Normal Echocardiographic Measurements in Uncomplicated Pregnancy, a Single Center Experience

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ABSTRACT

Background: Cardiovascular changes of pregnancy are well-known; however, parameters for accurately assessing these changes have not been refined as measurement tools have advanced. We sought to examine the range of echocardiographic parameters during normal pregnancy using current echocardiographic imaging modalities. **Methods:** We performed a retrospective analysis of normal echocardiograms in 121 women (97 pregnant, 24 non-pregnant) without evidence of cardiovascular disease. Linear, area, and Doppler flow measurements were made of commonly reviewed cardiac structures. Height-indexed measurements were compared between pregnant women and controls and between trimesters of pregnancy. **Results:** Compared to non-pregnant patients, all four cardiac chambers showed significant enlargement in the pregnant patients. The left atrium was the first chamber to enlarge. LV mass also increased in the third trimester (134.5 ± 31 vs. 112.3 ± 28.2 g, $P < 0.01$), with preservation of LV mass to volume ratio. LV ejection fraction was significantly larger (68% vs. 63%, $P < 0.036$) in the second trimester patients, but decreased into the third trimester (64.1% ± 6.8%, $P < 0.006$). When pulmonary artery systolic pressure (PASP) was calculated from the pulmonary artery acceleration time, observed pressures were significantly greater in the third trimester than second trimester (40.1 ± 10.3 vs. 45.5 ± 10.1 mmHg, $P = 0.029$). PASP calculated conventionally from tricuspid regurgitation gradient did not show similar significance. There was no significant change in diastolic parameters throughout pregnancy. **Conclusions:** This study provides data on echocardiographic parameters during normal pregnancy and is one of the largest sample sizes in the literature. The results will contribute to the current literature by helping to distinguish between normal and abnormal echocardiograms during pregnancy.

Keywords: Chamber quantifications, echocardiography, pregnancy

INTRODUCTION

Cardiovascular disease continues to be one of the most common causes of maternal death during pregnancy in developed countries. Pregnancy-related deaths from cardiovascular causes have risen in the last decade.[1,2] For individuals manifesting cardiovascular disease, hemodynamic stressors are accentuated by the normal physiologic changes that occur in the maternal heart.[1,3]

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The changes to cardiovascular hemodynamics during pregnancy are significant and well-studied. Cardiac output increases, rising gradually at 8-10 weeks of gestation and peaking at weeks 25-30.[4] This increase is due to a combination of increased preload, decreased afterload, increased heart rate (HR), increased compliance of conduit vessels, ventricular remodeling, and alterations in the renin-angiotensin-aldosterone system. Mean arterial pressure decreases due to the creation of a low-resistance placental vasculature, and maternal blood volume increases up to forty percent above non-pregnant values.

To accommodate these changes, the heart undergoes significant remodeling. Previous studies have shown that all four chambers enlarge.[4] Cardiac volume and mass increase concomitantly so that left ventricular (LV) function and ejection fraction (EF) remain unchanged. These changes

return to pre-pregnancy states approximately 6 months postpartum.[4]

These hemodynamic changes are well-understood; however, parameters for accurately assessing these changes have not been refined as our measurement tools have advanced. Echocardiography continues to be a safe and non-invasive technique to serially evaluate cardiac structures and function during pregnancy. Despite the wide availability of echocardiography, there are no recent, large studies using newer echocardiographic imaging technologies in the literature. Furthermore, the normal range of echocardiographic parameters that reflect these changes has not been thoroughly defined. We revisited the previously described values associated with normal maternal cardiovascular changes by applying newer modalities used in clinical practice today such as biplane Simpson's method of disks to derive EF, and pulmonary artery acceleration time (PAAT) to derive pulmonary artery systolic pressure (PASP). Overall, we attempted to further characterize the ranges of echocardiographic parameters in healthy pregnant women without cardiovascular disease by reporting a more comprehensive spectrum of cardiac measurements.

