Quantitative Analysis of Regional Systolic Function With Left Ventricular Aneurysm

Alfred C. Nicolosi, MD, and Henry M. Spotnitz, MD

Left ventricular aneurysm (LVA) remains a poorly understood entity, often resulting in congestive failure that is not consistently improved by standard resection with linear closure. Although other surgical approaches have been proposed, current methods to assess their effect on left ventricular function are not adequate. The purpose of the present study was to quantitatively define regional systolic function in patients with LVA and to assess acute changes in regional function after standard repair. Seven patients underwent resection of an anteropapical LVA. Intraoperative two-dimensional echocardiography was performed off cardiopulmonary bypass immediately before and after resection. In all patients, short-axis views at the papillary muscle (apex) level showed anteroseptal paradox and distorted geometry, whereas at the mitral valve (base), symmetric wall motion and geometry were preserved. Videotaped echo images were divided into octants by a floating axis fitted to internal landmarks. Myocardial area and midwall perimeter were obtained for each octant, and wall thickness was calculated at end diastole (ED), isovolumetric systole (IS), and end systole (ES). Wall thickening (Δt) for each segment was calculated as the percent increase in thickness from ED and averaged for all seven patients. At the apex level before resection, isovolumetric thinning occurred in the aneurysm as well as bordering segments, with Δt ranging from -17±5% (±SEM) in the anteroseptal segment to 12±6% posterolaterally (p<0.05). The isovolumetric bulge was followed by late-systolic thickening, however, with Δt ranging from 13±7% to 27±8% (NS). Systolic function at the base was symmetric, with Δt ranging from 12±3% to 18±3% at IS (NS) and from 20±6% to 26±6% (NS) at ES. After resection, isovolumetric function at the apex improved significantly, with thickening present in all segments. In the posteroseptal segment, Δt improved from -6±3% before repair to 11±6% after repair (p<0.05) and improved in the septal segment from -8±4% to 9±5% (p<0.05). End-systolic thickening was not significantly changed from prerepair values at either the apex or base. These data define the nature of regional systolic function with anteropapical LVA. Paradoxic wall thinning occurs not only in the aneurysm itself but in border zones as well. Thinning is maximal at isovolumetric systole and is followed by net end-systolic thickening. Normal systolic function is maintained in posterior segments and at the base of the heart. LVA resection eliminates the aneurysm itself and normalizes isovolumetric function of the border segments. This method of analysis will allow objective assessment of newer forms of LVA repair. (Circulation 1988;78:856–862)
Currently, it has been suggested by Jatene and Hutchins that standard aneurysmectomy with linear repair causes greater geometric distortion (and thus a greater mechanical disadvantage) than existed before surgery. This concern has led to proposals for geometric reconstruction of the left ventricle after aneurysm resection.

The heterogeneous nature of both the tissue properties and geometry of the aneurysmal ventricle have led to a poor understanding of this entity. The differences in systolic function of aneurysmal segment, border zones, and uninvolved areas must be better defined. Also, the effects of standard repair must be clear before alternate surgical approaches are adopted. The purpose of the present study is to define in a quantitative fashion regional systolic function in patients with left ventricular aneurysm with two-dimensional echocardiography and to assess the effects of standard aneurysm repair on regional performance.

**Patients and Methods**

The records and intraoperative echocardiograms of 19 consecutive patients who underwent repair of a left ventricular aneurysm by one surgeon during an 8-year period were reviewed. Twelve patients were excluded because echocardiographic studies were either incomplete or inadequate for analysis, leaving seven patients to form the basis of this report. Ages ranged from 42 to 72 years (mean, 61.3 years), and all seven patients had anteroapical aneurysms resulting from ischemic heart disease. Aneurysmectomy was accomplished under cardiopulmonary bypass (CPB) with crystalloid cardioplegia and moderate systemic hypothermia in the following manner: an incision was made lengthwise through the scar parallel to the long axis of the left ventricle, and the aneurysm was resected leaving a 1-cm rim of fibrous tissue. Mural thrombus was evacuated, and in four patients endocardial scar was removed as well. The edges of the ventriculotomy were then reapproximated in a linear fashion with interrupted horizontal mattress sutures and a two-layered continuous closure. Paired Teflon felt strips, 1-cm wide and equal in length to the ventriculotomy, were used to reinforce the suture line. Six patients had concomitant myocardial revascularization.

