Improvement in Midwall Myocardial Shortening With Regression of Left Ventricular Hypertrophy


Abstract—Despite normal indices of left ventricular (LV) chamber function, patients with LV hypertrophy (LVH) due to hypertension are thought to have depressed midwall systolic shortening compared with normotensives. The aims of the present study were (1) to confirm this observation and (2) to assess the effects of antihypertensive therapy that cause regression of LVH on LV systolic function assessed at both the midwall and endocardium. Thirty-eight previously untreated hypertensive subjects with LVH underwent echocardiography and were compared with 38 normotensive control subjects. Comparisons between the group with LVH and the control group revealed no significant differences in cardiac output (4.32±0.23 versus 4.55±0.21 L/min), ejection fraction (62.5±2% versus 66.4±1.07%), or endocardial fractional shortening (34.5±1.45% versus 37.0±0.82%), but shortening assessed at the midwall was significantly less in the group with LVH (17.9±1.11% versus 21.6±0.63%, P<0.01). Subsequently, 32 patients with uncontrolled hypertension (24 previously untreated and 8 on existing antihypertensive therapy) underwent treatment with ramipril, with the addition of felodipine and bendrofluazide if required, to reduce blood pressure to <140/90 mm Hg. These 32 patients underwent echocardiography at baseline, after blood pressure control, and after an additional 6 months of tight blood pressure control. Good blood pressure control was achieved after 6 months compared with baseline (143/86±2.8/1.4 versus 174/103±4.1/1.9 mm Hg; P<0.01) with significant regression of LV mass index (124±3.4 versus 145±3.8 g/m², P<0.01). LV fractional shortening assessed at the midwall improved with regression of LVH (21.9±0.84 and 18.7±1.19%, P<0.05), with posttreatment midwall shortening being similar to that of the normal control subjects evaluated in the first study. Hypertensive patients with LVH have depressed midwall systolic shortening despite normal indices of LV chamber function. Regression of LVH after good blood pressure control improved midwall shortening to normal levels. (Hypertension. 2000;34:755-759.)

Key Words: hypertension, detection and control • ventricular function • hypertrophy • blood pressure • systole • echocardiography

Discrepancies between experimental and human studies have resulted in controversy as to whether left ventricular (LV) systolic function is preserved in pressure-overload hypertrophy. Experimental studies suggest that systolic function is depressed in hypertrophy,1-3 whereas studies in humans suggest that it is preserved.4-6 However, most human investigations have assessed whole heart function through the use of endocardial measurements, whereas experimental studies have tended to involve the measurement of myocardial or myofibril function.

It is often assumed that the inner and outer parts of the LV wall thicken equally during systole. However, myocardial shortening in subendocardial layers is greater than that in subepicardial layers.7 Therefore, a theoretical mid point in the wall shows relative migration toward the epicardium throughout contraction (Figure). There may therefore be a discrepancy between shortening at the endocardium and the midwall, and this has prompted recent interest in the measurement of midwall shortening.7-12 There also are anatomic reasons why assessment of shortening at the midwall level may be preferred; circumferentially orientated fibers predominate here, unlike at the subendocardium and subepicardium, where most fibers are longitudinally orientated.13

Any discrepancy between shortening at the endocardium and the midwall may be increased during LV hypertrophy (LVH), when the ventricular walls are thickened, and it is suggested that in humans with LVH, fiber shortening is reduced but the geometric changes associated with the hypertrophy allow a maintained cardiac output despite this (ie, endocardial shortening is maintained when midwall shortening is depressed).11 This raises the question of whether regression of LVH and the associated geometric remodeling adversely influences systolic function. Previous studies have assessed serial changes in endocardial function with LVH.
Epicardial migration of theoretical midwall fiber during systole.

regression and have suggested that this is maintained. Our aim was to assess the effects of LVH regression on midwall shortening.

Initially, midwall shortening among our patient population with LVH was compared with that of normotensive control subjects. After this, systolic function measured at the midwall and endocardium was compared before and after antihypertensive medication in 32 hypertensive subjects who had been enrolled in a treatment study to assess the effects of drug therapy on LVH.

