Experimental Myocardial Ischemia

IV. Shape and Volume Changes During "Isovolumetric Relaxation" in Normal and Ischemic Ventrices

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SUMMARY  Shape and volume changes were studied in mongrel dogs between end-systole and mitral valve opening (MVO). Biplane left ventricular cineangiograms were performed at 200 frames/sec. The dogs were studied during the control state and during regional myocardial ischemia produced by balloon occlusion of the left anterior descending coronary artery. Outward wall motion, called pre-inflow relaxation (PIR), occurred in all 10 dogs in the control state, most frequently in the apical (seven of 10) and equatorial planes (seven of 10). PIR was seen less frequently during ischemia (13 of 40 measurements vs 19 of 40 measurements during control state), usually in the basal plane (five of 10).

The ventricular volume between end-systole and MVO increased in all 10 dogs during the control state (mean increment 4.4 ± 0.7 ml). Volume increased in eight of 10 dogs during ischemia (mean increment 3.0 ± 1.1 ml). The characteristic patterns of wall motion occurring between end-systole and MVO are altered by regional myocardial ischemia. During ischemia PIR occurs in segments of the myocardium with normal perfusion, but usually not in ischemic segments. Biplane ventricular volume between end-systole and MVO increased in 18 of 20 measurements (mean increment 3.6 ± 0.9 ml).

PORTIONS OF SYSTOLE and diastole during which large quantities of blood leave or enter the left ventricle have been studied often.1 These events occupy major portions of the cardiac cycle and are easily studied by standard angiocardiographic techniques.2 Little attention has been given to the changes that occur in shape and possibly volume between end-systole and opening of the mitral valve. This short segment of the cardiac cycle, lasting little more than 100 msec, may well involve major physiologic and metabolic changes needed for contraction and relaxation. Awareness of the normal pattern of wall motion during this period may alert angiocardiographers to subtle abnormalities of ventricular function.

Ventriculographic studies in humans have described a phenomenon of outward wall motion occurring between end-systole and opening of the mitral valve in both normal and abnormal ventricles.3,4 However, no studies have reported this phenomenon in normal ventricles and after an intervention such as regional myocardial ischemia. Accordingly, this study was designed to detect changes in specific areas of the myocardium before and during regional myocardial ischemia.

Materials and Methods

Fourteen mongrel dogs of both sexes weighing approximately 20 kg were studied during anesthesia with morphine sulfate (1.5–4.5 mg/kg), fentanylphropendorph (Inovar-Vet) (0.1–0.3 mg/kg) and α chloralose (100–150 mg/kg), tracheal intubation and assisted ventilation. During the study four dogs experienced ventricular fibrillation. Their data are excluded from the study. Catheters (USCI or KIFA, #7 or #8F) were inserted intravenously or intra-arterially into the central aorta, left ventricle, and coronary sinus (USCI, bipolar pacing catheter, #7F). Heart rate was controlled by electrical pacing (Physiologic Stimulator, Grass Instruments, Inc.), and atrioventricular nodal conduction was obtained in six of the 10 dogs.

Arterial pressures were measured from the central aorta (AoP) using a saline-filled catheter and an external transducer (Statham P23Db). Left ventricular pressure (LVP) was measured using a calibrated, transducer-tipped catheter (Millar Instruments) and a high-frequency-response preamplifier (Grass Instruments, Inc.). An endhole, balloon-tip catheter (Swan-Ganz #4 or #5F) was placed in the left anterior descending coronary artery (LAD) beyond the first major septal branch. Pressure in the distal LAD was monitored while the balloon was inflated with dilute contrast media (30% sodium meglumine diatrizoate) until the mean perfusion pressure was below 50 mm Hg. An estimate of the level of the left atrium was used as a zero reference. The first derivative of ventricular pressure, dP/dt, was measured using an electronic differentiating circuit.

Biplane cineangiograms of the left ventricle were recorded on 16-mm Plus-X film (contrast medium, 75% sodium meglumine diatrizoate; dose, 1.0 ml/kg; rate, 7 ml/sec; duration of injection, 3 seconds; shutter speed, 200 frames/sec). Eclere cameras were used to photograph the output phosphors of dual-mode 6–10-inch, high-resolution image intensifiers. To match the records from LVP and the left ventricular volumes from cine tracing, a single, standard-lead ECG was
simultaneously recorded and inscribed on the side of each cine frame. The equipment provided pulsed exposures which were recorded on top of the AoP, LV dP/dt and ECG tracing.

