Effects of physiologic load of pregnancy on left ventricular contractility and remodeling

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Left ventricular (LV) adaptation to the hemodynamic load of pregnancy has been studied with load-sensitive ejection-phase indexes, but the results of these studies are conflicting. The aim of this study was to examine the effects of the hemodynamic load of pregnancy on the contractile state of the left ventricle by using load-adjusted indexes of contractility. Thirty-four healthy women were prospectively studied by serial echo and Doppler examinations at six periods during pregnancy and after delivery. LV volume increased 10.5%, paralleling the change in stroke volume. End-systolic stress, an index of myocardial afterload, decreased 28.8% because of a decrease in end-systolic pressure and an increase in LV thickness/diameter ratio. Despite the increase in preload and the decrease in afterload, ejection phase indexes did not change during or after pregnancy. Although remaining within the normal range, the afterload-adjusted velocity of circumferential fiber shortening, an index of contractility that is relatively insensitive to preload, transiently decreased by 1.75 SDs during gestation, returning to non-pregnant values 2 to 4 weeks postpartum. Thus the increase in hemodynamic load that characterizes normal pregnancy is associated with preservation of global pump function. The transient decrease in contractile state may represent an adaptation phase of the contractile elements of the myocardium to the rapid changes in loading conditions observed during the first trimester of pregnancy. (Am Heart J 1997; 133:53-9.)

Pregnancy presents a unique opportunity to study the effects of a transient increase in hemodynamic load on the heart. Although a substantial amount of information regarding left ventricular (LV) adaptation to the physiologic load of pregnancy has been published, little is known about the interaction between altered loading conditions and LV systolic performance. The majority of publications related to cardiac changes during pregnancy have focused on describing structural and functional patterns at the ventricular level, the systemic vascular bed, and cardiac output.
Table I. Characteristics of study patients*  

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<tbody>
<tr>
<td>Age at enrollment (yr)</td>
<td>31.9 ± 4.4</td>
<td>(21.9-41.8)</td>
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<tr>
<td>No. of previous pregnancies</td>
<td>1.6 ± 1</td>
<td>(0-3)</td>
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<tr>
<td>No. of previous deliveries</td>
<td>0.6 ± 0.9</td>
<td>(0-3)</td>
</tr>
<tr>
<td>No. of previous abortions</td>
<td>0.4 ± 0.6</td>
<td>(0-2)</td>
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<tr>
<td>Length of gestation (wk)</td>
<td>39.6 ± 1.3</td>
<td>(37-41)</td>
</tr>
<tr>
<td>Mode of delivery</td>
<td></td>
<td></td>
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<tr>
<td>Vaginal</td>
<td>76.5%</td>
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<tr>
<td>Cesarean section</td>
<td>23.5%</td>
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<tr>
<td>Apgar score</td>
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<tr>
<td>1 min</td>
<td>8.62 ± 0.4</td>
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<tr>
<td>5 min</td>
<td>9.2 ± 0.6</td>
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<tr>
<td>Birth weight (kg)</td>
<td>3.47 ± 0.4</td>
<td>(2.5-4)</td>
</tr>
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</table>

*Data presented as mean ± SD (range).

Table I summarizes their demographic characteristics and pregnancy outcomes. Patients with pregnancy-induced hypertension, gestational diabetes, or other obstetric complications were not included in this study. Each participant was monitored with serial echocardiograms at six periods during and after normal gestation: at 10 to 12, 18 to 20, 28 to 30, and 36 to 38 weeks of gestation and at 2 to 4 and 12 to 14 weeks postpartum.

Echocardiographic protocol. Height and weight were measured before each echocardiogram, and body surface area was determined. Examinations were performed with the patients in the left lateral decubitus position with an Acuson XP 128 imaging system (Acuson Corporation, Mountain View, Calif.) equipped with 5.0, 3.5, or 2.5 MHz phased-array transducers. A complete two-dimensional M-mode tracing of the LV minor axis between the papillary muscles tips, the indirect carotid arterial pulse tracing, phonocardiogram, and electrocardiogram (ECG), were simultaneously recorded on a hard copy at a high paper speed (100 mm/sec). Right arm blood pressure was recorded at the same time with a Dinamap Vital Signs Monitor 8100T (Critikon, Inc., Tampa, Fla.). After discarding the first reading, the average of three blood pressure readings was used for data analysis.

