Regional Three-Dimensional Geometry and Function of Left Ventrices With Fibrous Aneurysms
A Cine–Computed Tomography Study

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Background. To assess the extent and nature of the dysfunction surrounding aneurysms of the left ventricle (LV), we examined the parameters of local and global three-dimensional shape, size, and function of LVs of eight patients with histologically confirmed anterior fibrous aneurysms.

Methods and Results. Three-dimensional reconstructions of each LV were made from 10–12 short-axis fast cine–angiographic computed tomography (cine-CT) slices encompassing the entire heart at end diastole and end systole. Regional three-dimensional wall thickness, thickening, motion, curvature, and stress index were calculated for 84 elements encompassing the entire LV. The aneurysmal border was defined by a sharp decrease in end-diastolic wall thickness and separated the LV into an aneurysmal zone and a normal zone that was further divided into adjacent normal (AN) and remote normal (RN) zones. As expected, thickening was negligible in both the aneurysmal and the border zones. Although both the AN and the RN zones had normal wall thickness (1.05±0.20 and 1.09±0.20 cm, respectively), thickening was depressed in the AN (0.22±0.08 cm) but not the RN (0.44±0.19 cm) zones. The size of the dysfunction zone (defined as less than 2 mm thickening) was found to be considerably greater than the anatomic size of the aneurysm (60.9±13.7% versus 33.6±7.6% of the left ventricular endocardial area, respectively; p<0.001). In addition, the AN zone had a smaller curvature and a higher stress index than the RN zone.

Conclusions. LVs with fibrous aneurysms are characterized by a relatively large region of nonfunction that encompasses the thin aneurysmal area and its transitional border zone, a normally functioning remote zone, and an intermediate region of normal wall thickness but with reduced function, which may be attributed to its low curvature and high stress index. (Circulation 1991;84:1072–1086)

Left ventricular (LV) aneurysm, a nonfunctioning dilated area of the left ventricle that often follows myocardial infarction, was first described by John Hunter1 in 1757. Since then, it has gained attention because of its numerous complications, most important of which is its effect on LV function. Numerous studies have analyzed the nature of the reduction in LV function,2–7 yet the mechanism by which this occurs is still unclear. It has been recognized that the function of the residual myocardium is of the utmost importance in determining the prognosis and, more specifically, in predicting and monitoring the outcome of aneurysmectomy.8–10 However, the effect of surgery on LV function in these patients is still controversial.

Many attempts to determine the size of the aneurysm5,7,11–14 were based on contrast or radionuclide angiography and relied on geometric simplification, usually modeling the left ventricle and the aneurysm as spheres or spheroids. The size of the aneurysm was usually based on the size of the akinetic or paradoxically expanding area and often overestimated compared with the size determined by surgery.15 Re-
gional function in aneurysmal left ventricles was relatively unexplored. Nicolosi et al. examined regional function relative to the borders of the aneurysm. Their study showed depressed LV function at the aneurysm border during isovolumic systole but with a significant amount of thickening in and around the aneurysm at end systole. However, all of these studies were subjected to the inherent limitation of the planar imaging techniques.

The modern evolution of three-dimensional imaging techniques such as cine-angiographic computed tomography (cine-CT) and magnetic resonance imaging (MRI) provides high-quality three-dimensional data for the entire left ventricle. Using cine-CT data, the present study sought to characterize the size, shape, and function of the aneurysmal region and the remaining “normal” regions of the LV wall in patients with anterior fibrous aneurysms. This is achieved by using our algorithm for three-dimensional reconstruction of the left ventricle to evaluate wall thickness and regional function by thickening and motion analyses. In addition, meridional and circumferential curvatures, surface normals, and surface area are evaluated for each left ventricle and used to define and map the regional stress index.

Methods

Patients

Eight patients (seven men and one woman) with fibrous LV aneurysms underwent cine-CT scanning at the University of Iowa Hospital before surgery. Patients varied in age from 46 to 65 years (mean age, 55.6 ± 7.8 years). Body surface area averaged 1.96 ± 0.18 m². The precise diagnosis of fibrous LV aneurysm was made histologically in seven patients after surgical removal of the aneurysm and in one patient by biopsy at the time of surgery. Two of the patients had a few remaining myocytes on histological examination. Three patients had significant (more than 80% obstruction) three-vessel disease, and five patients had one-vessel disease. The proximal left anterior descending coronary artery (LAD) was obstructed in all patients—in seven by 90% or more and in one by 70%. Intramural thrombi and diastolic distension were found in five patients. All patients demonstrated wall motion abnormalities of the left ventricle on angiography, exhibiting akinesis or dyskinesis. At surgery, aneurysms were resected in all except one patient because of its relatively small size. Coronary artery bypass grafts were performed in two patients. A control group of 10 normal volunteers was scanned for comparison.

Cine-CT Imaging

The imaging procedure has been presented in detail elsewhere; only a brief summary is provided here. Each patient was positioned in the scanner so as to yield a series of short-axis tomographic images from apex to base (Figure 1). This was accomplished by using a combination of cranial tilt and clockwise slew of the imaging table, the degrees of which were determined with the aid of previously obtained posteroanterior and lateral chest radiographs. Usually, 15–20° of clockwise slew and as much as 20–30° of cranial angulation were required. Localization scans were used to determine the location of the LV apex. The patient was then mechanically moved in the scanner to ensure that the most caudal tomographic image was obtained at a level close to the presumed apex of the heart.

A bolus injection of contrast agent was injected by means of a powered injector for a time roughly equal to the arm to tongue time, which was determined for each patient by injection of 0.2% magnesium sulfate solution. The patient was instructed to suspend respiration at about half the normal inspiratory volume before the initiation of scanning. Electrocardiographically triggered scanning, using the R wave or 80% of the RR interval, was initiated two thirds of the way through the contrast infusion. From six to eight tomographic levels were scanned throughout the cardiac cycle; either 10 or 14 images were obtained per cycle, at 70-msec intervals, in a sequence of images extending beyond end systole. The table was then moved by the distance required to line the imaging planes with the unscanned region of the heart toward the base, and the procedure was repeated after several minutes.

Data Processing

The files containing the digitized CT data were transferred on magnetic tapes to our VAX 3200...
FIGURE 2. Cine–computed tomography cross sections of an aneurysmal left ventricle at end diastole (left panel) and end systole (right panel). Endocardial and epicardial left ventricular tracings as well as endocardial right ventricular free wall tracings are presented as small + marks. Note very thin septum and anterior wall.

computer. The original data, which consisted of 11-bit (2,048) gray levels, were linearly converted to eight bits (256 gray levels). Image processing, carried out on a micro-VAX 3200/GPX workstation, included an option to adjust the minimum and maximum pixel values for optimal image quality. As shown in Figure 2, the cardiac zone was zoomed for tracing. The end-diastolic image was defined as the image coinciding with the R wave of the electrocardiogram. After reviewing all of the slices throughout the cycle, end systole was defined as the time (percent of RR interval) of the image with the smallest blood cavity area for each level. If this time varied at different levels, then end systole was determined by taking into account the relative contributions of each of the slices to the total cavity volume.