Dogs were strapped in position to minimize motion with the vertical x-ray beam perpendicular to a sagittal plane of the ventricle (approximately 33° right anterior oblique [RAO]) and the horizontal beam perpendicular to a coronal plane approximately 67° left anterior oblique [LAO]). After each set of experiments a grid of known dimensions was filmed in each plane with the same tube-to-image intensifier distance to calibrate cineangiographic images. One or more cardiac cycles after the completion of contrast injection were chosen for analysis. Premature ventricular complexes and the first three postextrasystolic contractions were excluded from consideration. The image obtained at the beginning of the rise of the +dP/dt was assumed to be end-diastole. The image obtained at the beginning of the fall of the −dP/dt was assumed to be end-systole. Opening of the mitral valve was clearly identifiable in every case. Identification of mitral valve was often aided by detecting the leaflet in the partially open state and running the film in reverse to make it look as though the valve was closing.

The corresponding end-diastolic, end-systolic and mitral valve opening (MVO) frames were then identified on the simultaneously obtained RAO and LAO projections in eight dogs by using the R wave of the ECG trace and the digital clocks for proper registration. In two dogs only the RAO projection was obtained.

Silhouettes of the RAO and LAO images of the left ventricle at end-diastole, end-systole and MVO were drawn; the length from the midpoint of the aortic valve to ventricular apex, the ventricular diameter at the midpoint of the length, the apical and basal chords, and the area of each image were measured. End-diastolic volume (EDV), end-systolic volume (ESV), and pre-inflow volumes (PIV)* were computed using a digitizing board employing a program based on Simpson’s rule. No regression formula was used to correct data. The computer volumes were tested against water displacement volumes of 13 canine ventricular molds, showing r = 0.97 and F = 126 (true volume = 0.80 [calculated volume] −0.38).

Stroke volume (SV) and ejection fraction (EF) were calculated in the usual manner from the ESV and EDV (SV = EDV − ESV, EF = SV/EDV × 100). Similar calculations of LV volume were made, substituting PIV for the ESV. The volume increment from end-systole to MVO was calculated (PIV − ESV); this was also expressed as a percentage of the SV (PIV − ESV/SV) × 100).

The time was recorded by a digital clock on the cine films in milliseconds and time interval was calculated by subtracting the time elapsed between end-diastole, end-systole and MVO. The apical and basal chords were chosen at the point midway between the mid-equatorial diameter and the apex or base (midpoint of aortic valve). Chords were drawn perpendicular to the major axis at these points. The circumferences in three planes were calculated using the standard formula. The circumferences at end-systole and MVO were calculated, and the percentage of change occurring during the period was calculated as:

\[
\frac{\text{circ ES} - \text{circ MVO}}{\text{circ ES}} \times 100
\]

Mean velocity of circumferential fiber length change (mean Vcf) in the equatorial (E), apical (AC), and basal (BC) chords was calculated by dividing the circumference in centimeters by the time in seconds from end-systole to MVO.

Fractional change of the segmental diameter (FC SD) was calculated at the equator, apical and basal chords in the RAO and LAO projections using the actual measurement of the diameters (dia) using the formula:

\[
\frac{\text{dia ES} - \text{dia MVO}}{\text{dia ES}} \times 100
\]

The phenomenon of outward LV wall motion occurring between end-systole (ES) and MVO is termed pre-inflow relaxation (PIR). Quantitatively, it is defined as an increment greater than 2 SEM values of the midequatorial diameter (E) or the diameter at either the basal or apical chord. An increment of greater than 2 SEM in apex-to-base length in the RAO projection between end-systole and MVO is likewise considered evidence of PIR in the major axis. Since end-systole is used as the baseline, numbers throughout these data that reflect outward wall motion have a negative sign.

