Stroke Volume Generation of the Left Ventricle and Its Relation to Chamber Shape in Normal Subjects and Patients With Mitral or Aortic Regurgitation

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The total stroke volume of the left ventricle (LV) is equal to the sum of the regional cavity shrinkage. Since nonuniformity of regional wall motion in LV has been well documented even in normal subjects, the extent of the contribution of each region to total stroke volume cannot be easily determined. To assess the left ventricular regional contributions to total stroke volume under normal conditions and in compensated chronic mitral or aortic regurgitation, LV cineangiograms were analyzed in 14 normal subjects (N), 8 patients with mitral regurgitation (MR) and 10 patients with aortic regurgitation (AR). We assumed that the LV cavity could be viewed as a stack of 30 half-cylindrical discs, 15 in the anterior and 15 in the inferior wall regions. LV chamber shape was more spherical in MR than in N, but was more conical in AR. Percent regional hemichordal shortening was significantly decreased in the anterobasal and anteroapical walls in AR, but was similar between N and MR. The regional contribution to total stroke volume showed a significant quadratic correlation with the end-diastolic regional shape index (N, r = 0.87; MR, r = 0.79; AR, r = 0.90), which was defined as the regional hemiaxial length divided by the LV long-axis length, but was not correlated with percent regional hemichordal shortening. Therefore, stroke volume is generated mainly in the mid-ventricular portion in N and MR, but in the basal portion in AR due to the characteristic change in cavity shape.

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INVESTIGATORS have recently stressed the importance of studying the structure, stress, shape, energetics and biochemistry of the left ventricular wall, particularly in the analysis or detection of wall motion abnormality. However, other than brief comments, little attention has been paid to the question of how each of the regional walls of the left ventricle contributes to the production of total stroke volume.

The total stroke volume of the left ventricle can be described as the sum of each of the regional shrinkages of the left ventricular cavity. Each of the regional shrinkages is equal to (regional end-diastolic volume × regional ejection fraction)/100. The distribution of regional end-diastolic volume is determined by left ventricular end-diastolic chamber shape, and regional ejection fraction reflects the degree of regional wall motion. Thus, stroke volume generation of the left ventricle is related not only to the

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distribution of regional wall motion but also to the chamber shape. Since nonuniformity of regional wall motion in the left ventricle has been documented even in normal subjects\(^2\)\textsuperscript{8} in that the apical region contracts more than other regions despite its smaller regional cavity volume, the extent of the contribution of each region to total stroke volume is not easily determined.

Therefore, in the present study, we used left ventricular cineangiography in an attempt to determine how total stroke volume is generated from cooperative regional wall motion in the left ventricle in normal subjects and in patients with mitral or aortic regurgitation, in whom larger stroke volumes must be generated.

MATERIALS AND METHODS

Patient Selection

Fourteen normal subjects (8 men and 6 women, aged 49±10 years [mean±SD]), 8 patients (3 men and 5 women, aged 35±11 years) with chronic isolated mitral regurgitation, and 10 patients (6 men and 5 women, aged 49±11 years) with chronic isolated aortic regurgitation were retrospectively selected from routine cardiac catheterization studies performed at Yamaguchi University Hospital. Patients with poor contrast opacification or excessive ventricular premature beats were excluded. The selection criteria were as follows:

1) Normal Subjects (N)

These subjects had undergone cardiac catheterization studies for evaluation of chest pain, but were found to have normal hemodynamics, coronary angiograms and left ventriculograms.

2) Mitral regurgitation (MR) and Aortic regurgitation (AR)

All 18 patients had evidence of chronic grade 3–4 isolated valvular regurgitation on left ventriculograms (MR patients) or on aortic root angiograms (AR patients). Examination of the clinical profiles revealed that the valvular lesions of these patients were primary, and that 7 of them were related to rheumatic fever. At the time of cardiac catheterization, all of these patients were well compensated at rest, and were considered to be in functional class 1–2 with a normal ejection fraction (50 percent or greater). Left ventricular end-diastolic volume index and total stroke volume index were matched for the MR and AR group; the values of these indices in these two groups were significantly greater (approximately twice as large) than those in the N group. Ejection fraction was matched for the three groups.

Patients with coronary artery disease, critical arrhythmias, such as atrial fibrillation, systemic or pulmonary hypertension other than systolic arterial hypertension in patients with aortic regurgitation, or idiopathic left ventricular wall hypertrophy, were excluded.

