnancy.

Noninvasive Monitoring

Echocardiography and urinary diagnostic indices are noninvasive monitoring methods that have been evaluated in obstetric patients in small studies. A high correlation between echocardiography and invasive techniques has been seen in the measurement of cardiac output [18, 19], stroke volume, ventricular filling pressure, and pulmonary artery pressures [18] in obstetric patients. In a study of 14 obstetric patients with refractory hypertension and oliguria, the authors reported that 12 of the reported cases were successfully managed without the need for invasive monitoring suggesting that echocardiography may be an effective alternative to pulmonary artery catheterization in pregnancy [20]. In a study evaluating seven oliguric preeclamptic patients [21], urinary indices of volume status including fractional excretion of sodium and urine sodium were inconsistent with findings on pulmonary catheterization. These data suggest that urinary indices alone may be misleading in guiding fluid management in preeclamptic patients. Given the increased hydrostatic pressure, lowered oncotic pressure, and capillary leak associated with preeclampsia, these patients are at very high risk for pulmonary edema.

Invasive Monitoring

The value of the pulmonary artery catheter (PAC) in critically ill patients has been debated for decades, and recent trials have not demonstrated a clear benefit [22]. Data in pregnancy are scarce. Although there are a few studies evaluating the

hemodynamic profiles of preeclamptic women [23], there are no randomized controlled trials evaluating the clinical usefulness of pulmonary artery catheters in pregnancy in general. Available data suggest a modest correlation between central venous pressures (CVP) and pulmonary capillary wedge pressure (PCWP) in untreated patients with preeclampsia. This is not true, however, in hypertensive patients that have undergone an intervention such as IV fluids or vasoactive agents, and wide variations in the range of the PCWP-CVP gradient have been reported in treated patients [24–26]. It is possible that this poor correlation following therapy is due to a delay in equilibration of intravascular fluid, with PCWP rising before CVP. These data suggest that although CVP may be an acceptable initial diagnostic tool in hypertensive gravidas, it may be less reliable in monitoring the hemodynamic status following therapeutic interventions.

Vasopressors

Etiologies of hemodynamic compromise in pregnancy are similar to those observed in the general population and include hemorrhage, infection and sepsis, or cardiogenic shock. However, circulatory collapse related to regional anesthesia is the most common cause in the pregnant population.

The overall principles of management of shock in the obstetric population should not differ much from the general population; however, it should take into consideration the physiology of pregnancy. Identifying pregnant patients in shock may be challenging as early signs of shock such as tachycardia and reduced blood pressure may also be observed in normal pregnancies. Changes in these measurements over time may be more helpful clues. One of the mainstays of the management of critically ill gravidas is maintaining adequate tissue perfusion and oxygenation of both the mother and the fetus. Placental blood flow is proportional to uterine blood flow. The latter is dependent on uterine perfusion pressure which is directly proportional to the maternal systemic blood pressure and cardiac output. Moreover, as uterine blood flow is at its maximal capacity under normal conditions, it is unable to adapt to low perfusion states.

Given the significant improvement in outcomes of sepsis with early goal-directed therapy, it would be reasonable to assume that similar interventions should be applied in pregnant women. However, it is not clear whether the same parameters should be used in pregnancy as in the nonpregnant population and whether this intervention truly improves outcomes in pregnant women. Until further data are available, it would be reasonable to intervene the same way, with some caveats. As discussed above, in the second trimester and beyond, the gravid uterus can hinder venous return and up to 25 % reduction in cardiac output by means of aortocaval compression. Hence, the first steps in the management of pregnant women with circulatory collapse should start by placing gravidas in a left lateral decubitus position which has been shown to improve cardiac output significantly [17]. Although fluid resuscitation is the first step in managing hemodynamic instability, the lower

oncotic pressures associated with pregnancy and the tendency of pregnant and postpartum women to develop pulmonary edema at lower hydrostatic pressures should be considered. Therefore, careful monitoring should be performed in pregnant women being resuscitated with intravenous fluids. Despite the lower physiologic oncotic pressures, it is unclear whether pregnant women would benefit from being resuscitated with colloids as opposed to crystalloids.

