Left ventricular chamber filling and midwall fiber lengthening in patients with left ventricular hypertrophy: overestimation of fiber velocities by conventional midwall measurements

Gen Shimizu, M.D., Michael R. Zile, M.D., Alvin S. Blaustein, M.D., and William H. Gaasch, M.D.

ABSTRACT Observations that the inner (subendocardial) half of the left ventricular wall contributes more to total left ventricular wall thickening than the outer (subepicardial) half may have important implications in the analysis of myocardial fiber length transients. Accordingly, we measured endocardial and midwall shortening and lengthening rates in normal and hypertrophic hearts and compared the results obtained with conventional methods of measurement with those obtained with a modified model that does not depend on use of conventional assumptions about the midwall. This modified (two-shell cylindrical model) method considers the substantial contribution of inner wall thickening and thus does not require the assumption of a theoretical midwall fiber that remains at the midwall throughout the cardiac cycle. Echocardiographic data from six normal subjects and six patients with concentric left ventricular hypertrophy (LVH) were examined; left ventricular wall thickness ranged from 8 to 10 mm in normal subjects and from 11 to 16 mm in the patients with LVH. By design, the standard measurements of left ventricular size (diastolic and systolic dimensions) and systolic function (fractional shortening and endocardial fiber shortening velocities) were equal in the two groups. Endocardial, conventional midwall, and modified midwall methods all indicate reduced fiber lengthening rates in patients with LVH; peak fiber lengthening rates for normal and LVH groups were 4.5 ± 0.7 vs 3.1 ± 0.8 sec⁻¹ (p < .02) at the endocardium, 2.3 ± 0.4 vs 1.6 ± 0.4 sec⁻¹ (p < .02) at the midwall (conventional method), and 2.1 ± 0.3 vs 1.4 ± 0.3 sec⁻¹ (p < .01) at the midwall (modified method). Chamber filling and fiber lengthening rates are depressed in patients with concentric LVH. Conventional measurements overestimate “true” fiber velocities more in those with LVH than in normal subjects; thus, the modified midwall method offers a theoretical advantage, especially in studies in which data from normal and hypertrophic hearts are compared.


INCREASED chamber stiffness in patients with left ventricular pressure overload hypertrophy has been largely attributed to increased myocardial mass; however, it is generally accepted that increased intrinsic myocardial stiffness and/or abnormal myocardial relaxation may also contribute to the abnormal diastolic properties of hypertrophic hearts.¹-⁹ Easily discernible alterations in left ventricular relaxation indexes have been observed in patients with left ventricular hypertrophy (LVH) (despite normal systolic performance) and it has been suggested that slow, delayed, or prolonged relaxation might be an early marker of left ventricular disease.¹⁰-¹⁵ Much of the published work in this area incorporates measurement of endocardial length (or volume) transients that reflect filling properties of the left ventricular chamber, not necessarily the lengthening properties of an average (i.e., midwall) unit of myocardium. In this report, chamber filling and midwall fiber lengthening will be assessed in normal and hypertrophic hearts and attempts will be made to refine the conventional method of determining midwall length transients.

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Experimental data indicate that myocardial shortening in subendocardial layers exceeds that seen in the outer (or subepicardial) layers. As a result, a theoretical midwall point (or end-diastolic midwall circumferential fiber) shows a relative migration toward the epicardium throughout contraction; the fiber then must move inward toward the endocardium during the filling phase of the cardiac cycle. This notion is directly supported by the observation that systolic thickening of the inner (subendocardial) half of the left ventricular wall exceeds that of the outer (subepicardial) half. A conventional and widely used assumption is that inner and outer wall thickening are equal and that a theoretical midwall fiber maintains its relative midwall position throughout the cardiac cycle. This assumption results in an overestimation of midwall length transients that is due to a transmural nonuniformity of thickening and thinning. For these reasons we developed a modified midwall model that allows expression of the relationship between left ventricular radius and wall thickness throughout the cardiac cycle; this two-shell cylindrical model does not require assumption of a theoretical midwall fiber that remains at the midwall throughout the cardiac cycle. The modified method was used to determine midwall length transients and in this fashion we compared and assessed endocardial filling and midwall fiber lengthening rates in normal and hypertrophic hearts.

