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Maternal Cardiac Hemodynamic in Pregnancy: An Echocardiographic Study

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Abstract

Background: The determinants of cardiac output in normal pregnancy remain incompletely defined. We prospectively characterized maternal hemodynamics and cardiac structure and function by echocardiography in healthy primigravid women.

Methods: In this prospective longitudinal study, 143 primigravid women underwent transthoracic echocardiography at 12–14 weeks' gestation, 30–32 weeks' gestation, and 6–8 weeks postpartum; the postpartum visit served as the within-subject baseline. Between-visit differences were analyzed by one-way analysis of variance (ANOVA) with Bonferroni post hoc testing.

Results: Cardiac output rose significantly from early gestation, peaked in the early-to-mid third trimester, and was sustained until term, with a 53% increase from baseline driven by a 20.3% rise in heart rate and a 29% rise in stroke volume. Left ventricular systolic function, assessed by ejection fraction and fractional shortening, was preserved throughout pregnancy. Total peripheral resistance fell by 29% in the first trimester and by 37% in the second half of pregnancy.

Conclusions: Total peripheral resistance and mean arterial pressure decline early in pregnancy, while cardiac output rises from early gestation and peaks in the mid-third trimester, remaining elevated until term. Left ventricular systolic function is preserved throughout normal pregnancy.

INTRODUCTION

Pregnancy is a physiologic state characterized by dramatic cardiac structural remodelling and enhanced functional performance.1 The cardiac adaptations of pregnancy can mimic, and must be distinguished from, pathological cardiovascular states.2–4

Maternal adaptation to pregnancy involves every organ system, but the circulatory system—together with its coupled respiratory function—undergoes the most pronounced changes. Understanding these physiological adjustments is essential both for recognizing normal adaptation and for managing pregnant women with pre-existing cardiorespiratory disease. Nonetheless, the mechanisms that underlie these adaptations remain incompletely understood, and it is unclear why pregnancy predisposes to oedema, hypertension, arterial dissection, and thromboembolism relative to the no pregnant state.

Evidence suggests that hemodynamic adaptation begins in the first trimester, well before the feto-placental unit could itself account for the magnitude of change observed—implying that these early changes are preparatory, and that their absence or attenuation may portend adverse pregnancy outcomes. Echocardiography allows non-invasive, serial assessment of cardio circulatory indices across gestation and the puerperium.5 reported echocardiographic adaptations include increased left ventricular (LV) end-diastolic and end-systolic dimensions and wall thickness, enhanced contractile function, and occasional functional

pulmonic, tricuspid, and mitral regurgitation with mild pericardial effusion. However, published data are heterogeneous across trimesters and ethnic groups. We therefore undertook a prospective study of two-dimensional echocardiographic hemodynamic adaptation in Indian primigravid women.

METHODS

Study Population

Women were excluded for multigravidas; hypertensive disorders of pregnancy; pre-existing heart disease, diabetes, anaemia, renal or thyroid disease, or tuberculosis; prior oral contraceptive use; history of infertility treatment; tobacco use; multifetal gestation; foetal growth restriction; oligohydramnios (amniotic fluid index [AFI] < 5 cm) or polyhydramnios (AFI > 20 cm); or antepartum haemorrhage.

Eligible participants were uncomplicated primigravidae who conceived within one year of regular unprotected intercourse and who attended the antenatal outpatient clinic at Nil Ratan Sircar Medical College and Hospital, Kolkata, West Bengal, over a one-year period. The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants. Baseline history, clinical examination, and sonographic confirmation of a singleton pregnancy without foetal or placental abnormality were completed at enrollment.

Echocardiographic and Hemodynamic Assessment

Transthoracic echocardiography was performed at 12–14 weeks' and 30–32 weeks' gestation and at 6–8 weeks postpartum, with the postpartum measurement serving as the baseline for comparison. Left ventricular systolic function was quantified by ejection fraction (EF) and fractional shortening (FS):

EF = (LV end-diastolic volume − LV end-systolic volume) / LV end-diastolic volume

FS = (LV end-diastolic diameter − LV end-systolic diameter) / LV end-diastolic diameter

Stroke volume (SV) was derived by Doppler echocardiography as the product of left ventricular outflow tract cross-sectional area and the Doppler velocity–time integral. Heart rate (HR) was recorded simultaneously by electrocardiography. Cardiac output (CO) was calculated as SV × HR, and cardiac index (CI) as CO normalized to body surface area.

