Changes in Cardiovascular System During Pregnancy

Dr.Snehal Ram Rathod ,
Associate Professor ,
Stree Roga-Prasutitantra Department
Ayurved Mahavidyalaya, Pusad

Abstract

Cardiovascular alterations during pregnancy are characterized by an increased vascular volume, cardiac output, and heart rate, with a marked fall in vascular resistance. Cardiac output is about 40-50% higher during the third trimester. Even higher values of cardiac output are observed during uterine contractions in labor. In general, arterial blood pressure remains unaffected or demonstrates some tendency toward lower diastolic pressure. The higher blood volume is associated with a slight increase in left ventricular dimensions. Left ventricular contraction force and its first derivative remain unchanged. Many symptoms and findings during pregnancy are caused by the described changes, such as dyspnea on exertion, presyncope due to pressure on the inferior vena cava resulting in a decreased venous return to the heart, prominent jugular venous pulsation, leg edema, and ejection murmurs over the aorta and pulmonary artery. Paroxysmal nocturnal dyspnea, anginal chest pain, syncopy, anasarca, and diastolic heart murmurs require further evaluation.

Introduction

The pregnancy induced changes in the cardiovascular system develop primarily to meet the increased metabolic demands of the mother and foetus. Despite the increased workload of the heart during gestation and labour the healthy woman has no impairment of cardiac reserve.

Physiological changes during pregnancy facilitate the cardiovascular system to the increased metabolic needs of the mother, enabling adequate delivery of oxygenated blood to peripheral tissues and the fetus. Changes occur in circulating blood volume (affecting preload), peripheral vascular compliance and resistance (affecting after load), myocardial function and contractility, heart rate.

Hemodynamic changes during pregnancy include increased blood volume, cardiac output (CO) and maternal heart rate; decreased arterial blood pressure; decreased systemic vascular resistance. CO increases up to 30% in the first stage of labor, primarily because of increased stroke volume; maternal pushing efforts in the second stage of labor can increase CO by as much as 50%.

Blood Volume increases progressively from 6-8 weeks gestation (pregnancy) and reaches a maximum at approximately 32-34 weeks with little change thereafter. The increase in plasma volume (40-50%) is relatively greater than that of red cell mass (20-30) resulting in haemodilution and a decrease in haemoglobin concentration. Intake of supplemental iron and folic acid is necessary to restore haemoglobin levels to normal (12g/dl.)

The increased blood volume serves two purposes

First, its facilities maternal and fetal exchanges of respiratory gases, nutrients and metabolites.

Second, it reduces the impact of maternal blood loss at delivery. Typical losses of 300-500 ml of vaginal births and 750-1000 ml for caesarean sections are thus compensated with the so-called "autotransfusion" of blood from the contracting uterus (cf. cardiac output below)

Blood Constituents : As mentioned above, red cell mass is increased 20-30% leukocyte counts are variable during gestation, but usually remain within the upper limits of normal. Marked elevation, however, develop during and after delivery. Fibrinogen, as well as total body and plasma levels of factors VII, X and XII increase markedly. The number of platelets also rises, yet no above the upper limits of normal. Combined with a decrease in fibrinolytic activity, these changes tend to prevent excessive bleeding at delivery. Thus, pregnancy is a relatively hyperacoagulable state, but during pregnancy neither clotting or bleeding times are abnormal.

Cardiac Output : Increase to a similar degree as the blood volume. During the first trimester cardiac output

is 30-40% higher than in the non-pregnant state. During labour, further increases are seen with pain in response to increased catecholamine secretion, this increase can be blunted with the institution of labour analgesia. Also during labour, there is an increase in intravascular volume by 300-500 ml of blood from the contracting uterus to the venous system. Following delivery this autotransfusion compensates for the blood losses and tends to further increases cardiac output by 50 % of pre delivery values.

Cardiac Size / Position : The heart is enlarges by both chamber dilation and hypertrophy. Upward displacement of the diaphragm by the enlarging uterus causes the heart to shift to the and anteriorly.