Data analysis

LV dimensions and stress. The M-mode tracing of the endocardial borders of the interventricular septum and the endocardial and epicardial borders of the LV posterior wall, the arterial pulse tracing, the onset of Q wave on the ECG, and the first high-frequency component of the second heart sound on the phonocardiogram were digitized with a computer-based digitizing station with customized software.15 Time delay of the carotid pulse tracing was corrected by electronically aligning the dicrotic notch with the first high-frequency component of the aortic component of the second heart sound. From the digitized data, the following instantaneous measurements were obtained by averaging three to six cardiac cycles. LV pressure during ejection was calculated by assigning the diastolic pressure to the minimum and the systolic pressure to the peak of the arterial pulse tracing and subsequently calculating the intervening values by linear interpolation. This technique has been previously validated against pressure measurements obtained by catheterization.17 LV posterior wall thickness was measured. LV internal dimensions in the short-axis plane was calculated. End-diastolic measurements were taken at maximal LV dimension, and end-systolic measurements were taken at aortic valve closure (first high-frequency component of the second heart sound on the phonocardiogram). LV meridional wall stress (WSm) in grams per square centimeter was calculated throughout ejection according to the formula of Brodie et al.18:

\[ WSm = \frac{(PD)(1.35)}{h(1 + \frac{h}{6})(4)} \]

where P is pressure in millimeters of mercury, D is dimension in centimeters, h is posterior wall thickness in centimeters, and 1.35 is a conversion factor from millimeters of mercury to grams per square centimeter. Meridional peak systolic stress is the maximal value of LV meridional wall stress. End-systolic meridional wall stress is measured at the onset of the aortic component of the second heart sound on the phonocardiogram. Total ejection meridional stress is calculated by integrating meridional stress over the ejection period. LV circumferential wall stress (WSc) in grams per square centimeter was calculated according to the formula of Mirsky19:

\[ WSc = \frac{(PD)(1.35)}{h} \times \left[ 1 - \frac{(D)(h)^2}{(2D + h)^{1.5}} \right] \]

where L is LV long-axis dimension in centimeters, D is short-axis dimension in centimeters, h is posterior wall thickness in centimeters, and P is pressure in millimeters of mercury. Heart rate adjusted ejection time (ETe) was measured from the arterial pulse tracing from the onset of upstroke to the onset of the dicrotic notch and adjusted to...
a heart rate of 60 beats/min by dividing the heart rate by
the square root of the R-R interval on the ECG. LV short-
tening fraction (SF) was calculated as \((\text{End-diastolic}
\dimen - \text{End-systolic dimension})/\text{End-systolic dimen-
sion}\). The rate-adjusted mean velocity of circumferential
fiber shortening (VCFC) was calculated as SF/ETc. LV vol-
ume was calculated by the area-length method.\(^2\) Ejection
fraction was calculated as \(100 \times (\text{End-diastolic volume}
- \text{End-systolic volume}) + \text{End-diastolic volume}\). LV
mass was calculated by the formula of Devereux and
Reich.\(^3\)

**Contractility index.** The relation between rate-adjusted
mean velocity of shortening and end-systolic stress (stress-
velocity index) was used as an index of LV contractility.\(^4\)\(^6\)
This inverse linear relation has been shown to be an after-
load-adjusted and a relatively preload-insensitive index of
contractility.\(^4\) The value of the stress-velocity index for each
subject was determined relative to the previously published
distribution of this index in unaffected subjects\(^4\) and calcu-
lated as a deviate from the group mean (Z score).

**Preload.** LV end-diastolic volume was used to assess
preload.

**Doppler.** Cardiac output was calculated with the stan-
dard echo-Doppler technique.\(^5\) Stroke volume was calcu-
lated as cardiac output divided by heart rate. Systemic
vascular resistance (SVR) was calculated as mean arterial
pressure divided by the echo-Doppler-derived cardiac out-
put and converted to dyne/sec/cm\(^{-5}\).