Tracing of the endocardial and epicardial borders and right ventricular insertions were performed on the VAX workstation. The criteria for identifying borders required looking for a sharp outline between the myocardium and the blood pool on the inside and the myocardium and various structures on the outside; if no sharp border was seen, then the middle of the intermediate area was defined as the border. Papillary muscles were cut at their insertions.

Correction for the movement of the table between the two sets of scans required alignment of the most basal image of the apical set to the lowest of the basal set. This was achieved by positioning the traced contour of the basal slice onto the raw image of the slice below it and searching for the best visual alignment. The displacement of the contour was regarded as the correction vector, and each of the traced points of the basal set of tracings was corrected by adding this vector.

The most apical point was defined as follows. If the image near the apex contained myocardium but no blood cavity, then the endocardial apical point was defined as being the point on the long axis passing through this tomographic level. If the lowest slice contained part of the blood cavity, then the most apical true myocardium was regarded as being at a level 0.4 cm (approximately half of a slice) below. The apical epicardial point was defined as the point d cm below the endocardial apical point on the long axis, where d represents the average wall thickness of the two most apical slices. The base of the heart was defined as the tomographic level with the most visible LV outflow tract. These points were different at end diastole and end systole.

Three-dimensional Reconstruction of Left Ventricle

Reconstruction of the left ventricle from these tracings was based on our helical coordinate system. Briefly, the three-dimensional reconstruction algorithm included the following features.

Long-axis definition. The long axis of the left ventricle was defined as the linear least-squares fit of the centroids of each of the slices, with extra weight given to the apical and basal slices. The long axis defined a new "center" for each tomographic level. The plane of the tomographic level was represented as the x,y plane, and
the z direction was perpendicular to this plane. The z value of each tomographic slice was taken as the value at the center of the 8-mm-thick slice. The long axis defined at end systole was used as a common axis for both end diastole and end systole. This was important for optimal matching of regions before and after contraction, especially when analyzing regional function in the presence of significant local pathology. The reason for this (illustrated in Figure 3) was that the normal slice centroid, and hence the long axis, may move significantly during contraction, whereas the pathological myocardial segment does not, resulting in poor tissue matching, when using separate long axis at end diastole and end systole.

Angular alignment. Angular alignment of the ventricles was obtained by defining the midseptal point—the center of the line segment joining the two right ventricular insertions to the epicardium—of each left ventricle as the angle of reference. The midseptal angle between the positive x axis and the line segment joining the midseptal point and the new slice center was given the value of 0°.

Angular segmentation. Twenty-four radii at equal angles, starting from the midseptal angle, were then radiated from the new slice center to define 24 points on each contour. Theta (θ) was the angle with respect to the midseptal angle.

Surface interpolation. Bivariate interpolation was used to identify the points of a normalized helix enveloping each surface. The helix was characterized by eight windings enveloping each left ventricle, with 24 nodes at equal angles apart on each winding. This procedure size-normalized the helix and allowed a comparison of different ventricles or of the same ventricle at different times. By consistently marking the helix at a number of points, one could then follow any point throughout the cycle or compare the corresponding position on another heart. Note that this normalization assumed homogeneous longitudinal contraction and the absence of torsion.

It was often difficult to trace the most apical slice. Similarly, it was rather difficult to trace the ventricle as a single complete contour when the atria appeared at the base. Consequently, these two extreme sections were ignored in the LV reconstruction and shape analysis, and the reconstruction was confined to 15–75% of the long-axis length. The apical and basal areas were, however, taken into account in calculation of the global volume and mass.

The endocardial and epicardial surfaces of the left ventricle were each represented by a helix of eight windings with 24 equiangular points on each winding. This defined 168 volume elements for each left ventricle (with the apex and base elements excluded as explained above). Each volume element was bordered by four points on the endocardium and four points on the epicardium. For curvature and stress calculations, every two adjacent elements were combined to yield a total of 84 (168 divided by 2) (double) elements for each heart.

Calculation of Geometric Parameters

The following local geometric parameters were calculated for each element.

Wall thickness. The perpendicular regional wall thickness was calculated for each three-dimensional volume element by dividing its volume by the average of its endocardial and epicardial surface areas.20,21 This procedure took into account the inclination of the surface with respect to the long axis of the ventricle, thereby correcting for errors created by the usual two-dimensional methods of calculating wall thickness.

Thickening. This index was best defined in the aneurysmal left ventricle as the difference between end-systolic and end-diastolic wall thicknesses. Note that the normal definition, in which thickening was expressed as a percentage by dividing by end-diastolic wall thickness, would create excess noise in the case of LV aneurysms because the end-diastolic thickness in the aneurysm was very small.

Curvature and surface normals. The normal to the surface was estimated at the center of the endocardial surface of each element. The meridional and circumferential curvatures of the endocardial surface were calculated using our algorithm, which is summarized in the “Appendix.”

Wall motion. The local endocardial wall motion during contraction was calculated by the difference

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** Schematic of division of left ventricular cavity into segments illustrating advantage of using same long axis when segmenting left ventricle at end diastole (ED) and end systole (ES) in cases of regional pathology. By defining end-systolic center point (obtained by local position of long axis obtained from all levels), segment AB at ED becomes segment A'B' at ES. However, using separate long axes for ED and ES makes CD at ED relate to A'B' at ES, with an error resulting from asymmetric contraction.
between the average position of the four corners of the endocardial surface of a volume element at end systole and the corresponding average position of the surface at end diastole.

**Endocardial surface area.** The curvilinear endocardial surface area was approximated by the sum of the two triangles constructed between the four corners that define the area. This procedure was used to evaluate the relative size of the aneurysm by expressing its area as a percentage of the total endocardial surface area. The following global geometric parameters were calculated.

**LV blood volume.** The volume was calculated at end diastole and end systole by a variation of Simpson's method. The area of the cavity was multiplied by the slice thickness, for each slice, from apex to base. The volume of the unscanned gaps between tomographic slices was estimated by linear interpolation. The calculations were repeated to include or exclude the intracavitary thrombus as part of the blood volume. LV stroke volume as well as ejection fraction were derived from these volumes.