Blood Pressure : Systemic arterial pressure is never increased during normal gestation. In fact, by mid-pregnancy, a slight decrease in diastolic pressure can be recognize. Plumonary arterial pressure also maintains a constant level. However, vascular tone is more dependent upon sympathetic control than in the non pregnant state, so that hypotension develops more readily and more markedly consequent to sympathetic blockage following spinal or extradural anaesthesia. Central venous and branchial venous pressures remain unchanged during pregnancy, but femoral venous pressure is progressively increased due to mechanical factors.

Aortocaval Compression : From mid-pregnancy, the enlarged uterus compresses both the inferior vena cava and the lower aorta when the patient lies supine (on the bank). Obstruction of the inferior vena cava reduces venous return to the heart leading to a fall in cardiac output by a much as 24 % towards term.

Cardiovascular Changes During Pregnancy, During labour and During Puerperium is as follows :

CHANGES DURING PREGNANCY	CHANGES DURING LABOUR	CHANGES DURING PUERPERIUM
- RR ↑ in pregnancy. - Breath more deeply event at rest. -Anterior posterior and transverse diameter ↑ about 2cm resulting in a 5-7 expansion of the chest circumference. - Progressively increase the subscostal angle from 68° to 103° at term. - Changes mediated by progesterone and relaxin which ↑ ribcage elasticity by relaxing ligaments. -By 8/52 gestation: Expansion of the ribcage cause the Tidal Volume (TV)↑ by 30-40%.	-Respiratory responses are greatly affected by stage of labour and the respond to pain and anxiety. - TV (tidal volume) range from 350 to 2250ml and minute ventilations from 7 to 90 L/min	Back to normal

Conclusions :

Improvements in diagnosis and treatment of patients with congenital heart disease along with an increasing number of women delaying pregnancy until later ages have increased the incidence of women at risk for cardiovascular complications during pregnancy. Several factors associated with increased maternal and fetal complications during pregnancy have been identified and include poor functional class (NYHA class III–IV) or cyanosis, history of previous cardiac events or arrhythmias, LV obstruction (moderate to severe MS or AS), and reduced LV systolic function (LVEF ≤ 40%). Patients with certain high-risk features such as pulmonary HTN, Marfan syndrome with aortic dilatation, or severely reduced LV systolic function should be counseled against becoming pregnant. Patients should be counseled on obtaining adequate rest, and correctable conditions such as anemia should be treated. Vaginal delivery usually carries the lowest risk of complications in most cardiac conditions but caesarean delivery may be indicated in women with certain conditions or with prolonged or difficult labor. To optimize outcomes in pregnant women with underlying cardiac disease, patients should be evaluated and monitored with a "team" approach by both an obstetrician and cardiologist.

References :

1. Christianson RE. Studies on blood pressure during pregnancy. I. Influence of parity and age. Am J Obstet Gynecol. 1976;125:509–13.
2. Moutquin JM, Rainville C, Giroux L, Raynauld P, Amyot G, Bilodeau R, et al. A prospective study of blood pressure in pregnancy: prediction of preeclampsia. Am J Obstet Gynecol. 1985;151:191–6.
3. Hunter S, Robson SC. Adaptation of the maternal heart in pregnancy. Br Heart J. 1992;68:540–3.
4. Poppas A, Shroff SG, Korcarz CE, Hibbard JU, Berger DS, Lindheimer MD, et al. Serial assessment of the cardiovascular system in normal pregnancy. Role of arterial compliance and pulsatile arterial load. Circulation.1997;95:2407–15.
5. D.C. Datta's, Texbook of Obstetrics, Hiralal Konar D.C. Datta's
6. D.C. Datta's, Texbook of Gynecologist, Hiralal Konar D.C. Datta's
7. Williams Obstetrics, 25th Edition ebook Jodi S.Dashe, Steven L.
8. Sladek SM, Magness RR, Conrad KP. Nitric oxide and pregnancy. Am J Physiol. 1997;272:R441–63.
9. Conrad KP, Novak J. Emerging role of relaxin in renal and cardiovascular function. Am J Physiol Regul Integr Comp Physiol. 2004;287:R250–61.