Catheterization Procedure

All of the patients gave their informed consent. The protocol was approved by the Research Protocol Committee at Yamaguchi University.

Patients had been premedicated with 10 mg diazepam intramuscularly and were studied in the rested, fasting state with the use of local anesthesia. Drugs that had been administered prior to cardiac catheterization were withdrawn 48–72 h before the study. After routine pressure recording by right and left catheterization, biplane left ventriculography was performed with a SIEMENS Cardoskop-U system in the 30 degree right anterior oblique and 60 degree left anterior oblique projections using an 8-Fr micromanometer-tipped catheter (Mikrotip, Millar Instrument) with a side hole for angiography from the brachial approach. The 60 degree left anterior oblique projection was used to verify the appropriateness of the projection of the left ventricle in the 30 degree right anterior oblique view. Urografin-76\textsuperscript{6} was injected at a flow rate of 13 ml/sec over 3 sec with an Angiomat 3000 injector (Viamonte /Hobbs) through the side hole. Cinefilm was exposed at a rate of 60 frames/sec. At the same time, electrocardiograms, phonocardiograms, left ventricular pressure and its first derivatives, and cine frame markers were recorded simultaneously on an Electronics for Medicine VR-12 recorder as guides for determining end-diastolic or end-systolic frames. An aortogram was also obtained under the same conditions when it was necessary to determine the grade of aortic regurgitation. Selective coronary arteriography was performed after left ventricu-
TABLE I  HEMODYNAMIC AND VENTRICULAROGRAPHIC PROFILES IN THE THREE GROUPS

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>MR</th>
<th>AR</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (beats.min⁻¹)</td>
<td>14</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>mPAP (mmHg)</td>
<td>12±2</td>
<td>14±5</td>
<td>14±4</td>
</tr>
<tr>
<td>LVSP (mmHg)</td>
<td>126±17</td>
<td>116±19</td>
<td>154±37*</td>
</tr>
<tr>
<td>LVEDP (mmHg)</td>
<td>8±2</td>
<td>11±3*</td>
<td>17±8*</td>
</tr>
<tr>
<td>LVESV (ml/m²)</td>
<td>78±12</td>
<td>145±67**</td>
<td>143±55**</td>
</tr>
<tr>
<td>SI (ml/m²)</td>
<td>31±8</td>
<td>61±27**</td>
<td>63±32**</td>
</tr>
<tr>
<td>EF (%)</td>
<td>61±7</td>
<td>57±6</td>
<td>58±9</td>
</tr>
<tr>
<td>%LS</td>
<td>17.0±5.5</td>
<td>16.5±4.7</td>
<td>15.1±5.1</td>
</tr>
</tbody>
</table>

Values are the mean ± SD. N, normal control; MR, mitral regurgitation; AR, aortic regurgitation; n, number of patients in each group; HR, heart rate; mPAP, mean pulmonary arterial pressure; LVSP, left ventricular peak systolic pressure; LVEDP, left ventricular end-diastolic pressure; LVESV, left ventricular end-diastolic pressure volume index; LVESVI, left ventricular end-systolic volume index; SI, stroke index; EF, ejection fraction; %LS, percent long-axis shortening. Values of LVEDV, LVESV, SI and EF were obtained using the conventional area-length method. *: p<0.05 vs N, **: p<0.01 vs N.

Ventriculographic Data Acquisition

The end-diastolic and end-systolic frames as viewed on a cine projector with single-frame capability (Vanguard XR-15) were determined by visual inspection as those with maximal and minimal areas, respectively, using the electrocardiogram, phonocardio-

gram, and left ventricular pressure and its first derivatives as guides. The outer margin was defined so as to include all opacified areas between trabeculations. These frames were manually traced onto sheets of paper using the projector, and were digitized into a computer (PDP11/24) for subsequent analysis.

Data Analyses

Left ventricular volumes and ejection fraction were calculated using the conventional area-length method. The midpoint of the aortic valve ring was estimated and the longest line between this point and any other point (apex) on the perimeter of the left ven-
tricular contour was defined as the long axis.

