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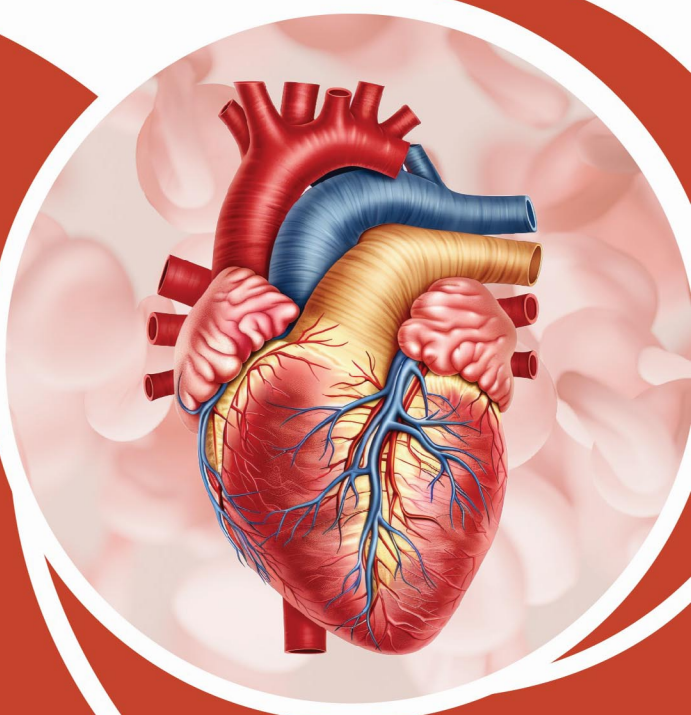
Obs and Gynae PG Focus Series

Cardiac Diseases in Pregnancy



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1

CHAPTER

Cardiovascular Physiology of Pregnancy

INTRODUCTION

Pregnancy is a dynamic process associated with significant physiological changes in the cardiovascular system. The increased metabolic demands of the mother and fetus are met through these changes by ensuring adequate uteroplacental circulation for fetal growth and development. Preeclampsia and intrauterine growth retardation result if the hemodynamic changes are insufficient.

Pregnancy is associated with vasodilation of the systemic and renal vasculature. Peripheral vascular resistance reduces significantly in the first trimester, reaches a low in the middle of the second trimester, and then plateaus or slightly increases for the duration of the pregnancy. The reduction is between 35 and 40% of the baseline. Systemic vascular resistance returns to prepregnancy levels 2 weeks after birth. Increased vascular compliance has been observed in normal human pregnancy starting in the first trimester. In the postpartum period, the systemic vascular resistance increases to the normal prepregnancy level. By the end of the first trimester there is a decrease in serum creatinine, urea, and uric acid values as renal blood flow and glomerular filtration rate is increased due to vasodilatation.[1]

Cardiovascular changes have been discussed under following subheading.

CARDIAC OUTPUT

There is increase in cardiac output throughout pregnancy. To measure cardiac output mother is generally made to lie in left lateral decubitus position to avoid variation due to position.

By the beginning of the first trimester, there is a significant increase in cardiac output, which continues until the second trimester. In a typical, singleton pregnancy, the increase in cardiac output can be up to 45% by 24 weeks.

When compared to singleton pregnancy, twin pregnancy results in a 15% increase in cardiac output, resulting in an increase in left atrial diameter, which is consistent with volume overload. Early in pregnancy, an increase in stroke volume is mediated by an increase in heart rate, while later in pregnancy is mediated by an increase in cardiac output. The stroke volume increases gradually during pregnancy until the end of the second trimester,

