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HEMODYNAMIC CHANGES AND CARDIAC FUNCTION DURING THE COURSE OF NORMAL
PREGNANCY AND THE PEURPERIUM

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Introduction

Pregnancy is associated with substantial physiologic changes that require adaptation of the cardiovascular system. Maternal heart disease can interfere with this process and, therefore, may have an important effect on both the mother and fetus. The increased circulatory burden of pregnancy can unmask previously unrecognized heart disorders and rapidly worsen heart disease toward a potentially lethal situation. Recognition and management of cardiac illness in pregnancy can be difficult at times. The absence of well-defined treatment guidelines and only scanty literature in the field represent further burdens for the practicing physician. A comprehensive understanding of cardiovascular adaptation during pregnancy, labor, and the puerperium is essential for the management of pregnant patients with cardiovascular disorders.

Hemodynamic Changes During Pregnancy

Blood Volume

One of the major circulatory changes during pregnancy relates to blood volume. Although there has been general agreement that blood volume increases significantly during pregnancy there has been disagreement about the magnitude of an increment and the pattern of change throughout pregnancy. The blood volume is pregnancy usually increases as early as approximately the sixth week of pregnancy and

continues to increase rapidly until some time in the mid-pregnancy and subsequently continues to increase but at a slower rate. There exists considerable individual variations in the degree of volume expansion during pregnancy and these variations range from 20 to nearly 100 percent of non-pregnant values. Most of the data, however, support the notion that at about the third trimester and between 33 and 36 weeks of pregnancy normal pregnant women have achieved a peak blood volume expansion of 50% above the pre-pregnancy level. From that point on the data are conflicting and some investigators suggest that there is a plateau in the volume expansion until the time of delivery and some others even suggest that there might be some slight decrease.

The rise in blood volume during pregnancy was found to correlate directly with fetal weight. Additional studies demonstrated a positive and good correlation between placental weight and mass and blood volume expansion. Another study also verified these finding when they demonstrated a significant correlation between total blood volume, weight of products of conception, and neonatal weight and suggested a correlation between maternal weight and total blood volume.

Normal women in subsequent pregnancies tend to show similar volume expansions by some investigators although others disagree and suggest that there is a higher degree of volume expansion in subsequent pregnancies. In addition, women with multiple gestations have higher increments of blood volume expansion. Subsequent normal pregnancies are associated with increasing fetal weights up to three pounds and then may plateau or even decline. Differences in parity among different studies could account for the conflicting conclusions.

Although the red blood cell mass increases during pregnancy concomitant with blood volume, the latter shows a more rapid and significant rise. The predominance of the plasma volume increase results in a fall in hemoglobin concentration during pregnancy, a situation that

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has been called "physiologic anemia of pregnancy". The levels can be as low as 33-38 percent

and hemoglobin levels can reach 11-12 grams

Mechanisms of Hypervolemia in Pregnancy

The mechanisms leading to hypervolemia in pregnancy are still not entirely understood

and seem to be multifactorial. Increased estrogen leads to increased amounts of renin, increased

angiotensin II, and increased amounts of aldosterone. Aldosterone increases the retention of

water and sodium and this leads to increased extracellular fluid volume. In addition, human

chorionic somatomammotropin and increased prolactin promote erythropoiesis which leads to

increased red blood cell mass. The combination of increased extracellular fluid volume and

increased red blood cell mass leads to increased blood volume. The role of atrial natriuretic

factor (ANF) in mediating changes in fluid balance during gestation is still unclear.

A fetus is not essential for the development of hypervolemia. A 50% increase in volume

has also been reported with hydatidiform moles. Postural changes effect plasma volume in late

pregnancy. Volume appears to decline in the supine position because the enlarged uterus

occludes the inferior vena cava and traps blood in the legs. The increase in blood volume with

pregnancy appears to meet the essential physiologic needs of both the mother and the fetus.

Therefore, a normal pregnant woman can withstand hemorrhage equal to the volume of blood

added to the circulation during normal pregnancy without decompensation.

Cardiac Output

One of the most significant hemodynamic phenomena during pregnancy is the change in

cardiac output which can reach as much as 50% above pre-pregnancy levels. It is generally

agreed that cardiac output begins to rise during the first trimester probably around the 10th week

of pregnancy. It increases rapidly until 20-24 and subsequently, it continues to increase at a

slower rate and up until 38 to 40 weeks gestation. Cardiac output continues to increase although

at a lower rate up to 38-40 weeks. After this point it remains rather stable with the exception of patients who are in the supine position when cardiac output drops.

Cardiac output is the product of stroke volume and pulse rate. The rise in cardiac output early in pregnancy is disproportionately greater than the increase in heart rate and, therefore, is attributable to augmentation in stroke volume. As pregnancy advances the heart rate increases and becomes the more predominant factor in increasing cardiac output. At the late stages of pregnancy the stroke volume declines to normal, non-pregnant values.