Methods

Model. The left ventricle is represented by a cylindrical model with uniform wall thickness. The cylinder contracts radially and longitudinally, and the wall volume (myocardial mass) is assumed to be constant throughout the cardiac cycle. Major advantages of the cylindrical model are that the calculated midwall parameters are independent of the length of the cylinder and that only measurements of left ventricular radius and wall thickness are necessary. Using echocardiographic measurements of left ventricular chamber diameter and wall thickness, we calculated the standard indexes of left ventricular size and function (endocardial and conventional midwall measurements). However, use of the conventional method requires the assumption that a theoretical midwall circumferential fiber maintains its relative midwall position throughout the cardiac cycle. We therefore also used a modified midwall method that does not require this assumption (see Appendix).

Clinical material. Echocardiograms from 12 subjects were selected for study; six were from patients with echocardiographic evidence of LVH (left ventricular wall thickness ≥11 mm) while six were from subjects with no echocardiographic or other evidence of heart disease. The basis for selection of the six patients with hypertrophy included normal left ventricular chamber size (normal left ventricular dimension at end-diastole and end-systole) and normal systolic function (normal extent and velocity of endocardial circumferential fiber shortening). Thus, by standard echocardiographic criteria, the only difference between the two groups was increased wall thickness in the hypertrophy group (table 1).

Echocardiography. M mode echocardiographic recordings of the left ventricle were obtained with a two-dimensional ultrasoundoscope (two-dimensionally directed M mode) and a 2.25 MHz transducer (Toshiba, model SSH-10A). The echocardiograms were processed by a semiautomated technique similar to that used in previous studies. Records placed on a digitizing tablet (Hipad, Houston Instruments) were manually traced with a cursor, the position of which was detected and converted to digital coordinates for processing by a microcomputer system (Apple Ile, Apple Computer Co., Ltd.). Left ventricular dimension and wall thickness (posterior wall and interventricular septum) were digitized with a sampling interval of 20 msec. Data were smoothed with use of a moving five-point third-order least square polynomial fit. The derivative (dD/dt) was obtained from the smoothed data, normalized by the instantaneous dimension (D), and expressed as circumferential fiber velocity (VCF). Peak VCF was derived as the maximum value (+) for (dD/dt)/D and expressed in inverse seconds. For the endocardial velocity D = left ventricular internal dimension, for the conventional midwall velocity D = left ventricular internal dimension + h/2, where h = left ventricular wall thickness; for the modified midwall velocity D = left ventricular dimension + a', where a' is the time varying thickness of the inner shell (see Appendix).

Data are presented as mean ± SD. Paired and unpaired t tests were used as appropriate and differences were considered significant if the value was less than .05.

Results

To validate the model and to test the hypothesis that modified midwall measurements more closely approximated true midwall fiber transients, we first compared the results from conventional and modified methods with wall thickening data obtained directly from the dog heart. This comparison is presented graphically in figure 1, which shows the contribution of inner and outer left ventricular thickening to total left ventricular wall thickening. In our normal group there was a 39% increase in left ventricular wall thickness from end-diastole to end-systole (9.0 ± 0.8 mm at end-diastole to 12.7 ± 0.5 mm at end-systole). The conventional assumption is that the inner and outer halves of the wall contribute equally to systolic thickening, but the modified midwall method indicated that 63% of the total thickening occurred in the inner wall while only 37% occurred in the outer wall. In the patients with LVH the fractional increase in wall thickness (30%) was less than normal; wall thickness increased from 13.3 ± 1.8 mm at end-diastole to 17.5 ± 1.9 mm at end-systole. As was true in the normal group, the inner and outer walls contributed 65% and 35%, respectively. Direct measurements of inner and outer wall thickening in the normal dog heart are shown for comparison; the data from the animal studies are in agreement with the results derived by the modified midwall method.