**Statistical analysis.** Data are reported as mean
value \pm SD for each group of measurements. For each
variable, the significance of differences during and after
pregnancy was examined by analysis of variance with a
repeated-measures model followed by Fisher’s protected
least significant difference test for post hoc analysis.\(^2\) \(^2\) \(^3\)
Linear and nonlinear regression analyses were used to ex-
amine the relations among continuous variables. Data
analysis was performed with a commercially available
statistical package (StatView 4.1 and SuperANOVA, Aba-
cus Concepts Inc., Berkeley, Calif.). For all tests, a \(p\) value
of \(<0.05\) was considered statistically significant.

**RESULTS**

One hundred thirty-two studies were performed in
the 34 women who enrolled in the study, averaging
3.9 studies per patient. Table I summarizes the de-
moregraphic characteristics and data regarding out-
come of pregnancies. No complications occurred dur-
dering any pregnancy, and all newborns were healthy.
Table II and Fig. 1 summarize the hemodynamic and
echocardiographic data.

**LV dimensions.** LV minor axis end-diastolic and
end-systolic diameters did not significantly change
during the study period, whereas end-diastolic length
increased 6.6\% (Table II). LV posterior wall thick-
ness and LV mass increased 30\% and 38\%, respec-
tively (\(p < 0.0001\)). After adjustment for the increase
in body surface area, LV mass index increased 24.6\%
(\(p < 0.0008\)). LV mass and posterior wall thickness de-
creased by the first postpartum examination and re-
turned to normal values by 12 to 14 weeks postpartum.
The increase in LV mass correlated linearly with car-
diac output (\(r = 0.37, p < 0.0001\)), and inversely with
peak systolic wall stress (\(r = -0.43, p < 0.0001\)).

**LV wall stress.** End-systolic stress, a measure of
myocardial afterload,\(^12\)\(^16\) decreased during gesta-
tion in both the meridional and circumferential planes (28.8\% and
26\%, respectively, \(p < 0.0001\)). The decrease in end-systolic stress was caused by a
combination of a 15.3\% decrease in end-systolic blood
pressure, a 30\% increase in posterior wall thickness,
and no significant change in end-systolic LV diame-
ter (Table II). Peak systolic wall stress was highest
in early gestation and progressively decreased to-
toward term. All stress measurements returned to
normal values by the first postpartum study.

**Systolic function, stress-velocity, and stress-shorten-
ing indexes.** Despite the decrease in afterload, the
following ejection phase indexes of global function
did not significantly change during the study period:
shortening fraction, ejection fraction, and VCFC (Ta-
ble II, Fig. 1). The stress-velocity index decreased by
1.75 SDs in the meridional plane and by 1.46 SDs in
the circumferential plane, reaching a nadir at 18 to
20 weeks of gestation and returning toward non-
pregnant values in the early postpartum period (Ta-
ble II, Fig. 1). This transient decrease in contractil-
ity index was caused by a decrease in end-systolic stress (28.8\%)
without the expected increase in
velocity of fiber shortening, which remained un-
changed. The afterload-adjusted fractional shorten-
ing (stress-shortening index, which is sensitive to
preload and contractility) did not significantly change
during the study period.

**Preload.** LV end-diastolic volume increased 10.5\%
during gestation, reaching its peak at 28 to 3 weeks.
Changes in LV volume during and after pregnancy
paralleled the changes in cardiac output with a sig-
nificant linear correlation (\(r = 0.42, p < 0.0001\)).

**Cardiac output and SVR.** Doppler-derived cardiac
output increased 44.8\%, peaking at 28 to 30 weeks
with a small decrease toward term, returning to
baseline values 2 to 4 weeks postpartum (\(p < 0.0001\)).
When adjusting for changes in body surface area,
cardiac index increased 34\% (\(p < 0.0001\)). The in-
crease in cardiac output was caused by a combination
of an 18.5\% increase in stroke volume and a
15.6 \pm 10.7 beats/min increase in heart rate. SVR
decreased 39.8% from 1118.3 \pm 217 to 672.8 \pm 176
dynes/sec/cm\(^{-5}\) at 28 to 30 weeks, returning to base-
line values 2 to 4 weeks postpartum.

