Perioperative Anesthetic Management of The High Risk Pregnant Patient

An Essay
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By

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Abstract

Pregnancy produces profound physiologic changes that alter the usual responses to anesthesia. It is important to know the effect of anesthetic agents on uterine activity and labor in order to get the safest anesthesia with the minimal side effects especially for the high risk parturient. Maternal problems include hypertensive disorders, obstetric hemorrhage, obesity, embolism, congenital or acquired cardiac conditions, endocrine and pulmonary disorders.

Obstetric Hemorrhage is a leading cause of maternal mortality. Morbid obesity also increases the risk of maternal and fetal mortality.
It is important for the anesthiologist to assess and manage the maternal problems before, during and after anesthesia.

➢ **Key Words:**

Anesthesia, pregnancy, heart disease, PIH, obesity, pulmonary embolism, diabetes mellitus, hemorrhage.

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List of abbreviations

ABP	Arterial blood pressure
ACE	Angiotensin converting enzyme
AFE	Amniotic fluid embolism
ALS	Advanced life support
aPTT	Activated partial thromboplastin time
AR	Aortic regurgitation
AS	Aortic stenosis
BSA	Body surface area
CAD	Coronary artery disease
CCBs	Calcium channel blockers
CPR	Cardio-pulmonary resuscitation
CTPA	Computed tomographic pulmonary angiography
DIC	Disseminated intravascular coagulopathy
DVT	Deep vein thrombosis
EDTA	Disoduim thylenediamine Tetra acetic acid
ERPF	Effective renal plasma flow
FEV_1	Forced expiratory volume in the 1^{st} second
FHR	Fetal heart rate
FRC	Functional residual capacity
GABA	Gamma amino butyric acid
GFR	Glomerular filtration rate
INR	International normalized ratio
IUFD	Intra-uterine fetal death
IUGR	Intra-uterine growth retardation
KPa	Kilo pascal
LAP	Left atrial pressure
LMA	Laryngeal mask airway
LVEDP	Left ventricle end diastolic pressure
LVEF	left ventricle ejection fraction
MAC	Minimum alveolar concentration
$MgSO_4$	Magnesium sulphate
MR	Mitral regurgitation
MRI	Magnetic resonance of Imaging
MS	Mitral stenosis
NSAIDs	Non steroidal anti-inflammatory drugs
PAC	Pulmonary artery catheter
$PaCO_2$	Arterial partial pressure of carbon dioxide

PCC	Pheochromocytoma
PE	pulmonary embolism
PEEP	Positive end expiratory pressure
PH	Pulmonary hypertension
PIH	Pregnancy induced hypertension
PT	Pothrombin time
PTU	Propyl thio-uracil
SVR	Systemic vascular resistance
TBG	Thyroid binding globulin
TEE	Transesophageal echocardiography
URI	Upper respiratory tract infection
VAE	Venous air embolism
V/Q	Ventilation-perfusion ratio
VTE	Venous thrombo-embolism

Physiological Changes During Pregnancy

Physiological Changes During Pregnancy

It is necessary to start with the physiological changes that occur during pregnancy to understand the hemodynamic changes occurring during labor and how to deal with it during anesthesia for labor. Pregnancy produces profound physiologic changes that alter the usual responses to anesthesia. Pregnancy affects organ system; many of these changes are useful to the mother in tolerating the stresses of pregnancy and delivery. (1)

I. Cardiovascular System

Alterations in circulating blood volume and other blood factors accompany the progressive growth of the fetus, placenta, and uterus. These cardiovascular changes begin early during pregnancy and are probably hormonally produced. Both the plasma volume and the red cell volume begin to increase between the sixth and 12th weeks of pregnancy, resulting at term in an increased plasma volume of 40% to 50%, and a total blood volume increase of 25% to 40%. Since red blood cell volume only increases by approximately 20%, there is a "physiologic" decline in the red blood cell count, hemoglobin, and hematocrit. The fibrinogen content increases throughout pregnancy both in absolute amounts and in relative concentrations, the latter from between 250 and 350 mg/dl to 450 mg/dl. There is also a marked increase in activity of several clotting factors, rendering the blood hypercoagulable and predisposing the pregnant woman to thromboembolic phenomena.(1)

The fibrinolytic system is usually reduced with the dilutional

anemia that often ensues, and administration of iron and folic acid supplement restores hemoglobin to relatively normal levels. As stated, blood volume first expands at 12 weeks gestation, increases rapidly during the second trimester, and then increases more slowly during the third trimester. This physiologic hypervolemia may mask volume loss, giving the clinician an unfounded sense of security about the patient's hemodynamic stability. (1)

1. Blood Pressure:

There is a gradual fall in blood pressure during pregnancy. Systolic blood pressure falls on average by 5–10 mmHg, whilst the fall in diastolic blood pressure is of the order of 10-15 mmHg. This difference results in an increase in pulse pressure. (2)

There is a decrease in peripheral vascular resistance in mid-trimester, yielding a mild drop in systolic pressure and a more significant drop in diastolic pressure. An increase in blood pressure over first trimester levels is not normal. (1)

2. Cardiac Output:

Cardiac output begins to rise during the first trimester of pregnancy and rapidly reaches a maximal increase of about 30%, usually by 30 to 34 weeks. The rise is produced by a 15% increase in heart rate and a 35% increase in stroke volume. Investigational work performed on women in the supine position indicated that the cardiac output appeared to decline by the 30th to 35th week, but this is apparently produced by the supine

hypotensive syndrome; the encroachment on the abdominal great vessels by the enlarged uterus. Cardiac output when measured in the lateral decubitus position during the last few weeks of pregnancy has been shown to equal that seen in the second trimester. Maximum reduction of cardiac output occurs in the supine position with less of a decrease when the mother is sitting or semi-recumbent. (1)