Statistical Analysis

With a sample size of 143 women followed at three time points, continuous variables are expressed as mean ± SD. Between-visit differences were assessed by one-way ANOVA with Bonferroni post hoc correction; a two-sided $P < 0.05$ was considered statistically significant.

RESULTS

This prospective observational longitudinal cohort comprised 143 primigravidae with a mean age of 22.9 ± 3.3 years (range, 18–30 years); age was most frequently distributed within the 18–19, 22–23, and 26–27-year bands. Hemodynamic values at 6–8 weeks postpartum (visit 3) served as the within-subject baseline against which visit 1 (12–14 weeks) and visit 2 (30–32 weeks) were compared.

Heart rate at baseline (visit 3) was 68.5 ± 3.4 beats/min (range, 61–76; 95% CI, 67.9–69.1). Heart rate rose to 72.7 ± 3.5 beats/min at visit 1 and 82.4 ± 4.0 beats/min at visit 2, both highly significant increases relative to baseline ($P < 0.0001$ for each).

Ejection fraction at baseline was 67.1 ± 2.9% (range, 61–79; 95% CI, 66.6–67.6%) and did not change significantly at visit 1 (67.1 ± 3.1%; P = 0.9043) or visit 2 (67.2 ± 3.3%; P = 0.1507).

Fractional shortening at baseline was 35.1 ± 2.0% (range, 31–38; 95% CI, 34.5–35.2%) and likewise showed no significant change at visit 1 (35.2 ± 2.4%; P = 0.2305) or visit 2 (35.3 ± 2.3%; P = 0.1408).

Left ventricular internal diameter at baseline was 37.8 ± 2.8 mm (range, 31–43; 95% CI, 37.3–38.3 mm), increasing significantly to 39.2 ± 2.9 mm at visit 1 (P = 0.0079) and further to 41.5 ± 3.2 mm at visit 2 (P < 0.0001).

Mean arterial pressure at baseline was 89.8 ± 5.4 mmHg (range, 80.7–99; 95% CI, 88.9–90.7 mmHg), falling to 80.5 ± 5.4 mmHg at visit 1 and 88.2 ± 5.6 mmHg at visit 2 (P < 0.0001 for each versus baseline).

Stroke volume at baseline was 64.4 ± 4.47 mL (range, 57–73; 95% CI, 63.7–65.2 mL), rising to 76.7 ± 5.07 mL at visit 1 and 83.2 ± 4.7 mL at visit 2 (P < 0.0001 for each).

Cardiac output at baseline was 4.3 ± 0.3 L/min (range, 3.7–5.1; 95% CI, 4.3–4.41 L/min), rising to 5.5 ± 0.3 L/min at visit 1 and 6.7 ± 0.4 L/min at visit 2 (P < 0.0001 for each).

Cardiac index at baseline was 3.1 ± 0.3 L/min/m² (range, 2.2–3.9; 95% CI, 3.04–3.16 L/min/m²), rising to 3.9 ± 0.4 L/min/m² at visit 1 and 4.6 ± 0.6 L/min/m² at visit 2 (P < 0.0001 for each).