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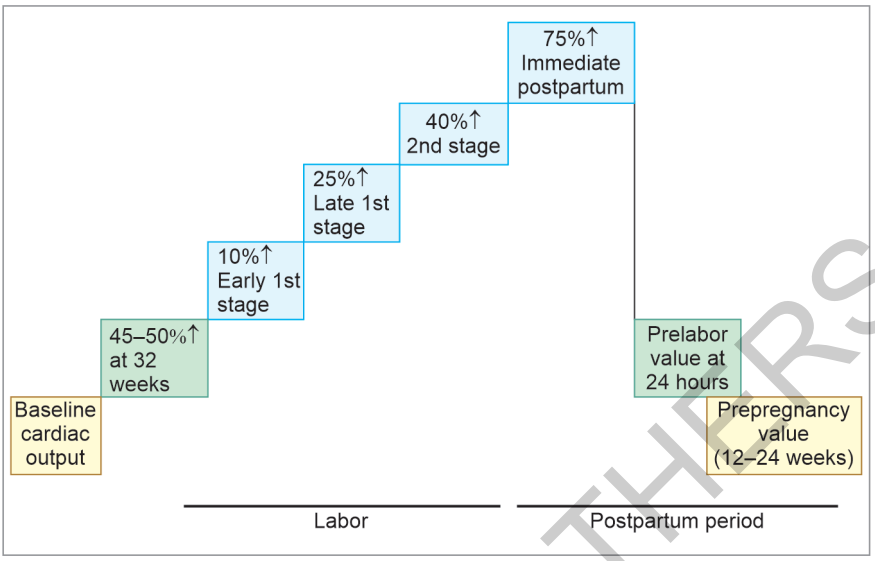

Fig. 1: Increase in cardiac output during pregnancy, labor, and postpartum period.
Source: Bhatia P, Chhabra S. Physiological and anatomical changes of pregnancy: Implications for anaesthesia. Indian J Anaesthesia. 2018;62(9):651-7.

after which it remains steady or declines late in the pregnancy. **Figure 1** shows the trend in increase in cardiac output during pregnancy, labor, and postpartum period.

BLOOD PRESSURE

Systolic blood pressure, diastolic blood pressure, and mean arterial pressure decreases during pregnancy. The fall in diastolic and mean arterial pressure is more than the fall in systolic blood pressure. Maximum fall in arterial pressure occurs in second trimester.

HEART RATE

During normal pregnancy, the heart rate rises by 10–20 beats per minute, peaking in the third trimester. The overall rise in heart rate is between 20 and 25% over baseline.

CONTRACTILITY

Although multiple cardiovascular parameters are altered during pregnancy, myocardial contractility, and ejection fractions do not appear to change during pregnancy.

SYMPATHETIC ACTIVITY AND BARORECEPTORS

Vasomotor sympathetic activity is increases, and this increase occurs very early in pregnancy. Normal pregnancy appears to be associated with

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increased maternal baroreceptor sensitivity and an attenuated responsiveness to α-adrenergic stimulation.

PREGNANCY HORMONAL CHANGES

Hormones (estrogen and progesterone) have been demonstrated to have an endothelium-dependent vasodilatory role in pregnancy that can influence small arterial resistance vessels. Although nitric oxide plays a crucial part in the reproductive system's physiology, its role in mediating the systemic vasodilation seen during human pregnancy is unknown.[2]

RENIN–ANGIOTENSIN–ALDOSTERONE SYSTEM

The renin–angiotensin–aldosterone system is activated throughout a normal pregnancy. The renin–angiotensin–aldosterone system is more active, with increases in plasma volume beginning at 6–8 weeks and continuing until 28–30 weeks. During pregnancy, the generation of renin substrate (angiotensinogen) rises, resulting in an increase in angiotensin levels. This helps retain salt and water in pregnancy, which maintains blood pressure, as maternal systemic and renal arterial dilation (with resulting salt and water loss) creates an "underfilled" cardiovascular system. Furthermore, during pregnancy, relaxin stimulates increased vasopressin secretion and drinking, resulting in increased water retention. There is hyponatremic hypervolemia in pregnancy in spite of increase in exchangeable sodium. Progesterone acts on the mineralocorticoid receptor as a potent aldosterone antagonist and helps in preventing sodium retention and to protects against hypokalemia. Maternal plasma atrial natriuretic peptide levels increases in the third trimester, suggesting a significant role in postpartum diuresis.[3]

CHANGES IN PLASMA VOLUME AND RED BLOOD CELL MASS

Total blood volume, plasma volume, and red cell mass increases during pregnancy to provide nutrition. Erythropoiesis maybe enhanced by effect of placental lactogen. This increase is more when red cell, hemoglobin is lower and subclinical iron deficiency exists. The decreased life span of red blood cells also results in increased production.