Methods

Patients
In the first study, 38 previously untreated hypertensive subjects with LVH were compared with 38 normotensive control subjects of a similar age. Hypertensive patients were recruited from the Peart-Rose Clinic at our institution. The normotensive subject groups consisted of healthy volunteers. All subjects had normal systolic function as determined with conventional echocardiographic methods and no clinical or Doppler evidence of valvular stenosis or regurgitation. Patients were excluded if they were obese (body mass index >30 kg/m²) or had a history of ischemic heart disease, peripheral vascular disease, congestive cardiac failure, diabetes mellitus, or alcohol abuse. Secondary causes of hypertension were excluded through standard clinical investigation. Each subject underwent echocardiography and a standard 12-lead ECG examination.

The second study involved 32 patients with uncontrolled hypertension (24 previously untreated and 8 patients on existing antihypertensive therapy). The 8 patients on treatment underwent a 4-week placebo washout period, during which all antihypertensive therapy was stopped. All subjects satisfied the same exclusion criteria as in the first study. Each patient entered a 4-week placebo run-in phase, during which they made 3 visits to the clinic. Baseline blood pressure (BP) was taken as the value obtained at the third visit just before the commencement of antihypertensive therapy. During this run-in phase, each patient underwent a baseline echocardiographic study and a standard 12-lead ECG. After the third visit, patients began a regimen of 5 mg/d ramipril. If BP remained “uncontrolled” (>140 mm Hg systolic or >90 mm Hg diastolic) after 2 weeks, the dosage was increased to 10 mg/d. If BP remained uncontrolled, felodipine was added, initially at a dosage of 5 mg/d and titrated up to 10 mg/d, if necessary. Thereafter, if further BP lowering was required, 2.5 mg bendrofluazide was added. After BP was controlled, each patient underwent repeat echocardiography. Thereafter, patients’ BPs were reviewed monthly in the clinic. Tablet counts were performed at each visit to monitor compliance. After 6 months of BP control, an echocardiographic study and a standard 12-lead ECG were repeated. Five patients achieved BP control on ramipril alone, 25 required the addition of felodipine, and 2 required the addition of both felodipine and bendrofluazide.

The study was approved by the St Mary’s Hospital Ethics Committee, and all subjects gave informed consent.

Blood Pressure
BPs were measured at each clinic visit with an automated monitor (Sentron; CR Bard Inc) and suitable cuff size after the patient had been seated for 5 minutes. The third of 3 sitting measurements made 2 minutes apart was taken as the BP. In addition, patients underwent 24-hour ambulatory BP monitoring at baseline (Spacelabs 90207). Measurements were made every 30 minutes throughout the day (6 AM to midnight) and hourly at night. Mean BP was calculated from the readings during the entire 24-hour period. Ambulatory monitoring was deemed acceptable if >90% of readings were recorded.

Echocardiography
Two-dimensional echocardiographic studies were performed with a phased array sector scanner (General Electric Pass II, 3.3-MHz transducer; General Electric Inc) with a standard examination protocol. 16 LV septal wall thickness, posterior wall thickness, and cavity size were measured from the LV short-axis view with 2-dimensionally guided M-mode echocardiography. Particular attention was paid to obtaining a precise cross-sectional “on-axis” image of the LV at the papillary muscle tip level. The papillary muscles were then bisected by the M-mode beam, and simultaneous 2-dimensional and M-mode images were obtained. Measurements of LV septal, posterior wall, and cavity dimensions were made at end diastole. In addition, posterior wall and cavity dimensions were measured at end systole. All measurements were made according to American Society of Echocardiography guidelines. 17 Three consecutive cardiac cycles were measured, and average values were obtained.

LV mass was determined with an area×length method that has been validated in humans. 18 For this calculation, 2 echocardiographic views are required: a parasternal short-axis view of the LV at the papillary muscle tip level to assess the area of the myocardium and an apical 4-chamber view that maximizes the distance from the mitral valve annulus to the LV apex to determine the length of the ventricle. LV mass is then calculated from the algorithm

\[
LV mass = 1.05 \times \left[ \frac{5}{6}(A1 \times L1) - \frac{5}{6}(A2 \times L2) \right]
\]

where A1 and A2 represent the epicardial and endocardial areas, respectively, measured with planimetry, and L1 and L2 represent the length of the LV from the mitral annulus to epicardial and endocardial borders, respectively. LV mass index (LVMI) was determined by dividing LV mass by body surface area.

