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Maternal Myocardial Performance in Various Stages of Pregnancy and Post-Partum

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Abstract: The aim of this study was to evaluate haemodynamic pattern during various stages of normal pregnancy and post-partum and study the effect of maternal factors. The study was conducted at Government Medical College and New Civil Hospital, Surat between August 2006 to October 2008. M-mode and Doppler echocardiography were performed in 19 normal pregnant women at 15-30 weeks, 31 normal pregnant women at 30-40 weeks and 8-12 weeks postpartum. Heart rate, stroke volume, cardiac output and left ventricular mass increased significantly, while total peripheral resistance decreased significantly during last trimester of pregnancy. Left ventricular contractility indices (percentage ejection fraction and fraction shortening) were within normal limit during pregnancy but fractional shortening was significantly higher post-partum than last trimester of pregnancy ($p < 0.05$). Maternal age was related to the transmitral peak velocity of early filling (E, $p = 0.001$) and the E to A ratio ($p < 0.001$), while height was related to heart rate ($p < 0.001$), stroke volume ($p = 0.003$), cardiac output ($p < 0.001$) and left ventricular mass ($p < 0.005$). This study express that along with gestation, maternal anthropometric profile may affect cardiac performance. Systolic performance was better in tall individuals and diastolic performance was better in younger individuals.

Key words: Maternal cardiac performance, echocardiography, pregnancy, post-partum

INTRODUCTION

Normal pregnancy is accompanied by maternal cardiovascular adaptations that include an increase in cardiac output with a decline in blood pressure and systemic vascular resistance (Desai *et al.*, 2004). Loading conditions change significantly during pregnancy (Fok *et al.*, 2006). In normal pregnancy the maternal left ventricle dilates and develops mild eccentric hypertrophy in response to volume load, increase in stroke volume and heart rate

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(Mabie *et al.*, 1994; Robson *et al.*, 1989). In Pregnancy left atrial diameter and left ventricular diameter are greater reflecting the increase in pre-load (Kametas *et al.*, 2003). The increase in heart rate and inotropic contractility imply that cardiovascular reserve is reduced. In pregnancy induced hypertension and fetal growth retardation, deviations from normal Left Ventricular (LV) systolic function may allow early detection of cardiac dysfunction, because of adaptation of the heart to the changing hemodynamic pattern in the prodromal period but abnormalities of diastolic function may be more sensitive than systolic (Mabie *et al.*, 1994; Kametas *et al.*, 2001b; Mesa *et al.*, 1999). Left ventricular subendocardial fibers are more susceptible than the circumferential fibers to the effects of pressure and volume load (Henein and Gibson, 1999a, b). Assessment of total vascular resistance, maternal hemodynamics and left ventricular geometry at mid term pregnancy can predict maternal and fetal complications (Barbara *et al.*, 2008). The increase in cardiac output early in pregnancy is predominantly due to augmentation in stroke volume, whereas in the 3rd trimester it is largely due to an accelerated heart rate and stroke volume contributes a little because of caval compression by the gravid uterus and decreased venous return to the heart. Increase in cardiac output seems to be enhanced in subsequent pregnancies (Clap and Capless, 1997). Heart rate peaks during the 3rd trimester with an average increase of 10 to 20 beat min⁻¹ (Clap and capless, 1997; Mesa *et al.*, 1999) although, on occasion it may be markedly faster. Reduced intrinsic function could occur as a result of subendocardial fibrosis consequent on high wall stress or possibly the effect of high catecholamine levels. By term pregnancy, an increase in ventricular wall stress is accompanied by deterioration in cardiac function (Zentner *et al.*, 2009). Few studies of diastolic and systolic function in normal pregnancies give variable results (Mabie *et al.*, 1994; Kametas *et al.*, 2001a, b; Mesa *et al.*, 1999). This variability may partly reflect the size of the study population, differences in study population characteristics such as maternal age, height, socioeconomic condition and BMI before pregnancy. Differences in methodology are also, likely to be important. Changes in long-axis systolic function and diastolic function occur in the presence of normal transverse systolic function in normal pregnancy and are partly dependent on maternal height, age and ethnicity. The changes do not resolve fully in the early post-partum period suggesting the possibility of intrinsic abnormalities of LV function (Jacqueline *et al.*, 2007). The aim of this study was to evaluate changes in maternal left ventricular performance in normal pregnancies and to study the effect of maternal characteristics on these measures.