To evaluate the regional characteristics of the left ventricle, the long axis for each frame was divided into 15 equal longitudinal segments, and 30 hemiaxies (15 in the anterior and 15 in the inferior wall region) perpen-
dicular to the long axis were constructed from the midpoint of each of the 15 longitudinal segments on the long axis to the border of the left ventricular contour. The lengths of these hemiaxies were considered to be regional hemichordal lengths. We assumed that the left ventricle could be modeled as a stack of 30 hemicylindrical discs of the indicated regional hemichordal lengths and segments along the long axis, as shown in Fig 1. Each region of the left ventricular cavity was assigned a number clockwise from the anterobasal region. We also assumed that discs of the same number at end-diastole and end-systole corresponded to the same re-
gional portion of the left ventricular wall.

We used a modification of the method described by Hooghoudt et al² to estimate the parameters of the regional dynamics of the left ventricle, particularly for calculations of regional volumes. Each regional volume was
calculated as:

Regional Volume = \pi \frac{C^2}{2} \times \frac{L}{15}

where C represents the regional hemichordal length and L represents the length of the left ventricular long axis. Regional stroke volumes were then calculated.

Based upon these assumptions, we defined the three major variables to be used in the study. The first variable was the regional contribution to total stroke volume (RCSV), defined as:

\[
RCSV \, \% = \frac{(\text{regional end-diastolic volume-\text{regional end-systolic volume})}}{\text{("total" stroke volume)}} \times 100
\]

This calculation was obtained by taking into account long-axis shortening.

The second variable was percent regional hemichordal shortening (%HS), which represents the degree of systolic regional wall motion:

\[
%HS = \frac{(\text{Ced-Ces})}{\text{Ced}} \times 100
\]

where Ced and Ces represent the end-diastolic and end-systolic regional hemichordal lengths of the same region.

The third variable was the end-diastolic regional shape index (RSI), defined as:

\[
\text{RSI} = \frac{(\text{regional hemichordal length}}{\text{(left ventricular long axis length)}
\]

This variable increases as the region extends further from the long axis. This index is referred to as the "regional shape index" because the intuitive global chamber shape of the left ventricle could be easily reconstructed from the value of this index for each region.

Statistics

Differences between means were statistically analyzed using unpaired t-tests. For analyses of correlation, linear, quadratic or other higher-dimensional polynomial and exponential equations were fitted to the data, and the equation associated with the highest correlation coefficient was adopted. The significance of differences between correlations was assessed by an analysis of covariance. \( p < 0.05 \) was considered statistically significant. Data are expressed as the mean ± standard deviation.

RESULTS

Inter- and Intra-Observer Variability

To determine interobserver variability, two experienced observers separately traced left ventriculograms and obtained the values for projected areas for all of the patients at both end-diastole and end-systole. Linear regression analysis was used to compare the two sets of values. Good linear correlations were found for both end-diastole (\( r = 0.96 \)) and end-systole (\( r = 0.95 \)). Similar results were obtained for intraobserver variability (\( r = 0.97 \) for end-diastole, \( r = 0.96 \) for end-systole). These findings indicate good reproducibility of the measurements.

Hemodynamic Profiles

Hemodynamic profiles for the three groups are provided in the Table I. There were no significant differences among the groups with regard to heart rate, mean pulmonary arterial pressure or ejection fraction, which were all within normal limits. Left ventricular peak systolic pressures were significantly higher in the AR group than in the normal subjects, but were comparable in the N and MR groups. Left ventricular end-diastolic pressure was significantly higher in the MR and AR groups than in the N group. Left ventricular end-diastolic volume index, end-systolic volume index and stroke index were each also significantly greater in the MR and AR groups than in the N group, but were comparable between the MR and AR groups. There were no significant differences in percent long-axis shortening among the three groups.

Left Ventricular Regional Wall Motion

Fig. 2 shows the distribution of the percent regional hemichordal shortening for each of the three groups. Even in normal controls, the apical region contracted more than the other regions of the left ventricle. There were no significant differences in percent regional hemichordal shortening between the normal control group and the MR group. However, in the AR group, regional hemichordal shortening was significantly decreased in the anterobasal and anteroapical regions relative to the corresponding regions in the normal control group (\( p < 0.05 \) in both regions).
**Left Ventricular End-Diastolic Regional Shape**

Fig 3 shows the "average" chamber shape of the left ventricle at end-diastole for each of the three groups reconstructed from the findings of the regional shape index, with normalization of the lengths of the long axis. Left ventricular chamber shape was more spherical in patients with mitral regurgitation than in normal control subjects due to an outward curvature of the midventricular portion (regional shape index: $p<0.05$ and $p<0.01$ in the anterior-mid and inferior-mid portions), and was more conical in patients with aortic regurgitation due to bulging of the anterobasal portion ($p<0.05$, vs N).