METHODS

Patient selection

A retrospective search of the Montefiore Heart and Vascular Center's urban and multiracial database of Bronx, New York patients was performed for consecutive normal echocardiograms of women with a singleton gestation from January 2008 to August 2011. For controls, the database was searched for all normal echocardiograms of non-pregnant women age 40 and under from June 2011 to August 2011. Normal echocardiograms were considered to be those with (1) normal biventricular systolic function, (2) normal appearing cardiac valves, (3) absence of valvular stenosis, (4) absence of moderate to severe valvular insufficiency, (5) absence of aortic root dilatation, (6) absence of congenital anomalies, and (7) absence of pericardial disease. Indications for these echocardiograms varied from evaluation of symptoms of shortness of breath, palpitations, peripheral edema, and auscultation of murmur on exam. Patients were excluded from the study if they had a history of congenital heart disease, coronary artery disease, diastolic dysfunction, significant ventricular hypertrophy (LV posterior wall [PW] or septal thickness >1.0 cm) or dilatation (LV end diastolic dimension >5.3 cm or >3.2 cm when indexed to body surface area [BSA] or height), previous cardiac surgery/procedures, or established maternal medical diseases known to have cardiovascular effects (diabetes mellitus, chronic hypertension, HIV, systemic lupus erythematous, sickle cell disease, rheumatoid arthritis, etc.). This study has been approved by The Institutional Review Board.

Echocardiography

All subjects underwent two-dimensional echocardiography with a commercially available system (Sonos or iE33, Philips, Andover, MA, USA). All images were acquired according to the American Society of Echocardiography guidelines.[5] These included parasternal, apical, and subcostal views along with spectral and color Doppler assessment of valvular regurgitation. All measurements were performed on the parasternal and apical images. These windows were acquired with the patient in the left lateral decubitus position. Chamber volumes and subsequent EF were calculated using Simpsons' biplane method of disks. Cardiac output was calculated using the stroke volume (SV) derived from the LV outflow tract (LVOT). Right ventricular (RV) systolic pressures were calculated from measured tricuspid regurgitation velocities. When the IVC was normal in size, 7 mmHg was used as the estimated right atrial (RA) pressure. Estimated peak PASP was calculated from the PAAT.[6] Volume and length measurements were normalized by patient height. All the echo studies were reviewed by independent investigators (GKL, HT, and CCT) who were blinded to the patients' prior imaging or clinical data.

Statistical analysis

Unpaired *t*-tests were performed to compare demographic characteristics and echocardiographic measurements between the pregnant and non-pregnant patient groups and also between trimesters. Analysis of variance was used when appropriate. In all tests, a $P < 0.05$ was considered as significant. Dimensional measurements are reported as height-indexed values unless otherwise specified. The resultant data are expressed as mean ± standard deviation To determine inter- and intra-observer variability of echocardiographic measurements, parameters for six randomly selected patients were analyzed by the three independent observers (interobserver variability) and on three different occasions 3 days apart, for each observer (intraobserver variability).

RESULTS

Baseline characteristics

Among the 97 pregnant patients, the mean age, gestational age, height, weight, and BSA were 27.2 ± 6.0 years,

26.4 ± 8.0 weeks, 162.2 ± 6.9 cm, 79.7 ± 17.0 kg, and 1.9 ± 0.2 m^2, respectively (Table 1). There was no significant difference between the pregnant patients and controls in terms of age, height, weight, or BSA. Five of the pregnant patients were in their first trimester, 52 were in their second trimester, and 40 were in their third trimester. The average gestational ages were 10 ± 2, 22 ± 4, and 34 ± 3 weeks, respectively for each trimester (Table 2).

Table 1 Comparison of echocardiographic and clinical characteristics between pregnant and control patients