**LV muscle volume.** This was calculated by subtracting the endocardial blood volume from the epicardial inner volume.

**Estimation of Regional Wall Stress**

Regional wall stress indexes ($\sigma/P$) were estimated by using the Laplace equation for a thick wall sphere as described by Janz:\(^{25}\)

$$\sigma/P = r_c^2/(2tr_c+t^2) \quad (1)$$

where $\sigma$ is the wall stress, $P$ is pressure, $r_c$ is the local three-dimensional circumferential radius of curvature at the endocardium, and $t$ is the local wall thickness. Comparison with other formulas such as Janz's\(^{25}\) circumferential stresses as well as with cylindrical and conical approximations are given in "Results" and "Discussion." It can be easily shown that the spherical stress in Equation 1 is identical to Janz's equation\(^{25}\) for meridional stress, $\sigma_m$. Because it has been indicated\(^{26}\) that the characteristic differences between the stress values in different diseases are not affected by the formula selected, even though different absolute stresses are obtained, the selection of simple spherical stress formula here should not affect the results of the present study.

**Identifying Physical Boundaries of an Aneurysm**

A chronic fibrous aneurysm is clearly distinguishable from the surrounding myocardium,\(^{27,28}\) and its border is often distinctly seen on the CT scans.\(^{29}\) Here we search for quantitative criteria that define the aneurysmal tissue and its borders on the three-dimensional reconstructed heart. As is well established, the diastolic wall thickness of fibrous aneurysms averages 4 mm,\(^{30}\) whereas the end-diastolic wall thickness in the normal left ventricles varies between 8 to 10 mm.\(^{31}\) Here we classified an element with an end-diastolic wall thickness of less than 5 mm to be part of the aneurysm and any element with an end-diastolic wall thickness of more than 7 mm as a nonaneurysmal tissue. The location of the border, with wall thickness between 5 and 7 mm, was defined as being the region with the maximal thickness.
gradient at end diastole (Figure 4). Note that this end diastole-based border definition is unrelated to regional function. Hence, a clearly defined border can be obtained by this method.

**Division of Ventricle Into Zones**

As seen in Figure 5, the border of the aneurysmal region was subdivided into an inner border (IB) zone and an outer border (OB) zone, each two elements wide (each element covers 15° of the circumference), aligning the border line. The remainder of the nonaneurysmal tissue was subdivided into two normal zones—the adjacent normal (AN) zone (two elements wide) and the remote normal (RN) zone, which includes the remainder of the myocardium.

**Comparison With Normal Baseline Properties**

To compare the aneurysmal hearts with the normal hearts, we defined equal subzones in the 10 normal hearts that corresponded to the average positions and sizes of the above-defined five zones in the aneurysmal left ventricles. Because all of the aneurysms included in the present study were anteroseptal, it was possible to obtain an average aneurysm location for the entire group. This was performed by assigning each volume element of each left ventricle a value of 1 if it was inside the aneurysm border and 0 if it was outside it. A summation of the values of the corresponding elements in the eight hearts was then performed. An element with a cumulative value of 4–8 was taken to be within the aneurysm border; a value of 0–3 was assumed to be outside the border of the representative aneurysm. This approach thus yielded a representative aneurysmal region that was an average of the eight hearts included in this study. The division into the five zones (aneurysm, IB, OB, AN, and RN) was then performed, as above, on this representative aneurysmal left ventricle, and these zones were then used to compare with each of the 10 normal left ventricles. Note that the cardiac helical coordinates used here are “adaptive” for each heart and that the use of the midseptum as a reference allows for regional matching between different hearts, regardless of the size of the left ventricle.

**Statistical Analysis**

Each of the measured geometric parameters was averaged within each zone for each patient at both end diastole and end systole. Data are presented as mean±SD. Statistical comparisons are made between zones by applying repeated measures analysis of variance among the different zones, followed by the Neuman-Keuls multiple range test. Differences between aneurysmal and normal hearts are demonstrated by unpaired t tests.

**Results**

**Global Parameters**

The calculated global results are presented in Table 1, once assuming that the thrombus is included within the cavity volume (five patients) and once with
TABLE 1.  Comparison of Global Parameters of Aneurysmal and Normal Left Ventricles

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Aneurysmal left ventricles</th>
<th>Normal left ventricles</th>
</tr>
</thead>
<tbody>
<tr>
<td>End-diastolic volume (ml)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excluding thrombus</td>
<td>328.6±83.2</td>
<td>156.5±21.4</td>
</tr>
<tr>
<td>Including thrombus</td>
<td>364.3±97.4</td>
<td></td>
</tr>
<tr>
<td>End-systolic volume (ml)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excluding thrombus</td>
<td>242.2±86.8</td>
<td>53.6±11.3</td>
</tr>
<tr>
<td>Including thrombus</td>
<td>279.1±91.0</td>
<td></td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>85.2±27.8</td>
<td>103.0±13.5</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excluding thrombus</td>
<td>27.2±9.6</td>
<td>65.9±4.0</td>
</tr>
<tr>
<td>Including thrombus</td>
<td>24.3±7.7</td>
<td></td>
</tr>
<tr>
<td>Left ventricular muscle volume (ml)</td>
<td>200.9±43.0</td>
<td>155.0±20.5</td>
</tr>
<tr>
<td>End-diastolic pressure (mm Hg)</td>
<td>31.0±6.4</td>
<td>NA</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>104.5±15.9*</td>
<td>116.6±7.7</td>
</tr>
<tr>
<td>End-systolic aneurysmal area (%)</td>
<td>36.7±7.3</td>
<td>NA</td>
</tr>
<tr>
<td>End-diastolic aneurysmal area (%)</td>
<td>33.6±7.5</td>
<td>NA</td>
</tr>
</tbody>
</table>

Values are given as mean±SD.
*See text.

the thrombus excluded. The end-diastolic and end-systolic volumes are markedly increased in all patients compared with the normal healthy left ventricle. End-diastolic LV pressure is high in the six patients with available data. Stroke volume is within normal limits, and ejection fraction is markedly reduced. LV muscle volume is increased (average, 200.9±43.0 versus normal value of 155.0±20.5 ml). The global accuracy of the tracings is reflected from the insignificant difference between the calculated end-diastolic and end-systolic muscle volumes (199.4±43.7 versus 202.4±42.7 ml, respectively). Surface areas of the endocardium and epicardium are also higher than normal, which is consistent with the pathological volume increase.