**Regional Contribution to Total Stroke Volume**

Fig 4 shows the distribution of the regional contribution (%) to total stroke volume in each of the three groups. In normal controls, the midventricular region was the primary contributor to stroke volume generation. The infero-midventricular region showed a significantly greater contribution to total stroke volume in the MR group than in the normal control group ($p<0.05$). However, in the AR group, the contribution was significantly increased in the anterobasal and infero-midventricular regions ($p<0.05$ in both regions, vs N), and significantly decreased in the anteroapical and infero-midventricular regions.
Fig 4. The distribution of the regional contribution to total stroke volume (RCSV) in the three groups. Upper panel: Normal control (N). Middle panel: Mitral regurgitation (MR). Lower panel: Aortic regurgitation (AR).
No., region number of the left ventricular cavity, as shown in Fig 1.
*: p<0.05 vs N

(p<0.05 in both regions, vs N). These findings indicate that left ventricular stroke volume is generated principally in the midventricular portion of the left ventricle in normal subjects and patients with mitral regurgitation. However, stroke volume is generated principally in the basal portion in patients with aortic regurgitation.

**Left Ventricular Regional Wall Motion Versus Stroke Volume Generation**

There was no correlation between the regional contribution to total stroke volume (RCSV) and percent regional hemichordal shortening (%HS) in any of the three groups (Fig 5). This Figure illustrates the contribution to total stroke volume of each region separately for the apical, midventricular and basal portions of the left ventricle. Although the apical region contracted more than the other regions, its stroke output was comparatively lower than that of the other regions.

**Left Ventricular Shape Versus Stroke Volume Generation**

Fig 6 illustrates the relationships between the regional contributions to total stroke volume (RCSV) and the end-diastolic regional shape index (RSI) for a representative example from each group. All of the patients in each group showed a significant degree of quadratic correlation between these two variables. Furthermore, an analysis of
covariance detected no significant differences in this relationship among the patients in any of the groups (p<0.01 in all groups). Therefore, we were able to assess this relationship by combining the results of all of the patients for each group, and similarly obtained good correlations between these two variables for each of the three groups (N, r=0.87; MR, r=0.79; AR, r=0.90), as shown in Fig 7. This Figure also illustrates the contribution to total stroke volume of each region separately for the apical, mid-ventricular and basal portions of the left ventricle. For each of the three groups, the finding of a quadratic relationship indicated that the further a regional segment of the wall extended from the long axis at end-diastole, the more it contributed to total stroke volume.

On the other hand, no correlations were found between the regional contribution to total stroke volume and the end-systolic regional shape index in any of the three groups.
DISCUSSION

This is the first detailed study to describe the left ventricular regional contribution to total stroke volume in normal subjects and patients with mitral or aortic regurgitation, and also to give an intuitive description of left ventricular global shape in the three groups. Left ventricular chamber shape was more spherical in patients with mitral regurgitation than in normal control subjects due to an outward curvature of the midventricular portion, and was more conical in patients with aortic regurgitation due to bulging of the anterobasal portion. Left ventricular stroke volume was generated principally in the midventricular portion of the left ventricle in normal control subjects and patients with mitral regurgitation. However, in patients with aortic regurgitation, stroke volume was generated principally in the basal portion. Although different types of nonuniformity of regional wall motion were observed among the three groups, regional contributions to total stroke volume were quadratically correlated with the regional shape index for all three groups. This suggests that the differences in stroke volume generation among the three groups were due to the characteristic changes in the shape of the left ventricle in affected patients. Therefore, the degree of percent regional hemichordal shortening was of relatively little importance in determining the regional contribution to total stroke volume in each of the groups.

Although numerous investigators have observed nonuniformity of left ventricular regional wall motion, even in the intact heart, few detailed studies have been made of the regional contributions to total stroke volume, other than brief comments in normal subjects. Herman et al using 30 degree right anterior oblique ventriculography, found that regional emptying in normal subjects was relatively uniform. However, base-to-apex shortening was ignored in that study and only four regions were selected for analysis. Since these regions had similar end-diastolic hemiasial lengths, the findings obtained by Herman et al were not inconsistent with our own. Shepertcyki and Morton using right anterior oblique ventriculograms divided into 5 portions, showed that the mid-ventricular portion contributed most to stroke output while the apical portion contributed least. This finding generally agrees with our present results. Haendchen et al using echocardiography, also obtained findings consistent with our own, in that the apical portion of the left ventricle contributed less to stroke volume than other regions. In their study, the regional contribution to stroke volume was studied using the absolute changes in regional cross-sectional area, and long-axis shortening was not considered. Moreover, no previous study has documented the distribution of the regional contribution to stroke output in a diseased group, such as mitral or aortic regurgitation.