BP was measured at the end of the echocardiographic examination. All echocardiographic measurements and analyses were carried out by a single observer.

Calculations
The following parameters were derived from the M-mode diastolic measurements of the interventricular septum (IVS), LV internal diameter (LVID), and posterior wall (PWT) and the systolic measurements of LV internal diameter (LVIDs) and posterior wall (PWTs).

Relative wall thickness (RWT) gives an indication of the ratio of wall thickness to cavity size. Conventionally, this is calculated with the following formula:

\[
RWT = \frac{PWT}{LVID}
\]

Endocardial fractional shortening (FS%) is calculated as follows:

\[
FS\% = \frac{100 \times (LVID - LVIDs)}{LVID}
\]

Stroke volume was derived from diastolic and systolic LV volumes calculated with Teicholz’s formula:

\[
\text{Stroke volume} = \left[ 7 \times (LVIDd)^3 / (2.4 + LVIDd) \right] - \left[ 7 \times (LVIDs)^3 / (2.4 + LVIDs) \right]
\]

This value was multiplied by heart rate to obtain a value for cardiac output (CO). Ejection fraction (EF%) was also derived:

\[
EF\% = \frac{100 \times (LVID diastolic volume)}{LV systolic volume} / LV diastolic volume
\]

Circumferential end-systolic wall stress (cESS) at the midwall was calculated from the M-mode measurements and the systolic BP measured at the end of the echocardiographic examination: 20:
To calculate fractional shortening at the midwall (mFS%), the position of a theoretical midwall fiber in systole must be known (see the Figure). This can be calculated by using a cylindrical model of the LV with the assumption that volume remains constant through the cardiac cycle.\(^7,21\) The LV in fact is considered to be 2 cylinders of equal wall thickness (at end diastole): 1 inside the other. If we consider the inner cylinder at end diastole, its inner radius is half of the LVID. Its wall thickness is half of the wall thickness of the LV. We have assumed that IVS = PWT, and all wall measurements are based on those of the PWT. Therefore, the thickness of the inner cylinder wall is taken to be half of the PWT.

The volume of the inner cylinder at end diastole is
\[ V_{\text{end-diastole}} = \pi \times (LVID_1)^2 \times 1/2PWT \]

where \( L1 \) is the diastolic length of the cylinder.

The volume of the inner cylinder at end systole is
\[ V_{\text{end-systole}} = \pi \times (LVID_2)^2 \times 1/2PWT \]

where \( a \) is the distance from the posterior wall endocardium of the theoretical midwall fiber at end systole and \( L2 \) is the systolic length of the cylinder.

If we now consider the entire LV as a cylinder rather than the inner shell, we can derive similar equations in diastole and systole for LV volume:

1. \[ V_{\text{end-diastole}} = \pi \times (1/2LVID + 1/2PWT)^2 \times 1/2PWT \]
2. \[ V_{\text{end-systole}} = \pi \times (1/2LVID + a)^2 \times 1/2PWT \]

By dividing equation 1 by equation 2, \( \pi \) and the diastolic length are cancelled out. Because LV volume is assumed to be constant throughout the cardiac cycle, this ratio is the same as equation 2 divided by over equation 4. Again, \( \pi \) and the length, this time systolic, are cancelled out.

Therefore
\[ \frac{V_{\text{end-diastole}}}{V_{\text{end-systole}}} = \frac{(1/2LVID + 1/2PWT)^2}{(1/2LVID + a)^2} \times \frac{1/2PWT}{1/2PWT} \]

All of the other factors are known, so \( a \) can be calculated. This is the distance from the posterior wall endocardium of the theoretical midwall fiber at end systole, so the distance from the midwall of the septum to the midwall of the posterior wall at end systole is \( LVID + 2a \). The distance at end diastole from the midwall of the septum to the midwall of the posterior wall is \( LVID + PWT/2 \), so the distance at end diastole from the midwall of the posterior wall to end systole is \( LVID + 2a \). Therefore, once \( a \) is known, midwall fractional shortening can be easily calculated.

\[ \text{mFS\%} = 100 \times \frac{(1/2LVID + PWT)(1/2LVID + 2a)}{(1/2LVID + PWT)^2} \]

### Electrocardiography

All 12-lead ECGs were performed at 25 mm/s with standard lead positions. Voltage height was derived from \( SV_i + RV_i \).

