Assessment Of Left Ventricular Structural And Functional Indices By Echocardiography In Relation To The Haemoglobin Levels In Second Trimester Of Pregnancy

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Abstracts: Background & objectives: Pregnancy is characterized by profound changes in functions of virtually every regulatory system in human body. Blood volume increases progressively from 6 – 8 weeks of gestation & reaches a maximum at approximately 32 to 34 weeks. The increase in plasma volume (40-50%) is relatively greater than that of RBC mass (20 -30%) resulting in hemodilution & decrease in haemoglobin concentration. Thus during pregnancy the mother is under risk of developing nutritional deficiency anaemia. Hence it is decided to determine the impact of altered haemoglobin levels in second trimester of pregnancy on left ventricular hemodynamic functions in present study. Method: In present study subjects were females in the age group of 20 – 30 years with the singleton midterm pregnancy (20 -28 weeks) attending antenatal clinics of New Civil Hospital, Surat whereas the control group comprised of purposive sample (33 healthy subjects) from those attending other outpatient department in reference to comparable age, height & accessibility. Pulse and Blood Pressure was measured by standard techniques and capillary blood was collected for Hb% estimation by cynmethhemoglobin method. Finally, Echocardiography was recorded using MEGAS CVX & MEGAS GPX equipped with ADV4 software from ESAOTE s.p.a Firenze, Italy and the frequency used for Doppler echocardiography was 2.0 -2.5 – 3.3 -5.0 MHZ & sweep time was 2 – 12 seconds. Results: The observations suggest volume overload during pregnancy is a risk factor for left ventricular contractility functions. Interpretation & conclusion: Increased values of stroke volume, cardiac output & cardiac index interpret the result of inability to compensate pressure overload or elevated venous return. Left ventricular diastolic dysfunction in the form of relaxation abnormalities is one of the first changes with left ventricular hypertrophy. The reduction of haemoglobin in second trimester; significantly and negatively correlates with the left ventricular cardiac function. The significant increase in CO and CI reflects a hyperkinetic heart in pregnancy. The increase in percent ejection fraction and percent fractional shortening in the study population can be best explained by Frank- Starling's law governing the heart. [Kariya D et al NJIRM 2012; 3(3) : 7-10]

Key words: pregnancy, left ventricular hemodynamics, second trimester, haemoglobin levels

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Introduction: The anatomical, physiological and biochemical adaptation to pregnancy are profound. These remarkable changes begin soon after fertilization and continue throughout gestation and most occur in response to physiological stimuli provided by the fetus. These alterations during pregnancy help to maintain healthy environment for the fetus without compromising mother's health, although sometimes, these alterations determine small discomfort to the mother. Many of these physiological adaptations could be perceived as abnormal in the nonpregnant woman. For example, cardiovascular changes during pregnancy normally include substantive increase in blood volume and cardiac output which may mimic thyrotoxicosis. On the other hand these same adaptations may lead to ventricular failure if there is underlying heart disease. Thus physiological adaptations of normal pregnancy can be misinterpreted as pathological and can also unmask or worse pre-existing disease.[1]

The maternal blood volume increases markedly during pregnancy. Blood volume increases progressively from 6 – 8 weeks of gestation & reaches a maximum at approximately 32 to 34 weeks. The increase in plasma volume (40-50%) is relatively greater than that of RBC mass (20 -30%) resulting in hemodilution & decrease in haemoglobin concentration.[1]

The maternal blood volume shortly before term is about 30 per cent above normal. This increase occurs mainly during the latter half of pregnancy. The cause of the increased volume is likely due, at least in part, to aldosterone and estrogens, which are greatly increased in pregnancy and to increased fluid retention by the kidneys. Also, the bone marrow becomes increasingly active and produces

extra red blood cells to go with the excess fluid volume. Therefore, at the time of birth of the baby, the mother has about 1 to 2 litres of extra blood in her circulatory system. Only about one fourth of this amount is normally lost through bleeding during delivery of the baby, thereby allowing a considerable safety factor for the mother.[1]

