Effects of right ventricular ischemia on left ventricular geometry and the end-diastolic pressure-volume relationship in the dog

YOICHI GOTO, JIN YAMAMOTO, MUNEYASU SAITO, KAZUO HAZE, TETSUYA SUMIYOSHI, KENICHI FUKAMI, AND KATSUKO HIRAMORI

ABSTRACT We studied the effects of right ventricular ischemia on left ventricular three-dimensional geometry and the end-diastolic pressure-volume relationship in 16 open-chest dogs before and after pericardectomy. Left ventricular volume was calculated from three internal dimensions measured with ultrasonic crystals. In one group of eight dogs, right coronary artery (RCA) occlusion for 2 min with the pericardium intact reduced aortic flow by 24 ± 9% (p < .001) and septal-lateral dimension by 8 ± 5% (p < .01), without changing anterior-posterior and apical-basal dimensions. However, parameters of left ventricular systolic function (aortic flow, left ventricular systolic pressure, peak dP/dt, and mean percent systolic shortening) were similar to those observed at a comparable level of left ventricular end-diastolic volume during inferior vena caval occlusion. In the other group of eight dogs, during RCA occlusion before pericardectomy the left ventricular end-diastolic pressure-volume relationship determined during rapid blood transfusion shifted leftward and upward significantly from the preocclusion relationship. After pericardectomy, RCA occlusion caused less significant changes in aortic flow and septal-lateral dimension as well as in the left ventricular end-diastolic pressure-volume relationship. We concluded that right ventricular ischemia causes a leftward shift of the interventricular septum in end-diastole and an alteration of the left ventricular end-diastolic pressure-volume relationship without changing left ventricular myocardial performance. These changes are enhanced by the intact pericardium.


AN INCREASE in right ventricular filling and the presence of the pericardium have been shown to cause a leftward and upward shift of left ventricular diastolic pressure-volume curves. Also, there is experimental evidence that the interventricular septum shifts leftward in end-diastole and left ventricular geometry is distorted when right ventricular loading is induced by pulmonary artery constrictions, atrial septal defect, the Mueller maneuver or positive end-expiratory pressure ventilation. However, the effects of right ventricular infarction or ischemia on left ventricular diastolic properties have not yet been reported. Although Goldstein et al. showed that elevated intrapericardial pressure after right ventricular infarction could reduce left ventricular preload and cardiac output, they neither evaluated left ventricular geometry nor measured left ventricular volume.

The goal of the present study was to elucidate the effects of right ventricular ischemia on left ventricular geometry and the end-diastolic pressure-volume relationship in dogs, and to consider the importance of the intact pericardium. To this end, we measured septal-lateral, anterior-posterior, and apical-basal dimensions of the left ventricle with three pairs of ultrasonic dimension gauges, during right coronary artery (RCA) occlusions with the pericardium intact and open.

Methods

Animal preparation and instrumentation. Sixteen mongrel dogs weighing 13 to 24 kg (average 16.9) were anesthetized with ketamine hydrochloride (5 to 7 mg/kg im) followed by intravenous α-chloralose (50 mg/kg) and urethane (500 mg/kg). After endotracheal intubation, ventilation was maintained, by a volume respirator, with room air. A bilateral thoracotomy was performed in each dog in the fifth intercostal space with transection of the sternum. The electrocardiogram was continuously monitored in a limb lead (lead II). Arterial blood was repeatedly sampled for measurements of pH, Po2, and Pco2, with a blood gas analyzer (Acid-Base Laboratory, ABL2d), and supplemen-
terial oxygen and intravenous sodium bicarbonate were given when necessary to maintain values for these parameters within their physiologic ranges.

Instrumentation was performed with the pericardium virtually intact. A micromanometer-tipped catheter with a fluid-filled lumen (Millar Instruments, PC-470) was inserted into the left ventricle through the right common carotid artery to measure left ventricular pressure. A fluid-filled polyvinyl catheter (1 mm internal diameter) was placed in the right ventricle through the right external jugular vein. Both the fluid-filled lumens were connected to Statham P23ID pressure transducers. The electronic micromanometer in the left ventricle, connected to a direct-current amplifier (NEC San-Ei, model 1170) and calibrated and balanced at 37°C with a mercury manometer before each experiment, was rechecked in vivo with the use of the left ventricular systolic and end-diastolic pressure signals obtained simultaneously through the fluid-filled lumen. An occlusive snare (3–0 silk thread) was placed around the RCA near its origin, and a pair of pacing electrodes was sutured to the right atrial appendage. The RCA snare and pacing electrodes were placed through two small incisions (2 to 3 mm in length) in the pericardium. When incisions were made in the pericardium, care was taken to minimize their size.

Placement of ultrasonic crystals. The three types of ultrasonic crystals used in this study are illustrated in figure 1. These crystals were made of 5 MHz piezoelectric crystals (Murata Mfg. Co., 7R-30-20-5000) that were coated with polyester resin in our laboratory. As shown in figure 1, three pairs of ultrasonic crystals (2.5 mm in diameter) were placed through small incisions (3 mm in length) in the pericardium to measure the three internal dimensions of the left ventricle. The septal-lateral dimension was measured with a pair of crystals between the left ventricular endocardial surface of the septum and the subendocardium of the lateral free wall. These crystals were placed by the following method. A small stab incision was made into the lateral wall of the left ventricle, a needle carrying a crystal was thrust through the incision to the right ventricle, and the wires were pulled through the interventricular septum and the right ventricular free wall until the crystal lay flat against the septum. A second crystal was inserted through the same stab incision into the subendocardium of the lateral wall. The anterior-posterior dimension was measured with another pair of crystals implanted in the subendocardium through small stab incisions adjacent to the anterior and posterior descending coronary arteries. These two pairs of crystals were on the equatorial plane of the left ventricle.

