التخدير العصبي المحورى في حالات ارتفاع ضغط الدم المصاحب للحمل

رسالة
توطئة للحصول على درجة الماجستير
في التخدير

مقدمة من
هاني ابراهيم احمد الباروجي
بكالوريوس الطب والجراحة

تحت اشراف
الأستاذة الدكتورة/ سهير عباس محمد صادق
أستاذ التخدير والرعاية والمركزة
كلية الطب - جامعة عين شمس

الدكتورة/ رشا سمير بندق
أستاذ مساعد التخدير والرعاية والمركزة
كلية الطب - جامعة عين شمس

د/ احمد سمير زيدان
مدرس التخدير والرعاية والمركزة
كلية الطب -جامعة عين شمس

كلية الطب
جامعة عين شمس
٢٠١٠

Hypertensive disorders are the most common medical complications of pregnancy, affecting 5% to 10% of all pregnancies ***(Habli and Sibai, 2008).***

Women who are hypertensive and pregnant must be subdivided into those with: A- Chronic hypertension B- Pregnancy-induced hypertension (PIH) ***(Hayman and Baker, 1997).***

Clinical manifestations of preeclampsia may affect maternal and fetal conditions, and may have severe complications such as HELLP syndrome and eclampsia ***(Solomon and Seely, 2006).***

An understanding of physiological changes during pregnancy is essential to optimize analgesic and anesthetic options for pregnant women during delivery ***(Campbell, 2000).***

Regional anesthesia is the method of choice for cesarean deliveries due to its proven record of maternal and fetal safety ***(Okafor and Okezie, 2005).***

General anesthesia for caesarean section in patients with severe pre-eclampsia is perceived to carry greater maternal risk and the cornerstones of management of pre-eclampsia remain seizure prophylaxis, fluid and antihypertensive therapy ***(Dyer et al., 2007).***

The aim of this work is to review the recent advances in neuraxial blocks (techniques, drugs and vasopressors used) related to pregnancy induced hypertension.

Chapter (I)

PHYSIOLOGICAL CHANGES OF PREGNANCY

The profound physiological changes that occur during pregnancy significantly impact the anesthetic management of the parturient. An understanding of the clinical importance of these physiological changes is essential to optimize analgesic and anesthetic options for pregnant women during delivery ***(Campbell, 2000)*.**

General changes:

Normal values of routine laboratory tests differ for gravid and nonpregnant states. Pregnant women adapt quickly to the gravid state because of changes in hormones such as human chorionic gonadotropin and progesterone. In general, pregnancy is characterized by progesterone-mediated hyperemia and edema of mucosal surfaces. These changes are evident in the nasopharynx and oropharynx. Pregnant women tend to have more nasal congestion. Accordingly, endotracheal and nasogastric tube size should be downsized. The diaphragm is displaced cephalad about 4 cm, and the lower chest wall widens about 5 to 7 cm. The upward movement of the diaphragm is important to consider when placing a chest tube in the intensive care

unit (ICU). At 12 weeks, the bladder becomes an abdominal structure and is more susceptible to blunt trauma. At 20 weeks, the fundus of the uterus is at the level of the umbilicus and can be injured directly in blunt or penetrating trauma ***(Chesnutt, 2004).***

Body water homeostasis

Maternal blood volume expands during pregnancy to allow adequate perfusion of vital organs, including the uteroplacental unit and fetus, and to prepare for the blood loss associated with parturition ***(Whittaker et al., 1996).***

Total body water increases from 6.5 L to 8.5 L by the end of gestation. Changes in osmoregulation and the renin-angiotensin system result in active sodium reabsorption in renal tubules and water retention. The water content of the fetus, placenta, and amniotic fluid accounts for approximately 3.5 L of total body water. The remainder of total body water is comprised of the expansion of maternal blood volume by 1500 mL to 1600 mL, plasma volume of 1200 mL to 1300 mL, and a 20% to 30% increase in erythrocyte volume from 300 mL to 400 mL. The rapid expansion of blood volume begins at 6 to 8 weeks' gestation and plateaus at approximately 32 to 34 weeks' gestation. The expanded extracellular fluid volume accounts for 6 to 8 kg of weight gain. The larger increase of plasma volume by 1000 mL to 1500 mL relative to

erythrocyte volume results in hemodilution and a physiologic anemia ***(Hill and Pickinpaugh, 2008)*.**