MATERIALS AND METHODS

The study was conducted at Government Medical College and New Civil Hospital, Surat between February 2006 to March 2008 on 46 healthy pregnant women with normal pregnancies and no history of cardiac disease. The study was approved by the Ethical Committee of Government Medical College and New Civil Hospital, Surat and written informed consent was obtained from all the participants. Predetermined exclusion criteria for the selection of the study population were: Diabetes, maternal cardiovascular disease, pre-eclampsia, defined according to the guidelines of the International Society for the Study of Hypertension in Pregnancy (Davey and MacGillivray, 1988). The women were enrolled at their routine first trimester scan. Gestation was confirmed by last menstrual period and ultrasound measurement of the fetal crown-rump-length in the first trimester. The women were examined twice during their pregnancy at 15-30 weeks and 30-40 weeks and once post-partum at 8-12 weeks. Growth was anticipated by serial ultrasound scans. One examiner performed all measurements.

Echocardiograms were recorded using MEGAS CVX and MEGAS GPX equipped with ADV4 software from ESAOTE s.p.a. Firenze, Italy. For Doppler echocardiography the frequency used was 3.5 MHz. Echocardiographic variables were calculated according to the American Society of Echocardiography (ASE) guidelines (Sahn *et al.*, 1978). Left Ventricular Internal Dimensions (LVIDs and LVIDd) at systole and diastole, Interventricular Septal dimension and Posterior Wall Thickness (IVSd and PWT) were measured. Stroke Volume (SV), Cardiac Output (CO), percentage Ejection Fraction (EF%), percentage Fraction Shortening (FS%) and Total Peripheral Resistance (TPR) were calculated from the measured dimension by the following formula of ASE convention:

$$SV = (LVIDd)^3 - (LVIDs)^3$$

$$CO (L \text{ min}^{-1}) = \text{Stroke volume (SV)} \times \text{Heart rate (HR)}$$

$$EF (\%) = \frac{(LVIDd)^3 - (LVIDs)^3}{(LVIDd)^3} \times 100$$

$$FS (\%) = \frac{(LVIDd - LVIDs)}{(LVIDd)} \times 100$$

$$TPR (\text{dyn} \times \text{sec} \times \text{cm}^{-5}) = (\text{mean BP} \times 80) / CO$$

Left Ventricular Mass (LVM) were measured using ASE measurements by the following equation (Devereux *et al.*, 1986):

$$LV M (ASE) = 0.8[1.04(IVS + LVIDd + PWT)^3 - (LVIDd)^3] + 0.6 \text{ g}$$

The transmitral peak velocity of early (E) and late atrial filling (A) were measured and the E to A ratio calculated (Kametas *et al.*, 2001a, b; Quinones *et al.*, 2002).

Transabdominal ultrasound examination was carried out for measurement of the fetal head circumference, abdominal circumference and femur length to ensure that the women had normal for gestational age size fetuses.

Statistical analysis was performed using the EpiInfo 6 and SPSS 12 package. Data were expressed as Mean $\pm$ SD. Unpaired Student's t-test was applied to the data of independent samples for equality of means, Levene's test for equality of variances and one-way analysis of variance (ANOVA). The probability value ($p < 0.05$) was considered statistically significant as this could be interpreted that the factor is less likely to occur due to chance, while a probability value ($p > 0.05$) was considered statistically not significant because such a difference could commonly occur due to chance and the factor under study may have no influence on the variables.

Reproducibility for a single examiner and between a second examiner was analyzed in 10 non-pregnant women. Intraobserver variability of Doppler imaging measurements ranged from 2-4% and interobserver variability from 4-8%.

RESULTS

Table 1 shows the subject characteristics of the study population. It has shown that, there was no significant difference observed in weight, blood pressure and heart rate based on food habits. Table 2 shows the comparison of echocardiographic parameters in the

Table 1: Demographic characteristic of the study populations (Mean±SD or No. (n) and percentage)

Variables	*Pregnant women, (n = 46)
Age (year)	26±6
Height (m)	1.64±0.06
Pre-pregnancy weight (kg)	54.2±8.2**
Non-smoker	46 (100%)
Non-alcoholic	46(100%)
Non-vegetarian	9(19.6%)
Vegetarian	37(80.4%)
Gestation at delivery (weeks)	36±3.5
Birth weight (g)	2880±552
Post-partum weight (8-12 weeks)	58±6.7