Histological findings consistent with ventricular aneurysm were noted in all cases and included transmural infarction, wall thinning, and fibrous replacement of myocardium. The degree to which viable myocardial tissue was present within the aneurysmal segment varied from patient to patient and within the same specimen. In some areas, a dense fibroblastic reaction entirely replaced muscle tissue, whereas in others, islands of viable myocytes were found.

Intraoperative two-dimensional echocardiography was performed off CPB with a hand-held, gas-sterilized, 3.5-MHz phased-array transducer (Diasonics CV3400R) immediately before and after LVA repair. Short-axis cross sections were obtained at four levels: base and tips of the mitral valve, midventricle, and papillary muscles. Images were recorded and stored on videotape for analysis. Echocardiograms were analyzed at three time points: end diastole (ED), which was defined as the onset of the QRS complex of the electrocardiogram; maximal paradox (IS), which occurred one or two video frames after the end of the QRS; and end systole (ES), which was defined as the smallest cavity diameter in association with the T wave.

The technique for regional analysis was adapted from a method described by Nieminen et al. Echocardiographic images were traced from the video monitor with tracing paper, and a reference line was drawn through the papillary muscles at the apex and mitral valve at the base. The image (excluding papillary muscles) and reference line were transferred to a computer (Apple Macintosh 512E) with an x-y digitizing tablet (MacTablet, Summagraphics Inc, Fairfield, Connecticut), and a floating eight-segment axis was superimposed on the image with commercial software (MacDraft). This axis was then centered on the cardiac image automatically by the software that aligns the midpoints of both the top-to-bottom and side-to-side margin of each object. Finally, the axis was rotated so that the 0° line coincided with the reference line to avoid errors in orientation (Figure 1). The anterior and anteroseptal segments correspond to the aneurysm itself, whereas the remainder of the ventricle is divided into anterolateral, lateral, posterolateral, posterior, posteroseptal, and septal regions. After resection of the anterior and anteroseptal segments, the circumference of the ventricle at the papillary muscle level was comprised of only the six remaining segments, with the edges of the septal and anterolateral segments in apposition. To reflect this, images through the papillary muscles after repair were divided into six segments. At the base,
which was not involved in the resection, images were divided into octants both before and after repair. The area of each segment was obtained by planimetry and divided by midoctantal chord length to obtain a mean segmental thickness. Systolic wall thickening was derived with the formula:

$$\Delta t = \left[ \frac{(tS - tED)}{tED} \right] \times 100$$

where $\Delta t$ is the percent change in wall thickness from end diastole, $tS$ is wall thickness during systole, and $tED$ is wall thickness at end diastole. Values for $\Delta t$ were averaged for the seven patients in each segment.

Systolic thickening of the various segments at a particular level were compared with ANOVA. Segments at the apex were also compared with those at the base with ANOVA. For each segment, after-repair systolic thickening was compared with the postrepair value by paired $t$ test. Statistical significance was defined as $p<0.05$.

**Results**

Qualitative review of the echocardiograms revealed paradoxic wall motion and geometric distortion on short-axis views through the papillary muscles (apex) in all seven patients. In comparison, sections through the base demonstrated normal wall motion and preservation of symmetric cross-sectional geometry. Maximum paradox occurred early in systole, shortly after the end of the QRS complex of the electrocardiogram. Intracavitary high-fidelity left ventricular pressure recordings available in one patient indicated that maximal paradox occurred at the end of isovolumetric systole.