The change in surface area from end diastole to end systole shown in Table 2 appears to be a good index of global function, correlating well with ejection fraction (r=0.88, p=0.001) and percentage of fibrous myocardium (r=-0.85, p=0.003). Mean changes of endocardial (16.3±5.7%) and epicardial surface areas (11.6±4.0%) are markedly reduced compared with our findings in normal hearts (48.3±4.4% and 23.8±3.1%, respectively). The LV long axis shortened by a mean of 6.7±2.7%.

Effect of Thrombus

Five of the eight left ventricles were found to have mural thrombi on CT. Four of these were confirmed at surgery. The volume of each thrombus was calculated by Simpson’s method. The surface area of the thrombus attached to the endocardial surface was determined in a manner similar to endocardial surface area calculation. The total volume of the thrombus averaged 57.1±42.5 ml (range, 16.5–108.5 ml). The thrombus constituted 14.2±9.9% of the LV volume at end diastole (range, 5–31%) and 19.0±14.1% at end systole (range, 7–43%), whereas the percent of endocardial surface area engaged by the thrombus was 20.2±11.7% at end diastole (range, 6–36%) and 24.2±14.5% at end systole (range, 8–44%). It is seen that the thrombus may be large, taking up as much as one third of the LV cavity volume.

Size of Aneurysms and Correlation With Local and Global Function

The relative sizes of the aneurysms are shown in Table 3 (column 1A). The surface area of the aneurysm plus its IB, (i.e., the zone within the bold curve in Figure 5) is expressed as a percent of the total surface area of the left ventricle. As noted above, all values exclude the two extremities of the left ventricle (i.e., apex and base). End-diastolic aneurysm sizes range from 23% to 43% of LV surface area (mean, 33.6±7.5%). The aneurysm size at end diastole has a high negative correlation with the ejection fraction, calculated with the thrombus included in the cavity (r=-0.86, p=0.002). A somewhat smaller correlation is found when the thrombus is excluded (r=-0.70, p=0.02). High correlations are also found between aneurysm size and end-systolic cavity volume (r=0.69, p=0.03), end-systolic endocardial surface area (r=0.68, p=0.03), and end-diastolic LV pressure (r=0.73, p=0.04). Correlations of the aneurysm size and regional function are described below.

Regional Geometric Parameters

To examine the relation between the size of the aneurysm and the zone of dysfunction, we calculated the percent of total LV surface areas of the elements with less than 1 mm thickening (Table 3, column 2A) and those elements with less than 2 mm thickening (Table 3, column 2B), representing akinetic and hypokinetic zones, respectively. These two areas of dysfunction are compared in Table 3 with the surface areas of the aneurysm (column 1A) and the aneurysm plus the OB zone (column 1B). As seen, the area of dysfunction with thickening of less than 2 mm occupies between 46% and 87% of the LV surface and is, in all cases, much larger than the area of the anatomic aneurysm (24–43% of the left ventricle). As seen, the size of the area of dysfunction based on thickening correlates closely with the size of the aneurysm (r=0.8, p<0.01). The nonfunctional zone of less than 1 mm thickening (column 2A) is seen, in most cases, to be almost identical in size to the anatomic aneurysm plus its border zones (column 1B), whereas the hypofunctional area (less than 2 mm thickening) extends much further.

Figures 6 and 7 and the data regarding thickness and thickening in Table 4 demonstrate the difference
between the anatomic and functional regions affected by the aneurysm. The blue areas in Figure 7 represent regions of decreased wall thickness and regional function assessed by thickening (less than 2 mm), respectively. It is clearly seen that the zone of dysfunction (abnormal thickening) is significantly larger than the size of the aneurysm (abnormal thickness).

With reference to Figures 5 and 6, it is seen that the anatomic aneurysm and the aneurysm's adjacent IB have very similar characteristics. By comparison, the other three zones exhibit highly different geometric characteristics. This pattern is consistent in all of the eight hearts investigated. The aneurysm and IB zones have average wall thicknesses of 3.7±0.4 and 5.0±0.3 mm, respectively, with practically no change from end diastole to end systole (Table 4 and Figure 6). The OB zone has a mean end-diastolic wall thickness of 7.9±1.0 mm and an insignificant mean thickening of 0.34±0.43 mm (3.4±5.9%). The two normal zones, AN and RN, exhibit almost identical end-diastolic wall thicknesses (10.5±2.0 and 10.9±2.0 mm, respectively; p=NS). However, their thickening differs markedly, ranging from 2.24 mm (22%) in the AN zone near the aneurysm to 4.43 mm (43%) in the remote RN zone (p<0.01). Wall motion is practically negligible in the aneurysm and its borders (=1 mm), increasing toward the remote RN zone, which exhibits normal wall motion (8.6±3.4 mm).

As evident from Table 4 and Figure 8, the circumferential curvature is small in all regions, averaging 0.26 cm⁻¹ at end diastole (compared with 0.42 cm⁻¹ in normal hearts) and 0.30 cm⁻¹ at end systole (compared with the normal value of 0.9 cm⁻¹). Highest curvature values are found in the aneurysm and the IB zones (=0.3 cm⁻¹), whereas the smallest curvatures are found in the AN zone (0.17 cm⁻¹ at end diastole) (Figure 8). In general, the change in curvature from end diastole to end systole is much smaller compared with normal, especially in the aneurysm zone.

Curvature depends on both the overall size and the local shape. The circumferential curvature was normalized for size by reference to the average global curvature. Because the normalized curvature is inde-

![Figure 6](http://circ.ahajournals.org/)

**Figure 6.** Plots of regional end-diastolic (ED) wall thickness and thickening in aneurysmal left ventricles. Note significantly decreased thickening but normal ED thickness extending into adjacent normal zone. There was a significant difference among the five zones (p<0.001) for both thickening and ED thickness as studied by analysis of variance.

### Table 3. Surface Areas of Aneurysm and of Zone of Functional Impairment as Percent of Total Left Ventricular Surface Versus Global Ejection Fraction

