Contractile Reserve and Left Ventricular Function in Regional Myocardial Ischemia in the Dog

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SUMMARY Contractile activity remaining in a region made ischemic by acute occlusion of the left anterior descending coronary artery (LAD) was assessed in dogs relative to its role in maintaining left ventricular (LV) function. Compensatory increases in contractility of normal myocardium were eliminated by treating all dogs with reserpine (3 mg/kg) to deplete their catecholamine stores. LV function was determined by measuring stroke volume while increasing the LV filling pressure with a shunt from the aorta to left atrium. Heart rate and mean aortic pressure were kept constant. LV function was studied after occlusion of the LAD alone and after the selective infusion of potassium chloride (1 mEq/ml) into the LAD to raise the regional extracellular potassium concentration to 30 mEq/ml. The reduction in LV function induced by LAD ligation was less than the reduction caused by abolishing contraction in the entire zone supplied by the LAD with infusion of potassium. The totally cardioplegic zone induced by potassium amounted to 20.3-39.8% of the LV mass. At an LV end-diastolic pressure of 12 mm Hg, stroke volume (SV) was reduced in proportion to the size of the cardioplegic zone: $-SV$ (SV volume) = $-1.55\%$ (of LV mass) + 120.1 ($r = -0.69, p < 0.005$). Thus, a dyskinetic zone of 35% of the left ventricle reduced stroke volume by 34% when adrenergic compensation was blocked. We conclude that residual transmural contractility exists in the ischemic region of myocardium subserved by an obstructed LAD and contributes significantly to LV function.

WHEN a coronary artery is occluded abruptly, loss of segmental shortening in the region of myocardium fed by this artery is generally observed. However, some collateral coronary blood flow remains and residual contractile function may remain in the region. The role of such contractile activity remaining in a region made ischemic by occlusion of a coronary artery on the overall function of the left ventricle has not been defined. Further, the relation between the size of the ischemic area and the extent of reduction in overall left ventricular (LV) function has not been precisely determined. When regional myocardial ischemia is induced, LV function is affected by the lateral dimensions of the ischemic region, the distribution of contractile activity transmurally in this region as determined by the remaining collateral coronary blood flow, the compliance of the ischemic zone, and compensatory increases in contractility in nonischemic zones of myocardium resulting from augmented sympathetic tone. This may explain the marked variation in the reported observations on this subject.1-9

We studied the effects of regional myocardial ischemia and total cardioplegia. The effects of involving varying amounts of the ventricle on overall LV function were also examined.

Methods

Experimental Preparation

Fifteen mongrel dogs that weighed 19-32 kg were treated with reserpine (3 mg/kg subcutaneously) 24 hours before study to deplete their catecholamine stores.10 This was done to abolish the compensatory increase in contractility that may occur in the nonischemic myocardium after coronary artery ligation.

Dogs were anesthetized with sodium pentobarbital (30 mg/kg i.v.). Additional pentobarbital was administered as required to maintain anesthesia. After intubation with a cuffed endotracheal tube, dogs were ventilated with an intermittent positive pressure respirator with 100% oxygen. A thoracotomy was performed in the fifth intercostal space and the heart was exposed by incising the pericardium. The left anterior descending coronary artery (LAD) was isolated and ligated 3-20 mm from its origin to vary the size of the ischemic region.

After ligation of the LAD, a cannula was inserted distal to the occlusion and perfusion was continued through a polyvinyl tube from the left carotid artery. Initiation of this procedure lasted 2-3 minutes. A T tube (9 mm i.d.) was inserted into the upper portion of the descending aorta and connected to a 2-liter reservoir (fig. 1). The pressure in the reservoir was adjusted to keep the mean aortic pressure constant. A shunt was created between the aorta and the left atrium. When this shunt was gradually opened, blood flow to the left atrium was increased, resulting in a rise in LV filling pressure.

LV pressure was measured through a short, stiff cannula through the apical dimple of the left ventricle and connected to a pressure transducer (Statham P23Db). Systemic blood pressure was measured in the ascending aorta (Statham P23Db transducer). Both transducers were placed at the midthoracic level and adjusted for equal sensitivity.

