Left Ventricular Performance before and after Removal of the Noncontractile Area of the Left Ventricle and Revascularization of the Myocardium

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SUMMARY
The left ventricular volume and the internal surface areas of noncontractile regions were measured by cineangiocardiology at 60 frames/sec in nine patients with a chronic localized noncontractile area of the left ventricle. Arteriosclerotic heart disease was proven in eight patients by means of coronary arteriography. Left ventricular end-diastolic pressure, stroke volume, ejection fraction, mean circumferential shortening, and cardiac output were also measured before and after removal of the noncontractile area and revascularization of the myocardium.

The noncontractile areas, measured at end-diastole, ranged from 12 to 40% of the internal surface area of the left ventricle. Generally, impairment of the left ventricular function depended on the size of the noncontractile areas. The end-diastolic volume was approximately 150 ml/m² when the size of noncontractile areas exceeded 20–25% of the left ventricular surface area (r = +0.72; P < 0.05). The ejection fraction decreased as the size of the noncontractile areas increased (r = −0.81; P < 0.01). Following surgery, the left ventricular function, as well as the clinical condition, improved significantly, although the cardiac performance remained in the abnormal range in most patients. The ejection fraction increased (P < 0.05), and the percent circumferential shortening also improved (P < 0.05).

Removal of the noncontractile area of the left ventricle and revascularization of the myocardium improved the cardiac performance and increased the sense of well being in these patients.

Additional Indexing Words:
Left ventricular akinesis  Left ventricular dyskinesis  Left ventricular volume
Left ventricular ejection fraction  Left ventricular excision
Left ventricular revascularization surgery

There is experimental and clinical evidence that the noncontractile area of the left ventricle interferes with the pumping action of the heart. Some authors have called these abnormal areas of the myocardium akinetic, dyskinetic, or asynergic regions; others have called them minor or major aneurysms, depending on their size. Some

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patients with a left ventricular aneurysm have remained well for years after their lesions were first discovered.\textsuperscript{9, 10} However, Schlichter and co-workers\textsuperscript{11} reported the prognosis for survival in patients with a ventricular aneurysm as being about half of that for the patient without an aneurysm. The heart with a dyssynergic area does not work effectively, and heart failure may ensue.\textsuperscript{3-7} Congestive heart failure has been reported as the cause of death in approximately 70\% of the patients with a ventricular aneurysm.\textsuperscript{12}

Excising the areas of infarction from the hearts of experimental animals had a beneficial effect on cardiac function.\textsuperscript{2, 5, 13, 14} Similarly, excising noncontractile areas of the left ventricle in patients appeared to improve these patients, as judged by clinical criteria.\textsuperscript{12, 15-17} This paper presents the measurements of left ventricular function as well as the clinical observations of patients before and after removal of the chronic postinfarction noncontractile area of the left ventricle and revascularization of the myocardium.

\textbf{Case Material}

The subjects of this report were nine patients with an akinetic or dyskinetic area of the left ventricle following documented myocardial infarction that occurred 3 months to 5 years prior to surgery. Patients with a concomitant postinfarction mitral regurgitation evidenced by clinical, hemodynamic, and angiographic findings were excluded from this series. The ages of the patients ranged from 45 to 58 years. Eight were men and one was a woman.

All patients complained of shortness of breath, decreased exercise tolerance, and anginal pain either as isolated symptoms or in various combinations. Two patients (cases 1 and 3) were in chronic congestive heart failure, and one patient had frequent attacks of ventricular tachycardia (case 9).

The preoperative electrocardiograms were compatible with an old, large myocardial infarction in the anterolateral or anteroseptal regions in all patients. The electrocardiogram in two patients (cases 1 and 3) revealed a left anterior hemiblock, and in four (cases 2, 3, 5, and 6) revealed persistent S-T segment elevations.

The cineangiograms showed paradoxical expansion of the damaged area of the left ventricle during systole (dyskinesis) in seven patients, and failure of a large area of the left ventricle to contract during systole (akinesis) in two (cases 4 and 7). All patients had noncontractile areas located in the anteroseptal portion of the left ventricle, and good contraction at the base of the heart was observed in the majority of the patients. Coronary arteriograms in eight patients showed significant narrowing or obstruction of two and sometimes three of the main coronary arteries. The anterior descending branch of the left coronary artery was invariably involved with an 80 to 100\% obstruction. The circumflex was involved least often.