Table1. Summary of Hemodynamic Changes Relative to Postpartum Baseline

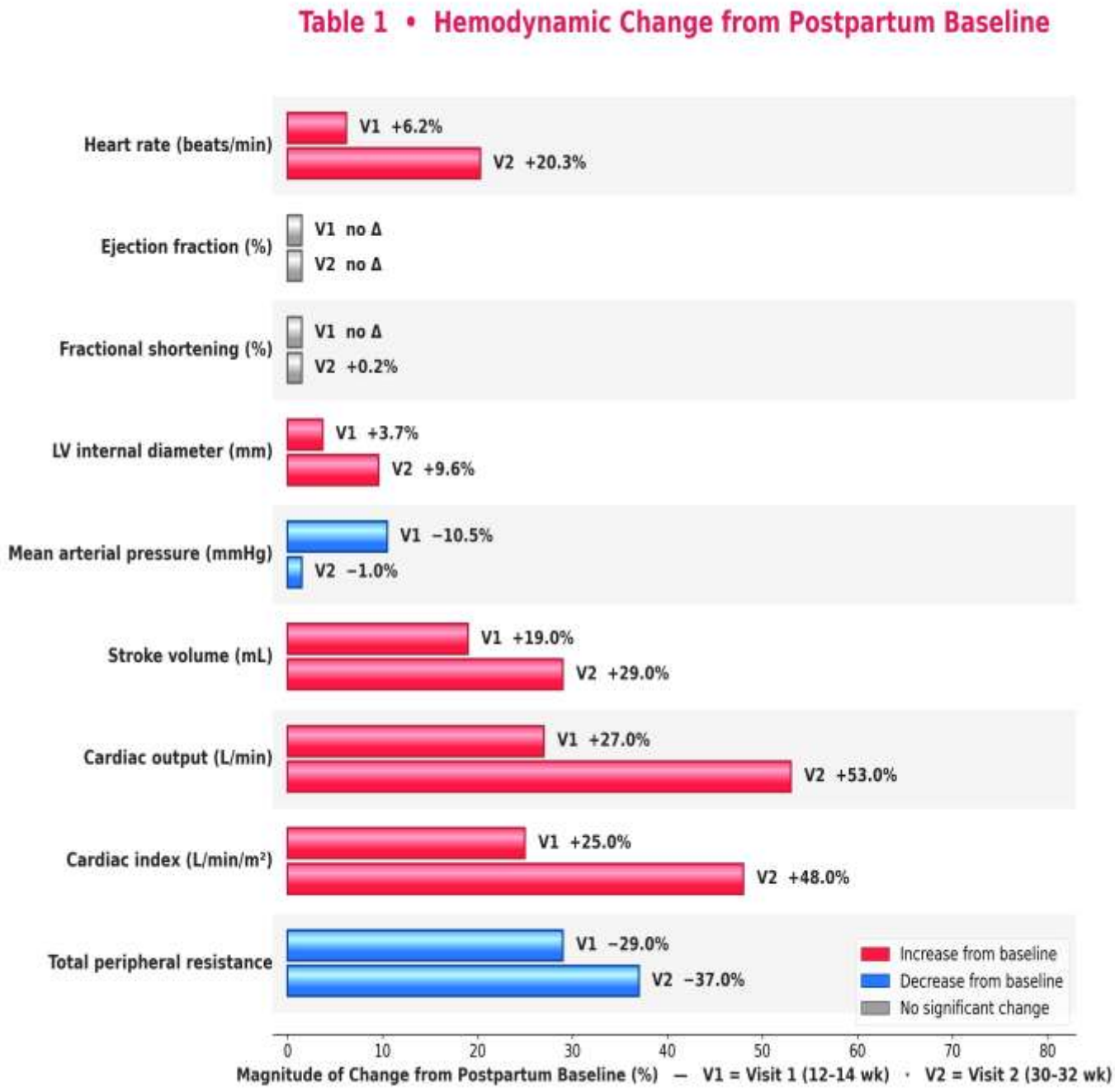

Table 1 (rendered graphically). Horizontal bars show the magnitude and direction of change from postpartum baseline at Visit 1 (V1, 12–14 wk) and Visit 2 (V2, 30–32 wk) for each hemodynamic parameter; magenta = increase, cyan = decrease, grey = no significant change. All P values are reported in the main text.