An electromagnetic flowmeter was placed around the proximal portion of the ascending aorta to measure aortic blood flow. Heart rate was controlled with wires sewn to the right atrium and connected to an electric stimulator (Grass S88).

A strain-gauge arch (John Warren) was sewn to the area of the LAD to be made ischemic. A standard lead
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The aorta to

sured

through the clamp

FIGURE 1. Experimental design. Clamp A was open to maintain a constant mean aortic pressure at a desired level (measured by the manometer connected to the volume reservoir) by adjusting the air flow into the reservoir. Gradual opening of clamp B created an aorta to left atrial shunt, resulting in an increase in left ventricular (LV) end-diastolic pressure. A carotid–left anterior descending coronary artery (LAD) cannula was used to maintain adequate coronary perfusion pressure and flow during the potassium infusion. A strain-gauge arch was sewn to the area of the LAD that became ischemic or cardioplegic. The aortic flow probe was placed around the ascending aorta to determine the stroke volume. LV pressure was measured through a short, stiff cannula inserted into the LV apex.

II ECG and an epicardial tracing were obtained. Aortic and LV pressures, aortic flow velocity, stroke volume, the epicardial ECG, and regional force development were recorded on a multichannel oscillographic recorder.

Experimental Procedure

The preparation is shown in figure 1. Clamp A was opened before any intervention and the reservoir solution containing dextran in 5% dextrose solution was allowed to mix with the dog’s blood to prevent significant drop in the hematocrit during the experiment. The heart was paced just above its spontaneous rate, generally at 100–130 beats/min. Clamp A was open and mean arterial pressure kept constant by adjusting the pressure in the reservoir. Clamp B was slowly opened to increase the blood flow to the left atrium; this resulted in a gradual increase of LV end-diastolic pressure (LVEDP). When LVEDP reached 16–20 mm Hg, clamp B was closed. LV filling pressure was rapidly lowered by bleeding into the reservoir. After a recovery period of about 20 minutes, the same procedure was repeated for another control run. After another 20 minutes of recovery period, potassium chloride (1 mEq/ml) was infused into the distal LAD with a Harvard infusion pump at a rate of 0.6 ml/min. Based on a flow of approximately 25 ml/min, this infusion rate would raise serum potassium concentration and presumably the extracellular potassium concentration in the myocardium perfused by the LAD to approximately 30 mEq/l, which would make this region electrically inexcitable. This resulted in a total loss of contractile activity (regional cardioplegia). During the infusion, LV stroke volume was continuously measured, while another determination of LV function was obtained by gradually increasing LVEDP to 18 mm Hg.

The potassium infusion lasted 10 minutes. During or immediately after the infusion, eight of 15 dogs developed ventricular fibrillation and could not be resuscitated. In seven dogs, the heart recovered completely from the effects of the regional cardioplegia as verified by the return of the hemodynamic indexes and ECG to control levels. After a 20-minute recovery period, the relationship of stroke volume to LVEDP was again determined. Five minutes later, standard carbonized microspheres, 15 ± 5 μ in diameter, labeled with the nuclide 14Ce (3M Company), were injected in the left atrium of seven hearts to measure myocardial blood flow and cardiac output.11 The microspheres were suspended in a 63% glucose solution and were dispersed before injection by mechanical agitation and sonication for 5 minutes in an ultrasonic bath. Ten milliliters of the solution containing 10^6 beads were injected over 20–25 seconds and flushed with 10 ml of physiologic saline. Withdrawal of an arterial blood sample was started just before administration of the microspheres and for 30 seconds beyond the end of injection by use of a Harvard pump with a withdrawal rate of 11.6 ml/min so that absolute tissue flows12 and the cardiac output3 could be calculated.

Eight to 10 minutes after occlusion of the distal LAD, right atrial pacing was resumed; clamps A and B were opened, and LV function was determined again.

At the end of each experiment, 5–8 ml of Evans blue dye solution was injected through the cannula inserted into the LAD. The heart was then arrested with potassium. The stained tissue was carefully dissected, separated from the nonstained tissue, and weighed. This stained tissue was expressed as the percentage of the total LV weight.

