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Physiology of a Typical Pregnancy

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ABSTRACT
The intensive care unit (ICU) pregnant patient presents a number of difficulties.Basic principles of maternal and foetal physiology must be understood by doctors and nurses. The doctors must treat the mother while taking into account any negative consequences that diagnostic and therapeutic procedures might have on the foetus. This article largely focuses on the typical pregnant state, with relevant allusions to how pregnancy may impact the patient in situations related to the intensive care unit. ICU doctors should be knowledgeable with how the major organs adjust physiologically to the pregnant state.

1. INTRODUCTION

For both gravid and nonpregnant states, regular laboratory test normal values are different. Due to changes in hormones like progesterone and human chorionic gonadotropin, pregnant women adjust to the gravid condition quickly. Edema of mucosal surfaces and progesterone-mediated hyperemia are the main characteristics of pregnancy. The oropharynx and nasopharynx both show this alteration.Women who are pregnant typically have increased nasal congestion. Therefore, smaller nasogastric and endotracheal tubes should be used.

General modifications

The lower chest wall widens by 5 to 7 cm, and the diaphragm is shifted cephalad by around 4 cm [1]. At 37 weeks of gestation, these alterations reach their apex. The electrocardiogram shifts Q waves inferiorly and T waves invert when the diaphragm is forced upward, which causes the heart to rotate slightly to the left [2]. When inserting a chest tube in the ICU, it's critical to take the diaphragm's upward migration into account. At 12 weeks, the bladder transforms into an abdominal structure and is more vulnerable to blunt damage. At 20 weeks, the uterus' fundus is level with the umbilicus and is susceptible to direct injury from blunt or piercing trauma.

Respiratory adjustments

Spirometry continues to be healthy during pregnancy [3]. Peak flows and flow volume loops are unaffected. There is a 4% to 5% reduction in total lung capacity [4]. The diaphragm's upward displacement is mostly to blame for this modification. Decreases in expiratory reserve volume and residual volume result in a 20% reduction in functional residual capacity (FRC) [1,5]. Early in pregnancy, diffusion capacity may stay the same or slightly increase before returning to normal values [1]. These modifications to pulmonary function.During pregnancy, the two main characteristics that alter are ventilation and oxygen consumption.

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Because of changes in respiratory mechanics, an increased metabolic rate, and an increase in progesterone levels, minute ventilation rises by around 50% during pregnancy [4]. With little to no change in respiratory rate, an increase in tidal volume results in an increase in minute ventilation. Before the end of the first trimester, this increase starts, and it stays pretty steady throughout the rest of the pregnancy. Before the gravid uterus presses up against the diaphragm, the first trimester of pregnancy is when pregnancy dyspnea first appears. Up to 60% of pregnant women may experience it. As a result of progesterone's actions on the respiratory centre, it is thought to be hormonally mediated [6]. Progesterone directly stimulates the respiratory system and makes the ventilatory response to variations in alveolar Paco2 more responsive [6]. When hyperventilation is prevalent during the luteal phase of the menstrual cycle, this impact is seen, which correlates with serum progesterone levels. From 25 ng/mL at 6 weeks' gestation to 150 ng/mL at 37 weeks' gestation, progesterone levels rise progressively during pregnancy [5]. The level of pregnancy-related hyperventilation and progesterone levels in the blood are correlated. The normal hyperventilation during pregnancy is caused by the increase in minute ventilation, which in turn causes a mild chronic respiratory alkalosis with a compensatory metabolic acidosis. A pregnant woman's normal arterial blood gas has a pH between 7.40 and 7.47, a Paco2 between 30 and 32 mm Hg, and a normal to slightly higher Pao2. By increasing renal bicarbonate excretion, the kidneys partially offset the alkalosis and maintain blood HCO3 levels between 18 and 21 mEq/L (base deficit, 3–4 mEq/L). The values of 7.47 for pH, 32 for Paco2, and 90 for HCO3 in the arterial blood gas may not always signify a pathologic state that necessitates additional investigation. Oxygen consumption rises by around 20% as minute ventilation rises. The larger uterus and growing foetus, together with increased maternal cardiac and respiratory work, are the causes of this shift. Maternal oxygen reserve is lowered as a result of the FRC decline and the rise in oxygen use. Pregnant patients in the ICU should be aware of these physiologic changes, especially as they pertain to endotracheal intubation. The FRC variable is important because it indicates how much oxygen reserve the lung has in the event of an apnea. Endotracheal intubation is linked to a more abrupt decline in Pao2 in pregnant individuals following a period of apnea due to the higher oxygen demand.

Cardiac modifications

Around six weeks into the pregnancy, the maternal cardiac output starts to rise. About 2 L, or 30% to 50% more than the volume in the nongravid state, is added to the maternal blood volume gradually throughout pregnancy [7]. Only 20% to 30% more maternal red blood cells are produced, which causes hemodilution and the relative anaemia of pregnancy. The increase in blood volume of 1000 to 1500 mL somewhat counteracts peripartum blood loss. After a straightforward vaginal delivery and after a caesarean delivery, the typical blood loss of 0.6 L and 1.0 L, respectively, is typically easily tolerated [8]. The finest study of typical hemodynamic measures in a typical pregnancy was written by Clark et al.[9] Between 36 and 38 weeks of pregnancy, these researchers examined 10 volunteers who were lying in the left lateral recumbent position. Each patient had their radial arterial line and pulmonary artery catheterized. A common thermodilution method was used to assess cardiac output. Between 11 and 13 weeks following delivery, the patients were restudied. Due to an increase in heart rate and stroke volume along with a roughly 20%–30% drop in systemic vascular resistance, cardiac output was nearly 40% higher than baseline values. In comparison to non-pregnant readings, the central venous pressure and pulmonary capillary wedge pressure were constant. During pregnancy, the colloid oncotic pressure, which measures the quantity of solute