*Parity of the study population were 0 (n = 26), 1 (n = 8), 2 (n = 4), 3 (n = 8). **Pre-pregnancy weight considered as per the information given by the subject during their routine first visit

Table 2: Cardiac performance at various stages of pregnancy and post partum in the study population (Mean±SD)

Parameter	15-30 weeks (n=19)	30-40 Weeks (n=31)	8-12 weeks post partum (n=22)	p-value
MAP (mmHg)	84.10±6.6	82.10±6.6	79.30±10.1	0.19
Heart rate (bpm)	82.60±11.7	88.60±12.1	72.70±9.4	<0.001
Stroke volume (mL)	92.80±11.7	89.80±16.7	72.76±13.7	0.003
Cardiac output (L min ⁻¹)	6.80±1.2	7.30±1.2	4.90±1.03	<0.001
LVIDd (cm)	3.82±0.3	4.17±0.31	3.93±0.34	>0.05
LVIDs (cm)	2.48±0.22	2.64±0.15	2.55±0.09	>0.05
PWT (cm)	1.05±0.07	1.12±0.08	1.07±0.07	>0.05
LVM (g)	107.70±13.3	145.50±20.6	119.45±15.8	<0.005
E velocity (cm sec ⁻¹)	80.80±7.4	71.80±11.4	76.24±14.9	0.38
A velocity (cm sec ⁻¹)	52.10±8.2	55.10±8.8	51.90±8.3	0.35
E/A ratio	1.50±0.4	1.30±0.3	1.40±0.31	0.29
EF (%)	71.06±5.7	68.60±6.9	70.95±7.7	>0.05
FS (%)	28.40±6.3	29.40±3.8	34.30±5.3	<0.05
TPR (dyn.sec.cm ⁻⁵)	1212.60±86.1	932.60±172.9	1320.30±287.5	<0.001

Out of the total 46 Pregnant women, enrolled at their routine first trimester scan, only 19 participated at 2nd trimester (15-30 weeks), 31 at 3rd trimester (30-40) of pregnancy and 22 post partum. MAP: Mean arterial pressure, LVIDd: Left ventricular internal dimension at diastole, LVIDs: Left ventricular internal dimension at systole, PWT: Posterior wall thickness, LVM: Left ventricular mass, E velocity: The rapid left ventricular filling phase peak velocity (early filling phase), A velocity: The atrial contraction phase peak velocity (late filling phase), E/A Ratio: The ratio of early to late filling velocity, EF (%): Percentage ejection fraction, FS(%): Percentage fractional shortening, TPR: Total peripheral resistance

women at 30-40 weeks of gestation and post-partum. Out of the 46 selected pregnant women only 35 attended at mid term pregnancy (15-30 weeks) for transabdominal ultrasound examination of fetal wellbeing. Echocardiogram was performed at 30-40 weeks of gestation (n = 31) and 8-12 weeks post-partum (n = 22). Heart rate, stroke volume, cardiac output and left ventricular mass increased significantly at last trimester of pregnancy compare to post partum (p<0.001, 0.003, p<0.001 and p<0.005), respectively, Table 2. Left ventricular contractility indices (percentage ejection fraction and fraction shortening) were within normal limit during pregnancy but fractional shortening was significantly higher post-partum than last trimester of pregnancy (p<0.05). A non significant increase in left ventricular internal dimensions (LVIDs, LVIDd and PWT; p>0.05) and significant decrease in total peripheral resistance (p<0.001) was observed at late third trimester of pregnancy (Table 2).

There was a statistically non-significant fall in transmitral E wave velocity and augmented A wave velocity at late third trimester of pregnancy leading to a decrease in the E/A ratio (Table 2).

DISCUSSION

Cardiac out put during pregnancy is estimated to increase by approximately 50% (Elkayam and Gleicher, 1998). Present study showed a 23.36% increase in stroke volume and