Respiratory changes

The diaphragm rises about 4 cm during pregnancy. The subcostal angle widens appreciably as the transverse diameter of the thoracic cage increases about 2 cm. The thoracic circumference increases about 6 cm, but not sufficiently to prevent a reduction in the residual volume of air in the lungs created by the elevated diaphragm ***(Cunningham et al., 2007)*.**

a- Upper airway:

Increased vascularity and capillary engorgement of the oral and nasal mucosa and larynx of the parturient begins at 8 to 12 weeks gestation. Mucous membranes also become increasingly edematous and the capillaries become more friable. Consequently, epistaxis caused by engorgement of the nasal mucosa is not uncommon. These alterations are likely the effect of the hormone progesterone ***(Campbell, 2000).***

Of importance, these alterations in the upper airway become more pronounced over the course of labor as shown by rapidly worsening modified Mallampati scores ***(Farcon et al., 1994).***

Increased weight gain, particularly in the third trimester, results in enlarged soft tissue of the neck and breasts, which may impair insertion of the laryngoscope. There is a paucity of information regarding the length of time after delivery it takes for this upper airway physiological alterations to resolve. However, it is unlikely that these changes will have completely resolved before 6 to 8 weeks after delivery ***(Campbell, 2000).***

b- Lower Airway:

Elevation of the diaphragm and hormonal factors alter the dynamics of lung mechanics and volumes. Although total lung capacity and vital capacity remain unchanged, residual volume and expiratory reserve volume decrease as pregnancy progresses ***(Campbell, 2000).***

Tidal volume rises by 30% in early pregnancy to 40–50% above non-pregnant values by term. Neither forced expiratory volume (FEV_1) nor peak expiratory flow rate are affected by pregnancy, even in women with asthma. The rise in tidal volume is largely driven by progesterone, which appears to decrease the threshold and increase the sensitivity of the medulla oblongata to carbon dioxide ***(Broughton-Pipkin, 2007).***

Minute ventilation increases between 28% and 45%, primarily because of a similar increase in tidal volume, and

respiratory rate increases minimally. Consequently, $PaCO_2$ decreases to 30 mmHg by the end of the first trimester and remains unaltered throughout the remainder of the pregnancy. To compensate for the changes in $PaCO_2$, plasma bicarbonate levels decrease to 20 mmol/L. The increased alveolar ventilation results in a much smaller proportional rise in PaO_2, from around 96.7 to 101.8 mmHg. This increase is offset by the rightward shift of the maternal oxyhemoglobin dissociation curve caused by an increase in 2,3-diphosphoglycerate (2,3-DPG) in the erythrocytes. This facilitates oxygen unloading to the fetus, which has both a much lower PaO_2 (25–30 mmHg) and a marked leftwards shift of the oxyhaemoglobin dissociation curve, due to the lower sensitivity of fetal haemoglobin to 2,3-DPG. Maternal oxygen saturation should be maintained at 95% to maintain a PaO_2 greater than 70 mmHg, thereby optimizing oxygen diffusion across the placenta. Fetal oxygenation is maintained when maternal PaO_2 remains above 60 mmHg to 70 mmHg. When it falls below this level, fetal oxygenation is compromised immediately ***(Hill and Pickinpaugh, 2008)*.**

