Effects of Acute Coronary Occlusion on the Motion and Perfusion of the Normal and Ischemic Interventricular Septum

An Experimental Echocardiographic Study

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SUMMARY To establish the effect of local and remote myocardial ischemia on interventricular septal motion, 27 open-chest dogs were studied using ultrasound and radioactive microspheres. In 14 dogs the left anterior descending coronary artery was ligated. If the ultrasound beam traversed ischemic septum (proximal LAD occlusion), significant ($P < 0.05$) declines in systolic septal velocity (26.4±2.9 to 6.4±1.8 mm/sec, and excursion (2.6±0.3 to 0.7±0.2 mm) occurred, and systolic thickening was reduced. Similar significant changes were seen when the ultrasound beam traversed non-ischemic septum adjacent to the ischemic area (distal LAD occlusion). In 13 additional dogs, circumflex coronary ligation produced posterior ischemia. The mean septal velocity for this group increased significantly (21.8±2.6 to 26.5±3.3 mm/sec), as did the septal excursion (2.5±0.2 to 3.1±0.4 mm). We conclude that acute LAD occlusion causes a reduction in systolic velocity, excursion, and thickening of both the involved ischemic and the adjacent nonischemic septum. When myocardial ischemia was produced in a part of the ventricle remote from the septum, septal velocity and excursion increased.

THE MOTION OF THE INTERVENTRICULAR SEPTUM, assessed echocardiographically in experimental and clinical studies, is altered by a variety of conditions including right ventricular volume overload, electrocardiographic conduction abnormalities, and cardiac surgery. Patients with coronary artery disease and acute or chronic myocardial ischemia also have changes in the motion of the septum. Such changes may take two forms: if the disease affects the septum directly, usually because of left anterior descending coronary artery stenosis or occlusion, the normal septal motion may be reduced or virtually abolished. On the other hand, if the myocardial ischemia is located elsewhere in the ventricle such as the inferior wall the septum may show "compensatory hyperactivity" — an exaggeration of normal septal motion.

Echocardiographic septal motion has not been studied in an experimental setting of coronary occlusion, and there is little information available on how abnormal septal motion in myocardial ischemia, the exact location of the coronary arterial lesions, and actual perfusion of the interventricular septum are interrelated. The purpose of this study was to evaluate systematically the effect of segmental myocardial ischemia on septal motion. Experiments were carried out in which the septum was either included in or excluded from the ischemic region, perfusion was determined, and the mechanism of "compensatory hyperactivity" was evaluated.

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Methods

Twenty-seven adult mongrel dogs weighing 15 to 26 kg were anesthetized with sodium pentobarbital — 25 mg/kg i.v. The dogs were ventilated by a Harvard respirator with room air and supplemental oxygen via a cuffed endotracheal tube. Tidal volume was adjusted to maintain arterial $pO_2$ and $pH$ within a physiologic range. A midsternal thoracotomy was performed, the pericardium incised, and the heart exposed. A small segment of the left anterior descending coronary artery was dissected free from the epicardial fat, and either an inflatable balloon cuff or a loose ligature was placed around it, either proximally, midcourse, or distally. In 13 dogs, the circumflex coronary artery was dissected free and a ligature placed around it. Heparin (250 units/kg, i.v.) was administered, and cannulae were placed in the right brachial and femoral arteries and the left atrial appendage. Polyurethane catheters (#8 French) were inserted into the ascending aorta and left ventricle by retrograde catheterization. Pressures were measured with Statham P23 strain gauges placed at the midst end position. Left ventricular dp/dt was determined from the left ventricular pressure tracing by an RC differentiation circuit. All recordings were made utilizing an Electronics for Medicine DR-12 multichannel photographic recorder.