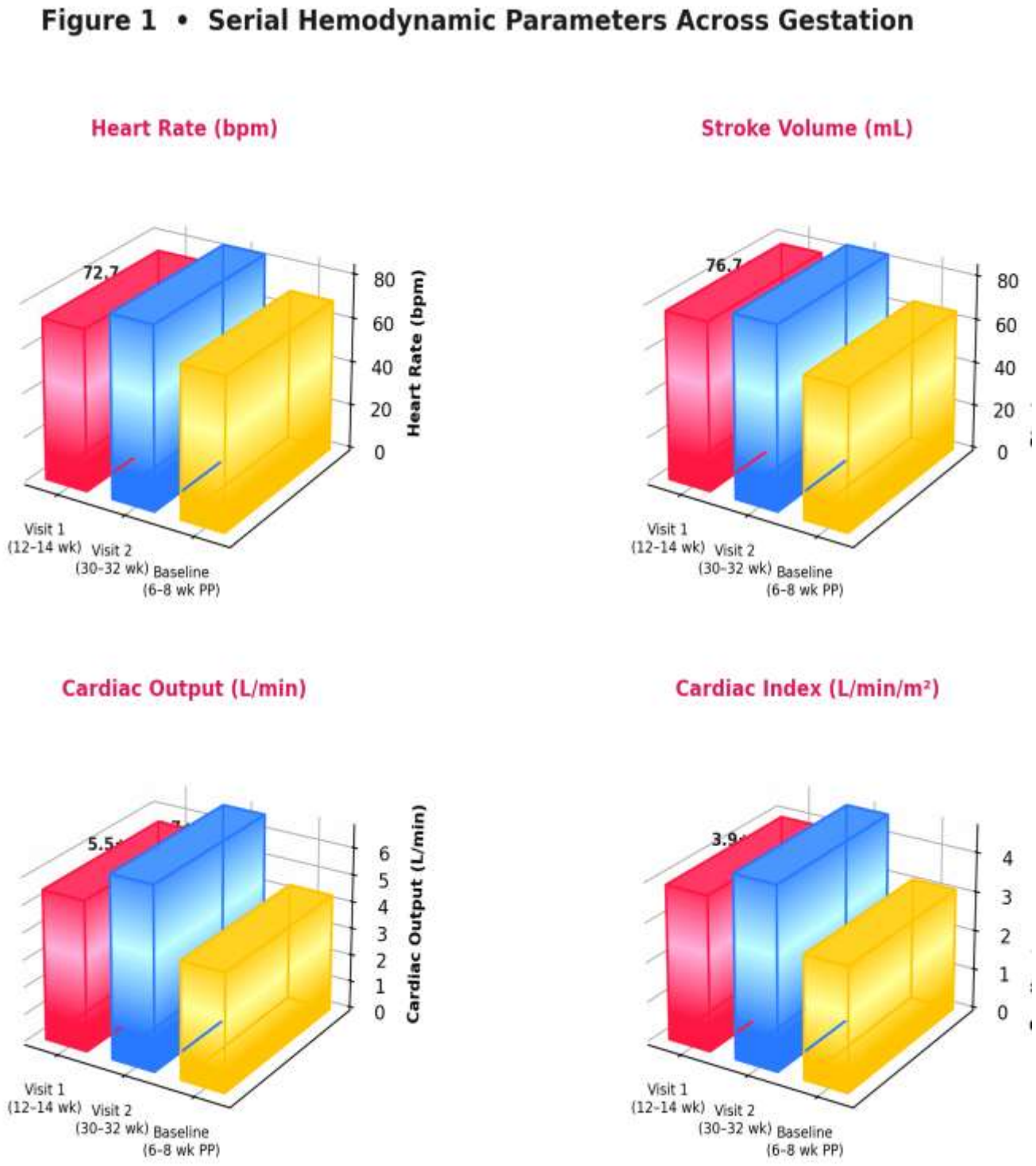

Figure 1. Serial changes in heart rate, stroke volume, cardiac output, and cardiac index across the two gestational visits relative to postpartum baseline (glossy gradient 3D bar chart).