A third pair of crystals was implanted to measure the longitudinal (apical-basal) internal dimension of the left ventricle. The apical crystal was placed in the subendocardium near the apical dimple, and the basal crystal was placed in the subendocardium just below the bifurcation of the left main coronary artery. In addition, to measure the right ventricular segment length, a fourth pair of crystals (2 mm in diameter) was placed in the mid portion of the right ventricular free wall in the circumferential direction. When the crystals were implanted, great care was taken not to injure any epicardial coronary arteries or veins.

The ultrasonic signals were continuously monitored with a four-channel ultrasonic dimension system (NEC San-Ei, model 4105). This instrument applied a pinging voltage to a piezoelectric crystal of 1024 times/sec/channel, measured the transit-time of ultrasound bursts across each pair of crystals, and generated a proportional analog voltage. Calibration was made electronically with gain (0.4 to 20 mm/0.5 V) and offset (0 to 100 mm) adjustments. The instantaneous distance between the crystals was calculated electronically according to the velocity of sound in blood (1.57 × 105 cm/sec). The linearity of this system in combination with 5 MHz crystals was ±0.5% over full scale (= 100 mm), and the noise level was below 0.05 mm.

The small incisions in the pericardium were not closed with sutures because suturing of the pericardium might have caused a leftward shift in the left ventricular diastolic pressure-volume relationship. Thee was no blood in the pericardial space at the end of the period of instrumentation.

Experimental protocol. After the instrumentation with the pericardium intact, the dogs were divided into two groups. In group I (eight dogs), we assessed the effects of right ventricular ischemia on left ventricular geometry before and after pericardiectomy and compared them with those observed during inferi-
or vena caval (IVC) occlusion. In group 2 (eight dogs), we assessed alterations in the left ventricular end-diastolic pressure-volume relationship during right ventricular ischemia before and after pericardiectomy. All experiments were carried out while the heart rate was held constant by right atrial pacing.

**Group 1.** In eight dogs, an electromagnetic flow probe was placed on the descending aorta because it was technically difficult to place it on the ascending aorta with the pericardium intact. The probe was connected to an electromagnetic flow-meter (Narco Bio-systems, RT-500). An occlusive snare (1-0 silk thread) was placed around the IVC to induce a transient reduction in left ventricular preload.

A first control recording was made, and then left ventricular preload was reduced for 30 sec by partial IVC occlusion with the IVC snare. The degree of IVC occlusion was adjusted to obtain a 20% to 30% reduction in aortic flow below the control level so that the end-diastolic volume would be similar to that during RCA occlusion. Hemodynamic and dimensional measurements were made, and then the snare was released.

After the hemodynamic and dimensional parameters had returned to the basal level, a second control recording was made. To produce right ventricular ischemia, the RCA was completely occluded with the RCA snare for 2 min. Measurements were made at 2 min of occlusion when hemodynamic and dimensional parameters became steady, and then the RCA snare was completely released.

When hemodynamic and dimensional parameters had completely returned to the control level, the pericardium was widely opened and a pericardial cradle was produced. When the hemodynamic values reached a new steady state, a third control recording was made. A second RCA occlusion was then induced, and the measurements were repeated.

**Group 2.** In the other eight dogs, a polyvinyl catheter (3 mm internal diameter) for blood transfusion was placed into the IVC through the right femoral vein. To prevent ventricular arrhythmias during transfusion, an intravenous infusion of lidocaine (0.05 mg/kg/min) was maintained throughout the experiments. A control volume loading with heparinized fresh blood withdrawn from donor dogs and kept at 37°C was carried out to obtain a control left ventricular end-diastolic pressure-volume relationship with the pericardium intact. Blood was rapidly transfused with a roller pump (Cole-Parmer Instrument, Masterflex PA-51B) at a constant speed of 400 ml/min through the IVC catheter until left ventricular end-diastolic pressure reached 16 to 18 mm Hg. Usually this level of left ventricular end-diastolic pressure was achieved within 35 to 50 sec of transfusion with the pericardium intact, and within 45 to 60 sec with the pericardium open.

During volume loading, pressure and dimensions were measured continuously. After left ventricular end-diastolic pressure reached 16 to 18 mm Hg, blood was withdrawn at a slower speed until left ventricular pressure returned to the control level. At least 15 min were allowed to elapse for restabilization of overall hemodynamics. A second control volume loading was performed to check the reproducibility of the hemodynamic responses to blood transfusion and to avoid errors due to an anaphylactic response to the first transfusion. Almost the same hemodynamic and dimensional responses were observed in all dogs and therefore we used the second control volume loading values for analysis.

After restabilization of hemodynamics, the RCA was occluded for 3 min, and during the last 1 min, rapid volume loading was performed as described above. After measurements were made, the RCA snare was released and blood was withdrawn. The pericardium was then opened widely to produce a pericardial cradle, and after hemodynamics had stabilized, a second series of control volume loading and volume loading during RCA occlusion was repeated in the same manner as in the first series.

After the completion of each experiment, the proper position of the ultrasonic crystals was verified by autopsy. In two dogs, mercury was injected into the RCA through a No. 5F Judkins’ catheter under x-ray fluoroscopic monitoring before they were killed, and in four dogs, barium sulfate suspension was injected into the RCA after they were killed. Postmortem x-ray films of the hearts were taken to assess the RCA distribution.