The enlarging abdominal uterus begins to elevate the diaphragm, culminating in a reduction by 20% of the functional residual capacity (FRC) at term gestation. Importantly, at term, FRC decreases a further 30% with the parturient in the supine position compared with the upright

position. This increase in minute ventilation appears to result from both increased carbon dioxide production and hormonal changes. FRC returns to prepregnancy values by 2 weeks postpartum, whereas minute ventilation remains elevated until 6 to 8 weeks postpartum ***(Broughton-Pipkin, 2007).***

Metabolism:

Oxygen consumption (mL/kg/min) increases significantly from 3.0 in the nonpregnant state to 4.3 (43% increase) at 8 to 11 weeks gestation and peaks at 5.0 (60% increase) in the immediate postpartum period. The increase in oxygen consumption results primarily from the metabolic demands of the fetus, placenta, and uterus, as well as increased pulmonary and cardiac work. Oxygen consumption further increases 40% during the first stage of labor and 75% during the second stage. Oxygen consumption remains elevated until 6 to 8 weeks postpartum ***(Campbell, 2000).***

Cardiovascular changes:

During pregnancy and the puerperium, the heart and circulation undergo remarkable physiological adaptations. The most important changes in cardiac function occur in the first 8 weeks of pregnancy. There is a significant fall in total peripheral resistance by 6 weeks gestation and

decrease about 40% by mid-gestation, resulting in a fall in afterload. This is 'perceived' as circulatory under filling, which activates the renin-angiotensin aldosterone system and allows the necessary expansion of the plasma volume ***(Ganzevoort et al., 2004)*.**

Systemic vascular resistance decreases by 20% by the second trimester because of the hormonal influences of estrogen and progesterone, which remain at this level at term. Maternal heart rate may increase as early as 4 weeks estimated fetal gestational age, with a 20% increase seen by 7 weeks. Stroke volume increases by 20% as early as 5 weeks and peaks at 30% above prepregnancy levels by the end of the second trimester. Cardiac output begins to increase at 5 weeks fetal gestation, likely because of the increase in heart rate and stroke volume, and continues to increase to 40% by the end of the first trimester, further increasing to 50% by term gestation. Much of the increased cardiac output is directed toward the enlarging uterus, with uterine blood flow increasing from 50 to 200 mL/ min in the prepregnant state to 700 to 900 mL/min at term. During active labor, cardiac output increases a further 25%, not only because of the pain of labor but, more importantly, as the result of autotransfusion of 300 to 500 mL of blood during each contraction. Even at 2 days postpartum, cardiac output remains 30% above prepregnancy levels.

Postpartum cardiac output returns to prepregnancy levels for some 3 to 6 months ***(Campbell, 2000)*.**

Mean arterial pressure has been shown to decrease by 10% at 7 weeks fetal gestation, reaching its lowest during the second trimester and returning to prepregnancy levels at term. Supine hypotension occurs in about 8% of women in late gestation. No changes have been observed in central venous, pulmonary artery, or pulmonary occlusion pressures throughout pregnancy**.** Tricuspid, pulmonary, and mitral valve areas increase, with the majority of parturients exhibiting trivial tricuspid and pulmonary regurgitation and 30% exhibiting mitral regurgitation ***(Mabie et al., 1994)*.**

There is progressive venodilatation and rises in venous distensibility and capacitance throughout a normal pregnancy, possibly because of increased local nitric oxide synthesis. Peripherally, the primary physiological alteration involves aortocaval compression by the enlarging extrapelvic uterus. Compression of the inferior vena cava occurs as early as the end of the first trimester. At term, distal inferior vena cava pressures are two to three times greater than prepregnancy values because of compression by the gravid uterus in the supine position. This increased venous pressure may adversely affect uterine artery perfusion pressure and uterine artery blood flow. In the supine position, at term, the gravid uterus may also

partially obstructs the aorta at the L3-L5 level ***(Broughton-Pipkin, 2007).***

Although the left uterine "tilt" position may only partially reduce inferior vena cava obstruction, repositioning the parturient in the lateral position completely relieves the obstruction ***(Hirabayashi et al., 1997).***