Echocardiography

Ultrasound recordings of the motion of the interventricular septum and left ventricular posterior wall were obtained serially from each of two 2.25 mHz transducers focused at 5 cm and an ultrasonoscope (Smith-Kline Ecoline 20). The ultrasound signals were displayed on the photographic recorder simultaneously with the pressure, dp/dt, and electrocardiographic signals. Two ultrasound transducers were used so that the motion of both the superior
(cephalad) and inferior (caudal) portions of the septum could be assessed. The transducers were placed lightly on the exposed anterior surface of the heart (fig. 1) in positions from which the right and left sides of the interventricular septum and the left ventricular posterior endocardium could be well defined. The transducer positions chosen were on the right side of the left anterior descending coronary artery, varying from 0.5 to 1.5 cm from the artery. Both transducers were aimed inferiorly to the mitral leaflet echoes so as to traverse and record the motion of the interventricular septum and left ventricular posterior wall. The sensitivity of the ultrasonoscope was manipulated so as to best define the ultrasonic signal from the left ventricular posterior and septal endocardium. The transducers were fixed in place by a rigid stationary bar to avoid transmitted motion from the heart and thereby to provide a fixed reference point. Once initially satisfactory position and fixation were achieved, the transducers were not moved throughout the study, to avoid changes in recorded wall motion due to altered ultrasound beam position or direction. Verification of the ultrasound identification of anatomic landmarks was achieved by rapid injection of 5 ml of normal saline through the left ventricular or left atrial catheters; the ultrasonic contrast reflections produced filled the chamber and outline the endocardial-blood interface, confirming delineation of the border of the ventricular cavity.4–6

Figure 2 illustrates a typical echocardiogram obtained in this study. Designations corresponding to specific points within the cardiac cycle were used for the labeling and description of left ventricular posterior endocardial motion.8 Point B, the posterior wall position at end-diastole, is approximately simultaneous with mitral valve closure. During isometric contraction the wall moves posteriorly from B to C, while the subsequent anterior motion from C to D is coincident with ventricular ejection. The mean posterior endocardial wall velocity (PWV) was obtained by calculating the slope of the line drawn from the onset (C) to the end (D) of ventricular ejection, in mm/sec. Posterior endocardial wall excursion was obtained by measuring the amplitude of posterior wall motion as the vertical distance from C to D, in mm.

The systolic septal velocity (SSV) was calculated as the slope of the line drawn from the left septal position at the onset of ventricular ejection (coincident with point C of the left ventricular posterior endocardium) to the furthest posterior point reached by the left side of the septum during systole. This latter point usually occurred slightly in advance of the D point of the posterior endocardium, a relationship also commonly encountered in echocardiograms recorded from human subjects. Systolic septal excursion was obtained by measuring the vertical distance between these two systolic septal points in mm. Systolic septal velocity and excursion thereby indicate septal motion during left ventricular ejection, not pre-ejection.

Diastolic septal velocity (DSV) was measured as the slope of the line extending from the furthest posterior point of the left septum during systole to the left septal position at the time of the nadir of the left ventricular pressure tracing after ventricular ejection and isovolumetric relaxation (fig. 2). We did not attempt to determine diastolic septal velocity during ventricular filling since the length of the R-R interval would be a major determinant of such a calculated velocity. The diastolic septal excursion was measured as the vertical distance between the latter two septal points in mm. End-diastolic septal thickness in mm was measured at the "R" wave of the ECG; end-systolic thickness at the point of maximum septal thickening. Left ventricular end-diastolic diameter was measured as the vertical distance between the left side of the interventricular septum and the posterior endocardium, at end-diastole (R wave of the ECG).

Perfusion

Left ventricular myocardial perfusion was determined by use of 7–10 microspheres labeled with $^{141}$Ce, $^{86}$Sr, $^{81}$Cr, and $^{46}$Sc. For each flow measurement $9.9 \pm 0.7 \times 10^4$ microspheres were suspended in saline and injected over a 10 sec
period into the left atrium, and the cannula was then flushed with 5 ml of saline. Previous investigators have demonstrated that injection of as much as $8 \times 10^4$ microspheres directly into the coronary circulation does not alter coronary vascular resistance.\textsuperscript{7} Left ventricular and aortic pressures were recorded during this period to assure that no hemodynamic alteration occurred. Prior to injection, the vial containing the microspheres and one drop of Tween-80 was vigorously agitated mechanically for at least 4 min. Microscopic examination of microspheres prepared in this manner showed dispersion of at least 98% of the spheres. Occasional small groups of three to five spheres were observed. Starting one minute before injection and continuing until three minutes after injection, blood for reference flow determinations was withdrawn simultaneously from the right brachial and right femoral arteries at 2.06 ml/min with a Harvard pump.

Location of the Ultrasound Beam

After all recordings were completed, metal needles were lined up on two sides of each of the two ultrasound transducers and passed through the heart in parallel to mark the path of the ultrasound beams (fig. 1). To minimize postmortem deformation of the left ventricular wall, we inserted #20 needles, with sharp points, through the beating heart. The points of intersection of the needles with the interventricular septum and posterior wall were noted and the myocardial segments (see below) between these points subsequently identified, in order to enable a calculation of the perfusion of the specific segments traversed by the ultrasound beam. The animals were then killed with an injection of potassium chloride.