**Data analysis.** All data were recorded on both a multichannel thermal pen recorder (NEC San-Ei, Polygraph 360) and magnetic tape. In group 1, we measured mean aortic flow, peak systolic and end-diastolic pressures of the right and left ventricles, dP/dt, and end-systolic and end-diastolic values for the dimensional data. These were averaged over 6 to 8 beats under steady-state conditions. Data obtained during the earliest phase of IVC occlusion were not used because decreases in right ventricular pressure in this phase often permit rightward displacement of the interventricular septum. We visually identified end-systole at the time when the left ventricular dimensions reached their nadir near peak negative dP/dt, and end-diastole at the time immediately before left ventricular pressure started its rapid upstroke. Percent systolic shortening of each dimension was calculated as the end-diastolic dimension minus the end-systolic dimension, divided by the end-diastolic dimension, times 100. Mean left ventricular percent systolic shortening was obtained by averaging percent systolic shortening of the three dimensions. In group 2, we measured left ventricular peak systolic and end-diastolic pressures and dimensions during blood transfusions, as well as right ventricular pressures and dimensions. Pleural pressure was not measured, and respiratory variation was neglected because these experiments were performed in an open-chest preparation.

Left ventricular volume was calculated from the three internal dimensions by a nonprolate ellipsoid model with the formula

\[ V_{\text{calc}} = \frac{\pi}{6} \times D_{\text{SL}} \times D_{\text{AP}} \times D_{\text{L}} \times 10^{-3} \]

where \( V_{\text{calc}} = \) calculated left ventricular volume (ml); \( D_{\text{SL}} = \) left ventricular septal-lateral dimension (mm); \( D_{\text{AP}} = \) anterior-posterior dimension (mm); \( D_{\text{L}} = \) longitudinal dimension (mm). This calculation was validated by comparing calculated volume and directly measured volume obtained in five excised hearts after the experiments in the following manner. The heart was suspended in a beaker filled with blood. A known amount of blood (directly measured volume) was injected into a well-fitted left ventricular balloon that was placed through the aortic valve with the mitral valve sutured, and simultaneously the three left ventricular dimensions were measured, for determination of the calculated volume, with the ultrasonic crystals. Using a total of 71 data points, we obtained the following regression equation:

\[ V_{\text{calc}} = 0.93 \times V_{\text{meas}} + 2.5 \]

where \( V_{\text{calc}} = \) calculated volume (ml); \( V_{\text{meas}} = \) measured volume (ml). The correlation coefficient of the pooled data was .94. Although there was a slight underestimation in the calculated volume, the correlation between the values obtained by the two techniques was highly linear for each of five hearts (correlation coefficients = .994, .996, .996, .998, and .998). These data were comparable to those of Rankin et al. On the basis of these results, we used calculated volume as left ventricular volume for subsequent analysis.

Alterations of the left ventricular end-diastolic pressure-volume relationship during RCA occlusion were evaluated in two ways. First, left ventricular end-diastolic volume during RCA occlusion was compared with the preocclusion value at several matched left ventricular end-diastolic pressures. By this analy-
sis, a leftward shift in the pressure-volume relationship was tested. Second, an upward shift in the left ventricular end-diastolic pressure-volume relationship was evaluated by approximating the pressure-volume data during blood transfusion as a fourth-order polynomial according to the method of Glantz:

\[ P = a_4V^4 + a_3V^3 + a_2V^2 + a_1V + a_0 + bS \]  

where \( P \) = left ventricular end-diastolic pressure (mm Hg); \( V \) = left ventricular end-diastolic volume (ml); \( S = 0 \) for data obtained during control volume loading and 1 for data obtained during volume loading with RCA occlusion; \( a_0, a_1, a_2, a_3, a_4, b \) = constants. The \( bS \) term shifts the entire pressure-volume relationship upward or downward in the presence of RCA occlusion, and thus the \( b \) value represents the amount of the shift of the relationship.

Statistics. In group 1, statistical comparison of data obtained during first control period, IVc occlusion, second control period, RCA occlusion before pericardiectomy, third control period, and RCA occlusion after pericardiectomy were performed by the analysis of variance for repeated measurements. In group 2 the same method was also used for comparisons of data obtained during control and RCA occlusion before and after pericardiectomy. When the analysis of variance offered \( F \) values for \( p < .05 \) among the measurements, the paired \( t \) test was used to determine the statistical significance of differences between corresponding control and intervention values. Statistical comparisons were also made by the paired \( t \) test between parameters of left ventricular systolic function during IVc and RCA occlusions, and between changes in the observed values during RCA occlusion with the pericardium intact and open. Probability values smaller than .05 were considered indicative of statistical significance. Data are presented as mean \( \pm \) SD unless otherwise indicated.

Results

In all of the six dogs in which postmortem radiographic findings were obtained, the mercury injected into the RCA was distributed only to the right ventricular free wall, not to the left ventricle or the interventricular septum (figure 2).

Group 1. Representative tracings obtained in one dog during RCA occlusion are shown in figure 3, and hemodynamic and dimensional data are summarized in table 1 and figure 4.

During partial IVc occlusion, there were significant decreases in aortic flow (−24.5 ± 5.8%), left ventricular systolic pressure (−15.5 ± 4.0%), left ventricular end-diastolic pressure (−1.3 ± 0.7 mm Hg), right ventricular systolic pressure (−7.9 ± 1.8 mm Hg), and right ventricular end-diastolic pressure (−1.2 ±

FIGURE 2. Postmortem x-ray of an excised heart that shows the distribution of the right coronary artery filled with mercury. After the picture of the whole heart (top) was taken, the heart was dissected into three separate sections (bottom): the right ventricular free wall (RV), the interventricular septum (IVS), and the left ventricular free wall (LV). Mercury was distributed only to the right ventricular free wall, not to the interventricular septum or the left ventricle. The pairs of ultrasonic crystals implanted in the heart can be seen.