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particles in the blood (mainly albumin), decreases. The supine position may significantly compress the inferior vena cava, or cause supine hypotension syndrome, when the uterus grows by about 20 weeks of gestation. A 20% to 30% drop in ejection fraction may arise from this uterine compression's ability to reduce venous return. The uterus can be moved to the left by laterally moving the patient and placing a foam wedge or pillow under the right buttock to improve cardiac output. When resuscitating critically ill pregnant patients in the ICU, this procedure is crucial. In the ICU, it's crucial to keep in mind that blood pressure, particularly the diastolic portion, tends to be lower during pregnancy. A third heart sound and systolic ejection murmur are frequently detected during physical examination. Echocardiography of healthy pregnant patients shows the following: (1) increases in all cardiac chamber dimensions, (2) increased left ventricular wall thickness, (3) small pericardial effusions, (4) mild tricuspid and pulmonic regurgitation in 90% of patients, and (5) mild mitral regurgitation in 30% [10].

Renal alterations

The glomerular filtration rate rises by 50% by the time a pregnant woman is 16 weeks along with remaining high [11]. Lower levels of serum creatinine, blood urea nitrogen, and uric acid are the outcome of increased creatinine clearance.Exceeding 0.8 mg/100 mL and 14 mg/100 mL of blood urea nitrogen in the plasma, respectively, may signify renal impairment [11]. Pre-existing renal illness heralds higher dangers of preterm delivery and declining renal function [12]. Premature births affect around 60% of children born to mothers with blood creatinine levels above 1.4 mg/dL [13].

Digestive system modifications

The majority of pregnant women frequently have gastroesophageal reflux. Starting in the first trimester, progesterone causes smooth muscle relaxation and lowers the pressure in the lower esophageal sphincter. The stomach is forced out of place as abdominal size expands, which further reduces the sphincter's efficiency. Always consider pregnant women to be at high aspiration risk. Increased plasma volume causes hypoalbuminema in the liver. A normal reading is 3.1 g/dL at the end of the term. During the fifth month of pregnancy, the placenta's contribution to serum alkaline phosphatase concentrations rises above the normal range and continues to rise to two to four times the typical values [14]. Cholecystitis is the second most typical nonobstetric pregnancy illness that necessitates surgery after appendicitis.

Fetal physiology

A formal understanding of the uteroplacental unit is necessary for the care of pregnant patients in the ICU. The placenta performs three key tasks: respiratory and gas exchange, foetus nourishment, and waste removal. Through a simultaneous exchange mechanism, the maternal and foetal circulations communicate with one another. Hemoglobin concentration and saturation, uterine artery blood flow, and the oxygen content of the uterine artery blood all have an impact on the transfer of oxygen from the mother to the placenta. At term, uterine blood flow is around 10% of cardiac output (600–700 mL/min), compared to 50 mL/min in the non-pregnant condition. Transfer of nutrients and oxygen is possible thanks to the placental membrane. The foetal umbilical vein transports oxygenated blood to the foetal circulation, and the foetal umbilical artery returns deoxygenated blood to the umbilical-placental interface. Vasoconstriction, hypotension, and uterine contractions can all reduce uterine blood flow. Hypovolemia, infection, drug use, and positioning in the supine position

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are just a few of the ICU conditions that can cause maternal hypotension. Preeclampsia and the delivery of the several inotropic support medications that are frequently used in the ICU can both cause vasoconstriction. The preferred vasopressor for treating pregnancy-related hypotension is ephedrine, which primarily possesses b-adrenergic action. The foetus has various defence systems to maintain its ability to absorb oxygen. The foetus has a greater haemoglobin level (15–16 g/dL). At a Po2 of 30 to 35 mm Hg, foetal haemoglobin is 80% to 90% saturated, whereas the main type of adult haemoglobin is only 30% saturated at this Po2. The prenatal oxygen dissociation curve's leftward shift is the cause of this variation. The ductus arteriosus gives the foetus two ventricles for circulation. Although the foetal umbilical vein Po2 is comparatively low at 35 mm Hg, the foetal oxygen content is considerably high due to the increased haemoglobin saturation and concentration [15]. Fetal Po2 is not significantly impacted by increasing the mother's fraction of inspired oxygen [16]. 100% oxygen in the ICU has very little impact on umbilical vein Po2 in pregnant patients. The foetus has 42 mL of oxygen reserves and uses 20 mL of oxygen every minute. A foetus with full hypoxia would only be expected to have 2 minutes of oxygen reserve; nevertheless, by diverting blood flow to important organs and reducing oxygen consumption, a foetus with this condition can survive for at least 10 minutes [8]. The uterine artery vasoconstricts in the presence of trauma-induced blood loss, which can cause foetal hypoxia despite generally normal vital signs [2]. The foetus suffers as a result of maintaining maternal blood flow. When the gestational age is between 24 and 25 weeks and the expected weight is above 750 g, the foetus is deemed viable [17]. Although there are no specific recommendations for perimortem caesarean delivery, it should generally be taken into consideration if the foetus is at least 24 weeks old. Less than five minutes should have passed since the mother's circulation stopped for the best possible outcome [7].

2. CONCLUSION

Obstetricians, critical care doctors, labour and delivery nurses, and ICU nurses must work together to provide treatment for severely sick pregnant patients. Understanding the pathophysiologic conditions that are covered in other articles in this issue requires knowledge of normal pregnant physiology. When doing diagnostic and therapeutic procedures, ICU doctors must take the mother and the foetus into account.

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