Regional Wall Motion in Patients With Mitral or Aortic Regurgitation

Osbakken et al found no significant difference in percent regional shortening of the left ventricle between normal control subjects and patients with compensated mitral or aortic regurgitation; however, patients in a decompensated state had an abnormal distribution of regional left ventricular wall motion. It has also been shown that the distribution of regional wall motion is preserved for a certain interval during compensated chronic volume overload. In our study, similar results were obtained for patients with compensated mitral regurgitation, but different results were obtained for those with compensated aortic regurgitation, who had wall motion abnormalities. DiDonato et al reported apical or anterolateral left ventricular hypokinesis in patients with compensated aortic regurgitation, which supports our findings. Johnson et al and Corya et al also obtained results similar to ours. When Osbakken's findings are reviewed in detail, it is found that anterior wall contraction was less in patients with compensated aortic regurgitation than in normal control subjects, although this difference was not statistically significant. These discrepancies may be due in part to the differences between the techniques used to assess regional wall motion.

Left Ventricular Chamber Shape in Patients With Mitral or Aortic Regurgitation

It has been reported that the globularity of the left ventricular chamber is increased in patients with mitral or aortic regurgita-

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tion using a parameter such as eccentricity. The parameter used in these studies, however, did not correspond to a unique shape, which limits the ability to describe regional shape. Fourier shape analysis, as performed by Kass et al., demonstrated that in patients with aortic regurgitation, the left ventricle has a globular outline at end-diastole but is banana-shaped at end-systole, while in patients with mitral regurgitation, the same end-diastolic outline changed to more of a light-bulb-like shape by end-systole. We did not study the instantaneous changes in the regional shape index during systole, but did find some differences in left ventricular chamber shape between patients with mitral and those with aortic regurgitation, even at end-diastole. In addition to technical differences, such that our assessment of chamber shape was more intuitive than Fourier shape analysis, the discrepancy between our results and those of Kass et al. in the description of end-diastolic chamber shape may also be due in part to differences in the methods used to select patients, since the AR and MR groups studied by Kass et al. included patients with left ventricles with very low ejection fractions.

The present study is the first study to intuitively describe “average left ventricular chamber shape” for normal subjects and patients with mitral or aortic regurgitation. The differences in left ventricular chamber shape between MR and AR patients can probably be attributed to the differences in loading conditions or differences in the directions of left ventricular inflow or outflow blood jets, as was pointed out by Kass et al.

**Left Ventricular Regional Wall Motion Versus Stroke Volume Generation**

Our present study demonstrated that the degree of regional wall motion was relatively unimportant in determining the regional contribution to stroke volume in all three groups. Lew and LeWinter using ultrasonic segment length gauges, demonstrated in anesthetized dogs that midwall shortening was nonuniform around the minor-axis circumference, but that different midwall sites around the circumference nevertheless appeared to make similar overall contributions to the ejection of blood. This finding was consistent with our conclusion that the nonuniformity of regional wall motion does not always determine the degree of regional contribution to total ejection, not only in normal subjects, but also in compensated patients with mitral or aortic regurgitation.

The preservation of the degree of long-axis shortening may also play a role in preserving the relationship between the regional contribution to total stroke volume and regional shape in patients with compensated chronic mitral or aortic regurgitation. Dumesnil et al. emphasized the contribution of long-axis shortening to efficient shrinkage of the left ventricular cavity.

