Regional Anesthesia for Cesarean Section in Preeclamptic Patients

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Preeclampsia, a pregnancy specific-disorder characterized by new-onset hypertension and proteinuria after 20 weeks of gestation, is the most frequently encountered medical complication during pregnancy, affecting 3–5% of pregnant women worldwide and still remains in the top three causes of maternal morbidity and mortality globally **(WHO, 2005)**.

Deaths are mainly due to intracranial hemorrhage, cerebral infarction, acute pulmonary edema, respiratory failure and hepatic failure. It is the leading cause of fetal growth retardation, intrauterine fetal demise and planned preterm birth **(Lewis, 2011).**

Obstetric management of preeclampsia relies on a high index of suspicion, careful observation, and early intervention. The method of intervention is logically a function of the severity of the disease, but ultimately the only definitive treatment is delivery of the fetus and placenta **(ACOG, 2002).**

Anesthetic management of a preeclamptic patient includes a detailed preanesthetic assessment that focuses on the severity of the condition, associated features and systemic involvement, evaluation of the airway, fluid status, and blood pressure control **(Judi, 2010).**

Anesthetic management of these patients remains a challenge. Although general anesthesia can be used in preeclamptic women, it is fraught with greater maternal morbidity and mortality. Currently, the safety of regional

anesthesia techniques is well established and they can provide better obstetrical outcome when chosen properly (**Mandal & Surapaneni, 2004).**

Regional anesthetic techniques have several advantages for pregnant women, especially preeclamptic patients, including a decreased risk of failed intubation and aspiration of gastric contents, avoidance of depressant agents, and the ability of the mother to remain awake and enjoy the birth experience. In addition, blood loss is reduced under regional anesthesia for cesarean delivery (**Visalyaputra et al., 2005).**

The purpose of this essay is to focus on the pathophysiological changes in preeclampsia, understanding the origins of this disorder and the regional anesthetic management for cesarean section in preeclamptic patients in an attempt to decrease perioperative morbidity and mortality.

Chapter 1

Physiological Changes During Pregnancy

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Caring for the pregnant patient is one of the most challenging and the most rewarding aspects of anesthesia because it involves two patients, the mother and the fetus. Both patients must be considered when making decisions. The mother is the primary patient, with the fetus secondary. Generally, optimal care of the mother provides good care of the fetus. Pregnancy alters maternal physiology. These changes must be considered when evaluating the pregnant patient **(Robert, 2008)**.

The physiologic, biochemical and anatomical changes that occur during pregnancy are extensive and involve systemic and local changes that maintain a healthy environment for the fetus without compromising the mother's health **(Pierce, 2005)**.

These modifications affect almost every organ system and influence the anesthetic and perioperative management of the pregnant woman. The physiologic changes of pregnancy, especially in the gastrointestinal and cardiovascular systems, directly influence the absorption, distribution, and elimination of drugs **(Goodman & Flood, 2006).**

Maternal changes in pregnancy occur as a result of hormonal alterations, mechanical effects of the gravid uterus, increased metabolic and oxygen requirements, metabolic demands of the fetoplacental unit and hemodynamic alterations associated with the placental circulation. Such changes become more significant as pregnancy progresses **(Birnbach & Browne**, **2005**).

It is important to understand the normal physiological changes occurring during pregnancy in order to predict the risks and effects of analgesic and

anesthetic intervention, and also to anticipate the impact of pregnancy on any coexisting medical condition **(Yentis et al., 2007)**.

I- Hormonal changes:

Following fertilization, the corpus luteum in the ovary secretes progesterone, estrogen and relaxin, and these hormones are secreted by the placenta when it takes over the function of the corpus luteum from 6–8 weeks gestation onwards. Progesterone is responsible for most of the hormonally mediated changes occurring during pregnancy **(Goodman & Flood, 2006)**.

II - Cardiovascular and haemodynamic changes:

Cardiovascular parameters are altered progressively throughout pregnancy and are accentuated in pregnancies subsequent to the first **Table 1.1**. Beginning as early as 4-8 weeks gestation and plateauing between 16 and 24 weeks **(Dean & Robert, 2002)**.