Prior to operation all of the patients except one (case 9) were given digitalis. Nitroglycerin or long-acting nitrates were given to the patients with anginal pain. A diuretic regimen was also added to the two patients in heart failure, and even though they responded poorly to the medical treatment it was thought they should be operated upon for anginal pain or symptoms suggestive of cardiac dysfunction.

In all patients the noncontractile areas were either excised or excluded, and saphenous vein grafts and/or internal mammary artery implantation were performed. Of the four patients with exclusion of the noncontractile myocardium, two (cases 1 and 2) had saphenous vein grafts; one (case 7) had two internal mammary arteries implanted in the myocardium; and the remaining one (case 4) had a vein graft and an internal mammary artery implant. Of five patients in whom the noncontractile region was excised, one (case 3) had a saphenous vein graft, and four (cases 5, 6, 8, and 9) had either one or two internal mammary arteries implanted in the myocardium.

Following surgery, a majority of these patients had excellent clinical results and were restudied. Other patients were reexamined because of mild angina pectoris which persisted postoperatively. Their pain, however, was less and they could do more work than before operation. All nine patients were maintained on digitalis following surgery. In the immediate postoperative period, diuretic therapy was used for some patients. The diuretics were successfully discontinued shortly thereafter without any evidence of heart failure. Coronary dilators were also given to those patients who had anginal pain following surgery.

\textbf{Methods}

Left heart catheterization and cineangiography were performed in nine patients and coronary arteriography in eight, prior to surgery. The ninth patient did not have coronary arteriography because of continual episodes of ventricular tachycardia. Between 3 weeks and 7 months following surgery, angiography was repeated in all patients and left catheterization in

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seven. All studies were done with the patients at rest in the supine position. The aortic and left ventricular pressures were recorded on a multichannel photographic recorder,† using strain-gauge transducers.‡ After measuring the pressures, approximately 60 ml of methylglucamine diatrizoate§ were injected through an angiocatheter into the superior vena cava or the right atrium with the use of a pressure injector. With the patients in 30° right anterior oblique position, the cineangiograms were recorded at 60 frames/sec, photographing the contrast medium as it passed through all four chambers of the heart. Although a slightly larger amount of contrast medium and a higher contrast are usually required, this forward method of cineangiography¶ allows a better mixing of contrast material and completely obviates the occurrence of ectopic beats so frequently seen with direct left ventricular injection. Additional information is derived from this method, such as detection of abnormal contraction of the right ventricle, circulation time through the lung, and state of contraction of the left atrium.

With this forward method, opacification of the left ventricle has not always been as good as with direct injection of contrast material into the left ventricular cavity; however, for the advantages described above, this method has been tried first, and it has been possible to trace the diastolic and systolic phases of the left ventricle in most patients with a noncontractile area. The opacification of the left ventricle by this method becomes poor in patients with a greatly dilated ventricle and very slow pulmonary circulation time. Also, this method is not suitable for precise detection of mitral insufficiency. Therefore, whenever the forward method fails to demonstrate the left ventricular cavity clearly or an associated mitral regurgitation is suspected, a direct injection is also carried out. Some patients in this series had both methods performed in order to prove the absence of mitral regurgitation. Ventriculograms taken by this method are demonstrated in figure 1.

Left ventricular volume (LVV) was calculated using the ellipsoid formula. The formula for this calculation is:

\[
LVV = \frac{4}{3} \pi \left(\frac{M}{2}\right)^2 \frac{L}{2}
\]

The longest axis (L) was measured directly from the tracing, and the short axis (M) was derived from L, and from the planimetered area (A) of the ventricle, \(M = 4 \frac{A}{\pi} L\). If the noncontractile area projected out from the ventricular wall, the volume of this area was calculated separately as a hemisphere and added to the volume of the remaining portion of the ventricle. Corrections for magnification of the left ventricular tracing were made in each case.

Stroke volume (SV) and ejection fraction (EF) were calculated. The SV was derived by subtracting the volume at the end of systole (ESV) from the volume at the end of diastole (EDV), and EF was obtained by using the formula \(EF = \frac{SV}{EDV}\). The EDV and SV were corrected for body size and expressed as milliliters per square meter of body surface area (BSA). Although there were some changes of BSA because of changes of body weight after operation, preoperative BSA was used for all calculations.