Gastrointestinal changes

The alteration in the normal orientation of the stomach by the enlarging gravid uterus decreases the tone of the intra-abdominal lower esophageal sphincter (LES). LES tone is further decreased by progesterone. Concurrently, intragastric pressure increases as the pregnancy progresses. Consequently, heartburn or gastric reflux is very common, particularly during the third trimester. Gastric acid secretion also increases because of increasing gastrin levels produced by the placenta ***(Campbell, 2000).***

Emptying studies have conclusively demonstrated a significant slowing of gastric transit times as well as increases in gastric volumes during labor. Hepatic synthesis of albumin, plasma globulin and fibrinogen increases, sufficiently to give increased plasma concentrations despite the increase in plasma volume ***(Broughton-Pipkin, 2007).***

Total albumin is increased, however, because of a greater volume of distribution. The concentration of serum albumin decreases during pregnancy. The reduction in albumin concentration, combined with a normal slight increase in serum globulin levels, results in a decrease in the albumin-to-globulin ratio similar to that seen in certain hepatic diseases ***(Cunnigham et al., 2007).***

Serum aspartate transaminase, alanine transaminase, Y-glutamyl transferase, and bilirubin levels are slightly lower during pregnancy compared with nonpregnant normal values. Intrahepatic cholestasis has been linked to high circulating levels of estrogen, which inhibit intraductal transport of bile acids ***(Girling and colleagues, 1997).***

Hematologic changes

a- Plasma volume

The physiological alterations of pregnancy result in a 15% increase in plasma volume as early as the first trimester to as high as 50% in the second trimester. This phenomenon is the direct result of maternal estrogen (which increases plasma renin) and progesterone (which increases aldosterone production) as well as fetal (dehydroepiandrosterone) hormones. Ultimately, these hormonal influences lead to a significant retention of sodium ***(Campbell, 2000).***

The plasma volume increases more than the red cell mass, which leads to a fall in the various concentration measures which include the plasma volume, such as the haematocrit, the haemoglobin concentration and the red cell count ***(Broughton-Pipkin, 2007).***

b- Immunological functions

Pregnancy has been assumed to be associated with suppression of a variety of humoral and cell-mediated immunological functions. Not all aspects of immunological function are depressed. But for example, there is upregulation of helper T lymphocytes type-2 (Th2) cells to increase secretion of interleukins such as IL-4, IL-6, and IL-13 ***(Thellin and Heinen, 2003).***

c- Coagulation

Continuing low-grade coagulopathy is a feature of normal pregnancy. Normal parturients develop a hypercoagulable state as shown on thromboelastograph (TEG) ***(Brenner, 2004).***

Evidence of activation includes increased concentrations of all clotting factors, except factors XI and XIII, with increased levels of high-molecular-weight fibrinogen complexes ***(Cunnigham et al., 2007).***

Studies of the fibrinolytic system in pregnancy have produced conflicting results, although the majority of evidence suggests that fibrinolytic activity is actually reduced in normal pregnancy, likely related to increased concentrations of plasminogen-activator inhibitors. There are a number of natural inhibitors of coagulation, including protein C, protein S, and antithrombin. Inherited or acquired deficiencies of these and other natural regulatory proteins, collectively referred to as thrombophilias, account for more than half of all thromboembolic episodes during pregnancy ***(Lockwood, 2002)*.**

Normal pregnancy also involves changes in platelets. The platelet count at term may be decreased 20% from the nonpregnant state, although the remaining platelets appear to possess an increased reactivity due to production of thromboxane A2, which induces platelet aggregation. For most pregnant women, this 20% decrease does not result in thrombocytopenia (platelet count <150,000mm^3) nor is it clinically significant. However, pregnancy-induced thrombocytopenia (platelet counts lower than 150,000 mm^3) and platelet counts lower than 100,000 mm^3 were observed in 7.6% and approximately 1% of pregnant women, respectively ***(Baker and Cunningham, 1999)*.**