a 21.85% increase in heart rate leading to a 48.9% increase in cardiac output during last trimester of pregnancy compare to post partum. This confirms earlier studies and is consistent with the expected effects of increased circulating volume and decreased total peripheral resistance (Mabie *et al.*, 1994; Kametas *et al.*, 2001a, b; Barbara *et al.*, 2008). Fok *et al.* (2006) observed a significant changes in vascular load during pregnancy. A small increase in left ventricular internal dimension was observed by late gestation. Present study showed a non significant change in percentage ejection fraction and a significant decrease in percentage fractional shortening during last trimester of pregnancy in confirmation of a previous cross-sectional study (Kametas *et al.*, 2001a, b). Zentner *et al.* (2009) also observed that by last trimester of pregnancy, an increase in ventricular wall stress is accompanied by deterioration in cardiac function. The fractional shortening was significantly increases 8-12 weeks pos-partum. These observations are consistent with an intrinsic abnormality of contractility associated with pregnancy. In response to stretch of atrial wall, Atrial Natriuretic Peptides (ANP) and B type natriuretic peptides (BNP), are secreted and regulate blood volume by producing significant natriuresis and diuresis. They also, promote vascular smooth muscle relaxation (Clerico and Emdin, 2004).

Other studies also, showed a fall in contractility function not explainable by changes in decrease in vascular resistance. Mone *et al.* (1997) studied the relationship between mean velocity of circumferential fiber shortening and end systolic stress as an after load-adjusted, preload-independent index of contractility. Both this measure and fractional shortening fell by term, decreased further 2-4 weeks postpartum and then returned to baseline by 8-10 weeks. One study reported a similar decrease in the after load-adjusted velocity during pregnancy with post-partum normalizations (Geva *et al.*, 1997). Jacqueline *et al.* (2007) found that myocardial changes do not resolve fully in the early post-partum period, suggesting the possibility of intrinsic abnormalities of LV function. In contrast to these studies, Poppas *et al.* (1997) found no change in fractional shortening or velocity of circumferential shortening. Gilson *et al.* (1997) found no change in ejection fraction or fractional shortening, but showed a non-significant increase in the velocity of circumferential fiber shortening and a 12% decrease in left ventricular wall stress, implying an increase in contractility. Decrease in left ventricular wall stress may be accountable for unchanged EF% and increase in circumferential fiber shortening.

Little previous work on diastolic function is available. In the mid term pregnancy there was increased preload as a result of increased blood volume causes an increased E velocity and a relatively low A velocity that was changed to a statistically non-significant fall in E velocity and an increase in transmitral A velocity. This confirms longitudinal observations by Valensise *et al.* (2000) and cross-sectional data by Kametas *et al.* (2001a, b), however several studies show discrepancies with these results. Mabie *et al.* (1994) found no change in E velocity and Mesa *et al.* (1999) reported a progressive increase in E velocity throughout pregnancy. Technical limitations or population characteristics including blood pressure might account for these differences or, as with measures of systolic function there might be a normal variation in the response of diastolic function to pregnancy. This confirms the study of Mesa *et al.* (1999), which indirectly estimated mean left atrial pressure and found that it did not change during pregnancy, which further suggests that left atrial pressure is normal in healthy pregnancy.

We investigated the impact of maternal characteristics on maternal cardiac performance where there has been significant independent contribution in the literature. Height is an important determinant of stroke volume, cardiac output and left ventricular mass. So, these

findings were expected in present study. However, present results might be influenced by our small sample size, maternal characteristics, socioeconomic status of the study population and the methods applied. The future larger studies may provide a better result with more consistency.

Changes in measures of diastolic and systolic function can occur as a result of physiological changes or pathology. These may be surprisingly hard to differentiate since, the changes in normal pregnancy are sometimes inconsistent with the expected effects of increased preload or decrease total peripheral resistance (Simmons *et al.*, 2002). The BNP has been evaluated as a marker for depressed left ventricular function (Heidenreich *et al.*, 2004). In spite of increased left ventricular internal dimension and left ventricular mass the mean arterial pressure is unchanged because of a decline in total peripheral resistance, probably mediated by gestational hormonal activity, increased levels of circulating prostaglandins, atrial natriuretic peptide, endothelial nitric oxide and increased heat production by the developing fetus (Poppas *et al.*, 1997).

The population size was small. Follow-up was incomplete, but this is common in such type of studies because of early delivery and drop out and the study participants came from a small city and rural population. However, it is imaginable that the women attending post partum evaluations may have been more aware of their general health than those not returning.

CONCLUSION

Pregnancy is a natural volume overload state, has important effect on cardiac dimensions and performance. Increase in heart rate, small increase in left ventricular dimensions, significantly increased left ventricular mass and significantly decreased total peripheral resistance contributes to improved maternal myocardial performance at last trimester of pregnancy. This study, also expresses that along with gestation, maternal anthropometric profile may affect cardiac performance.

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