By design, the standard echocardiographic indexes of left ventricular size and function were essentially the same in both groups of patients (table 1); left ventricular wall thickness in the group of patients with LVH...
Modified midwall fiber location by endocardial graphic parameters)

was substantially thicker than normal (p < .001). Shortening and lengthening rates for the two groups are shown in table 2 and figures 2 and 3. Peak (endocardial) shortening rate was nearly equal in the two groups. According to the conventional assumption, peak (-)VCF at the midwall was slightly lower in the patients with hypertrophy, but this difference did not achieve statistical significance. In contrast, use of the modified midwall method resulted in a significantly (p < .05) lower fiber shortening rate in the group with LVH. All three methods reflect low (+)VCF in the group with LVH. Thus, patients with increased left ventricular wall thickness (and otherwise normal left ventricular size and function by standard echocardiographic parameters) exhibited reduced shortening velocity at the midwall (modified) that was not apparent by endocardial or conventional measurements; there was also a tendency for modified midwall lengthening rates to show a greater change than those derived from endocardial measurements.

The absolute difference between conventional and modified midwall fiber location (i.e., the migration or shift of a theoretical midwall fiber during the cardiac cycle) was directly related to end-diastolic wall thickness (p < .02). This migration can amount to approximately 4% to 8% of the wall thickness. Thus, the effect of nonuniform wall thickening was greatest in hypertrophic hearts. Peak normalized lengthening rates are plotted against end-diastolic wall thickness in figure 4. These data indicate a strong inverse relationship between left ventricular wall thickness and fiber lengthening. This inverse relationship was highly significant (p < .001) for the normalized VCF data shown in figure 4; the relationship between wall thickness and non-normalized velocity (peak dD/dt) was equally significant (p < .001). Clearly abnormal lengthening rates were seen in the presence of only minimally increased wall thickness (i.e., >12 mm).

**Discussion**

In recent years our understanding of the pathophysiology of heart failure has evolved to the point where important differences between systolic and diastolic failure have been identified. In many patients, the signs and symptoms of pulmonary and/or systemic venous congestion are directly related to alterations in the diastolic properties of the left ventricular chamber; they are not necessarily a consequence of systolic dysfunction.\(^{(23)}\) Thus, congestive failure can be considered a diastolic disorder while systolic failure is a defect in contraction and systolic performance. The factors responsible for diastolic dysfunction have been outlined in a number of review articles\(^{(1-6)}\) and include abnormalities of left ventricular volume and mass (i.e., concentric or eccentric hypertrophy), altered intrinsic myocardial stiffness (i.e., fibroses or infiltrative disease), and delayed or prolonged left ventricular relaxation (i.e., ischemia or conduction disorders).

Relaxation, which refers to the process by which the myocardium returns to its initial tension and length, is influenced by a complex interaction between deactivation (the decay of active force generating capacity) and

**TABLE 1**
Echocardiographic data from normal and hypertrophied hearts

<table>
<thead>
<tr>
<th></th>
<th>D(_{end}) (mm)</th>
<th>D(_{es}) (mm)</th>
<th>Th(_{end}) (mm)</th>
<th>FS (%)</th>
<th>Peak endocardial (-)V(_{CF}) (sec(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal subjects (n = 6)</td>
<td>46 ± 2</td>
<td>30 ± 3</td>
<td>9.0 ± 0.8</td>
<td>35 ± 3</td>
<td>2.5 ± 0.4</td>
</tr>
<tr>
<td>Patients with LVH (n = 6)</td>
<td>46 ± 5</td>
<td>29 ± 4</td>
<td>13.3 ± 1.8</td>
<td>38 ± 5</td>
<td>2.4 ± 0.4</td>
</tr>
<tr>
<td>p value</td>
<td>NS</td>
<td>NS</td>
<td>&lt;.001</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

D\(_{end}\) = end-diastolic dimension; D\(_{es}\) = end systolic dimension; Th\(_{end}\) = end-diastolic wall thickness; FS = fractional shortening.
TABLE 2
Left ventricular peak ejection and filling rates in normal and hypertrophied hearts

