ISSN: 0975-3583,0976-2833 VOL13,ISSUE08,2022

Title

HEMODYNAMIC CHANGES IN NORMAL PRIMIGRAVIDA- AN ECHOCRDIOGRAPHIC ASSESMENT

AUTHORS

SWAPAN KUMAR HALDER[1], SAMIR KUMAR ROY[2],KUSUMITA MADAL[3] PREETHI MUTHUSAMY[4]

1 &2 Dept of Cardiology, NRS Medical College,3 IHBT,Medical College,Kolkata 4, Dept of G &O, NRS Medical College

Corresponding Author SAMIR KUMAR ROY, Dept of Cardiology, NRS Medical College,Kolkata SKRR80@YAHOO.IN, +919434439422

Abstract

Objective: The factors affecting cardiac output in normal pregnancy remain controversial. This study prospectively evaluates maternal hemodynamic and cardiac structure and function by echocardiography in healthy primi mother.

Methods: It's a prospective study, 143 primi gravida women were evaluated, Echocardiography had been done at 12 -14 wks gestation, 30-32 wk gestation and 6 -8 wks postpartum in which 6 – 8 weeks postpartum values were considered as baseline. The data was subjected to statistical analysis to observe the changes at different gestational periods by using ANOVA **Results:** Cardiac output increased significantly at the early to mid third trimester and was maintained until term. It increased predominantly in the latter half of pregnancy 53% occurred from baseline a 20.3% increase in heart rate and 29% increase in stroke volume. Left ventricular systolic function in terms of ejection fraction and fractional shortening was preserved until term. Total peripheral resistance also decreased in 29 % 1st trimester and 37% in later half of pregnancy.

Conclusion: The TFR and MAP decreases in the early pregnancy ,cardiac output increase in the early gestation and peak in mid third trimester and is maintained until term. Left ventricular systolic functions were maintained during pregnancy

Key Words : primi gravid, hemodynamic,echocardiographiy,cardiac output,ejection fraction

Abbreviations CO-cardiac output, EF-ejection fraction, TFR- total peripheral resistance, MAP- mean arterial pressure, SV- storke volume, L- left ventricle, OPD –out patient department, HR-heart rate, LVID-left ventricular internal diameter,FS –fractional shortening CI-cardiac index.

INTRODUCTION

Pregnancy is a physiologic state associated with a dramatic cardiac structural remodeling and an improved functional performance.[1] Cardiac adjustments to a pregnancy state may mimic abnormalities of the cardiovascular system.[2–4]

Maternal adaptation to pregnancy is apparent in all systems of the body. However,

ISSN: 0975-3583,0976-2833 VOL13,ISSUE08,2022

with the exception of the reproductive system itself, the circulatory system and its shared function with the respiratory system show the most striking changes, which have form the basis of this study. Knowledge of these physiological changes is essential for an understanding of both normal adaptation and maladaptation to pregnancy. Furthermore, such knowledge will be helpful in the clinical management of pregnant women with diseases of the circulatory and respiratory systems.

However, the basic mechanisms that cause physiological alterations of pregnancy are virtually unknown. For example, why are pregnant women more likely to suffer edema, hypertension, arterial dissection or thrombosis than their nonpregnant counterparts? At present, answers to such questions remain in the realm of teleology and hypothesis.

There is now good evidence, mentioned later, that normal adaptation to pregnancy occurs early in the embryonic period within the first trimester, long before the developing feto-placental unit is large enough to itself warrant the magnitude of the changes observed. Theoretically, this would suggest that the changes are preparatory to normal pregnancy and that if they do not occur or only occur partially, the future of the pregnancy may be in jeopardy. Indeed some studies have provided confirmation of this suggestion. Echocardiography can assess the hemodynamic changes noninvasively; thus, it is widely used to measure cardiocirculatory indices during pregnancy and after delivery.[5] Regarding the changes in echocardiographic parameters, left ventricular (LV) end-diastolic and end-systolic dimensions and wall thickness increase, and contractile function enhances. Functional pulmonary, tricuspid, and mitral regurgitation and mild pericardial effusion are occasionally seen in normal pregnancy. Cardiac output increases 30-40% from the pre-pregnancy levels [6,7]. However studies providevarying results as to the extent and timing of this increase. Particularly during the third trimester, reports on cardiac output diverge greatly [8]. Several hypotheses are postulated for the adaptive changes during pregnancy such as peripheral arterial vasodilatation hypothesis [9]. Renin-angiotensin system activation [10], sex hormone related mechanisms for sodium and fluid retention [11]. Concomitant increase in myocardial contractility may also contribute to increase in the cardiac output.[12] However, there was non uniformity in the published literature with various echocardiographic parameters and their alterations with each trimester. As there were differences in echocardiographic findings in pregnant women of differing ethnicity, the authors wanted to study two-dimensional (2D) echocardiographic features in Indian pregnant women.

