Ventricular Diastolic Pressure-Volume Relations and the Pericardium

Effects of Changes in Blood Volume and Pericardial Effusion in Dogs

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SUMMARY We investigated the role of the pericardium in the mechanism of shifts in the left ventricular (LV) diastolic pressure-volume relation produced by changes in circulating blood volume and by pericardial effusion. Twelve closed-chest anesthetized dogs were instrumented with pericardial and pleural balloons and intracardiac catheters for pressure measurements. We measured the volumes of the pericardium and the left and right ventricles by computed tomography (CT), integrating the area of CT cross-sections measured at 1-cm intervals from the cardiac apex to the aortic arch. The volumes of the pericardium and the cardiac chambers were changed by infusing 40 and 80 ml of dilute contrast medium into the pericardial space, by bleeding, and by rapidly infusing saline intravenously. Pericardial effusions of 80 ml reduced mean right ventricular volumes to 59% of control, whereas LV volumes were less severely compromised (81% of control). Total pericardial volume and pressure increased. Intravenous saline infusions, which raised right atrial pressure 10-15 mm Hg, produced increases of this magnitude in pericardial pressure. This was also the magnitude of the upward displacement in the LV diastolic pressure-volume relation after infusion. However, LV diastolic transmural pressure-volume coordinates fell along a single curve. Similar behavior was observed for the right ventricular diastolic pressure-volume relation. Pericardial transmural pressure-volume curves were described. When cardiac volume was altered by volume load and pericardial effusion, acute shifts in the LV diastolic pressure-volume relation were caused by changes in pericardial pressure, which, in turn, corresponded to changes in pericardial volume.

THE DIASTOLIC left ventricular (LV) pressure-volume relation can shift upward or downward acutely and to an important degree.1-3 These shifts are significant because the transmural diastolic pressure-volume relation of either ventricle determines the distending force or preload in that it relates diastolic transmural pressure to muscle fiber length. The strength of contraction is, in turn, dependent on this length according to the Frank-Starling mechanism. In this sense, the diastolic pressure-volume relation might be considered to be a determinant of ventricular performance. Some investigators have assumed that directly measured diastolic pressures are equal to transmural diastolic pressure, perhaps because many classic physiologic experiments have been performed after removal of the pericardium. LV function is commonly evaluated in terms of LV end-diastolic pressure or filling pressure. Hence, understanding acute shifts in the LV diastolic pressure-volume relation is of major clinical importance.

Many explanations for the shifts have been proposed. Some acute upward shifts, i.e., increases in LV diastolic pressure at a constant LV volume, have been associated with increased cardiac loading and decreased performance. Thus, these upward shifts have been observed under conditions in which the heart might dilate and stretch the pericardium. Conversely, downward shifts have been observed after administration of vasodilators when cardiac decompression might occur. Tyberg et al.8 proposed that these shifts were actually due to changes in pericardial pressure, in turn due to changes in heart size and pericardial volume; i.e., the change in absolute LV diastolic pressure may not represent a change in transmural pressure, but only the necessary effect of a change in the pressure external to the heart, i.e., the pericardial pressure. Since the presentation of this hypothesis, changes in pericardial pressure corresponding to the demonstrated shifts in the LV diastolic pressure-volume relation have been reported.9 The proposed central role of cardiac dilatation or decompression cannot, however, be established until simultaneous measurements of pericardial and cardiac volumes have been made.

We measured volume changes of the pericardium

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and the individual cardiac chambers by computed tomography (CT) while simultaneously measuring the pericardial pressure and intracardiac pressures. This study in anesthetized closed-chest dogs was designed to test the hypothesis that some acute changes in LV diastolic pressure are really caused by unobserved changes in pericardial pressure, which, in turn, are due to changes in pericardial volume. Saline infusion, hemorrhage and pericardial effusion were used to manipulate pressure-volume relations.