Supine Hypotension Syndrome of Pregnancy

Radiological studies have demonstrated an obstruction of the vena cava in approximately 90 percent of women who were studied in the supine position. Many women develop a paravertebral collateral circulation during pregnancy which permits blood from the legs and the pelvic organs to bypass the occluded inferior vena cava. When the inferior vena cava is compressed to fall in cardiac output is normally followed by a compensatory increase in peripheral resistance and, therefore, no significant change in systemic blood pressure or heart rate occurs. In addition, many patients who have collateral circulation can withstand the compression of the vena cava without any changes in the cardiac output. The patients, however, who do not have this collateral paravertebral circulation are vulnerable to acute changes in cardiac output that cannot be compensated and which lead to decreased blood pressure resulting in symptoms such as weakness, light-headnness, nausea, dizziness, and even syncope. This phenomenon is described as "supine hypotensive syndrome of pregnancy" or "the uterocaval syndrome" and is usually relieved promptly when the supine position is abandoned. The incidence of the supine hypotension syndrome ranges in different reports from 0.5 to 11 .2

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percent.

Usually the fall in cardiac output that happens with the supine hypotension syndrome is not followed by increase in the maternal heart rate. To the contrary when the compression of the vena cava is prolonged a profound bradycardia may develop just before the symptoms of the patient become intensified. The fact that despite supine caval compression and falling cardiac output usually no acceleration of the heart rate takes place, is interesting and probably unique to caval occlusion.

Heart Rate

Heart rate rises during pregnancy with a mean increase of about 10-20 beats per minute at term. Mean values vary from 78 to 89 beats per minute. Twin pregnancies are associated with an earlier acceleration of the heart rate and a peak increase of up to 40% above the non-pregnant level near term. The heart rate decreases slightly with a change from the supine to the lateral position. The rise in heart rate was found by some investigators to occur early in pregnancy and to remain similarly elevated until term. Some other studies, however, have suggested a gradual increase in pulse rate with a peak at the third trimester.

Systemic Arterial Blood Pressure

During pregnancy there is a slight fall of systolic arterial blood pressure and a considerable decrease in diastolic pressure. The decrease in blood pressure starts in the first trimester, reaches a peak in mid-pregnancy, and blood pressure returns toward nonpregnant levels before term. The magnitude of the changes varies from study to study. Both age and parity were observed to have significant effects on blood pressure during pregnancy. With increase in parity the blood pressure increases in subsequent pregnancies. Within each parity

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level, mean systolic arterial pressure is increased for pregnant women who are 35 and over in comparison to pregnant women who are less than 35.

The change in systolic and diastolic arterial pressure that is observed during pregnancy starts usually at about 8-10 weeks and is the result of decreased systemic vascular resistance. The mechanism for this phenomena is not entirely clear although it is believed that primarily the decline in the systemic vascular resistance is the result of a lower resistance circulation in the pregnant uterus. Other vascular systems are also affected by pregnancy with lower systemic vascular resistance. It seems that hormonal effects that take place during pregnancy have an impact of the systemic vascular resistance (estrogen, prolactin, PGF_2, and PGI_2). Finally, increased heat production by the developing fetus may also result in vasodilatation, especially in such heat losing areas such as the hands of the pregnant female and, thus, may contribute to the decrease in systemic vascular resistance.

<u>Uterine Blood Flow</u>

A significant increase in uterine blood flow occurs during normal human pregnancy. Uterine blood flow can reach a maximum of approximately 1000 ml/min. at term from a pre-pregnancy flow of about 30 ml to 50 ml/min. This rise in uterine blood flow is made possible due to a progressive fall in vascular resistance in the placental bed and the myometrium. Uterine blood flow correlates well with fetal size. The uterine blood flow may be affected by the myometrial contractility because of increased intramural pressure and compression of the vessels. During exercise most studies have shown a decrease in uterine blood flow due to diversion of blood to skeletal muscles. Similarly an increased incidence of premature labor and delivery, and of low fetal birth weights reported in women who performed endurance exercise may suggest a reduction in term blood flow during vigorous exercise. In general, normal uterine

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blood flow in pregnancy usually is associated with a good outcome of pregnancy as opposed to abnormal uterine blood flow that is associated with unfavorable outcome (pre-eclampsia, IUGR, and asphyxia).

Renal Blood Flow

In early stages of gestation renal blood flow increases significantly and reaches a peak of up to 30-80% above the non-pregnant state. As a result there is a 50% increase in glomerular filtration rate throughout pregnancy. Renal blood flow can be very much affected by positional changes of the patient and especially when the vena cava is compressed in the supine position. Changes in the renal flow during pregnancy are believed to be mediated by steroid hormones. Although prostacycline has hemodynamic effects during pregnancy it has not been found to affect renal blood flow.