The increase in cardiac output is progressive during the first and second trimesters and is detectable as early as 8 weeks gestation. Cardiac output does not change further in the third trimester. The increase in cardiac output it due to increase in both heart rate and stroke volume. Heart rate increases first and rises by up to 16 beats / min by term. Stroke volume increased from 65 ml before pregnancy to 83 ml in the second trimester. Combined Doppler and cross-sectional echocardiography have been used to assess cardiac function in pregnancy. There is great variation between and within individuals. Primarily, increased maternal vascular volume drives the hemodynamic changes. Because the maternal heart responds to an increased pre-load by increasing stroke volume, pathology that limits diastolic flow through the ventricles is poorly tolerated. (3)

During early labor, cardiac output increases a further 15% in response to catecholamine secretions associated with the pain. Augmented venous return also occurs with each contraction when 300 to 500 ml of blood is expelled into the circulation. In the second and third stages of labor, cardiac output is 45% and 80% respectively above pre-labor values, and returns to normal approximately two weeks postpartum. This immediate postpartum increase in blood volume represents a major hazard to patients at risk from

circulatory overload. [1]

3. Vascular Resistance:

Systemic and pulmonary vascular resistance both decrease significantly in pregnancy. SVR is in part related to the development of a low resistance utero-placental circulation and is in part due to progesterone driven vasodilatation. The blood vessels of pregnant women show increased refractoriness to angiotensin II. This may be mediated by endothelial and platelet derived prostaglandins. Reliable measurements of prostaglandins are difficult to obtain because of their low plasma concentrations, but metabolites of the vasodilator prostacyclin are markedly increased in pregnancy. Less information is available about the vasoconstrictor thromboxane, but one of its breakdown products has been found in decreased concentrations in pregnancy. Progesterone may modulate prostaglandin mediated vascular responsiveness to angiotensin. Nitric oxide is also likely to be an important regulator of maternal blood pressure. [4]

Although vascular resistance is lowered, the increased blood flow is not uniformly distributed. By term, the blood flow to the uterus has increased from a pre-pregnancy level of 50–190 to 700–800 ml/min. The uterus receives approximately 10% of the cardiac output. Blood flow to the breasts doubles, to make up 2% of the cardiac output. These increases are largely at the expense of blood flow to the splanchnic bed and skeletal muscle. The proportion of blood flow to the kidney, skin, brain and coronary circulations remains unaltered although the absolute flow is increased. Enhancer blood flow to the skin allows dissipation of heat from the uterine circulation, as the

fetus is 1°C warmer than the mother. [5]

4. Aorto-caval Compression:

The greatest rise in cardiac output is seen in the first and second trimesters. The amount and direction of change in the third trimester has been the subject of great controversy as values vary depending on the position of the subject when the measurement is made. Maternal hypotension associated with the supine position in late pregnancy was first recognized in 1942. That was due to inferior vena-caval compression, rather than displacement of the heart and diaphragm. Eleven percent of term pregnant women made to lie on injected radio-opaque dye and showed that the term uterus impeded venous return by obstructing the inferior vena cava. There was some collateral return via the para-vertebral and azygous systems and through the ovarian veins. Such collateral flow was often not sufficient to prevent a drop in tight atrial pressure and cardiac output. An additional factor was compression of the aorta by the uterus, increasing afterload and again reducing cardiac output. Angiography in women in the third trimester has shown that in the supine position the aorta is both compressed and displaced reducing placental perfusion in the supine position. As would be expected, there is evidence to suggest reduced fetal cerebral oxygenation if the mother adopts the supine position at term. [6]

Cardiac output studies in primiparous women who consented to pulmonary artery catheterization between 36 and 38 weeks gestation have been carried out. Cardiac output, which was 6.0 1/min in the supine position and 5.4 1/min in the standing position. The importance of avoiding

aorto-caval compression in late pregnancy cannot be overstated. The supine position must always be avoided though in a few women aorto-caval compression may still occur in the semi-recumbent position, or even whilst standing. It may be in these cases that the collateral venous system is poorly developed or that increased parasympathetic tone increases the likelihood of bradycardia. Aorto-caval compression is also more likely to develop in multiple-pregnancy, polyhydramnios, and maternal obesity, and can occur from as early as the beginning of the second trimester. (7)

If aorto-caval compression is allowed to persist, symptoms of the supine hypotension syndrome occur. The mother begins to feel anxious, sweaty, nauseated and eventually becomes profoundly hypotensive. Abnormal fetal heart rate patterns may develop, signifying fetal compromise through hypoxia and acidosis. Treatment consists of turning the patient from the supine to the lateral position with the administration of oxygen to the mother. (6)

If compensatory reflexes are obtunded or abolished by regional or general anesthesia, then marked maternal hypotension and fetal distress may develop in the supine position. If the full lateral position is impractical, for example, during a caesarean section, then 15° of lateral tilt must be achieved. During, cardiac arrest it is necessary to undertake caesarean delivery of a fetus in order to increase the chances of maternal survival if cardiopulmonary resuscitation remains ineffective after 5 minutes, to alleviate the effects of aorto-caval compression. (3)