	Pregnant	Non-pregnant	*P* value
Number	97	24	
Clinical characteristics			
Gestational age (weeks)	26.4±8.0		
Age	27.2±6.0	29.6±5.0	0.065
Height (cm)	162.2±6.9	161.6±7.4	0.702
Weight (kg)	79.7±17.0	73.0±18.7	0.101
BMI (kg/m^2)	30.3±6.3	27.9±6.6	0.096
BSA (m^2)	1.9±0.2	1.8±0.2	0.098
Right heart chambers			
RA area (cm^2)	13.7±2.7	12.0±2.2	0.006
RA area/height (cm^2/m)	8.5±1.7	7.4±1.3	0.008
RV DA (cm^2)	18.3±3.8	16.2±3.2	0.013
RV DA/height (cm^2/m)	11.3±3.8	10.0±1.9	0.013
RV SA (cm^2)	9.6±2.7	8.9±2.3	0.272
RV SA/height (cm/m)	5.9±1.6	5.5±1.4	0.302
RV FA change (%)	0.48±0.09	0.44±0.11	0.121
Left heart chambers			
LA AP (cm)	3.6±0.4	3.2±0.5	<0.001
LA AP/height (cm/m)	2.2±0.2	2.0±0.3	<0.001
LA Vol (cm^2)	48.7±13.9	40.4±11.2	<0.010
LA Vol/height (cm^3/m)	30.1±8.4	25.0±6.8	<0.010
LV EDD (cm)	4.7±0.5	4.5±0.4	0.052
LV EDD/height (cm/m)	2.9±0.3	2.8±0.2	0.067
LV ESD (cm)	2.9±0.3	2.8±04	0.254
LV ESD/height (cm/m)	1.8±0.3	1.7±0.2	0.291
LVOT diameter (cm)	2.00±0.14	1.87±0.16	<0.001
LV Dvol (cm^3)	100.7±20.7	90.4±17.5	0.027
LV Dvol/height (cm^3/m)	62.1±20.7	55.9±10.5	0.027
LV Svol (cm^3)	34.4±11.1	32.7±9.0	0.489
LV Svol/height (cm^3/m)	21.1±6.6	20.2±5.5	0.526
LV Mass (g)	121.6±33.5	112.3±28.2	0.215
LV Mass/vol (g/cm^3)	1.2±0.4	1.3±0.3	0.793
Ventricular function			
LV EF (%)	66.2±6.7	63.0±10.2	0.158
FS (%)	38.4±7.3	3.83±6.2	0.937
Wall dimensions			
IVS (cm)	0.78±0.12	0.77±0.11	0.644
IVS/height (cm/m)	0.48±0.08	0.48±0.07	0.722
PW (cm)	0.77±0.21	0.78±0.15	0.822
PW/height (cm/m)	0.47±0.13	0.48±0.09	0.773
Aortic measurements			
AA (cm)	2.5±0.3	2.4±0.3	0.488
AA/height (cm/m)	1.6±0.2	1.6±0.1	0.256

BMI: Body mass index, BSA: Body surface area, RA: Right atrial, RV: Right ventricular, LA: Left atrial, LV: Left ventricular, DA: Diastolic area, SA: Systolic area, AP: Antero-posterior, Vol: Volume, EDD: End diastolic diameter, ESD: End systolic diameter, OT: Outflow tract, Dvol: Diastolic volume, Svol: Systolic volume, EF: Ejection fraction, FS: Fractional shortening, IVS: Interventricular septum, PW: Posterior wall, AA: Ascending aorta

Table 2 Clinical and echocardiographic characteristics in second and third trimesters