In the seven hearts injected with radioactive microspheres, myocardial blood flow was determined in the subendocardial, midwall and subepicardial layers in the normal myocardium and in the center of the LAD region stained by Evans blue dye, as described previously.14 Analysis of variance and linear regression analysis were applied for statistical analysis of the data.

Results

Left Ventricular Function in Dogs Depleted of Catecholamine Stores

In all dogs, increasing LVEDP resulted in a significant increase in stroke volume (table 1, fig. 2). Increasing LVEDP from 8 to 16 mm Hg increased stroke volume from 38.7 ± 4.0 to 50.5 ± 4.3 ml. Thus,
TABLE 1. The Effects of Regional Cardioplegia and Ischemia on Left Ventricular Function at Constant Mean Aortic Pressure and Heart Rate

<table>
<thead>
<tr>
<th>LVEDP (mm Hg)</th>
<th>Procedure</th>
<th>Dog 2</th>
<th>Dog 5</th>
<th>Dog 8</th>
<th>Dog 9</th>
<th>Dog 13</th>
<th>Dog 14</th>
<th>Dog 15</th>
<th>Mean ± SEM</th>
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<tbody>
<tr>
<td>8</td>
<td>Control</td>
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<td>38.8</td>
<td>27.5</td>
<td>56.2</td>
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<td>47.4</td>
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<td>38.7 ± 4.0</td>
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<tr>
<td></td>
<td>Ischemia</td>
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<td>29.2</td>
<td>25.5</td>
<td>37.4</td>
<td>29.0</td>
<td>43.4</td>
<td>25.2</td>
<td>32.5 ± 2.7</td>
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<tr>
<td></td>
<td>KCl</td>
<td>36.0</td>
<td>27.8</td>
<td>21.9</td>
<td>34.1</td>
<td>23.4</td>
<td>*</td>
<td>18.5</td>
<td>26.9 ± 2.8</td>
</tr>
<tr>
<td>12</td>
<td>Control</td>
<td>49.3</td>
<td>43.3</td>
<td>31.4</td>
<td>61.9</td>
<td>42.3</td>
<td>56.2</td>
<td>33.6</td>
<td>45.4 ± 4.2</td>
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<tr>
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<td>Ischemia</td>
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<td>34.2</td>
<td>28.5</td>
<td>45.3</td>
<td>35.7</td>
<td>51.2</td>
<td>31.9</td>
<td>38.5 ± 3.1</td>
</tr>
<tr>
<td></td>
<td>KCl</td>
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<td>32.8</td>
<td>27.4</td>
<td>39.6</td>
<td>31.5</td>
<td>50.0</td>
<td>25.2</td>
<td>35.1 ± 3.2</td>
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<tr>
<td>16</td>
<td>Control</td>
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<td>45.2</td>
<td>36.2</td>
<td>65.6</td>
<td>48.6</td>
<td>65.1</td>
<td>40.3</td>
<td>50.5 ± 4.3</td>
</tr>
<tr>
<td></td>
<td>Ischemia</td>
<td>46.8</td>
<td>36.2</td>
<td>33.8</td>
<td>51.2</td>
<td>40.8</td>
<td>56.8</td>
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<td>31.8</td>
<td>45.6</td>
<td>34.3</td>
<td>55.1</td>
<td>31.9</td>
<td>38.7 ± 3.6</td>
</tr>
</tbody>
</table>

*KCl administration resulted in the increase of LVEDP to greater than 10 mm Hg.
Multiple range test (Scheffe procedure) shows the data from each procedure (control, ischemia, KCl) to be significantly different (p < 0.05) from each other.
Abbreviation: LVEDP = left ventricular end-diastolic pressure.

under these experimental conditions, which include control of aortic pressure and heart rate as well as the absence of myocardial catecholamine stores, the Frank-Starling mechanisms played a major role in increasing the performance of the left ventricle over the normal range of filling pressures.