Computerized images from a representative case are shown in Figure 2. Both the anterolateral border segment and uninvolved posterolateral segments have been shaded, and wall thickening is represented as the percent change in thickness from end diastole. Before repair, isovolumetric wall thinning ($\Delta t = -14\%$) is noted in the anterolateral region, followed by net end-systolic thickening ($\Delta t = 19\%$). In the uninvolved posterolateral segment, net thickening occurs throughout systole. After repair, wall thickening is seen at both isovolumetric ($\Delta t = 3\%$) and end systole ($\Delta t = 17\%$) in the anterolateral segment, while in the posterolateral segment systolic wall thickening is unchanged.

Mean results for the seven patients are presented in Table 1 and in Figures 3 (before repair) and 4.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Computerized short-axis two-dimensional echocardiographic sections through the papillary muscles in a patient with anterolateral left ventricular aneurysm. Wall thickening in the bordering anterolateral (AL) and uninvolved posterolateral (PL) segments is represented as the percent change from end-diastolic thickness. Other abbreviations are the same as in Figure 1. Before repair, isovolumetric thinning ($\Delta t = -14\%$) occurs in the anterolateral border zone and is followed by net end-systolic thickening ($\Delta t = +19\%$). Uninvolved posterolateral segment thickens throughout systole. After repair, isovolumetric thickening ($\Delta t = +3\%$) occurs in the anterolateral segment and continues to end systole ($+17\%$). Systolic function in the posterolateral segment is unchanged from before repair.
TABLE 1. Quantitative Analysis of Regional Wall Thickening Before and After Left Ventricular Aneurysm Repair

<table>
<thead>
<tr>
<th>Segment</th>
<th>Before repair</th>
<th>After repair</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apex</td>
<td>IS</td>
<td>ES</td>
</tr>
<tr>
<td>A</td>
<td>−11±6*</td>
<td>17±6</td>
</tr>
<tr>
<td>AL</td>
<td>−3±6*</td>
<td>21±7</td>
</tr>
<tr>
<td>±</td>
<td>5±6</td>
<td>24±5</td>
</tr>
<tr>
<td>PL</td>
<td>12±6</td>
<td>27±8</td>
</tr>
<tr>
<td>P</td>
<td>9±6</td>
<td>22±9</td>
</tr>
<tr>
<td>PS</td>
<td>−6±3*</td>
<td>16±5</td>
</tr>
<tr>
<td>S</td>
<td>−9±4*</td>
<td>13±7</td>
</tr>
<tr>
<td>AS</td>
<td>−17±5*</td>
<td>17±8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Base</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>12±3</td>
<td>23±5</td>
</tr>
<tr>
<td>AL</td>
<td>14±3</td>
<td>26±6</td>
</tr>
<tr>
<td>L</td>
<td>14±5</td>
<td>20±6</td>
</tr>
<tr>
<td>PL</td>
<td>15±4</td>
<td>24±4</td>
</tr>
<tr>
<td>P</td>
<td>16±5</td>
<td>25±8</td>
</tr>
<tr>
<td>PS</td>
<td>13±3</td>
<td>23±7</td>
</tr>
<tr>
<td>S</td>
<td>14±3</td>
<td>21±6</td>
</tr>
<tr>
<td>AS</td>
<td>18±3</td>
<td>25±6</td>
</tr>
</tbody>
</table>

Values represent percent change from end-diastolic thickness (mean ± SEM for seven patients).

IS, isovolumetric systole; ES, end systole.

*p < 0.05 vs. P and PL; †p < 0.05 vs. before repair IS.