FIGURE 3. Representative recording from one dog showing hemodynamic and dimensional changes during RCA occlusion with the pericardium intact. Decreases in aortic flow, left ventricular (LV), and right ventricular (RV) systolic pressures and an increase in right ventricular end-diastolic pressure occurred during 2 min of RCA occlusion. Interestingly, among the left ventricular dimensions, the septal-lateral dimension showed a striking decrease compared with other two dimensions. The right ventricular segment length extended markedly and showed systolic bulging, indicating right ventricular ischemia. After the release of RCA occlusion, the hemodynamic and dimensional parameter values returned to the control level within a few minutes.
TABLE 1

Effects of interventions on hemodynamics and dimensions in group 1 dogs

<table>
<thead>
<tr>
<th></th>
<th>C1</th>
<th>IVCO</th>
<th>C2</th>
<th>RCAO&lt;sub&gt;R&lt;/sub&gt;</th>
<th>C3</th>
<th>RCAO&lt;sub&gt;R&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>AoF (ml/min)</td>
<td>526 ± 74</td>
<td>400 ± 51&lt;sup&gt;c&lt;/sup&gt;</td>
<td>510 ± 92</td>
<td>387 ± 84&lt;sup&gt;c&lt;/sup&gt;</td>
<td>494 ± 154</td>
<td>429 ± 134&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>LVSP (mm Hg)</td>
<td>103 ± 15</td>
<td>87 ± 14&lt;sup&gt;c&lt;/sup&gt;</td>
<td>101 ± 17</td>
<td>85 ± 22&lt;sup&gt;b&lt;/sup&gt;</td>
<td>91 ± 17</td>
<td>83 ± 18&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>5.2 ± 1.8</td>
<td>3.8 ± 2.0&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.7 ± 1.9</td>
<td>4.8 ± 1.7</td>
<td>3.7 ± 1.1</td>
<td>3.4 ± 1.1&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>RVSP (mm Hg)</td>
<td>33 ± 8</td>
<td>25 ± 8&lt;sup&gt;c&lt;/sup&gt;</td>
<td>34 ± 10</td>
<td>29 ± 5</td>
<td>29 ± 5</td>
<td>27 ± 4&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>RVEDP (mm Hg)</td>
<td>4.7 ± 1.2</td>
<td>3.6 ± 1.0&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.7 ± 1.2</td>
<td>6.1 ± 1.0&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.6 ± 1.2</td>
<td>5.5 ± 0.7&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>EDD&lt;sub&gt;sl&lt;/sub&gt; (mm)</td>
<td>23.1 ± 3.4</td>
<td>22.1 ± 3.2&lt;sup&gt;b&lt;/sup&gt;</td>
<td>22.7 ± 3.5</td>
<td>20.9 ± 4.0&lt;sup&gt;b&lt;/sup&gt;</td>
<td>23.4 ± 3.1</td>
<td>22.6 ± 3.3&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>EDD&lt;sub&gt;ap&lt;/sub&gt; (mm)</td>
<td>31.3 ± 2.8</td>
<td>29.9 ± 2.7&lt;sup&gt;c&lt;/sup&gt;</td>
<td>30.9 ± 3.2</td>
<td>30.8 ± 3.3</td>
<td>30.7 ± 3.1</td>
<td>30.7 ± 3.0</td>
</tr>
<tr>
<td>EDD (mm)</td>
<td>46.0 ± 3.4</td>
<td>45.6 ± 3.7&lt;sup&gt;b&lt;/sup&gt;</td>
<td>45.8 ± 3.5</td>
<td>45.7 ± 3.4</td>
<td>46.3 ± 3.4</td>
<td>46.4 ± 3.5</td>
</tr>
<tr>
<td>RVEDL (mm)</td>
<td>15.4 ± 2.4</td>
<td>15.1 ± 2.3&lt;sup&gt;b&lt;/sup&gt;</td>
<td>15.4 ± 2.7</td>
<td>16.8 ± 3.0&lt;sup&gt;b&lt;/sup&gt;</td>
<td>16.2 ± 2.5</td>
<td>17.1 ± 2.8&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>RV %SS (%)</td>
<td>12.6 ± 4.0</td>
<td>11.3 ± 3.1&lt;sup&gt;b&lt;/sup&gt;</td>
<td>11.8 ± 3.8</td>
<td>-2.6 ± 5.8&lt;sup&gt;c&lt;/sup&gt;</td>
<td>15.1 ± 3.8</td>
<td>0.1 ± 5.9&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

AoF = aortic flow; LVSP = left ventricular systolic pressure; LVEDP = left ventricular end-diastolic pressure; RVSP = right ventricular systolic pressure; RVEDP = right ventricular end-diastolic pressure; EDD<sub>sl</sub> = end-diastolic septal-lateral dimension; EDD<sub>ap</sub> = end-diastolic anterior-posterior dimension; EDD = end-diastolic longitudinal dimension; RVEDL = right ventricular end-diastolic segment length; RV %SS = right ventricular percent systolic shortening; C1 = the first control; IVCO = inferior vena cava occlusion; C2 = the second control; RCAO<sub>R</sub> = right coronary artery occlusion with the pericardium intact; C3 = the third control; RCAO<sub>R</sub> = right coronary artery occlusion with the pericardium open.

<sup>a</sup>p < .05; <sup>b</sup>p < .01; <sup>c</sup>p < .001 compared with each control value by paired t test.