<table>
<thead>
<tr>
<th>Patient</th>
<th>Aneurysm (1A)</th>
<th>Aneurysm (1B)</th>
<th>Thickening (&lt; 1 mm)</th>
<th>Thickening (&lt; 2 mm)</th>
<th>Ejection fraction (3A)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(aneurysmal region border plus inner border)</td>
<td>(aneurysmal region border plus inner and outer borders)</td>
<td>(2A)</td>
<td>(2B)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>30.8</td>
<td>44.2</td>
<td>44.0</td>
<td>54.6</td>
<td>22.0</td>
</tr>
<tr>
<td>2</td>
<td>43.1</td>
<td>58.8</td>
<td>61.7</td>
<td>74.8</td>
<td>12.3</td>
</tr>
<tr>
<td>3</td>
<td>41.4</td>
<td>56.1</td>
<td>57.9</td>
<td>86.6</td>
<td>14.4</td>
</tr>
<tr>
<td>4</td>
<td>33.3</td>
<td>45.7</td>
<td>44.1</td>
<td>49.0</td>
<td>27.9</td>
</tr>
<tr>
<td>5</td>
<td>23.0</td>
<td>36.4</td>
<td>32.9</td>
<td>45.6</td>
<td>34.0</td>
</tr>
<tr>
<td>6</td>
<td>35.8</td>
<td>53.9</td>
<td>51.9</td>
<td>57.1</td>
<td>25.2</td>
</tr>
<tr>
<td>7</td>
<td>37.8</td>
<td>53.2</td>
<td>50.8</td>
<td>63.0</td>
<td>26.9</td>
</tr>
<tr>
<td>8</td>
<td>23.7</td>
<td>38.3</td>
<td>49.0</td>
<td>56.6</td>
<td>31.5</td>
</tr>
</tbody>
</table>

Mean±SD (%) 33.6±7.5 48.3±8.4 49.0±8.9 60.9±13.7 24.3±7.7

Correction coefficients: 1A versus 2A, r=0.78, p=0.009; 1A versus 2B, r=0.83, p=0.004; 1A versus 3A, r=-0.86, p=0.002; 1B versus 2A, r=0.77, p=0.011; 1B versus 2B, r=0.86, p=0.002; 1B versus 3A, r=-0.83, p=0.004; 2A versus 3A, r=-0.85, p=0.003; 2B versus 3A, r=-0.82, p=0.005.
pendent of the size of the left ventricle, it reflects the regional variations in curvatures. Compared with the normal heart value, the AN zone has a significantly lower normalized curvature ($p<0.05$), whereas the IB zone has a significantly higher normalized curvature ($p<0.01$).

The meridional curvature, on the other hand, is seen in Table 4 to increase (0.10 cm$^{-1}$) compared with the normal global value of 0.08 cm$^{-1}$ at end diastole ($p<0.05$). The RN zone has higher meridional curvatures than the remainder of the left ventricle.

As also seen in Figure 9 and Table 4, the regional stress indexes in the aneurysmal zone are significantly higher than those of the normal zones. Most interesting is the difference between the two normal zones. The AN zone close to the aneurysm has a significantly greater stress index than the more remote RN zone, at both end diastole ($p<0.01$) and end systole ($p<0.05$) (Figure 9). Also, it is noteworthy that the difference between $\sigma/P$ values at end diastole and end systole is significantly reduced at the aneurysmal zone compared with the RN zone.

Comparison of the wall stress at end systole, as calculated by some available equations, is presented in Table 5 (column A) for the different zones. The circumferential Janz stresses$^{25}$ are close to the cylindrical and conical approximations, and both are larger than the spherical approximation, which is identical to Janz's meridional stress. However, it is noted that despite the differences in the stress indexes calculated by the different formulas, the relative stress values of the different zones in the same left ventricle do not change significantly. Clearly, the qualitative conclusions regarding the load distribution presented in this article are essentially insensitive to the type of stress formula selected.

The absolute values of the end-systolic stress in the different zones have been calculated in six patients for whom we had end-systolic or peak systolic blood pressure from cardiac catheterization. In patients for whom only the peak pressure was available ($n=3$), it was assumed to approximate the end-systolic pressure. Table 5 presents the calculated values based on Janz's equations for circumferential and meridional (spherical) stresses. Absolute values of end-systolic stress were also calculated in the normal volunteers, using systolic cuff blood pressure as an estimate of end-systolic pressure and multiplying by $\sigma/P$ indexes for each patient. Spherical stress averaged 58±10 Kdyne/ cm$^2$, whereas Janz's circumferential stress yielded 158±20 Kdyne/cm$^2$. Note the markedly higher stresses in the aneurysmal heart at and around the aneurysm. In comparison, the modified Janz formula for circumferential stress yields values of 87–213 Kdyne/cm$^2$ in normal patients and 186–378 Kdyne/cm$^2$ in patients with dilated cardiomyopathy.$^{32}$

**Comparison With Regional Heterogeneity in the Normal Heart**

The regional heterogeneity of the various geometric parameters of the aneurysmal left ventricle is compared in Table 4 with the regional values in the corresponding zones in the group of normal hearts. Similar to the aneurysmal heart, the regional variations in the normal heart were studied by analysis of variance. In addition, comparisons between the matched zones in the normal and aneurysmal hearts

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**FIGURE 7.** Three-dimensional reconstruction of a left ventricle containing an aneurysm, in which end-diastolic (ED) thickness (left panel) and thickening (right panel) are color coded. Right ventricular endocardium is also shown for reference. An approximated left anterior oblique view is shown. Red indicates normal thickness or thickening, whereas blue signifies abnormal values. A color-coded scale is given in each picture. A schematic of coronary arterial tree is superimposed for orientation with site of 100% coronary obstruction in proximal left anterior descending coronary artery. Note that zone of abnormal thickening extends well beyond zone of reduced thickness. (Three-dimensional display algorithm was developed by Halmann et al.$^{42}$)
Table 4. Comparison of Characteristic Local Parameters in the Five Zones of the Eight Aneurysmal Hearts With Corresponding Matched Regions in a Group of Normal Hearts