### Statistical Analysis

All descriptive data are expressed as mean and SEM. Unpaired Student’s \( t \) test was used to compare the LVH group with control subjects. Repeated measures ANOVA was used to assess the significance of longitudinal changes in measured variables in the treatment study. The Tukey multiple comparison test was used to compare individual points in time. For data that were not normally distributed, the Mann-Whitney \( U \) test and the Kruskal-Wallis nonparametric ANOVA test with Dunn’s multiple comparison test were used.

### Results

The results of the initial study in which patients with LVH were compared with normotensive control subjects are displayed in Table 1. There were more men in the LVH group, and the subjects were slightly older. By design, the LVH group had a higher BP and a higher LV mass index. The mean cardiac output, ejection fraction, and endocardial fractional shortening were similar between the 2 groups, but shortening assessed at the midwall was significantly less in the LVH group. The same group had a slightly higher circumferential wall stress, but this difference was not significant. From our normal population data, the relationship between midwall fractional shortening and circumferential end-systolic stress is calculated as follows:

Midwall fractional shortening
\[ = \frac{24.0 - 0.019 \times \text{circumferential end-systolic stress}}{100} \times \text{predicted midwall FS\%} \]

When the observed midwall fractional shortening was expressed as a percentage of predicted shortening, this was 86% in the LVH group.

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TABLE 1. Patient Characteristics and Echo Results for Hypertensives With LVH and Control Subjects

<table>
<thead>
<tr>
<th></th>
<th>Hypertensives With LVH</th>
<th>Control Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, y</strong></td>
<td>46 ± 2.4†</td>
<td>40 ± 1.7</td>
</tr>
<tr>
<td><strong>Gender, M/F</strong></td>
<td>31/7</td>
<td>25/13</td>
</tr>
<tr>
<td><strong>Height, cm</strong></td>
<td>169 ± 1.4</td>
<td>176 ± 1.6</td>
</tr>
<tr>
<td><strong>Weight, kg</strong></td>
<td>78 ± 2.7</td>
<td>76 ± 1.5</td>
</tr>
<tr>
<td><strong>Body mass index, kg/m²</strong></td>
<td>26.9 ± 1.2</td>
<td>24.3 ± 0.7</td>
</tr>
<tr>
<td><strong>Systolic BP, mm Hg</strong></td>
<td>170 ± 3.7*</td>
<td>124 ± 2.3</td>
</tr>
<tr>
<td><strong>Diastolic BP, mm Hg</strong></td>
<td>100 ± 1.6*</td>
<td>77 ± 1.6</td>
</tr>
<tr>
<td><strong>Heart rate, bpm</strong></td>
<td>66 ± 1.9</td>
<td>63.2 ± 1.7</td>
</tr>
<tr>
<td><strong>ECG voltage, SVi + RVi</strong></td>
<td>39 ± 3.3*</td>
<td>27 ± 1.3</td>
</tr>
<tr>
<td><strong>IVS diastole, cm</strong></td>
<td>1.4 ± 0.04*</td>
<td>1.1 ± 0.02</td>
</tr>
<tr>
<td><strong>LVID diastole, cm</strong></td>
<td>4.7 ± 0.08</td>
<td>4.8 ± 0.08</td>
</tr>
<tr>
<td><strong>PWT diastole, cm</strong></td>
<td>1.3 ± 0.05*</td>
<td>1.0 ± 0.02</td>
</tr>
<tr>
<td><strong>LVID systole, cm</strong></td>
<td>3.1 ± 0.11</td>
<td>3.0 ± 0.07</td>
</tr>
<tr>
<td><strong>PWT systole, cm</strong></td>
<td>1.6 ± 0.05*</td>
<td>1.2 ± 0.02</td>
</tr>
<tr>
<td><strong>LVMI, g/m²</strong></td>
<td>140 ± 3.6*</td>
<td>92 ± 3.1</td>
</tr>
<tr>
<td><strong>RWT</strong></td>
<td>0.55 ± 0.02*</td>
<td>0.41 ± 0.01</td>
</tr>
<tr>
<td><strong>SBP after echo, mm Hg</strong></td>
<td>175 ± 5.0*</td>
<td>127 ± 1.8</td>
</tr>
<tr>
<td><strong>Cardiac output, L/min</strong></td>
<td>4.32 ± 0.23</td>
<td>4.55 ± 0.21</td>
</tr>
<tr>
<td><strong>Ejection fraction, %</strong></td>
<td>62.5 ± 2.00</td>
<td>66.4 ± 1.07</td>
</tr>
<tr>
<td><strong>Fractional shortening, %</strong></td>
<td>34.5 ± 1.45</td>
<td>37.0 ± 0.82</td>
</tr>
<tr>
<td><strong>Midwall fractional shortening, %</strong></td>
<td>17.9 ± 1.11*</td>
<td>21.6 ± 0.63</td>
</tr>
<tr>
<td><strong>Circumferential stress, kdynes/cm²</strong></td>
<td>166 ± 9.6</td>
<td>151 ± 6.7</td>
</tr>
<tr>
<td><strong>Observed/predicted midwall FS, %</strong></td>
<td>86 ± 5*</td>
<td>102 ± 3</td>
</tr>
</tbody>
</table>