0.5 mm Hg), compared with the respective control values. There were concomitant decreases in all three end-diastolic dimensions of the left ventricle, namely, septal-lateral (−4.3 ± 2.1%), anterior-posterior (−4.6 ± 1.4%), and longitudinal (−1.1 ± 1.3%) dimensions. As a result, the ratio of end-diastolic septal-lateral to anterior-posterior dimensions (D<sub>sl</sub>/D<sub>ap</sub>), which is considered to be an index of eccentricity of the left ventricular equatorial plane, did not change (0.74 ± 0.07 to 0.74 ± 0.07). Right ventricular end-diastolic segment length (15.4 ± 2.4 to 15.1 ± 2.3 mm) and its percent systolic shortening (12.6 ± 4.0% to 11.3 ± 3.1%) also decreased slightly during IVC occlusion.

During RCA occlusion with the pericardium intact, the right ventricular end-diastolic segment length increased (15.4 ± 2.7 to 16.8 ± 3.0 mm), its percent systolic shortening decreased markedly (11.8 ± 3.8% to −2.6 ± 5.8%), and right ventricular end-diastolic pressure rose (+1.4 ± 0.6 mm Hg), indicating right ventricular ischemia. Concomitantly, aortic flow (−24.0 ± 9.4%) and left ventricular systolic pressure (−17.2 ± 12.1%) decreased by similar degrees to those seen during IVC occlusion, but left ventricular end-diastolic pressure did not change. In contrast to IVC occlusion, among left ventricular end-diastolic dimensions, only the septal-lateral dimension showed a significant decrease (−8.2 ± 5.0%), while anterior-posterior and longitudinal dimensions remained unchanged (figure 4). As a result, D<sub>sl</sub>/D<sub>ap</sub> decreased significantly (0.73 ± 0.06 to 0.68 ± 0.08, p < .01).

After the pericardium was opened, RCA occlusion also increased right ventricular end-diastolic segment length (16.2 ± 2.5 to 17.1 ± 2.8 mm), markedly decreased the percent systolic shortening (15.1 ± 3.8 to 0.1 ± 5.9%), and elevated right ventricular end-diastolic pressure (+0.9 ± 0.5 mm Hg). Aortic flow (−12.9 ± 4.9%) and left ventricular systolic pressure (−9.6 ± 10.6%) decreased concomitantly, but these

![FIGURE 4](http://circ.ahajournals.org/)

Changes in left ventricular end-diastolic dimensions (LVEDD) during IVC or RCA occlusion with the pericardium intact and open (group 1). Data are expressed as the percentage of change from each control value. During IVC occlusion, septal-lateral (D<sub>sl</sub>), anterior-posterior (D<sub>ap</sub>), and longitudinal (D<sub>l</sub>) dimensions all decreased significantly. However, during RCA occlusion, only the D<sub>sl</sub> decreased significantly. The decrease in the D<sub>sl</sub> was significantly less with the pericardium open than with it intact in spite of the fact that the coronary occlusions were of the same duration (2 min). Means ± SE are indicated.
TABLE 2
Left ventricular end-diastolic volume and systolic function during IVC or RCA occlusion

<table>
<thead>
<tr>
<th>Parameter</th>
<th>IVCO</th>
<th>RCAO&lt;sub&gt;n&lt;/sub&gt;</th>
<th>p value (paired t test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDV (ml)</td>
<td>15.7 ± 2.6</td>
<td>15.4 ± 3.6</td>
<td>NS</td>
</tr>
<tr>
<td>AoF (ml/min)</td>
<td>400 ± 51</td>
<td>387 ± 84</td>
<td>NS</td>
</tr>
<tr>
<td>LVSP (mm Hg)</td>
<td>87 ± 14</td>
<td>85 ± 22</td>
<td>NS</td>
</tr>
<tr>
<td>Peak dP/dt (mm Hg/sec)</td>
<td>1980 ± 530</td>
<td>1904 ± 761</td>
<td>NS</td>
</tr>
<tr>
<td>LV %SS (%)</td>
<td>12.3 ± 2.6</td>
<td>11.4 ± 3.3</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean ± SD.
LVEDV = left ventricular end-diastolic volume; AoF = aortic flow; LVSP = left ventricular systolic pressure; mean LV %SS = mean left ventricular percent systolic shortening; IVC = IVC occlusion; RCAO<sub>n</sub> = RCA occlusion with the pericardium intact.

reductions were significantly less (p < .01) than those observed with the pericardium intact, despite the same duration (2 min) of occlusion. There were slight decreases in end-diastolic septal-lateral dimension (−3.6 ± 2.5%) and ΔD/L (0.76 ± 0.08 to 0.74 ± 0.09, p < .05) without changes in anterior-posterior and longitudinal dimensions. The decrease in end-diastolic septal-lateral dimension was significantly less (p < .01) with the pericardium open than with it intact, although the procedure was the same (figure 4).

To evaluate the effects of RCA occlusion on left ventricular myocardial performance, left ventricular end-diastolic volume and parameters of left ventricular systolic function during RCA occlusion with the pericardium intact were compared with those observed during IVC occlusion (table 2). Left ventricular end-diastolic volume was not different, and all parameters, i.e., aortic flow, left ventricular systolic pressure, peak dP/dt, and mean left ventricular percent systolic shortening, were similar in the two conditions.

Group 2. Hemodynamic and right ventricular segment length data determined during blood transfusion under control conditions and during RCA occlusion in group 2 dogs are summarized in table 3. When the data obtained during RCA occlusion were compared with the respective control values at matched left ventricular end-diastolic pressures (8 and 16 mm Hg), the changes were similar to those observed in group 1 dogs. Data observed at other end-diastolic pressures (6, 10, 12, and 14 mm Hg) were similar.