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Aneurysm</th>
<th>Inner border</th>
<th>Outer border</th>
<th>Adjacent normal</th>
<th>Remote normal</th>
<th>Analysis of variance (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>End-diastolic thickness (mm)</td>
<td>3.7±0.4*</td>
<td>5.0±0.3*</td>
<td>7.9±1.0</td>
<td>10.5±2.0</td>
<td>10.9±2.0†</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>7.5±0.9</td>
<td>8.0±0.7</td>
<td>8.8±0.6</td>
<td>9.2±0.5</td>
<td>9.3±0.5</td>
</tr>
<tr>
<td>Thickening (mm)</td>
<td>0.0±0.4*</td>
<td>0.0±0.4*</td>
<td>0.3±0.4*</td>
<td>2.2±0.8*</td>
<td>4.4±1.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>5.2±0.9</td>
<td>5.9±0.9</td>
<td>6.0±0.7</td>
<td>5.9±0.7</td>
<td>5.8±0.9</td>
</tr>
<tr>
<td>Motion (mm)</td>
<td>1.2±1.1*</td>
<td>1.1±1.0*</td>
<td>1.8±1.0*</td>
<td>4.6±1.4*</td>
<td>8.6±3.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>7.2±1.3</td>
<td>8.9±1.2</td>
<td>9.8±1.8</td>
<td>10.6±1.5</td>
<td>9.3±1.9</td>
</tr>
<tr>
<td>End-diastolic k (cm⁻¹)</td>
<td>0.28±0.05*</td>
<td>0.32±0.03*</td>
<td>0.26±0.04*</td>
<td>0.17±0.06*</td>
<td>0.25±0.02*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>0.50±0.06</td>
<td>0.42±0.06</td>
<td>0.39±0.03</td>
<td>0.36±0.05</td>
<td>0.41±0.03</td>
</tr>
<tr>
<td>End-diastolic normalized k</td>
<td>1.12±0.11</td>
<td>1.23±0.15*</td>
<td>0.97±0.12</td>
<td>0.65±0.19†</td>
<td>1.02±0.08</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>1.20±0.08</td>
<td>1.01±0.10</td>
<td>0.92±0.06</td>
<td>0.86±0.08</td>
<td>0.99±0.03</td>
</tr>
<tr>
<td>End-systolic k (cm⁻¹)</td>
<td>0.30±0.07*</td>
<td>0.35±0.06*</td>
<td>0.32±0.04*</td>
<td>0.23±0.05*</td>
<td>0.29±0.05*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>1.18±0.37</td>
<td>0.88±0.35</td>
<td>0.81±0.2</td>
<td>0.81±0.25</td>
<td>0.83±0.14</td>
</tr>
<tr>
<td>End-systolic normalized k</td>
<td>1.01±0.17*</td>
<td>1.17±0.13*</td>
<td>1.06±0.1*</td>
<td>0.76±0.11*</td>
<td>0.98±0.04</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>1.32±0.12</td>
<td>0.98±0.11</td>
<td>0.89±0.09</td>
<td>0.88±0.11</td>
<td>0.93±0.1</td>
</tr>
<tr>
<td>End-diastolic k (cm⁻¹)</td>
<td>0.06±0.07</td>
<td>0.09±0.03</td>
<td>0.08±0.05</td>
<td>0.08±0.04</td>
<td>0.15±0.05</td>
<td>0.020</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>0.07±0.03</td>
<td>0.07±0.02</td>
<td>0.05±0.02</td>
<td>0.06±0.03</td>
<td>0.11±0.04</td>
</tr>
<tr>
<td>End-systolic k (cm⁻¹)</td>
<td>0.02±0.10</td>
<td>0.10±0.03*</td>
<td>0.07±0.03†</td>
<td>0.05±0.04</td>
<td>0.11±0.06</td>
<td>0.050</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>0.02±0.04</td>
<td>0.03±0.06</td>
<td>0.04±0.03</td>
<td>0.05±0.04</td>
<td>0.10±0.09</td>
</tr>
<tr>
<td>End-diastolic σP</td>
<td>4.92±1.03*</td>
<td>3.40±0.45*</td>
<td>2.91±0.44*</td>
<td>3.09±1.05*</td>
<td>1.96±0.31*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>1.32±0.32</td>
<td>1.40±0.21</td>
<td>1.45±0.13</td>
<td>1.47±0.27</td>
<td>1.26±0.12</td>
</tr>
<tr>
<td>End-systolic σP</td>
<td>4.65±1.29*</td>
<td>3.16±0.71*</td>
<td>2.37±0.52*</td>
<td>2.23±0.53*</td>
<td>1.20±0.35*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>0.28±0.09</td>
<td>0.39±0.10</td>
<td>0.41±0.12</td>
<td>0.46±0.17</td>
<td>0.37±0.07</td>
</tr>
</tbody>
</table>

Values are expressed as mean±SD.
Analysis of variance is performed among the five zones in each row.
*p<0.01 and †p<0.05 indicate that value is significantly different from that expected in a normal left ventricle at same location using nonpaired t test.

were carried out by nonpaired t test and are given in the table. In general, although regional variations in end-diastolic thickness (p<0.0001), thickening (p=0.007), and motion (p<0.0001) exist in the normal heart, this variability is clearly different from that seen in the aneurysmal heart. Specifically, although the AN and the RN zones are equal in thickness in both normal and aneurysmal hearts (p=NS), there is no difference between them in motion or thickening in the normal hearts (p=NS) in contrast to a significant difference in motion (p<0.01) and thickening (p<0.01) in the aneurysmal hearts.

The comparison of the aneurysmal LV data with that of the normal hearts demonstrates clearly that the regional variations in the diseased hearts result from pathological distortion, not from regional variations in the typical shape of the left ventricle. Similarly, despite the evident regional variability in the circumferential curvature in the normal heart at end systole (p<0.0001), there are no significant differences between the AN and the RN zones in the normal heart. Also, there is no difference between the AN and the RN zones with regard to the end-systolic wall stress of the normal left ventricle, whereas a significant difference (p<0.01) between the two zones is seen in the aneurysmal hearts. Obviously, the stresses at all zones of the aneurysmal hearts are severalfold higher than those in the normal heart.
Discussion

The left ventricle with an aneurysm has commonly been regarded as having a localized nonfunctional LV wall section, whereas the residual myocardium exhibits either normal or depressed function. The impaired global LV function has been largely related to an exaggerated local effect of the aneurysm, assuming it to be a noncontractile and/or paradoxically expanding zone. It has also been recognized that a left ventricle containing an aneurysm that occupies more than 20% of LV surface area becomes dilated. This physiological change maintains a normal stroke volume by means of the Starling mechanism, consequent increasing the load on the left ventricle and its total work. In fact, little has been quantified and documented concerning the regional function in the aneurysmal left ventricle or the effect of the aneurysm on the surrounding, adjacent, and remote normal myocardial tissue.

Aneurysm Size Versus Regional Function

The assessment of the size of an aneurysm is presently based on emulating its impaired function, usually by determining the size of the zone of abnormal motion in the left ventricle. This method usually overestimates the size of the aneurysm compared with the size of the aneurysm as measured by echocardiography.

Table 5. Systolic Stress Values Based on Six Patients

<table>
<thead>
<tr>
<th>Model*</th>
<th>Aneurysm</th>
<th>Inner border</th>
<th>Outer border</th>
<th>Adjacent normal</th>
<th>Remote normal</th>
<th>Global</th>
<th>Analysis of variance (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Janz $\sigma_m$ (spherical)</td>
<td>584±157</td>
<td>417±84</td>
<td>325±47</td>
<td>320±76</td>
<td>166±38</td>
<td>339±76</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Janz $\sigma_c$</td>
<td>993±342</td>
<td>998±200</td>
<td>741±137</td>
<td>474±109</td>
<td>456±169</td>
<td>697±160</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values are given in Kdyn/cm².

In three patients, end-systolic pressures were available, whereas in three other patients, peak systolic pressures were available and used to estimate end-systolic pressures.