**Protocol**

To obtain a steady state, 20–30 minutes were allowed to elapse after onset of a paced, regular rhythm; then, pressure measurements, cardiac output and cineangiography of the left ventricle were obtained. Heart rate was held constant at a threshold 15–20% above the spontaneous rate during the experimental procedure. Data were obtained at two states, during the control period, and 20–40 minutes after balloon occlusion of the LAD.

**Results**

Physiologic data are presented for six paced dogs. Heart rate was maintained constant at 92 ± 12 beats/min throughout the study. Cardiac output was 2.2 ± 0.3 l/min during control and 1.9 ± 0.3 l/min during ischemia. The aortic pressure was 126/97 ± 5/
± 5 mm Hg during control and 130/102 ± 7/± 5 mm Hg during ischemia. The left ventricular end-diastolic pressure was 5 ± 1 mm Hg during control and 6 ± 0.7 mm Hg during ischemia. The data for the +dP/dt and −dP/dt are:

Control. +dP/dt, 2526 ± 449 mm Hg/sec; −dP/dt, 2564 ± 394 mm Hg/sec.

Ischemia. +dP/dt, 2118 ± 398 mm Hg/sec; −dP/dt, 2278 ± 349 mm Hg/sec.

Our results on shape and volume refer to the period between end-systole and MVO. The time elapsed was 115.5 ± 10.8 msec in control and 114.0 ± 7.4 msec during ischemia. At a rate of 200 frames/sec (5-msec interval between frames), there was an average of 23 ventriculographic silhouettes during isovolumetric relaxation.

Shape

Control

PIR occurred in all 10 dogs during the control state. Of the 30 chords and 10 axes measured, PIR appeared in 19 of 40. Outward wall motion was detected by measurements in the apical plane in seven dogs, the equatorial plane in seven dogs, the basal plane in one dog, and the major axis in four dogs. In seven dogs PIR was present in two or more chords or axes, occurring simultaneously in the apical and equatorial chords in six dogs (table 1).

Table 2 shows the mean circumference (cm) at end-systole and at MVO and the mean difference in centimeters. The mean percentage change in diameter from ES to MVO indicates that the major areas demonstrating outward wall motion were the equator (E = 12.99%) and the apical chord (AC = 13.44%). There was no significant change in length of the major axis from end-systole to MVO.

Table 3 shows the mean Vcf between end-systole and MVO; the negative values indicate outward wall motion at the equator (−19.77 cm/sec) and at the apex (−14.19 cm/sec).

In the same table the mean fractional change of segmental diameter (FCSD) values are shown. The negative values in the RAO projection indicate outward wall motion at the equator and apex (E = 13.2% and AC = −15.0%). The negative values in the LAO projection also indicate outward wall motion predominantly at the equator and apex (E = −13.5% and AC = −12.7%).

Ischemia

PIR was observed in nine dogs during ischemia due to distal LAD occlusion. Of the 30 chords and 10 axes measured, PIR was present in 13 measurements. Table 1 shows the altered location of PIR in each dog from the control to the ischemic state. PIR occurred in the apical plane in one dog, in the equatorial plane in three dogs, in the basal plane in five dogs and in the major axis in four dogs.

Of the five dogs with basal PIR during ischemia, none showed basal PIR during the control state. Four of these five had pronounced apical PIR during control that was not observed during ischemia. The three dogs with equatorial PIR during ischemia also had outward wall motion in this area during the control state. Only one of the seven dogs with apical PIR actually retained a significant degree of outward wall motion during ischemia.

Two of the four dogs with significant major-axis PIR during control retained this phenomenon during ischemia, two lost major-axis PIR, and two demonstrated such motion for the first time during ischemia.

Table 2 shows the mean circumference (cm) at end-systole and at MVO and the mean difference (cm) during LAD ischemia. All circumferences are greater during ischemia but the difference between end-systole and MVO is largest at the base (−1.52 cm) vs the control state, where the major differences occurred at the equator (−1.83 cm) and apex (−1.50 cm).