<table>
<thead>
<tr>
<th></th>
<th>Peak (-) ( V_{CF} ) (sec(^{-1}))</th>
<th>Peak (+) ( V_{CF} ) (sec(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Endocardial Midwall (C) Midwall (M)</td>
<td>Endocardial Midwall (C) Midwall (M)</td>
</tr>
<tr>
<td>Normal subjects</td>
<td>2.5±0.4 1.5±0.2 1.4±0.2</td>
<td>4.5±0.7 2.3±0.4 2.1±0.3</td>
</tr>
<tr>
<td>Patients with LVH</td>
<td>2.4±0.4 1.2±0.3 1.1±0.2</td>
<td>3.1±0.8 1.6±0.4 1.4±0.3</td>
</tr>
<tr>
<td>p value</td>
<td>NS NS &lt;.05</td>
<td>&lt;.02 &lt;.02 &lt;.01</td>
</tr>
</tbody>
</table>

Midwall (C) = conventional midwall measurements; Midwall (M) = modified midwall measurements.

loading conditions (forces that affect length). Attempts have been made to quantitate changes in left ventricular relaxation by examining the rate of tension decline during the isovolumetric relaxation period and/or by measuring the rate of fiber lengthening during the early diastolic filling period. Unfortunately, there are no available data on which to judge the relative merits of the ‘‘isovolumetric’’ and ‘‘filling’’ indexes of relaxation; however, after a period of interest in the isovolumetric relaxation period, many investigators are currently using measurements of change in volume or dimension during the diastolic filling period. The popularity of the filling phase indexes of relaxation is due in part to the ease with which these parameters may be derived with noninvasive echocardiographic or nuclear techniques.

The important differences between the diastolic properties of the left ventricular chamber (chamber stiffness) and the passive physical properties of the myocardium (myocardial stiffness) have been considered in previous publications. These articles emphasize chamber stiffness and myocardial stiffness, but we have not previously distinguished between the filling properties of the chamber and the lengthening properties of a midwall unit of myocardium. In the present study we considered indexes of chamber filling (endo-cardial measurements) and midwall fiber lengthening.

In the latter analysis we used the conventional assumption that a theoretical end-diastolic midwall fiber maintains a constant relative midwall position (it remains at the midwall throughout the cardiac cycle); we also used a modified midwall analysis that does not rely on this assumption.

In hypertensive patients, diastolic filling abnormalities have been described in the absence of systolic dysfunction. Thus, it has been suggested that filling abnormalities may be the earliest clinically detectable signs of LVH. A primary purpose of the present article was to suggest a refinement in the methods of assessing left ventricular filling, not merely to confirm the observation that ventricular filling is abnormal in cardiac hypertrophy. Accordingly, we developed a simple mathematical model of the left ventricle that allows comparison of chamber (endocardial) filling rates and myocardial (midwall) lengthening rates. It quickly became apparent that the conventional midwall assumption was not in keeping with the observation that endocardial shortening exceeds epicardial

![FIGURE 2](image_url)

**FIGURE 2.** Peak circumferential fiber shortening velocity in normal (N) and hypertrophic (LVH) hearts. There was no significant difference (NS) in endocardial or convention (C) midwall measurements of fiber shortening velocity in the normal and hypertrophic groups. The modified (M) midwall analysis reveals a significantly lower shortening rate in patients with LVH.

![FIGURE 3](image_url)

**FIGURE 3.** Peak circumferential fiber lengthening velocity in normal (N) and hypertrophic (LVH) hearts. Endocardial measurements clearly overestimated those made at the midwall, but results with all three methods indicated that peak fiber lengthening rate was lower than normal in patients with LVH. The level of significance (N vs LVH) was higher for the modified (M) than for the conventional (C) midwall analysis.
shortening and that it was not consonant with the recently published observation that inner wall (subendocardial) thickening exceeds outer wall (subepicardial) thickening.

As is shown in figure 1, the contribution of the inner half of the left ventricular wall to total wall thickening exceeds the contribution of the outer half. Also, the results from our cylindrical model are in agreement with detailed measurements in experimental animals. Based on these supporting data, we then used the model to assess differences in left ventricular chamber filling and midwall fiber lengthening in normal and hypertrophic hearts. Abnormal diastolic length transients were found at the endocardium and midwall. While there was a tendency for the modified midwall measurements to show the most significant differences between normal and hypertrophy, the differences were quite small. However, as is shown in figure 3, muscle lengthening rates derived from the conventional midwall assumption consistently exceeded those found with the modified midwall method.