MATERIALS &METHODS

STUDY POPULATION

Pregnant women with following conditions were excluded from the study Multigravida,Hypertensive disorders in pregnancy,Women with Heart disease,Diabetes,Anemia,Renal disease,Thyroid disorder,Tuberculosis,history of intake of oral contraceptive pills.history of treatment for primary,infertility,Tobacco users,Multifetal gestation.Intra uterine growth restriction of fetus.Oligohydraminos AFI < 5 cm,Polyhydraminos AFI>20 cm,APH

ISSN: 0975-3583,0976-2833 VOL13,ISSUE08,2022

After excluding as per criteria, the pregnant mother who were uncomplicated pregnancy and who become pregnant within one year of regular intercourse without medical illness attending Antenatal OPD in NRSMCH, Kolkata, West Bengal had been recruited for study over a period of 1 year.

Proper consent from Institutional Ethical Committee is taken. After enrollment, women attending antenatal clinic had been subjected to history, clinical examination.Sonological examination was done to confirm single ton pregnancy, fetal and placental abnormalities were excluded.

Echocardiography had been done at 12 -14 wks gestation, 30-32 wk gestation and 6 -8 wks postpartum in which 6 – 8 weeks postpartum values were considered as baseline. Considering baseline values changes in hemodynamic parameters in pregnancy were analyzed. Left ventricular systolic function was assessed by measuring ejection fraction and fractional shortening.

Ejection fraction = LV diastolic volume – LV systolic volume/LV diastolic volume.
Fractional shortening (FS) = LV end diastolic diameter – LV end systolic diameter/ LV end diastolic diameter. Doppler echocardiography was used to calculate the stroke volume

Stroke volume (ml) = left ventricular outflow tract area (m2) ×velocity time integral (mm2)

Simultaneously electrocardiograph determined heart rate in beats per minute was recorded. Cardiac output (l/min) = stroke volume (lit) ×heart rate (beats/min) Cardiac index (l/min/m2) =cardiac output (l/min)/body surface area (m2*)*.

Sample size of 143 followed up three times, continuous variables are presented as mean ± SD. Changes in parameters of each gestational period were compared by one way analysis of variance ANOVA followed by Bonferroni's as post-hoc test. in which $p<0.05$ is considered as significant.

Result

The present study is prospective observational longitudinal study. Hemodynamic changes in visit 3 at 6 -8 weeks postpartum is considered as baseline and values in visit 1 at 12 -14 weeks and visit 2 at 30 -32 weeks are compared with baseline visit 3 to delineate the gradual adaptation of hemodynamic changes in pregnancy and hypotheses behind these changes.
Mean age in the present study is 22.9 ± 3.3.It range is between 18 -30 years.

Most frequently distributed ranges are 18 -19 yrs, 22-23 yrs and 26 -27 years.

HR 3 at 6 – 8 weeks postpartum is considered as baseline. Thus Mean± SD of HR 3 is

68.5 ± 3.4.It range is between 61 -76 and its 95 % CI of mean is 67.9 – 69.1. Hence HR 1 mean is 72.7 ± 3.5 shows highly significant change($p<0.0001$) when compared to baseline HR 3 and also HR 2 mean is 82.4 ± 4 shows highly significant change ($p<0.0001$) when compared to HR 3.

ISSN: 0975-3583,0976-2833 VOL13,ISSUE08,2022

EF 3 at 6 – 8 weeks postpartum is considered as baseline. Thus Mean± SD of EF 3 is

67.1 ± 2.9.It ranges is between 61 – 79 and its 95 % CI of mean is 66.6 – 67.6.

Hence EF1 mean is 67.1 ± 3.1 shows a no significant change (p=0.9043) when compared to baseline EF3 and also EF2 mean is 67.2 ± 3.3shows no significant change (p=0.1507) when compared to EF3.

FS 3 at 6 – 8 weeks postpartum is considered as baseline. Thus Mean± SD of FS 3 is 35.1 ± 2.0. It range is between 31 -38 and its 95 % CI of mean is 34.5 -35.2. Hence FS1 mean is 35.2 ± 2.4 shows a no significant change (p=0.2305) when compared to baseline FS3 and also FS2 mean is 35.3 ± 2.3 shows no significant change (p=0.1408) when compared to FS3.

LVID 3 at 6 – 8 weeks postpartum is considered as baseline. Thus Mean± SD of LVID 3 is

37.8 ± 2.8 .It range is between 31- 43 and its 95 % CI of mean is 37.3 -38.3.

Hence LVID 1 mean is 39.2 ± 2.9 shows significant change(p=0.0079) when compared to baseline LVID 3 and also LVID 2 mean is 41.5 ± 3.2 shows highly significant change (p<0.0001) when compared to LVID 3.