The mean percentage change in diameters from

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**Table 1. Location of Pre-inflow Relaxation in Dogs During Control and Ischemia**

<table>
<thead>
<tr>
<th>Dogs</th>
<th>BC</th>
<th>E</th>
<th>AC</th>
<th>MA</th>
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<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>PIR</td>
<td>PIR</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
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<table>
<thead>
<tr>
<th>Totals</th>
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<th>7</th>
<th>7</th>
<th>4</th>
</tr>
</thead>
</table>

Abbreviations: PIR = pre-inflow relaxation; BC = basal chord; E = equator; AC = apical chord; MA = major axis.
end-systole to MVO is shown in Table 2. The negative value at the base (-7.22%) indicates a tendency toward net outward wall motion in this region vs the control state, whereas the positive value (+1.98%) indicates no significant net wall motion. The values at the equator and apex (-1.17 and 4.805) indicate no significant net wall motion in the ischemic zone. The mean values for major axis from end-systole to MVO indicate no change.

The mean $V_{cf}$ indicated no significant net wall motion during ischemia (Table 3).

Separate analyses of wall motion in the RAO and LAO projections using FCSD values (Table 3) demonstrate that outward wall motion at the base is best seen in the LAO projection (BC = -14.1%). The equatorial and apical planes demonstrate a loss of outward wall motion in both RAO and LAO projections during LAD ischemia.

**Volume**

**Control**

During the control state the mean EDV was $56.4 \pm 4.4$ ml; the mean ESV was $27.6 \pm 2.5$ ml; and the
mean SV was 28.8 ± 3.2 ml (table 4, columns 1, 2 and 3). The PIV was 32.0 ± 2.5 ml (column 6). The ventricular volume increased in all 10 dogs. From end-systole to MVO the mean increment in ventricular volume was 4.4 ± 0.7 ml. Substituting the PIV for ESV in the calculation of mean SV resulted in a reduction from 28.8 ± 3.2 ml to 24.4 ± 3.0 ml (corrected SV). Substituting PIV for ESV in the calculation of EF resulted in a decrease, from 0.50 ± 0.03 to 0.42 ± 0.03 (corrected EF) (table 4, columns 5 and 7). The mean decrement in corrected SV vs original estimate of SV was 16.6% (table 4, column 9).

**Ischemia**

During ischemia the mean EDV was 59.8 ± 0.8 ml; the mean ESV was 37.9 ± 3.4 ml; and the mean SV was 21.9 ± 1.9 (table 4, columns 1, 2 and 3). The PIV was 40.8 ± 3.6 ml. The ventricular volume increased in eight of 10 dogs, and the mean increment in ventricular volume was 3.0 ± 1.1 ml. Substituting PIV for ESV, mean SV for the 10 dogs was reduced from 21.9 ± 1.9 to 18.7 ± 2.3 ml (corrected SV). Substituting PIV for ESV in the calculation of EF resulted in a decrease from 0.37 ± 0.4 to 0.32 ± 0.04 (corrected EF) (table 4, columns 5 and 7). The mean decrease in corrected SV vs original estimate of SV was 15.1% (table 4, column 9).

**Discussion**

Our results show that in the left ventricle of the dog there is a definite change in shape between end-systole and MVO. This change in shape, which is predominantly outward wall motion, is referred to as preinflow relaxation, suggesting its occurrence before MVO. Other investigators have referred to the change in shape as a segmental early relaxation phenomenon (SERP).\(^8\)

**Shape**

Because of the complexity of the data, this section will deal separately with changes in shape and volume. First, outward wall motion (PIR) will be discussed, followed by changes in volume between end-systole and MVO.

**Preinflow Relaxation**

In our study, the distribution of PIR in the normal heart definitely favors the equator and apex, while occasionally occurring in the major axis and only rarely at the base. This distribution is similar to that described by Rutley et al., who found a definite predilection for the anterior wall in the normal and ischemic myocardium. Under ischemic conditions a change in the locations and frequency of PIR was observed. During myocardial ischemia the location of PIR changes from its apical and equatorial location in the normal heart and was observed most frequently at the base. This is the area of the heart that is presumably spared during distal LAD occlusion. There is a significant decrease in the occurrence of PIR at the equator and apex, the areas of ischemic myocardium. There is no change in the frequency of occurrence of PIR in the major axis, which is in distal LAD ischemia would presumably be partially ischemic and partially nonischemic. There was a good correlation in individual dogs between the development of ventricular asynergy at the equator and the apex and the loss of PIR in these areas. This suggests some correlation between the loss of PIR and