The heart was excised and the free walls of the right ventricle, the right and left atrium, great vessels, valves, surface vessels and epicardial fat were removed. Utilizing the posterior descending coronary artery as a starting point, the left ventricle was divided into four equal levels of eight segments each, and each segment was divided into three layers — endocardium, mid-wall, and epicardium. Thus the left ventricle was divided into 96 segments of about $1.6 \times 1.6 \times 0.3$ cm in size. Since the size of the needles was very small compared to the size of the segments, errors in beam localization should be minimal. The relative geometric position of each segment was constant from animal to animal.

Using scintillation counting and computer techniques previously described by us in detail,\textsuperscript{8} we determined the perfusion of every myocardial segment in each heart. Ischemic segments were identified using a statistical method which estimates the heterogeneity of perfusion to normally perfused segments and establishes the lowest level of perfusion associated with normal segments. Segments found to have abnormally low perfusion (seen only following coronary ligation) were classified as ischemic.\textsuperscript{9} In a separate study from this laboratory,\textsuperscript{9} we have shown that there is a close relationship between perfusion (determined in this manner) 5–10 min following coronary occlusion, and the extent of myocardial necrosis (determined histologically) 44–48 hours after occlusion.

Experimental Protocol

In the initial group of 14 dogs, the effects of left anterior descending coronary artery occlusion on the motion and perfusion of the interventricular septum were evaluated. The site of the coronary ligation was varied — proximal to both ultrasound transducers, between the two transducers, or distal to both — to vary the amount of the septum made ischemic. In this way the effects of ischemia on the motion and perfusion of both the ischemic and adjacent nonischemic septal myocardium could be ascertained. The initial judgment as to whether an ultrasound beam was traversing ischemic or nonischemic myocardium was in all cases verified by actually determining the perfusion of the segments traversed by the beam. These segments were identified by use of the marker probes described earlier. Hemodynamic and echocardiographic recordings were obtained, and labeled microsphere injections made in the control state, and 10 min after complete LAD occlusion. In each state
### Table 1. Effects of Acute LAD or Circumflex Coronary Artery Occlusion on the Motion of Ischemic and Nonischemic Septum