MAP 3 at 6 – 8 weeks postpartum is considered as baseline. Thus Mean± SD of MAP 3 is

89.8 ± 5.4.It range is between 80.7 -99 and its 95 % CI of mean is 88.9-90.7.

Hence MAP 1 mean is 80.5 ± 5.4 shows highly significant change(p<0.0001) when compared to baseline MAP 3 and also MAP 2 mean is 88.2 ± 5.6 shows highly significant change (p<0.0001) when compared to MAP 3.

SV 3 at 6 – 8 weeks postpartum is considered as baseline. Thus Mean± SD of SV 3 is

64.4 ± 4.47 .It range is between 57-73 and its 95 % CI of mean is 63.7 -65.2.

Hence SV 1 mean is 76.7 ± 5.07 shows highly significant change(p<0.0001) when compared to baseline SV 3 and also SV 2 mean is 83.2 ± 4.7 shows highly significant change (p<0.0001) when compared to SV 3.

CO 3 at 6 – 8 weeks postpartum is considered as baseline. Thus Mean± SD of CO 3 is 4.3 ± 0.3. It range is between 3.7-5.1 and its 95 % CI of mean is 4.3 – 4.41.

Hence CO 1 mean is 5.5 ± 0.3 shows highly significant change(p<0.0001) when compared to baseline CO 3 and also CO 2 mean is 6.7 ± 0.4 shows highly significant change (p<0.0001) when compared to CO 3.

CI 3 at 6 – 8 weeks postpartum is considered as baseline. Thus Mean± SD of CI 3 is 3.1 ± 0.3.It range is between 2.2-3.9 and its 95 % CI of mean is 3.04 -3.16.

Hence CI 1 mean is 3.9 ± 0.4 shows highly significant change(p<0.0001) when compared to baseline CI 3 and also CI 2 mean is 4.6 ± 0.6 shows highly significant change (p<0.0001) when compared to CI 3.

ISSN: 0975-3583,0976-2833 VOL13,ISSUE08,2022

SUMMARY STATISTICS

Considering visit 3 that is 6 – 8 weeks postpartum as baseline, various changes in hemodynamic parameters are depicted in regard to statistical significance, quantity and as well as quality of change.

Parameters analyzed	Visit 1 (12-14wks) Changes in parameters (increase /decrease /no change)	Visit 1 (12 -14 wks) Statistical significance	Visit 2 (30-32 wks) Changes in parameters (increase/decrease /no change)	Visit 2 (12 -14 wks) Statistical Significance
Heart rate (beats/minute)	6.2% ↑	P<0.0001	20.3% ↑	P<0.0001
Ejection fraction(%)	≈	p = 0.9043	≈	p=0.1507
Fractional shortening(%)	≈	p= 0.2305	0.2% ≈	p=0.1408
Left ventricular internal diameter (mm)	3.7% ↑	P<0.0079	9.6% ↑	P<0.0001
Mean arterial pressure(mm of Hg)	10.5% ↓	P<0.0001	1% ≈ / ↓	P<0.0001
Stroke volume(ml/minute)	19% ↑	P<0.0001	29% ↑	P<0.0001
Cardiac output (litres/minute)	27% ↑	P<0.0001	53% ↑↑	P<0.0001
Cardiac index(litres/minute/m²)	25% ↑	P<0.0001	48% ↑↑	P<0.0001
Total peripheral resistance(dyne.sec.cm-5)	29% ↓	P<0.0001	37% ↓	P<0.0001

Discussion

It was well known that the maternal cardiovascular system undergoes significant changes throughout pregnancy, which imposes considerable stress on the pregnant woman's heart. In this study, the authors demonstrated structural and functional changes in the maternal cardiovascular system during pregnancy. During early pregnancy, LV filling rates increase because of increased venous return to the left

ISSN: 0975-3583,0976-2833 VOL13,ISSUE08,2022

atrium (preload), represented by an increase in LA size and LV internal diameters from the first to second trimester. As gestation advances, the myocardium develops physiologic hypertrophy to withstand the chronic volume overload on cardiovascular system. In this study, there was a gradual increase in IVS and LV PWT as increasing gestational age. There was a progressive increase in these parameters across trimesters in other studies.[2,13,14] Mesa et al1 showed an increase in the LV end-diastolic volume as early as 10 weeks' gestation and a peak during the third trimester by echocardiography, whereas Geva et al[15] did not observe any change in LV end diastolic or end-systolic dimension throughout pregnancy. Other reports showed that left and right atrial and right ventricular diastolic dimensions increased throughout the gestational period.[14,16] LV end-diastolic septal and posterior wall thicknesses are increased similar to other studies.[13,14,18] This may be due to preload alterations.[18] Previous studies observed variable results with LVEF. In this study, there was no significant change in EF as gestation increases. Adeyeye et al[13] reported a gradual increase in LVEF with increasing gestational age. More recent studies showed no significant change in LVEF during pregnancy,[1,15] contrary to earlier report by Hunter and Robson[3] who found an increase in LVEF during the first two trimesters. A progressive decrease in LV fractional shortening (LVFS) was found by Schannwell et al,[14] whereas Mesa et al [1] and Clapp and Capeless[17] did not observe any change in LVFS during normal pregnancy