The marked increase in plasma volume associated with normal pregnancy causes dilution of many circulating factors. Of particular note is the hemodilution of red blood cells. Although pregnancy is associated with an increase in the production of erythrocytes, this increase is outstripped by the relative increase in plasma volume. The haematological indices which depend on the proportion of plasma in a measured blood sample tend to decrease. They are red cell count, hematocrit and haemoglobin concentration.[3] Thus during pregnancy the mother is under risk of developing nutritional deficiency anaemia. There is a steady reduction in systemic vascular resistance which contributes towards hyperdynamic circulation. The vast alterations in the hemodynamics occur as a result of increased metabolic demand of the fetus, the expansion of vascular channels & increase in steroid hormones. [1]

Pregnancy is characterised by peripheral vasodilatation. The precise cause of this phenomenon is still, to some extent, speculative but there is evidence to implicate vasoactive factors derived from the endothelium, such as nitric oxide. A significant increase in heart rate can be demonstrated as early as the 5th week of pregnancy and this contributes to an increase in cardiac output.[3]

About 625 milliliters of blood flow through the maternal circulation of the placenta each minute during the last month of pregnancy. This, plus the general increase in the mother's metabolism, increases the mother's cardiac output to 30 to 40 per cent above normal by the 27th week of pregnancy; then, for reasons unexplained, the cardiac output falls to only a little above normal during the last 8 weeks of pregnancy, despite the high uterine blood flow.

The iron requirement tremendously increased during pregnancy as – about 375 mg of iron is needed for fetal blood formation and 600 mg is needed to form mother's own blood and cell mass. [2]

Plasma volume expansion and decreased Haemoglobin concentration occur during pregnancy, the developing fetus puts the mother to greater risk of nutritional anaemia. Hence we have decided to determine the impact of altered haemoglobin levels in second trimester of pregnancy on left ventricular hemodynamic functions in the study population.

This study makes an attempt to describe the pattern of cardiovascular changes by echocardiography in nonanaemic and anaemic females in second trimester normal pregnancy.

Aims & objectives:

1. To compare cardiovascular functions in second trimester of pregnancy to the non – pregnant state
2. To evaluate the left ventricular hemodynamic profile in pregnant & non pregnant females
3. To evaluate & correlate left ventricular structure & function in the study group.
4. To assess left ventricular systolic & diastolic functions in the pregnant & non pregnant females.
5. To evaluate the effect of anaemia on cardiovascular functions during pregnancy

Materials & Methods: With the prior permission of the subjects and the concerned head of the departments the subjects were randomly selected from age group of 20 – 30 years with the singleton midterm pregnancy (20 -28 weeks) attending antenatal clinics of New Civil Hospital, Surat. The control group comprised of purposive sample (33 healthy subjects) selected from those attending other outpatient department in reference to comparable age, height & accessibility. Pulse & blood pressure were measured by using standard techniques. To prevent any modulation by the gravid uterus in the pregnant females leading to the aortocaval compression it was thought prudent to measure pulse & blood pressure in the sitting position. Capillary blood was collected for Hb

estimation by cyanmeth-hemoglobin method. Echocardiography was recorded using MEGAS CVX & MEGAS GPX equipped with ADV4 software from ESAOTE s.p.a Firenze, Italy. For Doppler echocardiography the frequency used was 2.0 -2.5 – 3.3 -5.0 MHz & sweep time was 2 – 12 seconds.

From the M – mode study performed, based on the recommendations of the American Society of Echocardiography, the following hemodynamic indices were calculated:

- Left ventricular end systolic &end diastolic internal dimensions (LVIDs & LVIDd)
- Interventricular septal dimension & posterior wall thickness (IVSD & PWT)
- Cardiac output (L/min) = stroke volume x heart rate
- Stroke volume = $(LVID\ d)^3 – (LVIDs)^3$
- Stroke volume index = stroke volume / body surface area
- Cardiac index = cardiac output / body surface area
- Left ventricular ejection function assessed by endocardial fractional shortening (FS) & ejection fraction (EF) using the Teichholz method.