In cases of severe hemodynamic compromise, where the patient remains unresponsive to volume repletion, vasopressors may be necessary to optimize the hemodynamic status. Owing to their potential to decrease uterine perfusion, the choice of vasoactive agents in pregnancy is important. However, the effect of these drugs on placental perfusion may not be an important factor in decision making since withholding such therapy will lead to persistence of severely low blood pressure, which will likely affect maternal morbidity and mortality and guarantee poor placental perfusion [27]. The optimal drug would be one with the least negative effect on placental perfusion but with equal or superior benefit to systemic pressures. There is a paucity of evidence about the optimal vasopressor in obstetric patients. The majority of data originate from animal studies as the majority of human studies have been performed in the setting of hypotension caused by regional anesthesia around labor and delivery. While these data are helpful, they are limited by the fact that cardiovascular physiology is different depending on the type of shock. Therefore, we may not be able to extrapolate data related to regional anesthesia to sepsis, for instance, or cardiogenic shock.

Dopamine is a dopaminergic, alpha-1 and beta-1 agonist. The action of this drug on these receptors is dose dependent. Multiple animal studies show that dopamine reduces uterine artery blood flow in pregnant baboons and sheep [28–30]. No studies looked at the possibility of fetal toxicity in humans, but cardiac abnormalities were reported in some animal studies [31, 32]. Though prolactin release is inhibited by dopamine [33], it is not clear whether exogenous dopamine impacts lactation significantly.

Dobutamine is mainly a beta-1 and beta-2 agonist. Though dobutamine use has been reported in human pregnancies following myocardial infarction or cardiopulmonary bypass [34, 35], there are no human studies looking its effect on placental perfusion. Based on experimental animal studies, dobutamine is not expected to increase the risk of congenital abnormalities.

Epinephrine is an alpha-1 and beta-1 and to a lesser extent a beta-2 agonist. The drug has been known to interfere with embryo development, likely through hemodynamic effects [36]. Epinephrine produced increased uterine activity and uterine vasoconstriction associated with impaired fetal gas exchange in monkeys [37]. In addition due to its beta-2 activity, epinephrine may induce uterine relaxation delaying the progression of labor. *Norepinephrine* is an alpha-1 and beta-1 agonist. No reproductive animal studies are available. The drug crosses the placenta, and thus its use in pregnancy may affect the fetus [38, 39]. Phenylephrine is a pure alpha-1 agonist. This drug is commonly used to treat maternal hypotension induced by spinal or epidural anesthesia during C-section. The use of this drug was not clearly associated with increased fetal risks, despite reports of small increase in unusual birth defects in

the Collaborative Perinatal Project which included more than 4,000 women exposed to this agent during pregnancy. *Ephedrine* stimulates the release of norepinephrine, but this drug is less potent than norepinephrine. It is also used at delivery for the prevention and/or treatment of maternal hypotension associated with spinal anesthesia during C-section. Though it has comparable effect on blood pressure to phenylephrine, ephedrine is associated with a higher incidence of elevated heart rate, maternal nausea, and vomiting [40]. Both drugs are associated with reduced fetal pH, but neonatal Apgar scores and the incidence of fetal acidosis defined as umbilical arterial pH <7.2 were better in women treated with phenylephrine [40].

Vasopressin is a direct vasoconstrictor without inotropic or chronotropic activity. No animal reproductive studies were conducted. Based on animal data, the drug can produce necrosis of embryonic extremities through vasoconstriction [41, 42]. This drug may be helpful in cases of severe acidosis where other agents may be ineffective. It is not clear whether higher doses of this drug need to be used in pregnant women given possible increased metabolism by the placenta-secreted enzyme vasopressinase. However, recent recommendations by the American Heart Association [43] do not suggest dose modification in pregnancy.

Based on the above data, when faced with persistent hypotension after adequate fluid resuscitation, phenylephrine and ephedrine have the best effect on placental perfusion judged by their effect on fetal acidosis during pregnancy. However, if another drug is thought to have superior effects on outcomes in a given context, that drug should be chosen instead of phenylephrine and ephedrine.

In summary, management of critically ill pregnant women may be challenging. However, a thorough understanding of pregnancy physiology can help clinicians make informed decisions in the absence of evidence-based guidelines to help direct the care. Multidisciplinary approaches to this complex and unique population would likely yield the best outcomes.

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