<table>
<thead>
<tr>
<th></th>
<th>Ischemic septum†</th>
<th>Nonischemic septum†</th>
<th>Nonischemic septum‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>LAD coronary occlusion</td>
<td>Control</td>
</tr>
<tr>
<td>Septal myocardial perfusion, ml/100g/min (segments traversed by beam)</td>
<td>73.4 ± 7.4 34.9 ± 5.6*</td>
<td>92.8 ± 10.1 78.0 ± 10.9*</td>
<td>68.8 ± 5.9 66.7 ± 5.7</td>
</tr>
<tr>
<td>Septal myocardial perfusion—% mean left ventricular perfusion</td>
<td>34.0 ± 3.0</td>
<td>43.7 ± 7.0*</td>
<td>105.5 ± 11.5 107.6 ± 15.1</td>
</tr>
<tr>
<td>Septal endocardial-pericardial perfusion ratio</td>
<td>1.06 ± 0.08 0.64 ± 0.11*</td>
<td>1.20 ± 0.05 1.19 ± 0.06</td>
<td>1.19 ± 0.09 1.23 ± 0.10</td>
</tr>
<tr>
<td>Septal myocardial perfusion, ml/100g/min</td>
<td>98.2 ± 17.6 73.1 ± 22.4*</td>
<td>88.8 ± 10.9 69.1 ± 7.8*</td>
<td>69.2 ± 5.8 16.7 ± 3.7*</td>
</tr>
<tr>
<td>Posterior myocardial perfusion—% mean left ventricular perfusion</td>
<td>95.2 ± 2.5</td>
<td>91.4 ± 1.6</td>
<td>100.7 ± 4.5 96.0 ± 4.1</td>
</tr>
<tr>
<td>Posterior endocardial-pericardial perfusion ratio</td>
<td>1.13 ± 0.17</td>
<td>1.17 ± 0.22</td>
<td>1.31 ± 0.08 1.22 ± 0.08</td>
</tr>
<tr>
<td>Septal end-diastolic thickness, mm</td>
<td>10.2 ± 0.9</td>
<td>9.7 ± 1.1</td>
<td>10.7 ± 1.0 10.4 ± 1.3</td>
</tr>
<tr>
<td>Septal end-systolic thickness, mm</td>
<td>12.8 ± 1.0</td>
<td>11.0 ± 1.6*</td>
<td>12.8 ± 1.2 11.9 ± 1.4</td>
</tr>
<tr>
<td>End-systolic/end-diastolic thickness</td>
<td>1.3 ± 0.0</td>
<td>1.1 ± 0.1*</td>
<td>1.2 ± 0.0 1.1 ± 0.0*</td>
</tr>
<tr>
<td>Systolic septal velocity, mm/sec</td>
<td>26.4 ± 2.9</td>
<td>6.4 ± 1.8*</td>
<td>24.2 ± 2.9 9.3 ± 1.7*</td>
</tr>
<tr>
<td>Systolic septal excursion, mm</td>
<td>2.6 ± 0.3</td>
<td>0.7 ± 0.2*</td>
<td>2.5 ± 0.4 1.1 ± 0.2*</td>
</tr>
<tr>
<td>Diastolic septal velocity, mm/sec</td>
<td>35.2 ± 3.4</td>
<td>23.4 ± 10.3</td>
<td>39.1 ± 3.4 38.5 ± 5.5</td>
</tr>
<tr>
<td>Diastolic septal excursion, mm</td>
<td>4.0 ± 0.5</td>
<td>3.0 ± 1.0</td>
<td>4.1 ± 0.3 3.8 ± 0.5</td>
</tr>
<tr>
<td>Posterior wall velocity, mm/sec</td>
<td>32.4 ± 2.3</td>
<td>30.8 ± 4.1</td>
<td>31.5 ± 3.2 31.3 ± 2.1</td>
</tr>
<tr>
<td>Posterior wall excursion, mm</td>
<td>3.5 ± 0.3</td>
<td>3.5 ± 0.3</td>
<td>3.5 ± 0.3 3.4 ± 0.2</td>
</tr>
<tr>
<td>B-C amplitude, mm</td>
<td>2.0 ± 0.2</td>
<td>1.5 ± 0.2*</td>
<td>2.5 ± 0.2 2.3 ± 0.2</td>
</tr>
<tr>
<td>LV end-diastolic diameter, mm</td>
<td>24.2 ± 1.3</td>
<td>26.5 ± 1.2*</td>
<td>20.2 ± 1.6 22.3 ± 1.3*</td>
</tr>
<tr>
<td>Aortic systolic pressure, mm Hg</td>
<td>134.3 ± 8.6</td>
<td>99.1 ± 10.1*</td>
<td>123.0 ± 11.9 108.6 ± 10.0*</td>
</tr>
<tr>
<td>Aortic diastolic pressure, mm Hg</td>
<td>92.8 ± 11.0</td>
<td>68.0 ± 11.7*</td>
<td>92.6 ± 10.8 77.7 ± 9.6*</td>
</tr>
<tr>
<td>Left ventricular end-diastolic pressure mm Hg</td>
<td>7.7 ± 0.7</td>
<td>12.2 ± 1.5*</td>
<td>8.9 ± 1.3 10.1 ± 1.3</td>
</tr>
<tr>
<td>Left ventricular dp/dt, mm Hg/sec</td>
<td>2600 ± 103</td>
<td>1661 ± 145*</td>
<td>2261 ± 274 1900 ± 196*</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>149.3 ± 4.7</td>
<td>137.2 ± 5.8*</td>
<td>159.6 ± 5.8 147.1 ± 3.3*</td>
</tr>
</tbody>
</table>

All values are means ± standard error of the mean.

†P < 0.05 occlusion vs control.

‡P < 0.01 occlusion vs control.
Echocardiographic recordings were obtained from each of the two ultrasound transducers used on each dog, so that two echo data points were obtained from each of these 14 animals for each state.

In a second group of 13 dogs, the effect of circumflex coronary artery occlusion (posterior ischemia) on septal motion was evaluated. Control hemodynamic and echocardiographic recordings and microsphere error. The circumflex coronary artery was occluded and the recordings were repeated 15 min later. In this group of dogs only one transducer, placed over the superior portion of the interventricular septum, was used to obtain echocardiographic recordings.

Data were analyzed utilizing a paired or unpaired Student's t-test. Results are expressed as the mean ± 1 standard error.

Results

Ischemic Septum (table 1)

Proximal occlusion of the left anterior descending coronary artery for 15 min caused a significant decline in the perfusion of the septal segments traversed by the ultrasound beam and in the endocardium-epicardium perfusion ratio of these segments. Perfusion of the nonischemic posterior segments traversed by the beam also fell, but percent of mean LV perfusion and endocardium-epicardium perfusion ratio did not. This indicates that the fall in perfusion of the posterior segments reflected a general decline in ventricular perfusion rather than a segmental perfusion deficit in that area.