Others have defined and described abnormal diastolic filling in patients with LVH and some have examined the inverse relationship between filling rate and left ventricular wall thickness or mass. The data shown in figure 4 reflect this inverse relationship. As left ventricular wall thickness approaches 18 mm, peak filling rate and/or midwall fiber lengthening rate are very low. Thus, in the presence of concentric LVH, chamber filling and muscle lengthening are likely to be abnormal unless some compensating mechanism (i.e., increased left atrial pressure) alters the relationship. Whether abnormal filling or lengthening in LVH is the result of abnormal function of an intrinsic cardiac relaxing system (sarcoplasmic reticulum) or of some other factor(s) remains to be determined.

An improved understanding of left ventricular systolic function might also result from the use of modified midwall measurements. For example, Quinones et al. calculated left ventricular stress-velocity relationships from endocardial and conventional midwall measurements of radius and wall thickness. In that study, midwall VC was approximately 60% of endocardial VC during short-term pharmacologic interventions, but it was suggested that “midwall VC does not offer advantage over endocardial VC.” In the current study, midwall shortening rate was also substantially less than endocardial shortening in both the normal and hypertrophied hearts. Although the conventional midwall shortening rate was also similar in the normal and LVH groups, the modified midwall measurements reflect a significantly reduced shortening rate in the patients with LVH. It therefore appears that the modified midwall method may identify shortening differences that are not evident at the endocardium. Thus, despite the previous conclusion, it is likely that the use of the modified midwall approach does offer an advantage over use of endocardial VC, especially in studies in which normal and hypertrophic hearts are compared.

In a study of ventricular function in patients with pressure overload hypertrophy, Gunther and Grossman assessed stress-shortening relationships by plotting endocardial shortening (ejection fraction) and midwall shortening (VC) against systolic wall stress. They emphasized a very important concept, namely the inverse relationship between stress and shortening. However, some of the patients with pressure overload hypertrophy and normal endocardial shortening had values for midwall VC that were decreased to levels found in patients with cardiomyopathy (some patients with near-normal systolic stress and normal ejection fractions had depressed VC). The use of the modified midwall method in these thick-walled ventricles would tend to exaggerate this difference (midwall VC would be reduced further) and it is likely that at least some of them would have had depressed VC that was clearly out of proportion to the level of wall stress. Since systolic shortening coordinates (modified method) may be lower than those calculated from the conventional midwall assumption, and since the difference is greater in hypertrophic than in normal hearts, it is possible that the use of modified midwall measurements will provide a more accurate assessment of left ventricular stress-shortening relationship.

Chronic pressure overload of the left ventricular results in an appropriate increase in myocardial mass.
(concentric hypertrophy) that is characterized by a low chamber radius/wall thickness (R/Th ratio). In contrast, the increase in myocardial mass that accompanies chronic volume overload (eccentric hypertrophy) is characterized by a normal or only minimally increased R/Th; as long as the ventricle remains compensated, the increase in thickness is proportional to the increase in radius and the R/Th remains normal. Krayenbuehl et al. developed the theory that (conventional) midwall shortening was less than normal in patients with concentric LVH and greater than normal in those with eccentric LVH. In Krayenbuehl’s analysis, endocardial (ejection fraction) shortening was equal in normal subjects and patients with concentric and eccentric hypertrophy. Based on the effect of non-uniform wall thickening, the results of Krayenbuehl appear to overestimate the extent of midwall shortening (or lengthening) in patients with concentric hypertrophy, and they may also overestimate shortening (or lengthening) in those with eccentric hypertrophy. Thus, use of the modified midwall model may offer an advantage not only in those with concentric, but also in those with eccentric LVH. These speculations, however, must be critically assessed with experimental data before they are directly applied in studies of patients with eccentric hypertrophy.