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**TABLE 4. Mean Differences in Stroke Volume and Ejection Fraction Based on End-diastolic, End-Systolic and Pre-inflow Measurements in Dogs during Control and Ischemia**

<table>
<thead>
<tr>
<th></th>
<th>EDV (ml)</th>
<th>ESV (corrected EDV)</th>
<th>Stroke Volume (EDV - ESV)</th>
<th>Corrected Stroke Volume (ESV - PIV)</th>
<th>Ejection Fraction (ESV - PIV) EDV</th>
<th>PIV (ml)</th>
<th>Corrected Ejection Fraction (PIV) ESV</th>
<th>PIV - ESV</th>
<th>PIV - ESV / EDV × 100</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong></td>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Mean</td>
<td>56.4</td>
<td>27.6</td>
<td>28.8</td>
<td>24.4</td>
<td>0.50</td>
<td>32.0</td>
<td>0.42</td>
<td>4.4</td>
<td>16.6</td>
</tr>
<tr>
<td>SEM</td>
<td>4.4</td>
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<td>3.2</td>
<td>3.0</td>
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<td>2.5</td>
<td>0.03</td>
<td>0.7</td>
<td>3.0</td>
</tr>
<tr>
<td><strong>Ischemia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>59.8</td>
<td>37.9</td>
<td>21.9</td>
<td>18.7</td>
<td>0.37</td>
<td>40.8</td>
<td>0.32</td>
<td>3.0</td>
<td>15.1</td>
</tr>
<tr>
<td>SEM</td>
<td>3.8</td>
<td>3.4</td>
<td>1.9</td>
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<td>3.6</td>
<td>0.04</td>
<td>1.1</td>
<td>6.2</td>
</tr>
</tbody>
</table>

Abbreviations: EDV = end-diastolic volume; ESV = end-systolic volume; PIV = pre-inflow volume (ventricular volume immediately before mitral valve opening); EDV − PIV = true forward stroke volume; PIV − ESV = difference in left ventricular (LV) volume between end-systole and immediately before mitral valve opening; PIV − ESV EDV − ESV × 100 = percentage difference in corrected stroke volume for original estimate of stroke volume; EDV − PIV = difference in LV volume between end-diastole and immediately before mitral valve opening.
depressed contractility due to regional myocardial ischemia.

This alteration in the distribution of PIR from the normal to the ischemic heart correlates with findings by Ruttley et al., who noted that in the asynergic ventricle, outward motion almost invariably occurred in the region of optimal contraction. In 27 of their 29 patients with ventricular asynergy, outward movement of the ventricular wall was noted precisely at the site of maximal systolic contraction. Alteri et al. also observed that in patients with contraction abnormalities segmental early relaxation usually occurred in the normally contracting areas.

Earlier workers studying the phenomenon of wall motion between end-systole and MVO used either exclusively or predominantly uniplane angiographic techniques. The question arose regarding what exactly was happening to those walls not seen in RAO projection, namely the septum and posterior wall. Was outward wall motion confined exclusively to the anterolateral wall, apex and diaphragmatic surface of the heart or were the septum and posterior wall also moving in the same direction?

Our data regarding magnitude, direction and velocity of wall motion were obtained from biplane techniques. The data in table 3 on mean FCSD are presented separately for the RAO and LAO projections. In both projections outward wall motion occurs at the equator and apex in the control state and is lost during ischemia. Clearly, PIR is not a focal change in shape accompanied by an equal but opposite change occurring in a nonvisualized segment of the ventricle.

PIR has been associated with the contractile state of the heart. If this assumption is true, any alteration of myocardial contractility would be reflected in the PIR phenomenon. Our results show that the mechanical parameters of cardiac function were altered by LAD occlusion. The velocity of myocardial contraction as reflected by peak dP/dt, the velocity of myocardial relaxation as reflected by peak dP/dt and the EF were significantly decreased during myocardial ischemia. During ischemia, PIR disappeared from the affected area. Absence of PIR during ischemia correlated with a decrease in the peak negative value for the first derivative of LVP (−dP/dt), which was used in this study as an index of LV relaxation rate. The depression of the EF indicates that myocardial contraction was altered. It thus appears that regional myocardial ischemia alters both myocardial contraction and relaxation, and such alteration can be shown angiographically.