Systolic and diastolic septal velocity and excursion fell and a general flattening of septal motion occurred (fig. 3); in three animals an anterior bulging of the ischemic septum during the pre-ejection period occurred, accompanied by a directional reversal of the septal motion in early diastole (fig. 3). Thickening of the septum during systole diminished. Posterior wall velocity and excursion changed little, but the end-diastolic ventricular diameter increased. Arterial pressures declined and left ventricular end-diastolic pressures rose.

Nonischemic Septum (table 1)

In this group of animals the coronary ligation was placed distally so that one or both ultrasound beams traversed nonischemic septum, proximal to the LAD occlusion. Data is given only from nonischemic septal segments. The perfusion of these septal segments did show a small decline after LAD occlusion, which is probably due to the reduction in metabolic demand secondary to the drop in systolic pressure. It was not a segmental perfusion deficit since the percent of mean left ventricular perfusion of the septal segments in this group of animals did not change, nor did the endocardium-epicardium perfusion ratio fall. Systolic septal velocity and excursion showed declines after occlusion (fig. 4). End-systolic septal thickness showed only a small and insignificant decline in this group, although the ratio of end-systolic to end-diastolic thickness did fall significantly. Diastolic septal motion for the whole group was not affected, as opposed to the change seen in the group of dogs where the beam traversed ischemic septum. Posterior wall motion was unchanged. End-diastolic ventricular diameter and pressure increased and arterial pressures decreased.

Figure 3. Echocardiogram illustrating changes in motion of ischemic septum after LAD coronary occlusion. A marked reduction of systolic and diastolic septal velocity occurred. In this animal an anterior bulging of the ischemic septum during the pre-ejection period occurred, and a reversal in the direction of early diastolic septal motion was also apparent. Posterior wall motion was only minimally changed.
Effects of Acute Circumflex Coronary Occlusion (Posterior Ischemia) on the Motion of Normally Perfused Septum (table 1)

Circumflex occlusion produced significant falls in the perfusion and endocardium-epicardium perfusion ratio of the posterior segments traversed by the beam; perfusion of the septal segments did not change. Posterior velocity and excursion fell and B-C amplitude increased. The septal systolic and diastolic velocity and excursions showed a significant rise (fig. 5). End-diastolic diameter and pressures increased and aortic pressures fell.

Compensatory Hyperactivity of Opposing Nonischemic Myocardium

In order to assess the effect of different locations of ischemia on motion of nonischemic opposing myocardium, the changes in posterior wall velocity of each dog with septal ischemia (fig. 6) were compared to the change in systolic septal velocity of each animal with posterior ischemia (fig. 7). To make the data comparable, we plotted posterior wall velocity from only the more superior of the two transducers used in dogs undergoing LAD occlusion.

In the septal ischemia animals "compensatory hyperactivity," an increase in posterior wall velocity, was registered by the ultrasound beam in six of the 14 dogs (43%); the remainder showed a decline or no change, and the mean posterior wall velocity for the whole group did not change significantly. In the posterior ischemia group, however, the septal velocity increased in ten of 13 animals (77%) and the mean septal velocity for the whole group showed a significant rise, from 21.8 ± 2.6 mm/sec to 26.5 ± 3.3 mm/sec (P < 0.05). Sixteen of 27 animals in these two groups showed compensatory hyperactivity.
In order to find out why this difference in compensatory hyperactivity occurred, we determined the size of the ischemic area (expressed as percentage of total left ventricular mass) in each dog and compared these two groups. Using an unpaired t-test, we found that percentage of left ventricular weight rendered ischemic was significantly larger in the posterior ischemia animals (33.4 ± 2.3% vs 26.0 ± 3.0%, *P* < 0.05) (fig. 8). The larger size of the ischemic area in these dogs might be expected to cause a greater increase in ventricular volume. To evaluate this we measured the left ventricular end-diastolic diameter in the two groups and found that the mean diameter in the posterior ischemia dogs was indeed larger: 35.7 ± 1.8 mm vs 26.7 ± 1.9 mm (*P* < 0.05) (fig. 8).

In the dogs undergoing LAD coronary occlusion the perfusion of the nonischemic posterior segments fell — presumably reflecting the decline in aortic pressures, since the percentage of mean LV perfusion remained constant. However, in the dogs undergoing circumflex coronary occlusion the perfusion of the nonischemic septal segments did not fall, despite a similar drop in aortic pressures.