**Left Ventricular Shape Versus Stroke Volume Generation**

There was a significant quadratic relationship between the regional contribution to total stroke volume and the end-diastolic regional shape index. A quadratic relation is reasonable when the definitions of these parameters are considered, and when regional wall motion is relatively homogeneous compared to regional chordal length; i.e., the variation in long-axis shortening is negligible. In this case, the regional shape index depends on chordal length and the regional contribution to stroke volume depends on the square of the chordal length, even though these parameters are obtained as dimensionless values after normalization by the long-axis length and total stroke volume, respectively. It is noteworthy that the proportion of regional stroke volume generated by the midventricular portion is greater than that expected intuitively from the left ventricular chamber shape. It should also be stressed that the quadratic relationship is maintained during compensated mitral regurgitation with normal regional wall motion and a different chamber shape, and even during compensated aortic regurgitation associated with a different pattern of regional wall motion and chamber shape. DiDonato et al. observed that the degree of regional wall motion in the midventricular portion had the highest degree of correlation with the left ventricular ejection fraction in normal subjects. Their finding is consistent with our own that the midventricular portion is the most important region in the generation of stroke volume.
It has been shown that additional sarcomeres may be replicated in series in chronically volume-overloaded heart, which gives the heart an enhanced stroke volume within the constraints of relatively normal sarcomere shortening.\textsuperscript{21} The finding of a preservation of the relationship between the regional contribution to global ejection and regional shape during compensated chronic volume overload in our study is consistent with the above findings at the regional wall level. In our study, this relationship was maintained even in patients with compensated aortic regurgitation with some disturbance of regional wall motion, suggesting that the increase in regional volume may develop earlier than the deterioration of regional wall contraction in states of chronic volume overload. Similar findings were obtained by Badke and Covell\textsuperscript{11} in anesthetized canines with aortocaval fistulae.

Our findings may also support a hypothesis regarding the mechanism by which stroke volume is generated efficiently. Regions with larger end-diastolic volumes can produce larger stroke volumes with less wall motion. Therefore, the ellipsoidal shape of the left ventricular chamber, which has been said to be suitable for the generation of pressure,\textsuperscript{22–24} may result in assignment of the responsibility for the generation of stroke volume to the midventricular portion.\textsuperscript{25} This mechanism may ensure minimal energy expenditure for a given stroke volume!

In the present study, although the overall contribution of the apical region to total stroke volume is small, its contribution may be different in each phase of systole, such as early-systole. The volume ejected in early systole is greater than that in late systole.\textsuperscript{26} There is an asynchrony in the regional wall motion during systole,\textsuperscript{27,28} which is modified by several diseased conditions.\textsuperscript{29} Therefore, a further study is necessary to clarify the significance of regional wall motion in generating stroke volume in each phase of systole.

\textbf{Limitations}

The results of the present study were obtained using only the right anterior oblique view in left ventriculographic studies, for which some assumptions regarding left ventricular geometry were made, while the left anterior oblique projection was used only to exclude subjects with apparent asynergy or left ventricular distortion. However, it has been documented that regional nonuniformity in segmental shortening of the left ventricle is much less in cross-sectional views than in apex-to-base views\textsuperscript{3} and that cross-sections of the left ventricle can be considered circular.\textsuperscript{17,30} Thus, the heart can be considered a prolate spheroid for which the major variability in regional wall dynamics can be assessed using right anterior oblique views.

We assumed that discs assigned the same number at end-diastole and end-systole corresponded to the same region of the left ventricular wall. Gelberg et al.\textsuperscript{31} demonstrated that this assumption may not be valid. However, the method we used to calculate regional and stroke volumes has already been shown to be useful and correct by Hooghoudt et al.\textsuperscript{32} In each of the three groups, none of the left ventricles were extremely distorted, and each had a smooth contour. Therefore, results obtained using measurements of regional hemichordal lengths might not significantly differ from those obtained using measurements of regional areas, which has been recommended,\textsuperscript{31} since the hemichord is constructed from the “midpoint” of each longitudinal segment along the long axis.

This study also assumed that regional long-axis shortening is uniform. However, a recent study revealed using magnetic resonance tissue-tagging that long-axis shortening in the human ventricle is nonuniform.\textsuperscript{32} Although it is unknown whether this nonuniformity can be considered as significant in our study, this limitation should be noted.

Another limitation of the present study is the absence of information regarding regional wall thickness and stress. It is nearly impossible to measure the wall thicknesses of all of the regions by left ventriculography, particularly of the anterobasal and inferobasal walls. Radiation exposure was applied to left ventriculography especially for intracavitary visualization, which made it difficult to identify left ventricular epicardial contour. Moreover, the low reliability of other methods that have been previously proposed to calculate regional wall stress has been discussed by Regen.\textsuperscript{33} Therefore, in the present

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study, we did not discuss our findings from the perspective of regional wall thickening and stress.

REFERENCES