CONCLUSION

1. Ejection fraction and fractional shortening remains unaffected throughout the pregnancy thus Left ventricular systolic functions are well preserved in pregnancy in our study.
2. Left ventricular diameter increases in the early gestation and continues to increase at later weeks of pregnancy.
3. Heart rate,Stroke volume, Cardiac output,Cardiac index increases in the early gestation and continues to increase at later weeks of pregnancy.
4. Mean arterial pressure decreases in early gestation then no significant change in late gestation.
5. Total peripheral resistance continues to decrease from early gestation to late gestation.

References

1 Mesa A, Jessurun C, Hernandez A, et al. Left ventricular diastolic function in normal human pregnancy. Circulation 1999;99(4):511–517

2 Duvekot JJ, Peeters LL. Maternal cardiovascular hemodynamic adaptation to pregnancy. Obstet Gynecol Surv 1994;49(12, Suppl):S1–S14

3 Hunter S, Robson SC. Adaptation of the maternal heart in pregnancy. Br Heart J 1992;68(6):540–543

ISSN: 0975-3583,0976-2833 VOL13,ISSUE08,2022

4 Maroo A, Raymond R. Pregnancy and heart disease. Related live CME: 9th Annual Intensive Review of Cardiology, August 17– 21, 2007

5 Kamiya C, Nakatani S, Hashimoto S, et al. Role of echocardiography in assessing pregnant women with and without heart disease. J Echocardiogr 2008;6:29–38

6. Mabie VC, DiSessa TG, Crocker LG, Sibai BM, Arheart KLA. Longitudinal study of cardiac output in normal pregnancy. Am J Obstet Gynecol 1994 March; 170 (3): 849-56

7.Robson SC, Hunter S, Moore M and Dunlop W. Hemodynamic changes during purperium: A Doppler and M mode Echocardiographic study. Br J Obstet gynecol 1987; 94: 1028-1039.

8. Van Oppen ACC, Stigter RH, Bruinse HW. Cardiac output in normal pregnancy: A critical review. Obstet Gynecol 1996; 87: 310-18.

9.Schrier RW, Brier VA. Peripheral arterial vasodilation hypothesis of sodium and water retension; Implication for pathogenesis of preeclampsisa-eclampsisa. Obstet Gynecol. 1991; 77: 632-39.

10. Maynard SE, Karumanchi SA, Thadani R. In Brenner and Rector Eds Hypertension and Kidney disease in pregnancy. The kidney Vol. 2, 8th ed. Saunders Eldevier.2004;1567-70

11. Weiner CP, Lizasoain I, Baylis SA, Knowles RG, Charles IG. Induction of calcium dependent nitric oxide synthases by sex hormone. Proc Natl Acad Sci May 1994; 91: 5212-16.

12. Katz R, Karliner JS, and Resnik R. Effects of natural volume overload state (Pregnancy) on left ventricular performance in normal human subjects. Circulation 1978; 58: 434-441.

13 Adeyeye VO, Balogun MO, Adebayo RA, et al. Echocardiographic assessment of cardiac changes during normal pregnancy among Nigerians. Clin Med Insights Cardiol 2016;10:157–162

14 Schannwell CM, Schoebel FC, Zimmermann T, et al. [Left ventricular diastolic function in normal pregnancy. A prospective study using M-mode echocardiography and Doppler echocardiography] [in German] Dtsch Med Wochenschr 2000;125(37):1069–1073

15 Geva T, Mauer MB, Striker L, Kirshon B, Pivarnik JM. Effects of physiologic load of pregnancy on left ventricular contractility and remodeling. Am Heart J 1997;133(1):53–59

16 Valensise H, Novelli GP, Vasapollo B, et al. Maternal cardiac systolic and diastolic function: relationship with uteroplacental resistances. A Doppler and echocardiographic longitudinal study. Ultrasound Obstet Gynecol 2000;15(6):487–497

17 Clapp JF III, Capeless E. Cardiovascular function before, during, and after the first and subsequent pregnancies. Am J Cardiol 1997;80(11):1469–1473

18 Melchiorre K, Sharma R, Thilaganathan B. Cardiac structure and function in normal pregnancy. Curr Opin Obstet Gynecol 2012;24(6):413–421