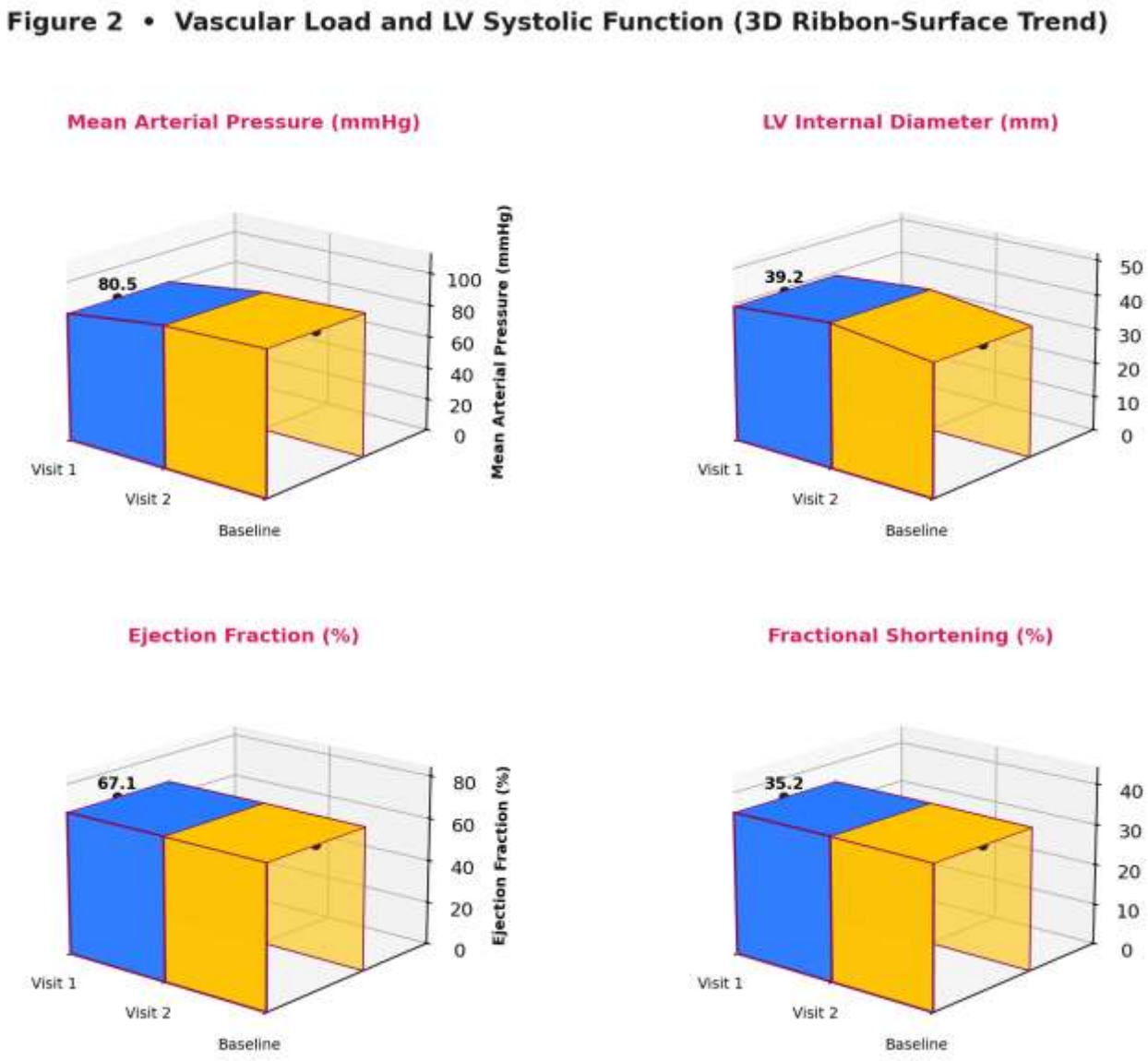

Figure 2. Serial changes in mean arterial pressure, LV internal diameter, ejection fraction, and fractional shortening across the two gestational visits relative to postpartum baseline (3D ribbon-surface trend chart).