**Heart rate and blood pressure.** Compared with the
Table II. Echocardiographic and Doppler data

<table>
<thead>
<tr>
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<th>Pregnancy</th>
<th>Postpartum</th>
<th>p Value</th>
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<tbody>
<tr>
<td></td>
<td>10-12 wk</td>
<td>18-20 wk</td>
<td>28-30 wk</td>
</tr>
<tr>
<td><strong>Timing intervals</strong></td>
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<tr>
<td>Heart rate</td>
<td>71.6 ± 13</td>
<td>77 ± 11</td>
<td>78.9 ± 10</td>
</tr>
<tr>
<td>Ejection time (msec)</td>
<td>315 ± 27</td>
<td>313 ± 20</td>
<td>299 ± 24</td>
</tr>
<tr>
<td><strong>Pressure data (mm Hg)</strong></td>
<td></td>
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</tr>
<tr>
<td>Systolic BP</td>
<td>102.9 ± 9.5</td>
<td>97.1 ± 7.4</td>
<td>96.5 ± 9.3</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>55.1 ± 4.5</td>
<td>53.6 ± 6</td>
<td>52.2 ± 7.9</td>
</tr>
<tr>
<td>Mean BP</td>
<td>75.6 ± 7.2</td>
<td>73.4 ± 6.5</td>
<td>71.4 ± 7.8</td>
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<tr>
<td>End-systolic BP</td>
<td>79.7 ± 9</td>
<td>76.1 ± 8</td>
<td>70.3 ± 9.5</td>
</tr>
<tr>
<td><strong>Dimensions</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>LVEDD (mm)</td>
<td>46.9 ± 5.7</td>
<td>47.1 ± 3.4</td>
<td>47.8 ± 3.5</td>
</tr>
<tr>
<td>LVESD (mm)</td>
<td>29.7 ± 5</td>
<td>30.1 ± 3.7</td>
<td>30 ± 4.4</td>
</tr>
<tr>
<td>LV length (mm)</td>
<td>74.1 ± 5.4</td>
<td>76.3 ± 3.3</td>
<td>78.5 ± 5.1</td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>112.6 ± 33</td>
<td>112.8 ± 19</td>
<td>121 ± 18</td>
</tr>
<tr>
<td>LV PWTh (mm)</td>
<td>8.7 ± 1.3</td>
<td>8.9 ± 1.5</td>
<td>9.3 ± 1.6</td>
</tr>
<tr>
<td>LV mass (gm)</td>
<td>134.4 ± 27</td>
<td>143.4 ± 36</td>
<td>153.7 ± 30</td>
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<tr>
<td>LV mass index (gm/m²)</td>
<td>80.5 ± 14</td>
<td>85.2 ± 19</td>
<td>88.6 ± 17</td>
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<tr>
<td><strong>Stress data</strong></td>
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<tr>
<td>ESS (merid) (gm/cm²)</td>
<td>35.8 ± 11.4</td>
<td>34.2 ± 11</td>
<td>31.6 ± 10.3</td>
</tr>
<tr>
<td>ESS (circ) (gm/cm²)</td>
<td>50.3 ± 13.8</td>
<td>48.9 ± 11.9</td>
<td>45.2 ± 12.4</td>
</tr>
<tr>
<td>Peak stress (gm/cm²)</td>
<td>154.8 ± 44</td>
<td>127.2 ± 32.5</td>
<td>120.2 ± 30.5</td>
</tr>
<tr>
<td>Total stress (gm/sec/cm²)</td>
<td>27 ± 9.2</td>
<td>25.1 ± 6.1</td>
<td>22.7 ± 6.5</td>
</tr>
<tr>
<td><strong>Contractility index</strong></td>
<td></td>
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<tr>
<td>Merid ESS-VCFc</td>
<td>-0.39 ± 1.65</td>
<td>-1.75 ± 2.5</td>
<td>-1.15 ± 2.6</td>
</tr>
<tr>
<td>Circ ESS-VCFc</td>
<td>-0.87 ± 1.86</td>
<td>-1.46 ± 2</td>
<td>-1.1 ± 2.1</td>
</tr>
<tr>
<td><strong>LV function</strong></td>
<td></td>
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</tr>
<tr>
<td>VCFc (circ/sec)</td>
<td>1.09 ± 0.16</td>
<td>1.03 ± 0.18</td>
<td>1.08 ± 0.18</td>
</tr>
<tr>
<td>Fractional shortening</td>
<td>0.37 ± 0.05</td>
<td>0.36 ± 0.06</td>
<td>0.37 ± 0.05</td>
</tr>
<tr>
<td>Merid FS-ESS (SD)</td>
<td>0.49 ± 1.99</td>
<td>0.6 ± 2.6</td>
<td>0.08 ± 2.3</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>0.69 ± 0.05</td>
<td>0.67 ± 0.07</td>
<td>0.67 ± 0.06</td>
</tr>
<tr>
<td><strong>Output results</strong></td>
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<tr>
<td>Stroke volume (ml)</td>
<td>82.4 ± 20.8</td>
<td>93.4 ± 20.4</td>
<td>100.8 ± 17.6</td>
</tr>
<tr>
<td>Stroke index (ml/m²)</td>
<td>49.2 ± 12.8</td>
<td>57.3 ± 11.4</td>
<td>58.1 ± 10</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>5.8 ± 1.6</td>
<td>7.1 ± 1.7</td>
<td>7.85 ± 1.5</td>
</tr>
<tr>
<td>Cardiac index (L/min/m²)</td>
<td>3.49 ± 1.1</td>
<td>4.3 ± 1.1</td>
<td>4.33 ± 0.86</td>
</tr>
<tr>
<td><strong>Vascular resistance</strong></td>
<td></td>
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<tr>
<td>SVR (dyne/sec/cm⁻⁵)</td>
<td>1076.1 ± 354</td>
<td>894.1 ± 138</td>
<td>672.8 ± 176</td>
</tr>
</tbody>
</table>