The mean end-diastolic and end-systolic diameter of the left ventricle (d) were calculated in centimeters by a method that treats the ventricular chamber as a sphere with the same volume as the calculated left ventricular volume; accordingly:

\[
d = \sqrt[\frac{6}{\pi}]\left(\frac{LVV}{4}\right)
\]

Internal circumferential fiber shortening (CFS) was calculated from the difference of circumferential distances (\(\pi d\)) at the end-diastolic and at the end-systolic periods. The CFS was corrected for ventricular diastolic size by dividing it by the circumference of the end-diastolic sphere, the result being expressed as percent CFS.

Mean velocity of circumferential fiber shortening (VCF) in centimeters per second was measured from CFS divided by left ventricular ejection time. This was also normalized by dividing it by the end-diastolic circumference, expressing the result as circumferences per second.

The percent CFS in the noninfarcted area extrinsic to the noncontractile area was estimated from the following formula:

\[
%CFS = \frac{1 - \frac{1 - EF}{1 - IS}}{100}
\]

where EF is ejection fraction and IS is the size of the noncontractile area expressed as a fraction of the total circumference of the ventricle at the end of diastole.

Cardiac output was measured using the dye-dilution technic in five patients. The SV measured by dye-dilution technic correlated within a range of approximately ±10% variation with that measured by angiographic method in these patients. In the remaining four patients the SV was measured only from the ventriculogram. This was multiplied by the heart rate at the time of the angiocardiography to give the cardiac output.
Figure 1

Ventriculograms taken by the forward angiocardiography in the right anterior oblique position in case 9. (Top) Preoperative, end-diastole. (Bottom) Postoperative, end-diastole. Note that the opacification of the left ventricle follows the opacification of the left atrium.

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The left ventricular end-diastolic pressure (LVEDP) was measured through a catheter passed through the aortic valve into the left ventricle. The internal surface area of the noncontractile region was calculated in each patient, using the central-angle method:  \[ 2\pi R^2 \left(1 - \cos \frac{\theta}{2}\right) f^2; \] or the graphic-integration method:  \[ \pi R^2 \sum_{k=1}^{n} W_k (R_k + R_{k+1}). \]

In the central-angle method, \( R \) represents the radius of the circle, the arc of which best fits the curvature of the noncontractile wall. The sides of the angle (\( \theta \)) were subtended by the chord of this circle drawn on the noncontractile area; \( f \) is a magnification factor. In the graphic-integration method, the noncontractile area of the left ventricle is assumed to be an integration of \( n \) disc slices, each of which has a radius of \( R_n \) and a slant height of \( W_n \). Because the curvature of the noncontractile wall is not symmetrical and therefore each disc is not a frustum of the right cone, the average slant height measured from the upper and lower curvatures was used. Application of the methods has been described by Klein et al.4 and Crawford et al.21 Also, this area was compared to the internal surface area of the entire left ventricle \( (\pi R^2) \) at the end of diastole, giving a relative size of the noncontractile area.

The average values were calculated with a standard error. Statistical comparisons were also made between the pre- and postoperative results using a \( t \) test.

**Results**

The ventricular performance before and after operation is presented in table 1. These measurements may be summarized as follows.

**Before Operation**

The end-diastolic volume of the left ventricle corrected for body surface area ranged from 86 to 211 ml/m², average 137 ± 12.9. Although in most patients the left ventricular chamber was enlarged, it was normal with a volume less than 100 ml/m² at end-diastole in three patients. Stroke index ranged from 17.1 to 55.0 ml/beat/m², average 38.3 ± 3.99. The EF ranged from 0.12 to 0.55, average 0.31 ± 0.047. This was less than half of the normal value for EF of the left ventricle.24 The cardiac index was low normal, average 2.85 ± 0.239 liters/min/m². The LVEDP was elevated in all but one patient and was elevated even in patients without a significant enlargement of the left ventricle. The LVEDP varied from 7 to 42 mm Hg, average 27 ± 3.5.