	Second trimester	Third trimester	Non-pregnant	*P* value
Number	52	40	24	
Clinical characteristics				
Gestational age (weeks)	22.1±4.6	34.0±3.1		
Age	27.4±5.7	26.9±6.5	29.6±5.0	NS
Height (cm)	162.0±7.2	162.1±6.8	161.6±7.4	NS
Weight (kg)	77.6±15.2	82.5±39.4	73.0±18.7	0.05[e]
BMI (kg/m^2)	29.7±5.6	31.3±6.8	27.9±6.6	NS
BSA (m^2)	1.9±0.2	1.9±0.2	1.8±0.2	0.047[e]
Right heart chambers				
RA area (cm^2)	13.3±3.0	14.3±2.3	12.0±2.2	<0.001[e]
RA area/height (cm^2/m)	8.2±1.9	8.8±1.3	7.4±1.3	<0.001[e]
RV DA (cm^2)	17.9±4.1	18.9±3.4	16.2±3.2	0.004[e]
RV DA/height (cm^2/m)	11.0±2.4	11.7±2.2	10.0±1.9	0.005[e]
RV SA (cm^2)	9.2±2.6	10.3±2.8	8.9±2.3	NS
RV SA/height (cm^2/m)	5.7±1.6	6.4±1.7	5.5±1.4	NS
RV SA change (%)	0.49±0.09	0.46±0.09	0.44±0.11	NS
Left heart chambers				
LA AP (cm)	3.6±0.4	3.6±0.4	3.2±0.5	<0.001[c]
LA AP/height (cm/m)	2.2±0.3	2.2±0.2	2.0±0.3	<0.001[c]
LA Vol (cm^3)	47.1±14.3	50.9±13.6	40.4±11.2	0.004[e]
LA Vol/height (cm^3/m)	29.0±8.5	31.4±8.3	25.0±6.8	0.004[e]
LV EDD (cm)	4.6±0.5	4.8±0.4	4.5±0.4	0.008[e]
LV EDD/height (cm/m)	2.9±0.3	3.0±0.3	2.8±0.2	0.008[e]
LV ESD (cm)	2.8±0.5	3.1±0.4	2.8±04	0.007[e], 0.008[d]
LV ESD/height (cm/m)	1.7±0.3	1.9±0.2	1.7±0.2	0.008[e], 0.008[d]
LV OT diameter (cm)	1.96±0.15	2.04±0.12	1.87±0.2	<0.001[e], 0.010[d]
LV Dvol (cm^3)	97.7±20.6	104.6±21.1	90.4±17.5	0.011[e]
LV Dvol/height (cm^3/m)	60.2±12.3	64.6±12.7	1.4±0.3	0.010[e]
LV Svol (cm^3)	31.6±10.4	37.8±11.4	32.7±9.0	0.009[d]
LV Svol/height (cm^3/m)	19.5±6.2	23.3±6.8	20.2±5.5	0.006[d]
LV mass (g)	113.1±33.7	134.5±30.9	112.3±28.2	0.002[d]
LV mass/Vol (g/cm^3)	1.2±0.4	1.3±0.4	1.3±0.3	NS
Ventricular function				
LV EF (%)	68.0±6.2	64.1±6.8	63.0±10.2	0.036[c], 0.006[d]
FS (%)	40.0±7.2	36.0±6.1	38.3±6.3	0.007[d]
Wall dimensions				
IVS (cm)	0.75±0.12	0.82±0.12	0.77±0.11	0.009[d]
IVS/height (cm/m)	0.46±0.08	0.51±0.07	0.48±0.07	0.013[d]
PW (cm)	0.73±0.23	0.82±0.19	0.78±0.15	0.045[d]
PW/height (cm/m)	0.45±0.14	0.50±0.12	0.48±0.09	0.006[d]
Aortic measurements				
AA root (cm)	2.7±0.4	2.3±0.2	2.4±0.3	NS
AA root/height (cm/m)	1.6±0.2	1.7±0.1	1.6±0.1	NS

BMI: Body mass index, BSA: Body surface area, RA: Right atrial, RV: Right ventricular, LA: Left atrial, LV: Left ventricular, DA: Diastolic area, SA: Systolic area, AP: Antero-posterior, Vol: Volume, EDD: End diastolic diameter, ESD: End systolic diameter, OT: Outflow tract, Dvol: Diastolic volume, Svol: Systolic volume, EF: Ejection fraction, FS: Fractional shortening, IVS: Interventricular septum, PW: Posterior wall, AA: Ascending aorta, T2: Second trimester, T3: Third trimester, NP: Non-pregnant, NS: No significance.[c]T2 versus NP, [d]T2 versus T3, [e]T3 versus NP