*Equations used:
Cylinder, $\sigma$: $\sigma/P = \pi/t$
Cone, $\sigma$: $\sigma/P = r_e/t$
Janz $\sigma_m$: $(\sigma/P)_m = r^2/[2t\sin\gamma(r + t\sin\gamma/2)]$
Janz $\sigma_c$: $(\sigma/P)_c = r_e \cdot r \cdot (2 - r_e \cdot \sin\gamma)/[2t\sin\gamma(r_e + t/2)]$

$r, r_e$, Three-dimensional circumferential and meridional radii of curvature; $\gamma$, angle between normal to endocardium and left ventricular long axis; $r$, two-dimensional circumferential radius of curvature; $r_e, r\sin\gamma, t$, wall thickness; $\sigma_c, \sigma_m$, circumferential and meridional stress.
with surgery. In the present study, the aneurysm's size is assessed anatomically, based on end-diastolic wall thickness, and compared with the left ventricle's regional function. The data show that reduced function extends well beyond the anatomic border of the aneurysm. For example, its OB (Figure 5) is practically akinetic despite having an average end-diastolic wall thickness of 8 mm, which is within the normal range. Furthermore, the two normal zones (AN and RN) have normal wall thicknesses but demonstrate vastly different functional characteristics, as evident by thickening and by wall motion. This finding may have prognostic and therapeutic importance because it may help clinicians in accurately assessing aneurysm size in relation to adjacent normal and remote region size and thus in assessing possible need for aneurysmectomy. A similar phenomenon is described in acute ischemia, where the zone surrounding the ischemic region has normal perfusion but reduced function. In their intraoperative, echocardiographic study, Nicolosi and Spotnitz noted reduced function outside the zone of the anatomic aneurysm, defined in terms of thinning of the wall close to the aneurysm border during isovolumic systole. However, they found no significant difference in thickening, at end systole, between aneurysm and border and remote zones. Arvan and Badillo found in patients with anterior aneurysms that percent fractional shortening is highest at the base (29±2%), decreasing toward mid left ventricle (22±2%) and the apex (3±1%).

The physiological mechanism that explains the phenomenon of reduced function beyond the border of the aneurysm is not clear. It may be a local tethering effect of the stiff aneurysm; it may be that the aneurysm does not end abruptly but sends out fibrous strands to the nearby tissues; and it may represent increased afterload secondary to LV dilation or to geometric remodeling of the left ventricle. The finding of four regions in the left ventricle with vastly differing geometric properties indicates that geometric remodeling occurs in these hearts. The two normal zones have remarkably different circumferential curvatures: the RN zone is more curved than the very flat AN zone close to the aneurysm.

To investigate the possibility that remodeling of the left ventricle can affect local function by increasing the regional myocardial load, we calculated the local stress indexes in each region by the Laplace equation, assuming spherical geometry in each zone. Although the calculation of stress in a diseased ventricle is controversial and has not been validated, comparison with calculated values using other formulas (Table 6) shows that the approach we used may reflect the actual differences between regions. Clearly, the aneurysm and its border showed high stress values. The two normal zones exhibit different radii of curvature and markedly different local stresses, with the larger values occurring in the AN zone (Figures 8 and 9). This suggests that an increased afterload in the AN zone may be the major mechanism responsible for the reduced function of this zone.

**Limitation of Stress Formulas**

As mentioned above, the local stress indexes are approximated by the Laplace equation for a sphere and yield only rough estimates of the regional stresses in the myocardium. The validity of this simplified stress formula to accurately predict the local wall stress is doubtful, and comparison with different stress formulas (Table 6) shows the variability of the stresses obtained by the different approaches. The spherical stress approximation is identical in magnitude to Janz's meridional stresses, whereas Janz's circumferential stress approximations as well as the cylindrical and conical based approximations are approximately double the spherical stresses. Because only relative regional values, not absolute values, are considered here, any calculation procedure is probably sufficient to give a reasonable comparison between the different zones in the myocardium. It is interesting that comparison of stresses predicted by Janz's equation with a finite element model of the left ventricle has shown that the simplified formulas can yield a good approximation despite their limitations.

**Comparison With Normal Hearts**

Because all of the aneurysms included in the present study were anteroseptal, it was possible to
define the average location of the aneurysms of the entire group and to then compare this representative location with each of the 10 normal left ventricles. The cardiac helical coordinates used here allowed us to normalize each heart; by using the midseptum as a reference, we can easily achieve regional matching between different hearts, regardless of the position and spatial orientation of the left ventricle. Differences in the volumes of the normal and aneurysmal hearts may also affect the curvatures. However, the use of normalized curvatures allows regional comparison between the two groups of hearts studied. Stresses, however, are not normalized for size because we are mainly interested in regional variations within the aneurysmal left ventricles. One can easily compare the regional variation in the aneurysmal left ventricle with that in the normal left ventricle by visual inspection. The results of the comparative analysis show that although some regional variations in both thickening and curvatures may exist in the normal heart, the regional changes observed in the aneurysmal hearts are clearly a result of pathological distortion and are not related to the specific location of the aneurysm.

**Paradoxical Aneurysm Expansion**

It is often noted in angiography that aneurysmal areas undergo paradoxical expansion (i.e., the endocardium appears to move outward during systole). However, it is questionable whether this is a true paradoxical expansion, especially in cases of stiff fibrous aneurysms, like those studied in the present report. Some exponents claim that the high pressure during systole stretches the passive aneurysm. However, fibrous aneurysms are significantly stiff than the normal myocardium, and it is therefore unlikely that they will stretch more than approximately 3% of their diastolic length. One possibility is that the poorly functioning yet viable muscle at the border of the anatomic aneurysm undergoes stretching, giving the impression that the entire aneurysm stretches. Other alternatives consider changes in the absolute position because of the movement of the left ventricle as a whole but with no movement of the aneurysmal zone relative to the remainder of the heart. The present data show that the average wall thickness of the aneurysm and its border do not change from end diastole to end systole, indicating the unlikelihood of any major stretching. It is interesting to note that some specific areas in the various hearts appear to undergo some thinning. Although this finding may be true, it is more likely that it is a consequence of tracing and normalization errors. Similarly, in these particular hearts, motion of the aneurysm wall averages zero, whereas various areas demonstrate small degrees of negative motion.