A cylindrical model of the left ventricle is obviously an oversimplified representation of a very geometrically complex structure. The simplicity and convenience of this model are appealing to the echocardiographer because the model essentially represents the routine short-axis (two-dimensional echocardiographic) view of the ventricle. Moreover, measurements or assumptions regarding the long axis are not necessary with a cylindrical model (Appendix). However, the potential limitations of these methods should be discussed. It is well known that small measurement errors can make substantial differences in calculated values for left ventricular mass and the R/Th ratio, as well as for midwall radius. This problem can be attenuated by careful attention to detail, the use of high-quality echocardiograms, and perhaps by use of the average (planimetric) radius and wall thickness derived from a two-dimensional short-axis (area) view rather than use of a single radius and thickness as was used in the present study. A second potential limitation of the modified midwall analysis is a lack of information regarding the factors that might influence the patterns of nonuniform wall thickening. For example, subendocardial ischemia or fibrosis might influence subendocardial thickening in hypertrophic hearts and the fractional contribution of inner wall thickening might therefore differ in normal and diseased hearts. Acute or chronic alterations in inotropic state and/or loading conditions might also influence the ratio of inner to outer wall thickening. Moreover, wall thickness (and thickening) varies from left ventricular apex to base and around the circumference and these nonuniformities limit any attempt to assess inner and outer wall thickening. Thus, although the modified midwall analysis has considerable theoretical appeal and despite the fact that there was close agreement between our clinical data and the published experimental data, it would seem that additional experimental work and validation are necessary before these concepts can be applied widely.

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Appendix

The geometric structure of the left ventricle is approximated by a cylindrical model with left ventricular (LV) wall volume (or muscle mass) represented by the equation

\[ \text{LV wall volume} = \pi \frac{L}{4} (R + h)^2 - R^2 \]  \quad (1)

where \( L \) is the long axis, \( R \) is the internal radius, and \( h \) is the wall thickness (at end-diastole). Assuming an incompressible and homogeneous myocardium with constant specific gravity, left ventricular mass (which is therefore constant throughout the cardiac cycle) can be represented at any instant in the cycle by the equation

\[ \text{LV wall volume} = \pi L' \left( \frac{R'}{2} + h' \right)^2 - R'^2 \] \quad (2)

where \( L', R', h' \) are the dimensions at a specific time in the cardiac cycle.

The left ventricular wall is divided into inner and outer (concentric) shells with thicknesses of \( a + b \), respectively; \( a + b = h \) and at end-diastole is \( R + h/2 \) or \( R + a \). The volume of each shell remains constant throughout the cardiac cycle and “midwall” radius can be represented at any instant in the cycle by \( R' + a' \).

The total left ventricular volume at end-diastole (equation 1) equals the left ventricular wall volume at any instant in the cardiac cycle (equation 2).

\[ \pi \frac{L}{4} (R + h)^2 - R^2 = \pi L' \left( \frac{R'}{2} + h' \right)^2 - R'^2 \] \quad (3)

Likewise, the volume of the inner shell at end-diastole equals the volume of the inner shell at any instant in the cardiac cycle.

\[ \pi L \left( R + a^2 - R' \right) = \pi L' \left( R' + a' \right)^2 - R'^2 \] \quad (4)

By dividing the result of equation 4 by that of equation 3, one can solve for the unknown parameter (\( a' \)): note that the long-axis parameter \( L \) and \( \pi \) (\( \pi \)) cancel.

\[ \frac{\pi L \left( R + a^2 - R' \right)}{\pi L \left( R + h^2 - R'^2 \right)} = \frac{\pi L' \left( R' + a' \right)^2 - R'^2}{\pi L' \left( R' + h'^2 \right)^2 - R'^2} \] \quad (5)

Since the value of \( R, h, a, R' \), and \( h' \) are known, the value for \( a' \) can be derived. This parameter is the time varying thickness of the inner shell; the ratio \( a'/h' \) is the instantaneous shifting ratio from the original end-diastolic midpoint of the left ventricular wall. Therefore, the “true” location of the original (end-diastolic) midpoint can be predicted at any instant in the cardiac cycle.
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