The internal circumference of the left ventricle shortened an average of 11.8 ± 1.93% (normal, 15–28). The mean \( V_{cr} \) averaged 10.3 ± 1.71 cm/sec \( (0.43 ± 0.082 \) circ/sec) \( (normal, 15–25 \) cm/sec; \( >0.95 \) circ/sec). The above values were below normal when compared with the measurements in our normal patients, as well as those published by Wilcken,22 Gault,23 Bristow24 and their co-workers.

The surface areas of noncontractile regions ranged from 20 to 87 cm², occupying from 12 to 40% of the internal surface area of the left ventricle at the end of diastole. There was a normal EDV in three patients, although the noncontractile area of the left ventricle occupied from 12 to 19% of the internal surface area. These patients had either a decreased EF or an elevated LVEDP or both. The EDV tended to be higher in patients in whom the noncontractile area occupied a larger fraction of the internal area of the left ventricle \( (r = +0.72; P < 0.05) \) (fig. 2). The EDV was 150 ml/m² or more when the noncontractile area exceeded 20 to 25% of the internal surface area of the left ventricle.

Figure 3 shows that the LVEDP was usually high in those patients in whom the noncontractile area occupied a large fraction of the total internal surface area of the ventricle. Figure 4 reveals that the EF was lower where the noncontractile area occupied a larger fraction of the internal surface area \( (r = -0.81; P < 0.01) \). The mean \( V_{cr} \) was lower when the noncontractile area occupied the larger fraction of the total ventricular internal surface area (fig. 5). From these results it was apparent that the localized noncontractile area impaired ventricular function, and the larger noncontractile area impaired ventricular function more than the small noncontractile area.
Comparative Studies of Left Ventricular Performance before and after Removal of the Noncontractile Area and Revascularization of the Myocardium

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<th>EDV/m² (ml/m²)</th>
<th>ESV (ml)</th>
<th>SV (ml)</th>
<th>SI (ml/m²)</th>
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*P < 0.05.
†Numbers in parentheses are circumferences per second.

Abbreviations: BSA = body surface area; EDV = end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; SI = stroke index; CI = cardiac index; EF = ejection fraction; CFS = circumferential fiber shortening; VCF = velocity of circumferential fiber shortening; LVEDP = left ventricular end-diastolic pressure; SALV = surface area of left ventricle; SANCA = surface area of noncontractile area; Cale CFS = calculated preoperative circumferential fiber shortening of the noninfarcted area; SEM = standard error of the mean.

After Operation
Following operation all nine patients improved by clinical criteria. The saphenous vein grafts were shown to be patent with the use of contrast medium at the time of postoperative study; however, internal mammary artery injection was not performed. Systolic expansion was not observed postoperatively, although small residual akinetic or hypokinetic areas remained at the suture line in all patients.

Figure 6a shows that the average EDV of the left ventricle decreased from 137 ± 12.9 to 108 ± 8.3 ml/m² (0.05 < P < 0.1). The left ventricular internal surface area decreased from 193 ± 12.1 to 166 ± 8.8 cm² after surgery; the average reduction was 27 cm². The average SV increased after surgery.
LEFT VENTRICULAR PERFORMANCE

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<th>% CFS</th>
<th>Mean V_CVF (cm/sec)</th>
<th>LVEDP (mm Hg)</th>
<th>CI (liters/min/m²)</th>
<th>SALV (cm³)</th>
<th>SANCA (cm²)</th>
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Figure 6b shows that LVEDP decreased after surgery, although it remained in the abnormal level in most patients. It averaged 27 ± 3.5 before and 18 ± 1.6 mm Hg after surgery.

Figure 6c shows that EF increased after operation. The average EF before operation was 0.31 ± 0.047, and after operation it increased to 0.44 ± 0.030, a significant increase (P < 0.05). The ventricle of one patient was normal in size before and after operation and revealed a decrease in EF following operation.

The mean $V_CVF$ averaged 10.3 ± 1.71 cm/sec (0.43 ± 0.082 circ/sec) before surgery and 14.4 ± 1.39 cm/sec (0.64 ± 0.063 circ/sec) after surgery (0.05 < P < 0.1) (fig. 6d). The percent CFS also increased from 11.8 ± 1.93 to 17.6 ± 1.45% after surgery (P < 0.05). Cardiac index was greater after surgery than before. The average was 2.85 ± 0.239 before and 3.47 ± 0.161 ml/min/m² after surgery.