Regional Cardioplegia

Cardioplegia of the region of myocardium supplied by the distal LAD was induced by perfusing potassium (1 mEq/ml) at a rate of 0.6 ml/min. The infusion of potassium into the distal LAD produced marked alterations in conduction (fig. 3). Epicardial electrograms recorded from other regions of the ventricle were unremarkable. Potassium infusion into the LAD led to a rapid fall in the regional contractile force to zero. The area then became dyskinetic, as demonstrated by its systolic expansion (fig. 4). The strain gauge also demonstrated passive stretching of the cardioplegic region. We did not quantitate the changes in the regional contractile force, and the strain gauges were only used as a qualitative indicator. Regional cardioplegia was associated with a decrease in stroke volume and aortic flow and a slight increase in LVEDP (table 1, figs. 2 and 4). In each state, an increase in LVEDP was associated with a significant increase in LV stroke volume. However, the stroke volume was significantly less at any LVEDP when the LAD zone was made cardioplegic compared with control. The size of the cardioplegic zone ranged from 20.3% to 39.8% of the LV mass (table 2). At an LVEDP of 12 mm Hg, LV stroke volume (SV) was reduced in proportion to the size of the cardioplegic zone (Z): −SV = −1.55 Z (% of LV mass) + 120.1 (r = −0.69, p < 0.005) (fig. 5). Thus, a cardioplegic zone involving 35% of the LV mass produced a 34% reduction in stroke volume when adrenergic compensation by the normal myocardium had been eliminated. Termination of the potassium infusion into the LAD, when not accompanied with uncontrollable arrhythmias, led to complete recovery of the remaining contractility in the LAD region as well as restoration of overall left ventricular function (fig. 6).

Regional Myocardial Ischemia

In the control state, coronary blood flow averaged 0.68, 0.65 and 0.63 ml/min/g in the subendocardial, midwall and subepicardial layers, respectively. These flows fell to 0.09, 0.08 and 0.13 ml/min/g in these
same layers after LAD occlusion (fig. 7). The size of the ischemic region varied between 24.5% and 34.0% of the total LV mass (table 2).

During the stable state of regional ischemia, increments in LVEDP produced significant increases in LV stroke volume (table 1, fig. 2). However, for each LVEDP, the increments in LV stroke volumes were significantly lower during regional myocardial ischemia compared with control conditions in the absence of ischemia. The mean reduction in stroke volume induced by ischemia was 16.0%, 15.2% and 14.7% at LVEDPs of 8, 12 and 16 mm Hg, respectively. The reduction in stroke volume was significantly greater at any LVEDP when precisely the same zone was made inexcitable by KCl infusion into the LAD than it was during LAD occlusion (table 1, fig. 2). Furthermore, when LVEDP was 12 mm Hg, LV stroke volume was reduced 15.2% by ischemia alone and 22.7% by regional cardioplegia.

**Discussion**

Regional mechanical dysfunction occurs quite rapidly after acute coronary occlusion. Such disorders of contractile function can be measured in the center of the ischemic region within approximately 10 seconds and before ST-segment elevation occurs on the epicardial ECG. Holosystolic expansion of the region develops over the next 1–2 minutes.

Regional dysfunction has been recorded in open-chest animals by force gauges and epicardial dimension gauges; recently, the use of implanted ultrasonic crystals has permitted precise measurements of regional dimensions simultaneously in several regions of the left ventricle in open-chest dogs and in the unanesthetized state. The paradoxical systolic expansion induced by regional myocardial ischemia acts as a slack elastic element in series with the contractile portion of the unaffected remaining muscle, and thus may reduce overall ventricular function. Parmley and Sonnenblick have shown experimentally that additional series elasticity causes a fall in the rate of ischemia.

### Table 2. Reduced Stroke Volume Induced by Cardioplegia and Ischemia: Its Relation to the Size of the Dyskinetic Region

<table>
<thead>
<tr>
<th>Dog</th>
<th>Dyskinetic region (% LV mass)</th>
<th>Reduction of SV from control condition at LVEDP = 12 mm Hg (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Cardioplegia</td>
</tr>
<tr>
<td>1</td>
<td>20.3</td>
<td>6.9</td>
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<tr>
<td>2</td>
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<td>7</td>
<td>39.8</td>
<td>55.3</td>
</tr>
<tr>
<td>8</td>
<td>29.2</td>
<td>12.7</td>
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<td>10</td>
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</tr>
<tr>
<td>15</td>
<td>34.0</td>
<td>25.0</td>
</tr>
</tbody>
</table>

Abbreviations: LV = left ventricular; LVEDP = LV end-diastolic pressure; SV = stroke volume.
of tension development, dT/dt, and developed tension of the isolated muscle in proportion to the added compliance. This observation has been applied in studying LV function in the canine preparation during coronary occlusion.