The end-diastolic pressure-volume relationship before and during RCA occlusions with the pericardium intact and open are presented in figure 5. Among the three end-diastolic pressure-volume relationships of the left ventricle, the most remarkable shift to the left was in the septal-lateral dimension whether the pericardium was intact or open. Furthermore, the leftward shift was greater with the pericardium intact or at high end-diastolic pressure than with the pericardium open or at low end-diastolic pressure. End-diastolic pressure-volume relationships of anterior-posterior and longitudinal dimensions showed only minimal or no changes.

Left ventricular end-diastolic pressure-volume rela-

TABLE 3
Effects of RCA occlusion on hemodynamics and right ventricular segment length in group 2 dogs

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pericardium intact</th>
<th>Pericardium open</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>RCAO</td>
</tr>
<tr>
<td>LVSP (mm Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP 8 mm Hg</td>
<td>116.4 ± 19.1</td>
<td>109.0 ± 20.2&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>LVEDP 16 mm Hg</td>
<td>139.8 ± 22.5</td>
<td>134.9 ± 21.6&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>RVSP (mm Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP 8 mm Hg</td>
<td>33.8 ± 6.3</td>
<td>31.9 ± 5.9&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>LVEDP 16 mm Hg</td>
<td>41.0 ± 6.7</td>
<td>37.1 ± 5.8&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>RVEDP (mm Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP 8 mm Hg</td>
<td>7.8 ± 0.7</td>
<td>8.6 ± 1.2</td>
</tr>
<tr>
<td>LVEDP 16 mm Hg</td>
<td>13.0 ± 1.6</td>
<td>15.9 ± 2.1&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>RVEDL (mm)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP 8 mm Hg</td>
<td>18.1 ± 4.1</td>
<td>19.0 ± 4.2&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>LVEDP 16 mm Hg</td>
<td>19.6 ± 4.4</td>
<td>20.9 ± 4.2&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>RV %SS (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP 8 mm Hg</td>
<td>13.7 ± 4.8</td>
<td>0.8 ± 4.7&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>LVEDP 16 mm Hg</td>
<td>17.4 ± 4.5</td>
<td>3.6 ± 5.1&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are means ± SD.
RCAO = RCA occlusion; other abbreviations as in table 1.
<sup>a</sup>p < .05; <sup>b</sup>p < .01; <sup>c</sup>p < .001 compared with each control value by paired t test.
tionships before and during RCA occlusion with the pericardium intact and open are shown in figure 6. At matched end-distolic pressures over 8 mm Hg, RCA occlusion with the pericardium intact significantly reduced left ventricular end-diastolic volume. Also, after the pericardium was opened, RCA occlusion reduced end-diastolic volume at matched end-diastolic pressure over 10 mm Hg. The reductions in end-diastolic volume were significantly greater with the pericardium intact than with it open except at end-diastolic pressures of 6 and 10 mm Hg (table 4).

Left ventricular end-diastolic pressure-volume data during control volume loading closely approximated the fourth-order polynomial (equation 3) in all animals, and the correlation coefficient averaged .998 ± .001 with the pericardium intact and .998 ± .002 with it open (table 5). Except in one dog with the pericardium intact (p = .060) and in one other dog with the pericardium open (p = .064), the b values during RCA occlusion were significant in all animals whether the pericardium was intact or open. However, compared with the values with the pericardium intact, the b values with the pericardium open were significantly smaller (p < .001), both by paired and pooled data analysis.

Discussion

The major new findings of the present study are (1) right ventricular ischemia shifts the interventricular septum to the left in end-diastole without changing left ventricular myocardial performance, (2) right ventricular ischemia causes a leftward and upward shift of the left ventricular end-diastolic pressure-volume relationship, and (3) the intact pericardium augments these effects.

The hemodynamic and dimensional changes during IVC occlusion in group 1 dogs indicate that the reduction of stroke volume was primarily due to the reduction of left ventricular end-diastolic volume. These findings are consistent with previous findings that
IVC occlusion simply reduces left ventricular preload, with no change in geometry except in the earliest phase when the interventricular septum often shifts rightward. In contrast, the hemodynamic and dimensional changes during RCA occlusion indicate that ischemic right ventricular dysfunction causes a disproportionate decrease in septal-lateral dimension, and hence distorted the left ventricular end-diastolic geometry, despite a reduction similar to that during IVC occlusion in end-diastolic volume, aortic flow, and left ventricular systolic pressure. These geometric changes, as well as the reductions of aortic flow and left ventricular systolic pressure observed during RCA occlusion, were significantly attenuated after the pericardium was opened. Because the degree of right ventricular ischemia probably remained unchanged, these attenuations can be attributed to opening of the intact pericardium.

In group 2 dogs, the left ventricular end-diastolic pressure-volume relationship during RCA occlusion shifted leftward and upward significantly (figure 6; tables 4 and 5). These changes can be attributed to the leftward shift of the end-diastolic pressure-diameter relationship of the septal-lateral dimension during RCA occlusion (figure 5). These findings indicate that an acute dilatation and end-diastolic pressure elevation of the right ventricle causes encroachment on the left ventricle by the interventricular septum, resulting in a leftward and upward shift of the pressure-volume relationship. The intact pericardium augments these effects. We can assume that, in this situation, the right and left ventricles not only are aligned in series, but also interact directly through the interventricular septum.

On the basis of these results, we can at least partly attribute the absent or minimal circulatory derangement in previous studies in which the right ventricular free wall was damaged by cauterization or RCA.