Ventricular relaxation appears to be an important determinant of LV diastolic pressure-volume relations. Impairment of ventricular relaxation may be partly responsible for the apparent decrease in LV diastolic compliance observed during regional myocardial ischemia.

Volume

Our results show that in the left ventricle of the dog, under the experimental design of this study, the volume of the ventricle increased from end-systole to MVO. Volume increase was also found by Alteri et al. in 39 patients with segmental early relaxation phenomenon. A volume increment of 6.1 ml in his study represented 6.3% of his listed EDV of 97 ml/m². The 3.6-ml mean increment in our animal data represents 6% of our mean EDV of 60.3 ml.

Ruttley et al. also showed a volume increment during isovolumetric relaxation ranging from a 13% increase over ESV in normal subjects to 18% in subjects with coronary artery disease and abnormal ventriculograms. Our animal data reveal a 16.6% increment during the control state but only a 15.1% increment during ischemia. The magnitude of the volume changes, however, corroborate the findings of both Ruttley et al. and Alteri et al.

Our volume data differ from those of Ruttley et al., who showed larger increases in abnormal ventricles, while the acutely ischemic ventricle in dogs showed less of an increase vs our control state. One explanation may be that Ruttley’s data in the abnormal ventricle were obtained not while the ventricles were acutely ischemic, but under conditions of chronic ischemia and possibly even after myocardial infarction with well-established zones of myocardial fibrosis.

Another major question arising from the volume data is how ventricular volume can increase between end-systole and MVO. We defined end-systole as the point at which the −dP/dt departed from baseline and end-diastole as the point at which the dP/dt departed from baseline. These definitions obviated the occasional discrepancies among observers when defining end-diastole and end-systole as the largest and smallest silhouette, respectively.

At frame rates of 200/sec the 5-msec interval between images provides a number of frames that visually could be interpreted as the smallest or largest. Thus the definition of these points based on the first derivative of pressure change within the ventricle as measured with a transducer-tipped catheter provided the most precise data as to end-systole and end-diastole.

Using this definition of the end-systole and the angiographic end-point for MVO our measurement of the isovolumetric relaxation period (mean 113.5 ± 10.5 msec) correlated with Alteri’s angiographic method in man of 100 ± 23 msec. The measurements also correlate with that of other authors who used the graphic method measuring the time from the aortic component of the second heart sound to the “0” point of the apexcardiogram (105 ± 14 msec and 103 ± 22 msec).

The most credible hypothesis for the volume increase during isovolumetric relaxation is that blood is returned to the LV cavity from the aortic root during closure of the aortic valve. The fact that the catheter and sometimes the contrast agent can be seen moving toward the ventricular apex at the time of the volume increase lends some support to this explanation. In 1961, Hawthorne demonstrated a sudden increase in
the base-to-apex length of a dog's left ventricle at end-systole. Lynch and Bove reported an increase in length immediately after aortic valve closure with ballooning of the apex in canine hearts. Neither Hawthorne nor Lynch and Bove detected any concurrent decrease in any other measured intraventricular distance; therefore, we must conclude that an increment in ventricular volume occurred in their investigation.

It is equally important to seek corroboration of the ventricular volume changes in aortic flow phenomena. Spencer and Greiss demonstrated that at or about the time of aortic valve closure, blood flow reverses in the aorta. Fabian and Abrams demonstrated the presence of minimal aortic "reflux" after the termination of ventricular ejection in dogs with normal aortic valves under varying conditions of intrathoracic pressure and varying volumes of contrast injection.

In conclusion, specific patterns of ventricular wall motion occur during isovolumetric relaxation. These patterns are altered by regional myocardial ischemia. Abnormal patterns of relaxation may be related to the decreased diastolic compliance of the left ventricle during regional myocardial ischemia.

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Experimental myocardial ischemia. IV. Shape and volume changes during "isovolumetric relaxation" in normal and ischemic ventricles.
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