**Effect of Thrombus**

Five of the eight left ventricles contained thrombi. Of these, two were large (31% and 20% of the cavity volume at end diastole) and three were small (taking up an average of 8% of the cavity). Although the effect of thrombi on global LV function is unknown, it is clear that they form a dead space inside the left ventricle, thereby increasing LV volume and hindering blood flow. On the other hand, the thrombus may effectively block up a nonfunctioning or even paradoxically expanding portion of the left ventricle and decrease the cavity volume without affecting stroke volume, thereby improving the ejection fraction. Comparing these two possibilities, our calculations of volumes and ejection fractions (Table 1) show that a better correlation exists between ejection fraction and the size of the aneurysm when the thrombus is considered part of the LV cavity. This suggests that a large thrombus may give misleading results concerning ventricular function when assessing volumes and ejection fractions by angiographic techniques that rely on blood volume.

**Effect of Left Ventricular Twist**

LV twist is not accounted for in the present or any other study of regional aneurysm. Theoretically, the twist can affect the measurements that depend on local changes from end diastole to end systole (i.e., thickening or wall motion). Curvature and stress do not depend on the twist. As has been observed in the hearts of normal dogs and humans, the twist is largest toward the apex, where the angular rotation is maximal (10–15°). The angular rotation near the equator is usually minimal; therefore, the measurement error at the midventricular zones resulting from this factor is quite negligible. As is well known, the twisting motion depends on fiber shortening. Not surprisingly, the angle of twist is reduced in acute ischemia, where shortening is reduced. Similarly, the twist is expected to be very small in the aneurysmal hearts, where shortening is markedly reduced.

The twist could not be measured in the present study, nor has it been measured in aneurysmal hearts elsewhere. However, the visual impression is that the borders of the aneurysm, which could be clearly seen in most patients, did not move from end diastole to end systole, and it is reasonable to expect the effects of the twist to be very small in these patients.

Each volume element in the present study takes up 15° of the circumference. This means that the maximal shift resulting from torsion is half an element for the normal heart (where each region in this study is at least two elements wide, or 30°) and much less than that for the aneurysmal heart. Therefore, the twist of the aneurysmal heart should have only slight effects on the measurements of thickening and motion in the different regions; this effect may be of some importance in the normal heart, particularly toward the apex.

**Study Limitations**

Although the imaging and quantitative techniques we described can be applied to any type of LV pathology, the results of the present study are limited to a very specific, homogeneous group of left ventri-
circles with chronic fibrous anterior wall aneurysms. Thus, one should be careful in extrapolating the results quantitatively to LV aneurysms in general.

The studied images of each left ventricle undergo extensive processing. Each stage may add error. For example, tracing of the endocardial and epicardial borders may be erroneous where the border is unclear, such as when mural thrombi are present. Pixel resolution is either 1.37 or 1.86 mm/pixel, and these are the orders of magnitude of errors that can be expected because of tracing. The reconstruction algorithm involves interpolation, especially in the longitudinal direction (i.e., perpendicular to the image plane). This step creates relatively smooth surfaces but may eliminate small local geometric details. Fortunately, most of the needed information is at a frequency range lower than the interslice gap.

The rather good accuracies of the calculated global and regional geometric parameters were previously validated.20-22 The errors of the helical three-dimensional reconstruction procedure in assessment of thickness and thickening were found to be negligible. The accuracy of the values obtained depends mainly on the correctness of the tracing, which was based on general agreement among three trained observers. Intraobserver and interobserver variabilities of approximately 0.6 mm for the radial distance in any point on the contour were found. The maximal errors in thickness and thickening estimation between these observers were 8% and 16%, respectively. The exception is the meridional curvature, which is associated with a low spatial resolution, and the results that include the meridional curvatures should therefore be interpreted with caution.

Although following a material point from end diastole to end systole is still a goal to be pursued, the procedure described in the present study provides a general algorithm for end diastole to end systole matching. Some noise is inevitable, but the amount of noise is reduced considerably by using an average for a number of elements; this procedure ensures that a larger portion of the areas being compared would be followed from end diastole to end systole.

**Conclusions**

The present study analyses the regional three-dimensional geometry and function of a group of fibrous LV aneurysms by using noninvasively obtained cine-CT scans to reconstruct the three-dimensional geometry of each left ventricle. The analysis provides new understanding and detailed insight into the shape and function of the aneurysmal left ventricle. The study demonstrates a quantitative procedure that accurately relates and maps the anatomic entity and the regional function associated with pathological constraints. The regional function, mapped at different distances from the anatomic aneurysmal zone, is shown to be significantly abnormal in the apparently normal myocardium bordering the aneurysm. This abnormality extends up to a distance of approximately 3–4 cm from the border despite the fact that this area has a normal baseline wall thickness. It is suggested that this phenomenon is at least in part related to some geometric remodeling of the left ventricle, which produces a zone of low curvature in the muscle adjacent to the aneurysm border, with resultant high myocardial stresses that serve as a high afterload and dampen normal myocardial contraction. The size of the abnormally functioning zone is almost twice the size of the aneurysm, and high correlations exist among the size of the aneurysm, the size of the dysfunctional zone, and the global LV function, indicating that these three factors are closely related to one another.

The technique we presented can eventually be used to present accurate and detailed anatomic and functional information in a compact, visual form to the cardiologist and cardiac surgeon. Surgical and clinical management may be improved by assessing the residual myocardial size and function. Not the least exciting is the potential to stimulate surgery on the computer and predict functional outcome from knowledge of the residual regional function and projected LV surgical remodeling.

**Acknowledgment**

Particular thanks are due Dr. David Gutterman, Division of Cardiology, Iowa City University Hospital, Iowa, for his tireless help in obtaining the cine-CT data.

**Appendix**

**Calculation of Principal Radii of Curvature**

The principal radii in the circumferential ($r_c$) and meridional direction ($r_m$) are calculated as follows.

Local tangents to the endocardial surface are approximated and used in the following equation:

$$K_j = \frac{|\Delta t_i|}{\Delta s}$$

where $K_j$ is curvature in the j direction ($j$ is c when circumferential and m when meridional), $t_i$ is tangent vector at points $i=a, b$, $\Delta s$ is segment length between points $a$ and $b$, and $\Delta t$ is the vector difference between tangents at points $a$ and $b$.

The curvature in a plane perpendicular to the endocardial surface is then given by the following equation:

$$K_{B_i} = K_j \cos \theta$$

where $\theta$ is the angle between the normal ($n_i$) in the plane containing the data points and a local normal ($N_i$) to the endocardium. The latter is the vector (A, B, C) where A, B, and C are the coefficients of the local tangent plane at the surface:

$$Ax + By + Cz = D$$

This local tangent plane is estimated by least-squares fitting of four neighboring points on the
surface. The respective radius (r) is then given by the following:

\[ r_j = \frac{1}{K_j} \]

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**Key Words**: wall thickness, thickening, curvature, left ventricular wall stress
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