**TABLE 4**

Reduction in left ventricular end-diastolic volume (ml) during RCA occlusion in group 2 dogs

<table>
<thead>
<tr>
<th></th>
<th>Pericardium intact</th>
<th>Pericardium open</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDP 6 mm Hg</td>
<td>0.6 ± 1.0</td>
<td>0.0 ± 1.1</td>
</tr>
<tr>
<td>LVEDP 8 mm Hg</td>
<td>1.9 ± 1.7</td>
<td>0.5 ± 1.0^A</td>
</tr>
<tr>
<td>LVEDP 10 mm Hg</td>
<td>2.4 ± 1.6</td>
<td>1.6 ± 1.2</td>
</tr>
<tr>
<td>LVEDP 12 mm Hg</td>
<td>2.9 ± 1.6</td>
<td>1.5 ± 0.9^A</td>
</tr>
<tr>
<td>LVEDP 14 mm Hg</td>
<td>3.3 ± 1.4</td>
<td>1.7 ± 0.8^A</td>
</tr>
<tr>
<td>LVEDP 16 mm Hg</td>
<td>4.1 ± 1.7</td>
<td>1.7 ± 0.6^A</td>
</tr>
</tbody>
</table>

Values are mean ± SD.
LVEDP = left ventricular end-diastolic pressure.
*p < .05; *p < .01 compared with the pericardium intact value by paired t test.
TABLE 5
Effects of RCA occlusion on the left ventricular end-diastolic pressure-volume relationship in group 2 dogs

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Pericardial condition</th>
<th>Control n</th>
<th>r</th>
<th>RCAO n</th>
<th>b ± SD</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Intact</td>
<td>7</td>
<td>.999</td>
<td>7</td>
<td>2.55 ± 1.64</td>
<td>&lt;.01</td>
</tr>
<tr>
<td></td>
<td>Open</td>
<td>7</td>
<td>.995</td>
<td>8</td>
<td>0.88 ± 0.85</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>2</td>
<td>Intact</td>
<td>7</td>
<td>.995</td>
<td>6</td>
<td>3.45 ± 1.57</td>
<td>&lt;.01</td>
</tr>
<tr>
<td></td>
<td>Open</td>
<td>7</td>
<td>.998</td>
<td>7</td>
<td>1.38 ± 1.15</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>3</td>
<td>Intact</td>
<td>9</td>
<td>.999</td>
<td>9</td>
<td>1.63 ± 1.73</td>
<td>&lt;.05</td>
</tr>
<tr>
<td></td>
<td>Open</td>
<td>9</td>
<td>.998</td>
<td>8</td>
<td>0.88 ± 0.62</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>4</td>
<td>Intact</td>
<td>9</td>
<td>.999</td>
<td>8</td>
<td>2.66 ± 1.81</td>
<td>&lt;.01</td>
</tr>
<tr>
<td></td>
<td>Open</td>
<td>10</td>
<td>.999</td>
<td>8</td>
<td>1.10 ± 1.07</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>5</td>
<td>Intact</td>
<td>6</td>
<td>.999</td>
<td>6</td>
<td>0.60 ± 0.60</td>
<td>.060</td>
</tr>
<tr>
<td></td>
<td>Open</td>
<td>6</td>
<td>.996</td>
<td>6</td>
<td>0.51 ± 0.43</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>6</td>
<td>Intact</td>
<td>9</td>
<td>.997</td>
<td>9</td>
<td>1.88 ± 2.11</td>
<td>&lt;.05</td>
</tr>
<tr>
<td></td>
<td>Open</td>
<td>8</td>
<td>.998</td>
<td>10</td>
<td>0.78 ± 1.00</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>7</td>
<td>Intact</td>
<td>11</td>
<td>.999</td>
<td>11</td>
<td>2.21 ± 2.23</td>
<td>&lt;.01</td>
</tr>
<tr>
<td></td>
<td>Open</td>
<td>10</td>
<td>.997</td>
<td>10</td>
<td>1.00 ± 1.06</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>8</td>
<td>Intact</td>
<td>8</td>
<td>.998</td>
<td>8</td>
<td>2.13 ± 2.28</td>
<td>&lt;.05</td>
</tr>
<tr>
<td></td>
<td>Open</td>
<td>8</td>
<td>.999</td>
<td>8</td>
<td>1.00 ± 1.29</td>
<td>.064</td>
</tr>
</tbody>
</table>

Paired data

| Pericardium intact (n = 8) | 2.14 ± 0.83 |
| Pericardium open (n = 8)   | 0.94 ± 0.25  |

Pooled data

| Pericardium intact (n = 64) | 2.13 ± 1.91 |
| Pericardium open (n = 65)   | 0.94 ± 0.96  |

Left ventricular end-diastolic pressure-volume data were approximated as fourth-order polynomials (equation 3). See text for details.

RCAO = RCA occlusion; r = correlation coefficient of fitting to equation 3; b = coefficient in equation 3 that indicates the amount of the upward shift of the end-diastolic pressure-volume relationship during RCAO; p value = significance of b during RCAO compared with each control by unpaired t test.

^Significance of difference in b for intact and open pericardium by paired t test.

^Significance of difference in b for intact and open pericardium based on analysis of pooled data (unpaired t test).

ligation\textsuperscript{19} to the use of a preparation with an open pericardium. Recently, Goldstein et al.\textsuperscript{9} produced right ventricular infarction by RCA occlusion with the pericardium intact and observed an elevation of intrapericardial pressure and significant reductions of mean aortic pressure and cardiac output. However, they did not evaluate left ventricular geometry or the diastolic pressure-volume relationship. Our present study is therefore the first one showing that right ventricular ischemia is a pathologic condition that causes a leftward shift of the interventricular septum and a leftward and upward shift of the left ventricular diastolic pressure-volume relationship.