*\( P < 0.01 \) compared with control group. †\( P < 0.05 \) compared with control group.
midwall function in hypertrophy is that because the inner layer of the ventricle thickens more than the outer layer, a thickened inner layer provides significantly more inward movement compared with the inner layer of a normal ventricle. This allows total wall shortening to remain normal despite a depression in fiber shortening (ie, the change in LV geometry allows the chamber function to remain normal). The cylindrical model used predicts that the inner half of the LV contributes about two thirds of the total shortening. This is consistent with experimental data.

The primary aim of this analysis was to assess the effects of antihypertensive therapy on midwall function as it regresses LVH. Significant regression of LVH occurred with good BP control, and this regression was associated with a significant improvement in midwall shortening. In parallel with this, endocardial measurements of LV systolic function also improved but not significantly. It is important when cardiac systolic function is assessed that this is conducted in parallel with afterload, because this can influence shortening independently of myocardial factors. In the present study, cardiac afterload was considered through assessment of circumferential end-systolic wall stress. The normal relationship between midwall shortening and circumferential wall stress was examined in 156 normal subjects, and in the application of these data to our patients, the small reduction in wall stress that was observed (which was not statistically significant) would not have had an important impact on shortening. In our smaller normal population, the relationship between midwall fractional shortening and circumferential end-systolic wall stress was similar to that of the larger study. When stress-shortening relationships were directly examined by expressing the observed midwall fractional shortening as a percentage of predicted shortening derived from the circumferential end-systolic stress data, the observed shortening had improved to predicted levels by the end of the study. This is consistent with the assumption that the small, nonsignificant differences in circumferential wall stress at the beginning and end of the study were not important in the interpretation of changes in midwall fractional shortening.

It is somewhat surprising that an improvement in midwall fractional shortening occurred without a significant improvement in relative wall thickness; however, endocardially measured systolic function also improved, and although this change was not statistically significant, it may explain this discrepancy. In animal studies, ACE inhibitors have been shown to reduce the increased interstitial fibrosis that occurs in hypertensive LVH as well as to reduce myocyte hypertrophy.

If this fibrosis were implicated in the reduction in midwall shortening and it were improved with ACE inhibitor treatment, this would provide a potential mechanism for some improvement in systolic function independent of geometric changes.

The observed improvement in midwall shortening is an important finding. A reduced midwall shortening has been shown to be associated with a lower exercise performance, and although a parallel improvement is not necessarily implied, this is clearly a possibility. Furthermore, depressed midwall shortening has been shown to be an independent predictor of an adverse outcome in hypertensive subjects.