Reduced plasma volume expansion has been linked to preeclampsia and other pathological diseases, and there is a clear link between plasma volume expansion and fetal growth. Blood volume rises dramatically in the first few weeks of pregnancy and continues to rise steadily throughout the pregnancy. The overall blood volume increase ranges from 20 to 100% above prepregnancy levels, with the average being about 45%. There is a 40% increase in red blood cell synthesis via erythropoiesis. Physiological anemia occurs when the volume of plasma expands higher than the volume of red blood cells, resulting in physiological dilution.

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REMODELING

Left ventricular wall thickness and left ventricular wall mass increase throughout pregnancy. Increased vascular distensibility, aortic augmentation index, a marker of aortic stiffness, decreases significantly early during pregnancy, due to systemic vasodilation, reaching a nadir in the second trimester and gradually increasing in the third trimester.

TRANSTHORACIC ECHOCARDIOGRAPHY AND CARDIOVASCULAR MAGNETIC RESONANCE

Mild four-chamber dilatation (changes in the right atrium and ventricle are often higher than those in the left atrium and ventricle) is common during pregnancy, along with trivial mitral regurgitation and physiological tricuspid and pulmonary regurgitation. The most common imaging technique used in pregnancy is echocardiography, which is safe in all three trimesters.

A cardiac MRI is a better indication of left ventricular volume. When compared to cardiovascular magnetic resonance, echocardiography underestimates left ventricular mass, cardiac output, and stroke volume.

Finally, in pregnant patients with suspected cardiovascular problems, cardiovascular magnetic resonance of the atria, right ventricle, and aorta is probably more accurate than echocardiography and should be explored on a case-by-case basis.[4]

LABOR AND DELIVERY

Many factors contribute to the greatest increase in cardiac output during labor and immediately after delivery, including an increase in heart rate and preload caused by uterine contraction pain, increased circulating catecholamine, and auto transfusion of 300–500 mL of blood from the uterus into systemic circulation.

Spinal anesthesia can have serious side effects on the heart and blood vessels. A considerable drop in systemic vascular resistance and compensatory increases in heart rate and stroke volume are the most common responses to spinal anesthesia during caesarean section. In obstetrics, phenylephrine is preferred over ephedrine as a vasopressor.

CLINICAL CARDIOVASCULAR FINDINGS IN NORMAL PREGNANCY

Shortness of breath and increased fatigue can be experienced by normal woman during pregnancy. As resting cardiac output is increased in pregnancy, maximal cardiac output induced by exercise is achieved at lower level of work. During rest or weight bearing exercise, there is significant increase in maternal oxygen consumption.

In the respiratory system, the tidal volume and resting minute ventilation are increased and expiratory reserve volume and functional residual capacity is reduced.

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TABLE 1: Changes in electrocardiogram and cardiac auscultation during pregnancy.[5]

Changes in cardiac auscultation	*Changes in electrocardiogram*
• Loud S_1 • Ejection systolic murmur • S_3 and S_4 (no clinical significance)	• Shortened PR and QT interval • Shifted QRS axis • Rightward (first trimester) • Leftward (third trimester) • ST segment depression • Isoelectric low-voltage T waves in both precordial and limb leads

Source: Bhatia P, Chhabra S. Physiological and anatomical changes of pregnancy: Implications for anaesthesia. Indian J Anaesthesia. 2018;62(9):651-7.

Physiological anemia of pregnancy develops as a result of increase in plasma volume more than that of red blood cell mass with increased vasopressin secretion causing water retention leading to hyponatremic hypervolemic state of pregnancy. As a result, gestation-dependent edema can be found in up to 80% of healthy pregnant women.

In 90% of cases, an ejection systolic flow murmur is heard, a third heart sound is heard in 80% of cases, and an atrioventricular diastolic flow murmur is heard in 20% of cases.

In a normal pregnancy, electrocardiogram (ECG) changes include an increase in resting heart rate and a little shift in the QRS complex (left or right). **Table 1** highlights the ECG changes in pregnancy.

CONCLUSION

The cardiovascular system undergoes significant structural and hemodynamic changes which are necessary for fetal growth and development, failure to achieve which can lead to fetal and maternal morbidity.

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