The effects of right ventricular ischemia on left ventricular geometry and hemodynamics have some resemblance to those of pulmonary artery constriction during which the interventricular septum shifts leftward, the diastolic pressure-volume curve also shifts leftward, and left ventricular stroke volume decreases.\textsuperscript{5,14} However, when our data are compared with those from studies of acute pulmonary artery constriction,\textsuperscript{5,14} the increase in right ventricular end-diastolic pressure during our RCA occlusion seems smaller (1.4 vs 3.0 and 3.7 mm Hg), despite a similar decrease in left ventricular stroke volume. This might be attributable to the difference in the degree of right ventricular dilatation between the two conditions. Using two-dimensional echocardiography, Goldstein et al.\textsuperscript{9} reported a 110% increase in right ventricular end-diastolic size after RCA occlusion in the dog, while Stool et al.\textsuperscript{4} reported only a 46% to 52% increase in right ventricular end-diastolic volume during acute pulmonary artery hypertension that reduced stroke volume by 19% to 39%. In addition, although a significant increase in intrapericardial pressure has been reported during RCA occlusion,\textsuperscript{6} Kenner and Wood\textsuperscript{20} found no increase in this pressure during acute pulmonary artery obstruction. Thus, during right ventricular ischemia, the acute and marked right ventricular dilatation within the relatively stiff pericardium might affect left ventricular geometry and hemodynamics by increasing intrapericardial pressure even when the increase in right ventricular end-diastolic pressure is relatively small.

One potential limitation in our study is related to the accuracy of the left ventricular volume measurement. Although we validated our crystal method in excised hearts, we could not validate the method in a beating heart. Also, there seems to have been a slight underestimation in our calculated volume, probably because the position of the basal crystal had to be slightly lower than the level of the aortic valve. However, the correlation between calculated and directly measured volume was highly linear in each animal, and changes in left ventricular geometry would not have affected the measurement.\textsuperscript{14} Therefore, the present method provides a reliable index of left ventricular volume.

Another potential problem is that the brief periods of right ventricular ischemia have resulted in persistent diastolic abnormalities in the right ventricle. However, right ventricular systolic pressure, end-diastolic pressure, and percent systolic shortening during the third control period were not significantly different (p > 0.1) from those during the second control period (table 1). Therefore, the prolonged effects of brief ischemia, if any, were negligible.

The leftward and upward shift of the left ventricular end-diastolic pressure-volume relationship could be caused by changes in viscous properties or by acute
ischemia of the left ventricle. In group 2 dogs use of the rapid blood transfusion itself might raise the possibility that the changes in viscous properties of the left ventricle altered the end-diastolic pressure-volume relationship. However, we infused blood at the same rate during both control and RCA occlusion periods. The choice to read end-diastolic values also tends to minimize viscous effects since ventricular filling is relatively slow near end-diastole. Therefore, the observed shift in the relationship during RCA occlusion could not be attributed to changes in viscous properties. Also, we found that the RCA territory was confined to the right ventricular free wall, as Blair reported, and that the indexes of left ventricular systolic function during RCA occlusion were not different from those observed at a comparable level of left ventricular end-diastolic volume during IVC occlusion (table 2), so that left ventricular ischemia would not have occurred during our RCA occlusion.

The results of the present study indicate that the primary mechanism by which aortic flow is decreased during right ventricular ischemia is a reduction of left ventricular end-diastolic volume, because parameters of left ventricular systolic function remain relatively constant when compared with those observed during IVC occlusion (table 2). Olsen et al. reported that the parameters of left ventricular systolic function measured during pulmonary artery and IVC occlusions were similar at a matched left ventricular end-diastolic volume, despite significant interventricular septal shift and alteration of left ventricular shape. Our results suggest that their conclusion that end-diastolic volume or mean myocardial fiber length is the major preload determinant of left ventricular systolic function independent of chamber pressure and shape also holds true under conditions of right ventricular ischemia.

In addition, our results suggest that the reduction of left ventricular end-diastolic volume during right ventricular ischemia can itself be attributed to two mechanisms. One is right ventricular systolic dysfunction, which reduces right ventricular output. This was suggested in our study by the decrease in right ventricular systolic pressure during RCA occlusion. The other mechanism is the leftward and upward shift of the left ventricular end-diastolic pressure-volume relationship. When right ventricular systolic function is constant, i.e., when left ventricular filling pressure is constant, the leftward and upward shift of the pressure-volume relationship itself results in a decrease in left ventricular end-diastolic volume for a given end-diastolic pressure. The presence of the latter mechanism was suggested by our finding that the greater shift in the pressure-volume relationship during RCA occlusion was accompanied by a greater decrease in aortic flow with the pericardium intact than with the pericardium open, because the degree of ischemic right ventricular dysfunction was similar and left ventricular myocardial performance would be the same under the two conditions. Thus, series ventricular interaction, together with substantial direct interaction, seems responsible for the reduction in left ventricular end-diastolic volume during right ventricular ischemia.

The findings shown in the present study cannot be applied directly to the clinical setting, because clinical right ventricular infarction is usually accompanied by left ventricular inferior or posterior infarction. However, there is one case report of a patient with right ventricular infarction who had interventricular septum convexity toward the left ventricle during diastole on the two-dimensional echocardiogram. Therefore, the present results merit further studies of ventricular interaction in clinical right ventricular infarction.

In summary, right ventricular ischemia causes a leftward shift of the interventricular septum in end-diastole, distorts left ventricular geometry, and also causes a leftward and upward displacement of the left ventricular end-diastolic pressure-volume relationship, without changing left ventricular myocardial performance. The presence of the intact pericardium augments these changes. The reduction of aortic flow during right ventricular ischemia seems to be primarily due to the reduction of left ventricular end-diastolic volume, which may be attributable to right ventricular systolic dysfunction and the leftward and upward shift in the left ventricular end-diastolic pressure-volume relationship.

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