All pregnant patients versus non-pregnant

In all pregnant patients compared with the non-pregnant patients, all four chambers showed enlargement during pregnancy (Table 1). Indexed left atrial (LA) volumes were larger compared to non-pregnant patients (30.1 ± 8.4 cm^3/m vs. 25.0 ± 6.8 cm^2, $P < 0.01$). Indexed LV diastolic volumes (Dvols) (62.1 ± 20.7 cm^3/m vs. 55.9 ± 10.5 cm^3/m, $P = 0.027$) and indexed LVOT diameter (1.23 ± 0.10 cm/m vs. 1.16 ± 0.09 cm/m, $P < 0.001$) were also significantly larger. Similarly, the indexed RA area was larger (8.5 ± 1.7 cm^2/m vs. 7.4 ± 1.3 cm^2/m, $P = 0.008$), as was the indexed RV diastolic area (11.3 ± 3.8 cm^2/m vs. 10.0 ± 1.9 cm^2/m $P = 0.01$). Table 3 shows Doppler-derived measurements in this comparison. SV and HR were increased. As a result, cardiac index (CI) was increased (3.0 ± 0.7 L/min/m^2 vs. 2.4 ± 0.4 L/min/m^2, $P < 0.001$). There were no significant differences in valvular regurgitation, the amount of pericardial effusion, or LV fractional shortening (FS). There was no significant difference in diastolic function between the pregnant and non-pregnant subjects.

Second and third trimester patients versus non-pregnant

Table 2 shows the clinical and echocardiographic measurements comparing the patients in the second and third trimester of pregnancy with the non-pregnant controls. There were significantly larger indexed-LA anteroposterior diameters (2.2 ± 0.3 cm/m vs. 2.0 ± 0.3 cm/m, $P < 0.001$) and non-indexed LVOT diameters (1.96 ± 0.15 cm vs. 1.87 ± 0.2 cm, $P = 0.012$) in the second trimester patients. These remained constant throughout the third trimester. HR also remained constant in the second trimester and increased during the third trimester. SV significantly increased in the second trimester (64.2 ± 11.8 mL vs. 56.2 ± 11.7 mL, $P = 0.008$) along with CI (3.0 ± 0.6 L/min/m^2 vs. 2.4 ± 0.4 L/min/m^2, $P < 0.001$), and both continued to increase significantly into the third trimester. LV EF increased significantly in the second trimester and decreased with progression to the third trimester (Table 4).

The significant changes that occurred solely during the third trimester include, a larger indexed-LA volume (31.4 ± 8.3 cm^3/m vs. 25.0 ± 6.8 cm^3/m, $P = 0.004$, larger indexed-LV Dvol (64.6 ± 12.7 cm^3/m vs. 55.9 ± 10.5 cm^3/m, $P = 0.01$) and increased LV mass (134.5 ± 31 g vs. 112.3 ± 28.2 g $P < 0.01$), with preservation of mass/volume ratio. Indexed RA and RV volumes also only increased significantly during the third trimester. PASP estimated from the PAAT was increased (40.1 ± 10. 3 mmHg vs. 45.5 ± 10.1 mmHg, $P = 0.029$) during the third trimester, but this was non-significant when comparing all pregnant women with non-pregnant women. Both E and A velocities were greatest in the second trimester; however, there were no overall significant changes observed in the diastolic parameters measured throughout pregnancy as manifest by an insignificant increase in mitral inflow velocity in early diastole (E) and at atrial contraction (A) and an insignificant increase in E/A ratio (Tables 2 and 4).

Reproducibility of measurements

Interobserver reproducibility was strong with an intraclass correlation coefficient higher than 0.7 with no significant

Table 3 Comparison of Doppler-derived variables between pregnant and control patients

	Pregnant	Non-pregnant	*P* value
TR vel (ms)	2.2±0.3	2.3±0.2	0.954
LVOT VTI (cm)	20.7±4.1	20.6±3.2	0.920
E (cm/s)	93.3±21.1	87.0±11.8	0.072
A (cm/s)	58.9±14,1	54.6±12.8	0.201
E' (cm/s)	16.0±2.8	16.6±3.0	0.387
A' (cm/s)	7.9±2.4	7.6±2.5	0.616
E/A	1.6±0.5	1.7±0.4	0.867
E/E'	6.0±1.7	5.4±1.4	0.200
HR	87.8±14.7	79.3±18.1	0.018
SV (mL)	64.0±12.6	56.2±11.7	0.008
CI (L/min/m^2)	3.0±0.7	2.4±0.4	<0.001
RV SP from TR (mmHg)	27.5±5.3	27.4±3.8	0.974
PAAT (ms)	121.1±29.4	129.36±32.3	0.258
PASP from PAAT (mmHg)	42.7±10.6	39.9±12.4	0.299