Blood Flow to Extremities and Skin Perfusion

Although there are reports conflicting as to whether there is a change in the blood flow to the extremities during pregnancy it is most likely that there is a progressive rise of blood flow through the hands from the early weeks of pregnancy until delivery. Similarly perfusion of the skin is increased during pregnancy. This increase continues up to 30 weeks and then it remains stable. Vascular spiders and palmar erythema are seen during pregnancy in about 60% of white women and are an additional sign of vasodilatation. Another area in which blood flow increases during pregnancy is the nasal mucosal membrane. This explains the common finding of nasal congestion in pregnancy and sometimes spontaneous nasal bleeds.

Oxygen Consumption

In pregnancy a progressive increase in resting oxygen consumption occurs with a peak

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increase of 20-30% near term. The rise in oxygen consumption can be attributed to the increased metabolic needs of the mother and her growing fetus. While most of the increases in cardiac output occurs during the early part of pregnancy with a peak increase around the 20th week, oxygen consumption shows a gradual rise throughout pregnancy reaching its peak near term. It has been speculated that this discrepancy serves the fetus by supplying well oxygenated blood during the early stages of organogenesis before the fetal-placental circulation is fully established. At this stage of pregnancy the arterial venous oxygen difference is small and later on with advancement of gestation the arterial venous oxygen difference increases gradually reaching non-pregnant levels in the latter part of the gestation.

Flow to Other Organs

Early studies showed no significant changes in hepatic and cerebral blood flow during pregnancy. The effect of pregnancy on coronary blood flow is still unknown. It is assumed, however, to increase due to the augmentation in cardiac output.

Cardio-Circulatory Changes During Labor And Delivery

Labor and Delivery

Significant hemodynamic changes observed during labor and delivery can be attributed to several factors. Pain and anxiety has been implicated as well as uterine contractions that are followed by significant increase of 300-500 ml in central blood volume. Cardiac output is higher in the lateral position than in the supine position in patients in labor. When the patients are laboring in the supine position there is a significant increase in the cardiac output during contractions. This can be in part explained by the squeezing of blood out of the uterine vasculature and into the systemic circulation and in part by the fact that a well contracting uterus

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is raised above the inferior vena cava and thus the compression effect is eliminated. Measurement of inferior vena cava pressure during and in between contractions has demonstrated significantly lower pressure in the vena cava during contractions and significantly higher in between contractions.

During contractions and just a few seconds prior to the contraction the blood pressure increases above baseline. The systolic can increase as much as 35 mmHg and the diastolic as much as 25 mmHg. As labor advances during the second stage the blood pressure is gradually increasing. Since there is no change observed during the same period in the systemic vascular resistance it is assumed that this change in the blood pressure is the result of increasing cardiac output. In patients with hypertension this may lead to severe changes(severe pre-eclampsia/eclampsia) in a very short period of time.

Hemodynamic changes during labor are markedly influenced by the form of anesthesia or analgesic employed. In general caudal anesthesia does not affect the hemodynamic changes caused by uterine contractions. The decrease in pain and apprehension abolishes, however, the progressive rise in cardiac output which is seen between contractions and limits the absolute increase of cardiac output at delivery. Both caudal and local anesthesia do not significantly alter the cardiovascular response to the uterine contractions. Patients in labor respond differently to local and caudal anesthesia. With local anesthesia both the systolic and diastolic blood pressures show a mild gradual rise during the first stage of labor and a significant increase during the second stage. These changes are associated with a progressive increase in stroke volume towards a peak immediately following delivery. In contrast, caudal anesthesia is associated with no significant change in heart rate and both the diastolic and systolic blood pressures are maintained constant throughout labor and delivery. The stroke volume is also maintained throughout labor

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but rises rapidly after delivery.

Cesarean Section

During delivery with Cesarean section the hemodynamic changes that may take place are mostly related to the anesthesia used. Usually caudal anesthesia with intra-thecal injection is more troublesome with fluctuations in the hemodynamic condition of the patient. In contrast, general anesthesia with thiopental and maintenance with nitrous oxide and succinyl choline as well as epidural anesthesia with appropriate volume expansion and without epinephrine are associated with more stable hemodynamic conditions.

Postpartum

The hemodynamic changes that take place in the postpartum period can be summarized as follows. The blood volume decreases and the magnitude of this decline is about 10% with vaginal deliveries and about 5 - 30 % with Cesarean sections. The cardiac output increases by as much as 60-80% immediately postpartum and then drops to levels slightly higher than non-pregnant state, and it may remain at this level for a few weeks postpartum. The stroke volume increases and the heart rate decreases. The magnitude of decrease of the heart rate has been reported to be anywhere from 4 -17 beats per min. shortly after delivery. The blood pressure can be variable depending on the blood loss and other hemodynamic parameters. The peripheral vascular resistance is usually increases or it may remain unchanged.

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