Methods
Preparation of Experimental Animals

We prepared the dogs partially 2 days before the experiment. Ten mongrel dogs that weighed 16-30 kg, of either sex, were anesthetized with sodium pentobarbital (Nembutal, Abbott Laboratories), 25 mg/kg i.v., intubated and ventilated with a positive-pressure respirator (model 607, Harvard Apparatus Company). A thoracotomy through the fourth left intercostal space was performed. A 2 × 3-cm silastic balloon and an open catheter were placed in the pericardial cavity. Care was taken to limit the pericardial incision to 1-2 cm. The pericardial incision was then sutured securely. A similar silastic balloon was placed in the pleural space next to the pericardium and near the intrapericardial balloon. Both balloons were fixed at the level of the mid-left ventricle when the dogs were supine. Radiolucent catheters were also placed in the right atrium and the right ventricle through the left external jugular vein. The chest was then closed and the dogs were allowed to recover. Air was removed from the chest by suction. The catheters were flushed with 2 ml of 250 USP units/ml heparin in saline twice daily. The dogs received intramuscular injections of procaine penicillin, 400,000 units, and dihydrostreptomycin sulfate, 500 mg (Combition, Pfizer Laboratories Division), once daily.

On the day of the experiment the dogs were again anesthetized with sodium pentobarbital, 5-10 mg/kg i.v. (with additional doses of 1 mg/kg every 30-60 minutes to maintain anesthesia), and ventilated with a Harvard respirator. To control the heart rate, the dogs were first premedicated with xylazine (Rompun, Bayer Division, Cutter Laboratories, Inc.), 1-2 mg/kg i.m. Radiolucent catheters were placed in the left ventricle through the left femoral artery and the aorta through the right femoral artery. Two milliliters of saline were added to each balloon. (The maximal unstressed volume of these balloons was 8 ml.) All pressure catheters and balloons were connected to pressure transducers (model P23Db, Statham Instruments) positioned at the same level.

At the beginning of each experiment a so-called scout view (a lateral picture of the dog's chest) was constructed. For this scan, a metal needle was placed on the outside of the dog's chest at the level at which the pressure transducers had been zeroed. This allowed pressures to be corrected accurately to the level of the mid-left ventricle. Pressures were calibrated to 200 or 40 mm Hg full-scale on a recorder (Model 350, Sanborn Co.). LV pressure was also recorded at 40 mm Hg full-scale for the accurate measurement of diastolic pressure. All pressure recordings were made at a paper speed of 100 mm/sec. LV diastole was defined as the interval between the time of the minimum diastolic pressure and the end of the a-wave. The average during this interval was determined by a line above which and below which were equal areas of the pressure-time curve. For the same interval of LV diastole, average diastolic pressures for the other chambers were determined similarly.

To rapidly increase the circulating blood volume and to maintain an elevated systemic venous pressure, we placed a large catheter connected to a Lampson bottle into the right external jugular vein. In four dogs, a Swan-Ganz triple-lumen balloon catheter (Edwards Laboratories) was introduced through the right femoral vein for cardiac output measurements and connected to a cardiac output computer (model 9520, Edwards Laboratories). In addition, a catheter for infusing contrast medium (Renografin-76, E. R. Squibb & Sons, Inc.) was placed in the left femoral vein and a catheter in the right brachial vein to administer anesthesia and succinylcholine chloride (Anectine, Burroughs Wellcome Co.), 600 mg/hr. The ECG was monitored on an oscilloscope.

Cardiac Computed Tomography

The dogs were placed head-first into the scanner and were fixed in a supine position using a plexiglass cradle. A General Electric CT/T 7800 whole-body scanner equipped with minor software and hardware modifications was used to determine pericardial and cardiac volumes. The instrument was capable of completing a 360° scan exposure in 2.4 seconds. Approximately 1 second was required between scans. Twelve cross-sectional scans at 1-cm intervals were taken from just below the cardiac apex to the aortic arch. The cardiac chambers and myocardium could be identified well only with contrast medium enhancement. Contrast medium (Renografin-76, E.R. Squibb & Sons, Inc.) was infused into the left femoral vein with an infusion pump (model 600910/920 VDC, Harvard Apparatus Co.) at an infusion rate of 7 ml/min for 10 minutes. Steady-state levels could then be maintained over the course of the study at an infusion rate of 2 ml/min. Pericardial and cardiac volumes were calculated by tracing the outline of the structure from each cross-sectional scan (fig. 1) and measuring the area with a planimeter. This area was multiplied by the appropriate correction factor determined for each experiment to express the area in absolute square-centimeter units. Because scans were taken at 1-cm increments, each cross-sectional area was multiplied by 1 cm to give the volume within the slice. These incremental volumes were measured throughout all the scans to give the total volume.