(averaged). At the apex before repair, isovolumetric thinning occurred in the anterior (Δ t = −11 ± 6%) and anteroseptal (Δ t = −17 ± 5%) segments as expected because these represent the aneurysm itself (Figure 3). However, significant paradox was also seen in the bordering anterolateral (Δ t = −3 ± 6%), septal (Δ t = −9 ± 4%), and posteroseptal (Δ t = −6 ± 5%) segments as well. The isovolumetric thinning in these segments was statistically different from the thickening that occurred in the posterior (Δ t = 9 ± 6%) and posterolateral (Δ t = 12 ± 6%) segments (p < 0.05). By end systole, however, net thinning occurred in all eight segments (Δ t = 13 ± 7% to 27 ± 8%), regardless of the degree of thinning seen during the isovolumetric period, with no statistical differences among the eight segments. At the base, thickening occurred in symmetric fashion at both isovolumetric systole (Δ t = 12 ± 3% to 18 ± 3%) and end systole (Δ t = 20 ± 2% to 26 ± 6%), with no statistical differences among the eight segments at either point in time. The overall asymmetry at the apex during isovolumetric systole was statistically different from the base by ANOVA (p < 0.01), although no particular segment showed a statistical difference between the two levels. By end systole, overall function in the two regions was not significantly different. At the apex after resection, IS thickening improved in the posteroseptal segment from −6 ± 3% before repair to 11 ± 6% after repair (p < 0.05) and in the septal segment from −9 ± 4% to 9 ± 5% (p < 0.05). In the anterolateral segment, thickening increased from −3 ± 6% to 2 ± 5%, but this was not statistically significant. Net wall thickening progressed to end systole in all segments (Δ t = 10 ± 8% to 28 ± 7%), and the overall pattern more closely resembled the base, where systolic thickening was statistically unchanged from before repair.

A qualitative analysis of regional function with a summary of coronary anatomy is shown in Table 2. At the apical level before repair, a total of 22 segments from the seven patients showed isovolumetric paradox in addition to the anterior and anteroseptal segments that were to be resected. This was followed by end-systolic paradox in only one patient (four segments). All patients had significant atherosclerotic disease in the left anterior descending coronary artery, and all but one had stenoses of more than 50% of luminal diameter in the circumflex coronary artery or its branches. In addition, two patients had stenoses in the right or posterior descending coronary artery. After repair, a total of nine segments showed isovolumetric paradox, but of these, only four were among the original 22. Thus, 18 (82%) of the apical segments that were paradoxic before repair functioned normally.
afterward. Interestingly, eight (44%) of the segments did not fall in the distribution of a bypassed artery; hence, their improvement cannot be explained simply as a result of increased perfusion. Isovolumetric wall thinning disappeared completely in four patients. In two segments (one patient), persistent isovolumetric paradox was associated with new end-systolic paradox, suggesting acute ischemia in these segments, despite attempted revascularization. In the other two segments with persistent paradox, no revascularization was attempted. New isovolumetric paradox was seen in a total of five segments (four patients) and was associated with new end-systolic paradox in two, again suggesting new ischemia despite attempted revascularization. New end-systolic paradox was seen in a total of seven apical segments after resection.

Wall thickening in the separate segments at a particular level was summed and averaged to estimate global function at that level (Figure 5). Despite improved isovolumetric function at the apex with repair (1 ± 5% before repair increased to 10 ± 5% after repair), there was no parallel change in end-systolic function (20 ± 5% before repair decreased to 19 ± 5% after repair) nor did any significant changes in systolic function occur at the base with resection.

Discussion

The aneurysmal ventricle is at a mechanical disadvantage for several reasons. The curvature and thickness of the ventricular wall are determinants of afterload, and as changes in these parameters occur, significant changes in performance can be expected. A left ventricular aneurysm causes obvious localized geometric changes but leads to global cardiac remodeling with generalized dilatation as well. In addition, there are variations in the intrinsic tissue properties of scar, normal muscle, and bordering tissue that influence both active and passive function. Finally, the presence of a paradoxic segment reduces the efficiency of the ventricle as a whole because systolic work is wasted on expansion of this segment.