Figure 7 shows the extent to which the surgical operations decreased the EDV and increased the EF in each patient. As a whole, there was an inverse relationship observed between the EDV and the EF (r = -0.83; P < 0.001), suggesting that an increase in EF was mainly due to a decrease in EDV.

Before operation the internal circumferential shortening was calculated in the active parts of the left ventricles, and following

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The end-diastolic volume (EDV) is plotted against the surface area ratio of the noncontractile area to the ventricle at the end of diastole. The EDV increased as the ratio increased ($r = +0.72; P < 0.05$). When this ratio exceeded 20–25%, EDV was 150 ml/m$^2$ or more.

![Figure 2](image)

**Figure 2**

Comparison of LVEDP with the size of the noncontractile area is shown. All patients but one had an elevated LVEDP. Generally there was a rise in end-diastolic pressure with an increase in the size of the noncontractile area.

![Figure 3](image)

**Figure 3**

In this study, the left ventricular volume was measured by single-plane angiocardiography performed with the patient in the right anterior oblique position. Several investigators have shown that this method is reliable in determining the left ventricular volume with the assumption that the left ventricular chamber can be represented as an ellipsoid revolution figure. An irregular and complex geometric configuration of the left ventricular cavity, as occurs with aneurysm, makes the method less accurate. However, Klein, Herman, and Gorlin showed that measurements of ventricular volume by this method correlated well with measurements by the thermodilution method, even in patients with a ventricular aneurysm.

The use of a spherical model for simplifying the estimation of circumferential shortening and the method for measuring the surface operation as the best part of the left ventricle shortened prior to the operation ($r = +0.73; P < 0.05$).

**Discussion**

In this study, the left ventricular volume was measured by single-plane angiocardiography performed with the patient in the right anterior oblique position. Several investigators have shown that this method is reliable in determining the left ventricular volume with the assumption that the left ventricular chamber can be represented as an ellipsoid revolution figure. An irregular and complex geometric configuration of the left ventricular cavity, as occurs with aneurysm, makes the method less accurate. However, Klein, Herman, and Gorlin showed that measurements of ventricular volume by this method correlated well with measurements by the thermodilution method, even in patients with a ventricular aneurysm.

The use of a spherical model for simplifying the estimation of circumferential shortening and the method for measuring the surface

*Circulation, Volume XLV, May 1972*
patients with marked dilatation of the left ventricle.

The left ventricle with a chronic localized noncontractile area was shown to function poorly in the assessment of left ventricular performance. The myocardial fibers in the best portion of the left ventricle can shorten to some extent more than the normal amount and thereby maintain an overall ventricular function near normal when the noncontractile area is small. In one (case 4) of three patients whose left ventricular volume was normal, the best part of the myocardium contracted with increased vigor, with a calculated circumferential shortening in the noninfarcted area greater than 28%. The noncontractile areas in these three patients were less than 20% of the internal surface area of the left ventricle. However, the LVEDP was elevated or the EF was decreased. Local abnormal contraction reduced the effective function of the ventricle in the absence of significant dilatation of the ventricle.

Klein, Herman, and Gorlin reported that when the internal surface area of the noncontractile region exceeds 20 to 25% of the left ventricular internal surface area, increased fiber shortening in the best portion of the ventricle cannot maintain a normal stroke volume. If the left ventricle enlarges in such a patient, the stroke volume can increase...
because the larger heart can deliver a given stroke volume with less fiber shortening. However, the enlarged ventricle with a large noncontractile area usually ejects an abnormally small stroke volume, because a large fraction of the ventricular volume is surrounded by the noncontractile and/or paradoxically expanding wall. This results in an inappropriate enlargement of the left ventricle, characteristic of myocardial injury. According to the law of Laplace, the disadvantage of ventricular enlargement is that a greater contractile tension is required in order to generate any given intraventricular pressure. This requirement for increased tension leads to a decrease in the rate of myocardial fiber shortening and an increase in myocardial oxygen requirement. Paradoxical expansion during systole reduces the mechanical effectiveness for the ejection of blood because of the backward flow of blood into this area.