Experimental Protocol

At the beginning of the experiment, all pericardial fluid was removed (usually about 20 ml of clear liquid) through the pericardial catheter. A series of 12 control
scans from the cardiac apex to the aortic arch was taken. The respirator was turned off at end-inspiration during the scanning procedure. The anesthetized, paralyzed dogs were slightly hyperventilated to better sustain the 20-second pause required to record six of the 12 cross-sectional scans. Three respiratory cycles were interposed before the remaining six scans were recorded. Pressures were recorded at rapid paper speed immediately before and after each series of scans.

Pericardial effusion, hemorrhage and saline infusion were then used to manipulate pressure-volume relationships. Forty milliliters of a warmed 5% solution of meglumine diatrizoate (Renografin-76) in saline were infused into the pericardium (episode 2 on figures 2–5). Cross-sectional scans and pressure measurements were repeated as above. Another 40 ml (total of 80 ml) were added before the next set of measurements (episode 3). Then, the pericardial fluid was removed. In six dogs, 200–300 ml of arterial blood was collected in heparinized syringes until ventricular diastolic pressure approximated 0 mm Hg (episode 4). After scans and pressure measurements, the blood was reinfused and the dogs were volume loaded. The fluid level in the Lampson bottle was maintained at 15–20 cm above the dog. When aortic and intracardiac pressures had stabilized after the volume load, scans and pressure measurements were repeated (episode 5). As before, scans and pressure measurements were repeated after 40 ml (episode 6) and 80 ml (episode 7) had been added to the pericardium. After completing the last set of measurements, the dogs were killed by injecting a saturated solution of potassium chloride. A postmortem pressure-volume curve of the pericardium was obtained by adding fluid to the pericardium until desired pressures were achieved. (The dead heart tended to collapse, but the total pericardial volume and transmural pressure were measureable.)

Statistics

A two-way analysis of variance was done using the Student-Neuman-Keuls test.

Results

By using CT with contrast medium enhancement, we could measure pericardial and cardiac volumes. Figure 2 shows the volume of the total pericardium and its contents throughout one of our experiments. We measured the volume of the total pericardium, the pericardial effusion, and the contents of the left and right ventricles. The volume of the myocardium was obtained by weight, assuming a density of 1.05 g/ml. The volume of the left and right atrium was obtained
by difference. Pericardial effusion decreased the LV volume, but decreased the right ventricular (RV) volume to an even greater extent (table 1). When we removed the effusion and bled the dogs, the LV volume decreased further in all experiments, whereas the RV volume increased in four experiments and decreased in two experiments. When we volume-loaded the dogs and produced pericardial effusion, the total pericardial volume increased only slightly (fig. 2). Instead, the LV and RV volumes decreased.

To evaluate our volume measurements, we plotted the difference between the total pericardium and the volume of the heart against the amount of fluid we added (fig. 6). There has been no method to measure pericardial volumes. To corroborate our method of outlining the ventricular lumen, we reanalyzed some of our experiments using a more objective, computerized technique. In this procedure we measured the average radiodensity of the myocardium and of the ventricular lumen by defining appropriate regions of interest. The program then automatically recorded the area of the lumen by defining the boundary as the locus of volume elements having a radiodensity halfway between those of the myocardium and the blood. Although the measurements sometimes differed absolutely, we always found an excellent correlation (r = 0.95–0.99).

The amount of fluid infused into the pericardial cavity (40–80 ml) was not sufficient to produce the classic signs of cardiac tamponade. Table 2 shows the hemodynamic effects of pericardial effusion and changes in blood volume. Infusion of 40 and 80 ml fluid into the pericardial cavity induced a nonsignificant increase in mean diastolic pericardial pressure, but decreased the LV volume significantly. Mean aortic blood pressure and cardiac output decreased. Bleeding the dogs by removing 200–300 ml of arterial blood (after removing the pericardial effusion) induced a nonsignificant decrease in pericardial pressure, and further decreased LV volume, mean aortic blood pressure and cardiac output. When the dogs were rapidly infused with the previously withdrawn blood plus saline, all these variables increased. Subsequent infusion of 40 and 80 ml of pericardial fluid again decreased the LV volume, mean aortic blood pressure and cardiac output. Throughout the course of the experiments the changes in heart rate were small and nonsignificant.