**Discussion**

In this study acute ischemia of the interventricular septum produced a flattening of both the systolic and diastolic echocardiographic motion of the left side of the septum, and also impaired the ability of the septum to thicken during systole. Jacobs et al. demonstrated that reductions in septal motion occurred in chronic coronary artery disease with obstructive lesions in the left anterior descending coronary artery and usually a previous transmural infarction. This implied that a perfusion deficit produces a reduction in septal motion. This was confirmed by Beaver et al. who injected Indium selectively into the left coronary artery and found that patients with reduced septal motion generally had a septal perfusion deficit and probable old infarct by ECG criteria. The present study extends these observations on patients with old infarction and chronic myocardial fibrosis to a setting of an experimentally induced acute septal perfusion deficit and provides support for the clinical observation that electrocardiographically diagnosed acute anterior myocardial infarction in man may be associated with a reduction of normal septal motion because of septal ischemia.

Ischemia of the inferior portion of the septum only (distal LAD occlusion) produced significant reductions of motion and systolic thickening in the superior portion of the septum, even though this area was shown to have normal perfusion. In a previous investigation we demonstrated similar motion abnormalities in nonischemic left ventricular posterior wall areas which were immediately adjacent to acutely ischemic areas. Recently, Wyatt et al. using epicardial length gauges, also found that reductions in systolic shortening occurred in areas of the left ventricle close to an acutely ischemic area but perfused by patent vessels. These motion abnormalities may be due to passive changes induced by the severe dyskinesia of the ischemic myocardium. Wyatt et al. found that the motion changes of adjacent nonischemic myocardium diminished in magnitude as distance from the...
occluded vessel increased. Changes in right ventricular size and contractility or changes in left ventricular geometry and contractility induced by the fall in aortic pressure after coronary occlusion are other influences on both ischemic and nonischemic septal motion. These parameters cannot be fully assessed by the methods used in this study.

The reduction of proximal septal motion with distal LAD occlusion has potential diagnostic implications. Echocardiographic septal motion abnormalities have been said to reflect the location of the LAD lesion in relation to the first septal perforator branch. But if distal LAD obstruction produces proximal dyskinesis in humans, as we observed in these animal experiments, the presence of septal dyskinesis may not be useful in localizing LAD lesions. Further clinical studies will be necessary to settle this question.

Diastolic velocity of ischemic septum fell whereas diastolic velocity of normally perfused septum did not. McLaurin et al. found that a defect in myocardial relaxation was an early and sensitive manifestation of regional myocardial ischemia; such a relaxation abnormality would be compatible with the reduction in diastolic septal velocity which we observed in ischemic septum. Diastolic motion changes may provide a means of distinguishing between ischemic and nonischemic septal myocardium, since the systolic motion abnormalities were similar in both areas following LAD occlusion.

Increased motion of opposing cardiac wall — compensatory hyperactivity — occurred in 16 of 27 (60%) of the animals. A noteworthy finding was that septal hyperactivity was more likely to occur than posterior wall hyperactivity. This propensity of the septum for exaggerated motion, as compared with that of the posterior wall, has previously been noted in clinical and experimental studies. Corya et al. found left septal amplitude increased in 12 of 31 patients with acute inferior wall infarction, whereas posterior endocardial amplitude was above normal in only four of 25 patients with acute anterior infarction. Heyndrickx et al. found that velocity of shortening of nonischemic segment length increased with acute experimental left circumflex coronary artery occlusions of 5 or 15 min, but did not change significantly after LAD occlusion. In this study we found that the percentage of left ventricular mass rendered ischemic was significantly higher in the dogs with posterior ischemia than in those with septal ischemia, and that their left ventricular end-diastolic diameter was correspondingly larger (fig. 8). This suggests that a Starling mechanism was occurring in the nonischemic myocardium: as the size of the ischemic zone became larger and the ventricle dilated, diastolic length in the normal area increased and this produced an increase in shortening. A similar conclusion was reached by Theroux et al. who used implanted ultrasound dimension gauges to study regional myocardial function after acute coronary occlusion.

Finally, it should be noted that in the dogs undergoing posterior ischemia, perfusion of the nonischemic septal segments did not fall despite a decrease in aortic pressure. This is contrary to the decline in nonischemic segment perfusion seen in other instances where blood pressure fell. This preservation of perfusion to the septal segments may be a consequence of increased septal motion with resultant increased metabolic requirements of the hyperactive myocardium.

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