Because of the asymmetries involved, evaluation of left ventricular function is difficult in patients with LVA. Standard measurements of global function normally assume homogeneity and are thus not reliable for presurgery evaluation and prognostic assignment. Methods to assess regional function have therefore been used in an effort to improve overall evaluation. With conventional ventriculography to define contractile segment ejection fraction (CSEF) separate from global EF, the state of the residual myocardium has been correlated with presurgery global function as well as postsurgery result. Although CSEF correlates with postsurgery global function in large groups of patients, it cannot be relied on to predict outcome in individual cases. In addition, techniques that rely on standard contrast radiography do not relate important three-dimensional information, which is essential with LVA. Many recently developed techniques that combine newer imaging modalities with computer analysis may prove useful in evaluating these patients.

The present study uses an echocardiographic method of regional analysis in which short-axis images are divided radially into octants and segmental wall thickness is measured. Wall thickening is a more accurate measure of regional function than wall motion. In addition, short-axis sections can be analyzed at multiple apex-to-base levels, thereby providing the important three-dimensional information not available by contrast radiography. There are several potential problems with this particular method, however. The anatomic confines that exist with intraoperative echocardiography, especially with a large LVA that can be adherent to the sternum, provide one source of difficulty in that images must often be taken proximal to the greatest dimension of the aneurysm. In the present study, the apical sections always included the most proximal extent of resection as evidenced by an obvious suture line with Teflon strips on postrepair sections. Sections through the center of the aneurysm might...
be obtained in the future with a retrocardiac or transesophageal transducer. Second, we assumed that because all patients had the same general area of paradox (all had anteroapical LVA), the resection uniformly excised the anterior and anteroseptal segments. However, there was some individual variation in the exact dimensions of the aneurysm. After the two aneurysmal segments were resected, the edges were reapprroximated so that the remaining myocardium comprised the entire ventricular circumference. We approximated postresection geometry by dividing images at the papillary muscles into only six segments after repair. Although this may not have provided exact correspondence to prerepair segments, maintaining an eight-segment axis would have caused similar inaccuracy.

The present study defines the nature of regional systolic function with LVA. The aneurysmal segments, as well as border zones, displayed early systolic paradox but had developed net thickening by end systole. In the uninvolved lateral and posterior segments, net wall thickening was observed throughout the systolic cycle. Maximal paradox occurred slightly after the end of the QRS of a simultaneous electrocardiogram; correlated with other cardiac events, this coincided with opening of the aortic valve, when wall stress is normally peaking. In acute ischemia models, maximal paradox has also been noted to occur at isovolumetric systole26-28; however, in these models, some degree of net bulging remains at end systole. It appears then that with a healed scar, paradox occurs in the scar and bordering segments under high-systolic pressure (isovolumic contraction). Subsequently, under lower loads, the same segments exhibit net thickening and apparently contribute to ejection.

The underlying mechanism for this late-systolic recovery is uncertain. It may reflect a completely passive process in which early-systolic expansion of the scar is followed by strong elastic recoil as the load decreases. Histological examination of the resected specimens showed variable amounts of viable muscle cells imbedded within the scar. Areas with only scattered cells would not be expected to contribute significantly to systolic function; however, large nests of intact cells possibly could. A combination of these active and passive processes may explain late-systolic thickening. Although the surgical implications require further investigation, this phenomenon suggests that afterload reduction may be of benefit in the medical management of these patients in that it could reduce isovolumic paradox and enhance late-systolic thickening.