Observations: Study group comprised of pregnant females and control group includes nonpregnant healthy females

Table:1. Results of haemoglobin concentration:

Subject status		Haemoglobin (gm%)
Study group	Mean	9.54
	SD	0.34
Control group	Mean	11.34
	SD	5.76

Table:2 Results of mean pulse rate in study and control group:

SUBJECT STATUS		Pulse rate (/min)
Study group	mean	88.57
	SD	5.79
Control group	Mean	76.85
	SD	6.86

The above tables are suggestive of that the mean values of haemoglobin concentration were significantly lower ($p < 0.005$) in study group than control group. Table 2 summarizes that pulse rate in study group were significantly higher ($p<0.001$) than control group.

Table: 3 Results of echocardiographic Left ventricular functional indices:

Subject status		CO (liters/min)	CI (liters/min/m^2
Study group	Mean	4.88	3.46
	SD	2.07	1.57
Control group	Mean	3.76	2.68
	SD	1.67	1.16

Table 3 summarizes the mean parameters of CO & CI were significantly higher amongst anaemic group than control group.

Table: 4 Results of echocardiographic left ventricular contractility indices:

Subject status		EF	FS
Study group	Mean	76.95	39.48
	SD	8.4	7.03
Control group	Mean	71.96	35.78
	SD	13.78	8.9

The mean values of left ventricular contractility indices were significantly higher among pregnant anaemic group compared to control group.

Discussion: Pregnancy is associated with profound adaptive changes in the maternal hemodynamics. Pregnancy induces physiological changes that often confuse the diagnosis of haematological disorders. The maternal blood volume increases markedly during pregnancy. Pregnancy induced hypovolemia has several important functions:

a. To meet the demands of enlarged uterus.
b. To protect the mother against the adverse effects of blood loss associated with parturition.

During pregnancy the heart and circulation undergo remarkable physiological adaptation. Cardiac output is increased as early as 5^{th} week of pregnancy and this initial increase is a function of reduced systemic vascular resistance and an increase in heart rate. Multiple factors contribute to

these changes in overall hemodynamic function, allowing the cardiovascular system to adjust to the physiological demands of the fetus while maintaining maternal cardiovascular integrity.[4]

Aim of present study was to examine how physiological preload alteration influence left ventricular hemodynamic parameters and left ventricular systolic & diastolic functions. The study also evaluates the effect of anaemia on cardiovascular functions during pregnancy.

In severe anaemia blood viscosity may fall to as low as 1.5 from the normal value of about 3. This decreases the resistance to blood flow in the peripheral blood vessels. So that far greater quantity of blood passes through the tissue and returns to the heart, there by greatly increasing cardiac output. Moreover tissue hypoxia also causes peripheral vasodilatation and increase venous return and cardiac output. Thus one of the major effects of anaemia is greatly increased cardiac output as well as pumping workload on the heart.[1]

In our studies mean values of haemoglobin concentration were significantly lower ($p < 0.005$) in study group than control group. Observations suggest that volume overload during pregnancy is a risk factor for left ventricular contractility functions. Increased values of stroke volume, cardiac output & cardiac index in the study group compared to the control group interpret the negative correlation between reductions in haemoglobin level and left ventricular functions.

In present study left ventricular contractility was assessed with the use of ejection fraction & fractional shortening %. The mean values of left ventricular contractility indices were significantly higher among pregnant anaemic group compared to control group.

Conclusion: The reduction of haemoglobin in 2nd trimester significantly and negatively correlates with the left ventricular cardiac functions. The significant increase in cardiac output and cardiac index reflects a hyperkinetic heart in pregnancy. The increase in percent ejection fraction and percent fractional shortening in the study population can best be explained by the Frank – Starling's law governing the heart.

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