TR vel: Tricuspid regurgitant velocity; LVOT VTI: Left ventricular outflow tract velocity-time integral, HR: Heart rate, SV: Stroke volume, CI: Cardiac index, PAAT: Pulmonary artery acceleration time, PASP: Pulmonary artery systolic pressure, NP: Non-pregnant

Table 4 Doppler-derived variables during pregnancy and in non-pregnant patients in each trimester

	Second trimester	Third trimester	Non-pregnant	*P* value
TR vel (ms)	2.3±0.3	2.2±0.3	2.3±0.2	NS
LVOT VTI (cm)	21.6±4.0	19.6±4.2	20.6±3.2	0.015[d]
E (cm/s)	95.5±19.2	91.9±24.5	87.0±11.8	0.025[c]
A (cm/s)	61.2±11.9	56.4±16.4	54.6±12.8	0.040[c]
E' (cm/s)	16.1±2.6	15.6±3.1	16.6±3.0	NS
A' (cm/s)	8.0±2.7	7.3±1.6	7.6±2.5	NS
E/A	1.6±0.4	1.7±0.6	1.7±0.4	NS
E/E'	6.1±1.6	6.0±2.0	5.4±1.4	0.022[b]
HR	86.6±13.7	90.7±15.8	79.3±18.1	0.014[e]
SV (mL)	64.0±11.8	63.7±14.1	56.2±11.7	0.008[c], 0.035[e]
CI (L/min/m^2)	3.0±0.6	3.0±0.7	2.4±0.4	<0.001[c], <0.001[e]
RV SP from TR (mmHg)	28.5±5.4	26.3±5.0	27.4±3.8	NS
PAAT (ms)	129.4±32.3	113.8±28.3	129.36±32.3	NS
PASP from PAAT (mmHg)	40.1±10.3	45.5±10.1	39.9±12.4	0.029[d]

TR vel: Tricuspid regurgitant velocity, LVOT VTI: Left ventricular outflow tract velocity-time integral, HR: Heart rate, SV: Stroke volume, CI: Cardiac index, NP: Non-pregnant, PAAT: Pulmonary artery acceleration time, PASP: Pulmonary artery systolic pressure. [a]T1 versus NP, [b]T1 versus T2, [c]T2 versus NP, [d]T2 versus T3, [e]T3 versus NP

difference between reviewers. LA anteroposterior and RA area measurements between observers were in moderate agreement with correlation coefficients above 0.44. The intraobserver correlation coefficients of the three investigators were 0.97, 0.96, and 0.97, respectively.

DISCUSSION

To the best of our knowledge, our study is one of the largest to provide data on the cardiovascular changes that occur during uncomplicated pregnancy. We demonstrated physiological increase in all four chamber sizes, LV mass, and CI throughout pregnancy. We further outlined at which stage of pregnancy these changes were most significant. We also noted that despite the geometrical changes that occur, there is no significant change in diastolic function.

HR, SV, and CI increased in the second trimester patients. While HR was greater in the third trimester patients, the SV and CI were not seen to be significantly different between the second and third trimesters. This had been previously described by Hunter *et al.* and Savu *et al.*, but not observed by Mesa *et al.,*[7-9] Our study demonstrated a 25% greater CI during pregnancy as compared to non-pregnant patients. While some studies have demonstrated 30-50% increase in cardiac output, the general consensus is that cardiac output rises significantly during pregnancy. Varying methods used for measurement of cardiac output including thermodilution, Fick, M-mode, and Doppler echocardiography could account for this disagreement.[10] Similar to our study, others used Doppler echocardiography which estimates cardiac output using LVOT cross-sectional area derived from LVOT diameter. Variation in the cross-section used to measure LVOT diameter can have a great effect on cardiac output.[11] Analysis of our data demonstrated that LVOT diameter had the greatest influence on differences in cardiac output. The studies showing greater increases in cardiac output reported larger LVOT diameters. The methodology of calculating LVOT area possibly had a significant effect on their measurements of cardiac output.[12-14]

Previous studies calculating chamber volumes used formulas that made assumptions in shape.[12,15] We used Simpson's biplane method of disks, which allows for a more accurate measurement of the altered chamber geometry during pregnancy. As a result of chronic volume overload from increased maternal blood volume, the left atrium was first to respond with significant enlargement by the second trimester and a non-significant increase into the third trimester. Other chambers did not notably enlarge between trimesters.