Standard aneurysm resection appears to affect isovolumetric function mainly by eliminating paradox in the border zones. It is unclear whether the effect is due to more favorable geometry in these segments, a reduction in global wall stress related to decreased chamber size, or simply better perfusion secondary to coronary bypass. As noted above,

---

**TABLE 2. Regional Paradox Before and After Resection of Left Ventricular Aneurysm**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Native coronary disease</th>
<th>Segments with paradox before repair</th>
<th>Segments with paradox after repair</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IS</td>
<td>ES</td>
<td>Isovolumetric systole Persistent</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>End systole Persistent New</td>
</tr>
<tr>
<td>1</td>
<td>RCA, LAD, Cx</td>
<td>L, AL, S, PS, P</td>
<td>M1, M3, PDA, LAD</td>
</tr>
<tr>
<td>2</td>
<td>LAD, Cx</td>
<td>L, AL, S, PS, P</td>
<td>D1, M1</td>
</tr>
<tr>
<td>3</td>
<td>LAD, M1</td>
<td>L, S, PS</td>
<td>M1</td>
</tr>
<tr>
<td>4</td>
<td>LAD, M1</td>
<td>L, AL, S, PS</td>
<td>AL, PL, P, S</td>
</tr>
<tr>
<td>5</td>
<td>LAD, PDA, M1, M2</td>
<td>L, AL, S, PS</td>
<td>PDA, M1</td>
</tr>
<tr>
<td>6</td>
<td>LAD, RI, Cx</td>
<td>S</td>
<td>LAD, RI, Cx</td>
</tr>
<tr>
<td>7</td>
<td>LAD, D1, M1</td>
<td>PS</td>
<td>LAD, D1, M1</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td>22</td>
</tr>
</tbody>
</table>

*Not including anterior and anteroseptal segments.

IS, isovolumetric systole; ES, end systole; RCA, right coronary artery; LAD, left anterior descending; Cx, circumflex; M, obtuse marginal branch; PDA, posterior descending artery; RI, ramus intermedius; D, diagonal branch; L, lateral; AL, artero-lateral; S, septal; PS, posteroseptal; P, posterior; PL, posterolateral; NE, norepinephrine; DA, dopamine.

**FIGURE 5.** Global function in seven patients with anteropical left ventricular aneurysm. Global wall thickening is estimated by summing and averaging wall thickening of the separate segments. Function is compared before and after repair at the apex and base. (Error bars represent the pooled standard deviation. Labels and axes are identical to Figure 3.)
eight of 18 segments in which isovolumetric function improved were not in regions that were revascularized. Thus, factors other than perfusion contribute to the salutary effect of LVA repair. Although inotropic agents were used after repair in four patients, it is unlikely that this was a major factor because function at the base remained unchanged. Furthermore, some patients whose paradox resolved completely were not on pressor support.

The elimination of paradox suggests that some degree of mechanical efficiency has been restored to the ventricle and that this may in fact account for the symptomatic relief that many patients experience. It should be kept in mind that this study documents only the short-term changes in regional function associated with LVA repair. The degree and resultant effects of cardiac remodeling that may occur over time in these patients is unknown, but obviously long-term changes in geometry may contribute to the ultimate effects of aneurysm surgery.

Whether proposed techniques of geometric reconstruction would improve on the current standard of linear repair remains untested. However, the lack of benefit that has been documented in other studies8-10 and the fact that end-systolic function was not improved by standard repair in this study support investigation of new methods. As of yet, no animal model of true left ventricular aneurysm has been reported, but the development of such a model should be pursued because of its obvious use in exploring new operative techniques. This form of regional analysis would provide an excellent method for assessing function both clinically and in the laboratory in the development of such alternatives.

Acknowledgments

The authors thank Mr. Renald von Muchow for his technical assistance and Robert Sciaccia, Eng Sc D, for his valuable assistance with statistical analysis.

References


KEY WORDS • left ventricular wall thickening • left ventricular geometry • echocardiography
Quantitative analysis of regional systolic function with left ventricular aneurysm.
A C Nicolesi and H M Spotnitz

Circulation. 1988;78:856-862
doi: 10.1161/01.CIR.78.4.856

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1988 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/78/4/856

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the
Editorial Office. Once the online version of the published article for which permission is being requested is
located, click Request Permissions in the middle column of the Web page under Services. Further
information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/