### TABLE 2. Treatment Study: Hypertensives With LVH

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>BP Control</th>
<th>6 mo After BP Control</th>
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</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>50±2</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Gender, M/F</td>
<td>28/4</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Height, cm</td>
<td>170±1.5</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>76±1.8</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>26.3±0.6</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>174±4.1</td>
<td>138±1.8*</td>
<td>143±2.6*</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>103±1.9</td>
<td>84±1.2*</td>
<td>86±1.4*</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>66±2.0</td>
<td>66±2.1</td>
<td>65±1.9</td>
</tr>
<tr>
<td>ECG voltage, SV₁+RV₁</td>
<td>39±2.4</td>
<td>...</td>
<td>36±2.4</td>
</tr>
<tr>
<td>IVS diastole, cm</td>
<td>1.48±0.03</td>
<td>1.41±0.03</td>
<td>1.35±0.03*</td>
</tr>
<tr>
<td>LVID diastole, cm</td>
<td>4.71±0.10</td>
<td>4.61±0.07</td>
<td>4.64±0.08</td>
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<tr>
<td>PWT diastole, cm</td>
<td>1.30±0.03</td>
<td>1.28±0.02</td>
<td>1.26±0.02</td>
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<tr>
<td>LVID systole, cm</td>
<td>2.98±0.12</td>
<td>2.88±0.08</td>
<td>2.82±0.07</td>
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<tr>
<td>PWT systole, cm</td>
<td>1.62±0.04</td>
<td>1.54±0.05</td>
<td>1.51±0.04</td>
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<tr>
<td>LVMI, g/m²</td>
<td>145±3.8</td>
<td>133±3.5</td>
<td>124±3.4*</td>
</tr>
<tr>
<td>RWT</td>
<td>0.56±0.02</td>
<td>0.56±0.02</td>
<td>0.55±0.02</td>
</tr>
<tr>
<td>SBP after echo, mm Hg</td>
<td>174±4.5</td>
<td>146±3.3*</td>
<td>148±2.4*</td>
</tr>
<tr>
<td>Cardiac output, L/min</td>
<td>4.48±0.26</td>
<td>4.37±0.27</td>
<td>4.34±0.19</td>
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<tr>
<td>Ejection fraction, %</td>
<td>65.6±2.22</td>
<td>67.0±1.81</td>
<td>69.3±1.66</td>
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<tr>
<td>Fractional shortening, %</td>
<td>37.0±1.69</td>
<td>38.0±1.4</td>
<td>39.5±0.13</td>
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<tr>
<td>Midwall fractional shortening, %</td>
<td>18.7±1.19</td>
<td>20.0±0.68</td>
<td>21.9±0.84†</td>
</tr>
<tr>
<td>Circumferential stress, kdynes/cm²</td>
<td>152±9.7</td>
<td>131±8.1</td>
<td>130±7.3</td>
</tr>
<tr>
<td>Observed/predicted midwall FS, %</td>
<td>88±5</td>
<td>93±3</td>
<td>101±4</td>
</tr>
</tbody>
</table>

*P<0.01 compared with baseline. †P<0.05 compared with baseline.

The results of the treatment study are displayed in Table 2. The group consisted of 28 men and 4 women whose mean age was 50±2.0 years. Good BP control was achieved with significant regression of LV mass index. LV fractional shortening assessed at the midwall improved with regression of LVH, and posttreatment midwall shortening was similar to that of the normal control subjects in the first study. When the observed midwall fractional shortening was expressed as a percentage of predicted shortening, this improved from 88% to 93% to 101% at the end of the study. The wide confidence intervals meant that this improvement did not reach statistical significance.

### Discussion

The results of the first study suggest that in patients with pressure overload, hypertrophy indices of LV chamber function may be normal while there is depressed midwall shortening. This is not due to the increased afterload; when stress-shortening relationships were taken into account by comparing actual and predicted midwall shortening, actual midwall shortening was still significantly depressed. These results confirm previous observations and provide a link between experimental data that suggest reduced systolic function in hypertrophy and studies in humans that indicate chamber function is normal. A possible explanation for normal LV chamber function in the presence of depressed...
particularly in subjects with additional LVH. There is evidence that improvements in LVH are related to an improvement in subsequent prognosis in hypertensive subjects, and the demonstration that midwall shortening can also be improved provides a further potential goal for antihypertensive treatment.

In summary, hypertensive patients with LVH have depressed midwall systolic shortening despite normal indices of LV chamber function. Regression of LVH after good BP control improved midwall shortening to normal levels.

Acknowledgments
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References
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