BP, Blood pressure; LVEDD, LV end-diastolic diameter; LVESD, LV end-systolic diameter; LVEDV, LV end-diastolic volume; LV PWTh, LV posterior wall thickness (in diastole); ESS, end-systolic stress; merid, meridienal; circ, circumferential; VCFc, rate-corrected velocity of circumferential fiber shortening; FS, fractional shortening.

In this study of LV adaptation to the physiologic load of pregnancy, we found that myocardial mechanics substantially change in a reversible fashion with preservation of global systolic function and a transient decrease in contractility. LV structure also changes in response to the hemodynamic load with an accrual of physiologic hypertrophy that regresses after birth.

**DISCUSSION**

In this study of LV adaptation to the physiologic load of pregnancy, we found that myocardial mechanics substantially change in a reversible fashion with preservation of global systolic function and a transient decrease in contractility. LV structure also changes in response to the hemodynamic load with an accrual of physiologic hypertrophy that regresses after birth.

**LV function and contractility.** Many investigators have studied LV systolic function during pregnancy, most commonly with ejection phase indexes such as fractional shortening, ejection fraction, and VCFc. The results of these studies have been inconsistent—some authors found enhanced function, whereas others found no significant change. In this study, ejection-phase indexes of LV function remained unchanged despite an increase in LV end-diastolic volume and a decrease in afterload. When LV contractility was assessed by the stress-velocity...
Fig. 1. Changes in loading conditions, LV global function, contractility, and mass during and after pregnancy. Top, Percent change in heart rate-corrected VCFc, cardiac output, SVR, and LV mass compared with 12- to 14-week postpartum study. Bottom, Changes (in SDs) for stress-shortening index (an ejection-phase index of LV systolic function adjusted to afterload) and contractility index. Changes in meridional end-systolic stress ($ESS_m$), an index of myocardial afterload, are presented as percent change from 12- to 14-week postpartum study. PP, Postpartum.

Index, a transient reversible decrease in contractility was found in the second trimester. LV contractility, however, remained within 2 SDs of the normal limits throughout gestation and returned toward baseline values by the early postpartum period. The transient decline in contractility index was caused by a decrease in end-systolic stress that was not matched by an increase in rate-corrected VCF. Lang et al.16
used the stress-velocity index to assess LV contractility before and 24 hours after birth in patients with preeclampsia. In corroboration with the findings of this study, contractility at term was normal and changes in global function were attributed to an increased afterload related to preeclampsia. The significance of a mild transient decline in contractility (that remained within normal limits) is unclear. Because contractility was assessed with a relatively load-insensitive index, this observation suggests that factors unrelated to preload or afterload may be responsible for the observed changes in contractility. The transient decrease in contractility index may conceivably be related to other factors of pregnancy, such as endocrinologic or autonomic changes, which were not measured in this study. Alternatively, the observed changes in contractility index may represent an adaptation phase of the contractile elements of the myocardium to the rapid changes in loading conditions during early gestation. Data from clinical investigations of the volume-overload state support the contention that in some circumstances of acute or severe volume overload, indexes of LV contractility may decrease. As pregnancy progresses, the contractile elements adapt to the volume overload and return to baseline values. Such adaptation of the contractile elements to chronic volume overload has been demonstrated in experimental models of isolated papillary muscle and intact canine heart.