In eight of our nine patients the LVEDP was elevated, although in two of these eight the size of the left ventricle was normal. Low diastolic compliance of the left ventricle even in those patients with uncomplicated arteriosclerotic heart disease has been reported previously.

The ejection fraction correlated well with the size of the localized noncontractile area ($r = -0.81; P < 0.01$). A low EF disproportionate to the size of the noncontractile area may indicate that generalized myocardial impairment predominates over localized dys-synergy.

Considering that removal of the noncontractile area relieves the heart theoretically of several mechanical disadvantages, one would expect the heart to work more efficiently following surgery. In the present series, ventricular function studies revealed improvement in the EF, percent CFS, mean $V_C$, cardiac index, ventricular volume, and LVEDP after elimination of the noncontractile area and revascularization of the myocardium, although some of these parameters remained in the abnormal range. It is also suggested that the ventricular compliance may increase after removal of the noncontractile area, since both increased stroke volume and decreased LVEDP were observed in some instances. These patients who had successful operation were improved not only by clinical criteria but also by hemodynamic and angiocardio graphic measurements.
Postoperative deaths may be attributable to poor contraction of the remaining myocardium, or too small volume of the remaining ventricle. The calculated internal CFS of the contractile myocardium before operation correlated fairly well with the actual CFS of the remaining left ventricle after operation (fig. 8). Two patients (cases 1 and 3) who had calculated CFS of the functioning area of less than 10%, apparently had a difficult postoperative course. Examining the amount and vigor of the contractile myocardium has been helpful in the assessment for operation.

The left anterior descending coronary artery was invariably involved with an 80 to 100% obstruction. Obstruction of this artery is most significant in the formation of the noncontractile area located in the anteroapical portion of the left ventricle. Similar observations were reported in larger series of patients. Six of eight patients who underwent coronary arteriography had one main coronary artery with insignificant or not more than 50% obstruction. This fact appears to be important for successful surgical results. Indeed, the postoperative average EDV decreased to a level slightly higher than normal (108 ± 8.3 ml/m²) with a normal stroke volume (45.4 ± 2.71 ml/m²), indicating good functioning muscle in the remaining ventricle. Significant hemodynamic improvement was attained in such patients.

At surgery the excised noncontractile area was smaller than the noncontractile area estimated in the preoperative study. In two patients (cases 3 and 6) with reliable data, the excised wall measured 4.0 × 9.5 cm (38.0 cm²) and 4.0 × 7.5 cm (30.0 cm²), compared to the estimated area of 69 and 50 cm², respectively. The akinetic or dyskinetic area which was excised was less than the total size of the noncontractile area observed during operation, because a considerable amount of myocardium which looked viable was observed in the noncontractile area, and some of this was left intact. In addition, some parts of the noncontractile area were left for suture line. The postoperative angiocardiograms showed that the akinetic or hypokinetic areas remained in the region of the stump of excision or exclusion.

A variety of combinations of surgical procedures were performed on these nine patients who differed to some extent in their clinical status. This makes a detailed analysis of the surgical results difficult. Improvement of the ventricular function demonstrated by this study appeared mainly attributable to excision or exclusion of the noncontractile area rather than revascularization surgery. This was noted in other studies with excision of the noncontractile area without revascularization surgery. There was no significant difference demonstrated in left ventricular function between the small groups of patients with saphenous vein grafts and patients with internal mammary artery implants, in this series.

Considering the results in this study together with those reported by other investigators, removal of the noncontractile area appears to be an effective surgical procedure for patients with a localized dyssynergic area of the left ventricle. Concomitant myocardial revascularization with removal of the noncontractile area as performed in our patients should be considered with the hope of prolonging life and improving the function of the remaining myocardium.

References
8. Harrison TR: Some unanswered questions concerning enlargement and failure of the heart. Amer Heart J 69: 100, 1965
10. Master AM, Jaffe HL: Complete functional recovery after coronary occlusion and insufficiency. JAMA 147: 1721, 1951
20. Hood WB Jr: Experimental myocardial infarction: Recovery of left ventricular function in the healing phase; Contribution of increased fiber shortening in noninfarcted myocardium. Amer Heart J 79: 531, 1970
33. Schimert G, Falsetti HL, Bunnell IL, Deane DC, Gage AA, Grant C, Greene DG: Excision of akinetic left ventricular wall for


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