Changes during pregnancy

Blood volume increases throughout pregnancy reaching 45–50% above pre-pregnant values by term. This represents an increase in both red cell volume and plasma volume with the latter being relatively greater, thus causing the so-called 'physiological anemia of pregnancy'. The magnitude of the increase is greater in women with multiple pregnancy and greatly reduced in women with preeclampsia (**Yentis et al., 2007**).

Cardiac output rises gradually, beginning by 8 to 10 weeks gestation. By the end of the second trimester, cardiac output is elevated by 50% of nonpregnant values. The elevation in cardiac output is a result of both an increase in heart rate and stroke volume . Heart rate begins to increase by 5 weeks gestation to a maximum of about 20% at term. Stroke volume reaches a maximum of a 25% increase by 20 weeks gestation **(Goodman & Flood, 2006)**.

Systemic vascular resistance falls as a result of peripheral vasodilatation mediated by progesterone, prostacyclin and oestrogens, and there is a decrease in both systolic and diastolic blood pressures, which reach a nadir during the second trimester and then increase gradually towards term, although remaining lower than pre-pregnancy values (**Yentis et al., 2007)**.

Central venous pressure and pulmonary artery pressures do not change with pregnancy and are similar to the values in the nonpregnant state (**Robert , 2008).**

Aortocaval compression (supine hypotensive syndrome) was first reported in 1931. The inferior vena cava and aorta become compressed by the pregnant uterus (the vena cava may be totally occluded), causing reduction in venous return and cardiac output and thus compromising the mother, fetus, or both. Vasovagal syncope may follow aortocaval compression. Maternal symptoms and signs vary from asymptomatic mild hypotension to total cardiovascular collapse, partly dependent on the efficacy of the collateral circulation bypassing the inferior vena cava. Onset of symptoms and signs is associated with

lying in the supine or semi-supine position, and is relieved by turning to the full lateral position in most cases (**Yentis et al., 2007).**

Table 1.1: *Normal Hemodynamic Values in Pregnancy.*

Parameter	**Normal value**	**Change at term**
Heart rate	90 bpm	20% increase
Cardiac output	7 L/min	40–50% increase
Central venous pressure	4	No change
Pulmonary capillary wedge pressure	7	No change
Systemic vascular resistance	1200 dyne/cm/sec	20% increase
Systemic blood pressure	Slight decrease	Midtrimester 10–15 mm Hg, then rises
Pulmonary vascular resistance	78 dyne/cm/sec	30% decrease
Pulmonary artery pressure	Slight decrease	

[*From* ***Goodman & Flood,*** **2006**].

Changes during labor and delivery

Cardiac output increases by 25–50% in labor, with an additional 15-30% increase during contractions. This increase in cardiac output is mediated through increased sympathetic nervous system activity, and is therefore significantly attenuated by epidural analgesia. Central

venous pressure increases during contractions, partly due to sympathetic activity and partly from the transfer of up to 500 ml of blood from the intervillous space. The latter is unaffected by epidural analgesia, as is the increase in central venous pressure which occurs when the Valsalva maneuver is performed during pushing. Autotransfusion of blood (from the placenta) occurs during the third stage. The effect of this may be significant in women with cardiac disease. After delivery there is a sustained increase in cardiac output and central venous pressure for several hours, which is associated with hypervolemia. The implications of these changes for women with cardiac disease are significant **(Yentis et al., 2007)**.

III - Pulmonary changes:

Changes during pregnancy

Progesterone increases the sensitivity of the respiratory center to carbon dioxide and also acts as a primary respiratory stimulant. These effects are enhanced by estrogens, and the combined hormonal effect causes an increase in minute ventilation of 45–50% **(Yentis et al., 2007).**

Oxygen consumption and carbon dioxide production increase by approximately 60% over prepregnant values. PaO_2 is increased in early pregnancy due to a decrease in $PaCO_2$. Functional residual capacity, expiratory reserve volume, and residual volume are decreased at term **Figure. 1.1.** These changes are related to the cephalad displacement of the diaphragm by the large gravid uterus. Inspiratory capacity increases somewhat because of increase in