**Alterations in loading conditions.** Although difficult to measure directly, preload has been assumed to be increased during pregnancy. The increases in blood volume and cardiac output have been well documented by invasive and noninvasive methods, and, consistent with previous reports, a 44.8% increase in cardiac output and a 34% increase in cardiac index were also found in our study. LV end-diastolic volume increased in a parallel fashion, indicating an increase in preload until term with a return to normal values by the first postpartum examination.

A progressive decrease in myocardial afterload was demonstrated throughout gestation. A nearly 40% decrease in SVR was found in this study, similar to other investigations. Afterload, however, may be misrepresented by SVR because it fails to account for the geometric relation between fiber shortening and force generation. This limitation is particularly relevant to pregnancy because the left ventricle remodels throughout gestation with accrual of physiologic hypertrophy. Wall stress measured at end systole has been proposed as a sensitive index of myocardial afterload. In this study, meridional end-systolic stress decreased 28.8%, and circumferential end-systolic stress decreased 26%, reaching its lowest values in late gestation and returning to normal values in the early postpartum examination. As expected, changes in end-systolic stress did not exactly parallel changes in SVR. In the third trimester SVR increased 17.8%, whereas end-systolic stress remained unchanged at its nadir. The continued decrease in myocardial afterload in late gestation despite a rise in end-systolic blood pressure and SVR is explained by the continued increase in wall thickness during that period that counterbalances the increase in end-systolic blood pressure.

**Structural adaptation to the hemodynamic load of pregnancy.** The results of this study show that the left ventricle remodels during and after gestation in response to the physiologic load of pregnancy. The physiologic hypertrophy of pregnancy resembles other forms of physiologic hypertrophy, such as those found with exercise training, except for the transient decrease in contractility. LV remodeling during gestation is characterized by an increase in end-diastolic volume, posterior wall thickness, and mass index. These findings are consistent with findings in experimental preparations of volume overload in a canine model with a large aortocaval shunt.

The trigger for accrual of LV hypertrophy during normal pregnancy is not entirely clear. A significant correlation was found between the increase in cardiac output and the increase in LV mass. Hence speculating that the substantial increase in volume load triggered the increase in LV mass is reasonable. Peak systolic stress, a parameter that closely correlates with hypertrophy, was at its highest level in early pregnancy. Nonmechanical factors may also play a role in accrual of LV mass during pregnancy, including hormonal and other humoral substances. It should also be kept in mind that the increase in wall thickness and mass may not all be from an increase in contractile proteins, and that myocardial edema or an increase in extracellular matrix may contribute to the apparent hypertrophy. Regardless of the mechanism and content of the physiologic hypertrophy of pregnancy, a regression to normal values was found in all measured parameters of LV dimensions by 12 to 14 weeks postpartum.

**Study limitations.** Pregestation measurements were not obtained in the study subjects because the women were enrolled after pregnancy was confirmed. Having preconception values to compare with gestational and postpartum measurements and to ascertain return of cardiovascular parameters to baseline would be optimal. However, the temporal relation between changes in hemodynamic load, indexes of ventricular function and contractility, and LV remodeling do not rely on any assumptions re-
garding pregestation conditions and are made on the basis of observations made during the study period. The echo-Doppler method used to assess cardiac output has inherent limitations. However, the same technique was used in all study subjects, and the relative changes throughout the study period are likely to be accurate. Also, although all measurements were performed by one investigator, interobserver and intraobserver variability exist.

In conclusion, the physiologic increase in hemodynamic load that characterizes normal pregnancy is associated with LV remodeling and preservation of global LV pump function. Although myocardial contractility remains within normal limits during and after pregnancy, a transient reversible decrease in contractility index occurs during gestation and subsequently returns to nonpregnant values. The transient decrease in contractility during a physiologic state such as pregnancy warrants further study, particularly with regard to potential implications concerning peripartum cardiomyopathy.

REFERENCES