In this study, the overall LV function measured as stroke volume was markedly reduced by regional myocardial ischemia. The reduction in stroke volume was significantly greater at any LVEDP when precisely the same zone was made inexcitable by potassium infusion. Although the local contractile activity was not quantitated in these two experimental interventions, we infer that in the absence of any other variables, the improved LV function during regional ischemia (compared with regional cardioplegia) is due to residual contractile activity in the ischemic zone which is abolished by potassium infusion when the region was made inexcitable and acontractile. Regional ischemia and regional cardioplegia could affect ventricular compliance to a different degree, and thus alter the stroke volume/end-diastolic pressure relationship. However, no data are available on the effects of regional cardioplegia on ventricular compliance.

During the first few minutes (and up to 1 hour) of myocardial ischemia, ventricular compliance is increased, and the stiffening of the ischemic segment occurs only after several hours. Thus, the reduced stroke volumes at identical LVEDPs during regional ischemia were associated with larger end-diastolic volumes than in the preischemic period. Whether this also occurs during regional cardioplegia is not known; in any case, the effect of changes in segmental diastolic

![Figure 5](image1.png)

**Figure 5.** The effects of cardioplegic size on left ventricular (LV) function. The relationship between the cardioplegic zone (expressed as percent of LV mass) and the percent reduction of stroke volume (SV) is expressed as a linear equation with 95% confidence limits of the mean. The SV at an LV end-diastolic pressure (LVEDP) of 12 mm Hg in the control state is considered to be 100%. The reduction of SV is in proportion to the extent of the cardioplegic size. The open circle and its bars are the mean ± SEM of the data obtained during ischemia from seven hearts and is above the mean value with cardioplegia.

![Figure 6](image2.png)

**Figure 6.** The recovery of left ventricular function and segmental contraction after termination of potassium infusion into the left anterior descending coronary artery (LAD). After termination of the infusion, SV gradually increases and left ventricular end-diastolic pressure decreases. The systolic expansion of the cardioplegic zone gradually decreases and full recovery of the regional contractile force follows (compare with figure 4). The frequently observed overshoot of contractile force is unexplained.
compliance on the relationship between stroke volume and diastolic pressure is not easily determined.

However, regional myocardial dysfunction may not always reduce overall LV function. With a somewhat augmented ventricular filling pressure, an increase in resting fiber length produces augmented shortening of normal segments. The ischemic region is also stretched to a stiffer portion of its diastolic length-tension curve. The increased fiber shortening in the nonischemic myocardium may compensate for dysfunction of the ischemic area and the overall LV function may remain unchanged. The physiologic compensatory mechanisms that enhance contractility in the nonischemic portion of the heart during acute myocardial ischemia may also be attributed to the increase of catecholamines discharge. In these experiments, pretreatment with reserpine was used to deplete myocardial catecholamines. The dose used in the present study was higher than that previously used in the dog that was found to deplete myocardial norepinephrine stores. Therefore, we feel that compensatory increases in contractility of the nonischemic tissue were eliminated. However, residual catecholamine responses would tend to reduce the magnitude of the observations. The size and location of the ischemic region may also significantly influence the effect of acute myocardial ischemia on ventricular function. These factors may explain the marked variation in ventricular function during acute myocardial ischemia.

When coronary blood flow to the region of the ventricular wall is reduced, subendocardial flow decreases more than subepicardial flow. Concomitantly, contractile force is reduced to a greater extent in the deeper layers of the ventricular wall. Since significant collateral flow remains in a region after an acute coronary occlusion, contractile activity might have persisted in the epicardial layers of the wall where the flow is highest, although reduced relative to the normal state. Indeed, the present study suggests this possibility.