LV mass significantly increased between the second and third trimesters. However, after mass was indexed by ventricular Dvol, there was no significant difference. This suggests that mass increase is a result of myocyte elongation rather than myocardial thickening (eccentric rather than concentric hypertrophy). Previously studied measurements of interventricular and LV PW thickness showed significantly larger measurements when comparing third trimester values with those of non-pregnant patients.[8,16]

Using the biplane Simpson's method, we found that LV EF significantly increased in the second trimester and decreased between the second and third trimesters. Savu *et al.* also used the biplane Simpson method and observed similar trends.[9] Previous studies using the Teichholz and M-mode formulas showed conflicting results on EF changes during pregnancy.[11,12,17,18] The inconsistent results can be attributed to the use of different formulas. Correlating with decreased EF in the third trimester, FS also decreased with an increased LV systolic volume as compared to second trimester patients. This resulted in a lower SV. Mone *et al.* showed that a fall in afterload allowed for relative preservation of systolic ventricular function during most of the pregnancy.[19] However, there was an observed decrease in EF near term caused by decreased preload, attributed to compression of the inferior vena cava by the uterus as well as to a reduction in placental blood flow. This accounts for the decrease seen in load dependent parameters, EF and FS late in pregnancy.

As for Doppler imaging parameters, our data showed that transmitral inflow velocity (E) was higher during pregnancy, increasing through the second trimester. This is likely due to increased venous return. As the left ventricle wall hypertrophies in response to chronic strain, LV compliance and early diastolic filling rate decreases.[20] This, along with a reduction in preload, accounts for the decrease in E. Flow from the atrial kick (A) increased and reached a maximum in the second trimester and decreased in the third trimester. We did not, however, observe a change in the overall E/A ratio and did not we observe any evidence of diastolic dysfunction. E/E' was insignificantly higher in our study and remained stable throughout pregnancy. Previous studies, with smaller sample sizes, had observed slightly decreased or normal E/E'.[15,20] This inconsistency in could be attributed to the small sample sizes of these studies.

Accurate assessment for pulmonary hypertension is especially important in pregnancy due to increased mortality in women with pulmonary arterial hypertension. We calculated the peak PASP during pregnancy from PAAT

and observed pressures significantly greater in the third trimester.[21] This increase in PASP was not observed when it was calculated from the tricuspid regurgitation gradient. The accuracy of standard transthoracic echocardiographic measurements of pulmonary artery pressures using the tricuspid regurgitation maximal velocity has been questioned in pregnancy.[22] In our study, PASP was measured in 81.4% of the pregnant patients using PAAT and was 10 mmHg higher than the PASP calculated from the tricuspid regurgitation velocity. Although calculating PASP from PAAT has been shown to have similar accuracy as using tricuspid regurgitation in non-pregnant patients, it has not been evaluated in pregnancy.[6] If validated, the usage of PAAT could allow for a more accurate calculation of PASP in pregnant patients.

Limitations

In this retrospective study, changes observed were not the result of serial observations and patients were unpaired. Echocardiographic changes followed longitudinally in the same patient would increase the study's power by reducing inter-subject variability. First trimester data were limited by the small sample size of the first trimester echocardiograms. However, the number of total pregnant patients and of patients in the second and third trimesters is among the largest in the existing literature.

CONCLUSIONS

We provided observational data on echocardiographic parameters during each trimester of normal pregnancy, using the most current measurement modalities. Confirmation of our results with larger, longitudinal studies using 2D/3D assessments should be performed, and future studies comparing normal and abnormal echocardiograms would improve diagnostic criteria for cardiovascular pathology.

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