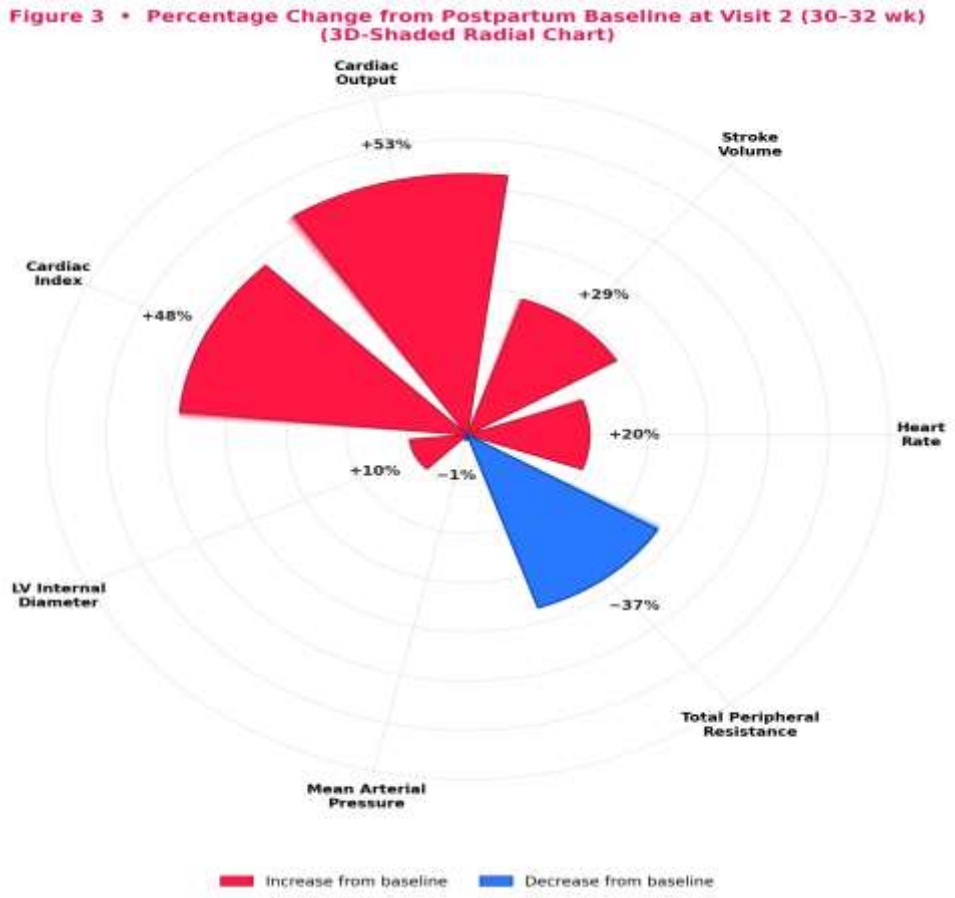

Figure 3. Percentage change from postpartum baseline for each hemodynamic parameter at visit 2 (30–32 weeks) (3D-shaded radial bar chart).

DISCUSSION

The maternal cardiovascular system undergoes substantial change throughout gestation, imposing a chronic volume load on the pregnant heart. In this cohort, early pregnancy was characterized by increased LV filling consequent to augmented venous return (preload), reflected in increasing left atrial size and LV internal diameter from the first to second trimester. As gestation advanced, the myocardium developed physiologic hypertrophy to accommodate this chronic volume overload; we observed a progressive increase in inter-ventricular septal and posterior wall thickness with advancing gestational age, consistent with prior reports.2,13,14

Mesa et al[1] observed an increase in LV end-diastolic volume as early as 10 weeks' gestation, peaking in the third trimester, whereas Geva et al[15] did not detect a change in LV end-diastolic or end-systolic dimension across pregnancy. Other investigators have reported progressive increases in left and right atrial and right ventricular diastolic dimensions throughout gestation.14,16 The increase in LV end-diastolic septal and posterior wall thickness observed here parallels earlier reports13,14,18 and likely reflects preload-mediated remodeling.18

Reported changes in LV ejection fraction across pregnancy have been inconsistent. We found no significant change in EF with advancing gestation. Adeyeye et al[13] reported a gradual rise in LVEF with gestational age, and more recent studies similarly found no significant change in LVEF during pregnancy,[1,15] in contrast to the earlier report by Hunter and Robson[3], who observed an increase in LVEF during the first two trimesters. A progressive decline in LV fractional shortening was reported by Schannwell et al[14], whereas Mesa et al[1] and Clapp and Capeless[17] found no change in fractional shortening during normal pregnancy.

CONCLUSIONS

1. Ejection fraction and fractional shortening remain unaffected throughout pregnancy; left ventricular systolic function is well preserved.
2. Left ventricular internal diameter increases from early gestation and continues to increase through late pregnancy.
3. Heart rate, stroke volume, cardiac output, and cardiac index increase from early gestation and continue to rise through late pregnancy.

4. Mean arterial pressure decreases in early gestation, with no further significant change in late gestation.
5. Total peripheral resistance continues to decline from early to late gestation.

DISCLOSURES

Conflict of Interest

The authors declare that they have no conflict of interest.

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REFERENCES

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

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

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

4. Maroo A, Raymond R. Pregnancy and heart disease. Related live CME: 9th Annual Intensive Review of Cardiology, August 17–21, 2007. (CME activity; not indexed in PubMed.)

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(2):29–38. DOI: 10.2303/jecho.6.29

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. PubMed

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. PubMed

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. PubMed

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. PubMed

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. DOI: 10.1016/S0002-9149(97)00738-8

18. Melchiorre K, Sharma R, Thilaganathan B. Cardiac structure and function in normal pregnancy. Curr Opin Obstet Gynecol 2012;24(6):413–421. DOI: 10.1097/GCO.0b013e328359826f