The induction of regional cardioplegia by the infusion of potassium into the LAD warrants further comment. The rate of infusion of potassium was designed to raise the extracellular potassium to approximately 30 mEq/l. With this extracellular potassium, the resting membrane potential would have been approximately −40 mV. When the resting potential is reduced to about one-half of its normal value, the fiber becomes inexcitable. Thus, a region of the left ventricle was made inexcitable and acontractile during potassium infusion into the LAD. The infusion did not appear to induce permanent damage to regional myocardial function.

We assume that there is a discrete region of dysfunction due to ischemia and that the identical volume of tissue is made noncontractile by the potassium infusion. This assumption is supported by other studies that indicate that there is little, if any, capillary interconnection between differing arterial sources of coronary blood. This view is also supported by recent fluorophotographic studies by Harlan et al., which show a very sharp (< 50 μ) transition from anoxic to perfused tissue after coronary ligation. Collateral blood flow arises from relatively large arterial interconnections. Thus, when potassium is infused into the LAD under conditions of equal pressure in all coronary arteries, no collateral blood flow occurs and the potassium remains in the LAD region. Leakage by blood flow and subsequent depolarization of other regions is not likely. Indeed, the end-capillary loop anatomy of the coronary circulation permits discrete regional ischemia and cardioplegia to occur. Diffusion of potassium or oxygen at the sharp interface between two discrete capillary beds may give rise to a small discordance between the boundary of the tissue affected by potassium cardioplegia and ischemia. The transition zone resulting from diffusion will be limited to a few microns, however, and will certainly be limited to one or two intercapillary spaces. Consequently, the volume of tissue involved will be minute and would have no detectable effect on ventricular performance.

In this study, LV function during ischemia was always assessed after cardioplegia. One may question if regional cardioplegia could have affected LV function during regional myocardial ischemia. This is probably not the case. LV function always returned to normal levels after cessation of potassium infusion (fig. 6) and LV function during regional cardioplegia was always significantly more depressed than during ischemia (fig. 2, table 1). Thus, the improved LV function during regional ischemia (compared to cardioplegia) could not be explained by the potassium infusion. If the last experimental intervention (ischemia in the
present study) would have caused more reduction in LV function compared with previous interventions, one may speculate that it could be due to a deterioration of the experimental preparation. Since in the present study, the last intervention (ischemia) was always associated with improved LV function (compared with previous cardioplegia), deterioration of the experimental preparations seems less likely. Thus, the deleterious effects of cardioplegia on the overall LV function compared with regional ischemia is more pronounced.

The present study supports the view that complete transmural loss of contractile activity does not occur when an acute coronary ligation is induced. Thus, some degree of transmural contractile activity remains during ischemia, while potassium infusion, which totally depolarized the region of the ischemic myocardium, abolishes all contractile activity. The area that remains contractile during acute ischemia is probably in the epicardial layers and is related to residual collateral blood flow. However, this residual contractile activity is not enough to prevent acute paradoxical bulging of the area, but bulging would presumably be even greater in the absence of residual contractile activity. This residual contractile activity was lost during potassium-induced cardioplegia and its effect was shown by the resultant fall in stroke volume at any LVEDP.

Theoretical considerations of LV function, as well as laboratory experiments, support the contention that LV failure is related to the amount of LV damage. Klein et al., in a theoretical analysis of nonfunctioning regions of LV muscle, concluded that the Frank-Starling mechanisms of functional compensations would fail when such region of akinesia approached 20–25% of the LV surface area. Survival of dogs after excision of part of the LV wall and replacement with a patch was considered to be related to the amount of heart muscle excised. Glass et al. and Stein and Cordell reported removal of over 30% or 35% of the canine LV myocardium would not permit survival. We quantitated the relation between the size of the noncontractile zone and reduction in the overall LV function. When the involved area varied from 20.3–39.8% of the LV mass, the reduction in ventricular function was proportionate to the loss of functioning myocardium. This relation is expressed by a regression line (fig. 5). Our findings support the clinical observations that extensive myocardial damage of 35–70% of LV mass is associated with severely depressed myocardial function.41-43

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