The LV pericardial and RV diastolic pressure-volume relations are best shown by studying the results of a single experiment (figs. 3–5). As fluid was added to the pericardium, the LV diastolic pressure increased slightly as LV volume decreased (fig. 3). The pericardial effusion shifted the pressure-volume coordinates to the left and upward, not to the left and downward along a single curve. Measurements made after acute hemorrhage produced the minimum LV and pericardial volumes. After the jugular vein catheter was opened to the elevated saline bottle, the LV pressure-volume relation was shifted upward conspicuously. The relationship of the LV transmural pressure (pressure$_{LV}$ – pressure$_{pericardium}$) to the volume appeared to be close to a single curve. As LV volume decreased, the LV transmural pressure also decreased.

Figure 4 shows the pericardial transmural pressure-volume relationship. The pericardial transmural pressure was calculated as the difference between the pericardial pressure and pleural pressure. An increase in pericardial pressure corresponds to an increase in pericardial volume. Beyond a certain pericardial volume, a small increase in volume produces a large increase in pressure.

The RV diastolic pressure-volume relationship is shown in figure 5. As volume was added to the pericardium, the RV pressure increased slightly as RV volume decreased. After volume loading, the RV pressure-volume relationship was shifted upward grossly. The RV diastolic pressure-volume curve shifted upward similar to that of the left ventricle. Also, the RV transmural pressure-volume relation remained close to a single curve. The right ventricle is very compliant; thus, the curve is essentially flat in this range.

LV and RV diastolic pressure-volume relationships were obtained in six other experiments and are shown in figures 7 and 8. The essential features already ex-
**FIGURE 4.** Pericardial transmural pressure (pressure_{pericardium} - pressure_{pleura}) vs pericardial volume for the experiment shown in figures 3 and 5. Numbers indicate the sequence of the experiment. Symbols are the same as in figures 3 and 5. Asterisks represent data recorded post-mortem with infusion of fluid into the pericardial cavity.

**FIGURE 5.** Right ventricular (RV) pressure-volume relations for the experiment shown in figures 3 and 4. Open symbols represent RV mean diastolic pressure. Closed symbols represent RV transmural pressure (pressure_{RV} - pressure_{pericardium}). Numbers indicate the sequence of the experiment. Symbols are the same as in figures 3 and 4.

**FIGURE 6.** Difference between the total volume of the pericardium (Volume_{P}) and the total volume of the heart (Volume_{H}) plotted against the amount of fluid added to the pericardial cavity. The two larger symbols with error bars represent the mean ± SD of volumes measured after pericardial effusions of 40 and 80 ml (n = 14). The smaller symbols represent individual volume measurements of various amounts of pericardial effusion.

**TABLE 1.** Effect of Pericardial Effusions on Left Ventricular and Right Ventricular Volumes

<table>
<thead>
<tr>
<th></th>
<th>Control, volume load</th>
<th>Pericardial effusion 40 ml</th>
<th>Pericardial effusion 80 ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV volume</td>
<td>1.00</td>
<td>0.91</td>
<td>0.81</td>
</tr>
<tr>
<td>RV volume</td>
<td>1.00</td>
<td>0.74*</td>
<td>0.59*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;0.001</td>
<td>p&lt;0.001</td>
</tr>
</tbody>
</table>

The absolute values for the LV and RV volume at control and after volume load were set to 1.00 in order to compare the effect of pericardial effusions of 40 and 80 ml on LV and RV volumes. The values are means of 14 experiments. The statistical analysis was done using the non-normalized data. The effects of pericardial effusion of 40 ml are compared with control, volume load. The effects of pericardial effusion of 80 ml are compared to those of 40 ml. *p < 0.001, LV vs RV volume.

Abbreviations: RV = right ventricular; LV = left ventricular.
considerable evidence has been adduced in its support. However, it remained for the current investigation to demonstrate that some shifts in the LV pressure-volume relation equal the changes in pericardial pressure. Furthermore, these changes in pericardial pressure correspond directly to changes in pericardial volume. We demonstrated this in anesthetized dogs whose cardiac volumes were manipulated by saline infusion, hemorrhage and pericardial effusion.

Our investigation demonstrates the complexity of the interaction between individual cardiac chambers and the inadequacy of the concept that LV diastolic pressure-volume relations can be approximated by a single curve. As expected from the hypothetical model, any intervention that increases the volume of the pericardium and thus increases pericardial pressure will shift the LV diastolic pressure-volume

**Table 2. Hemodynamic Effects of Pericardial Effusion and Changes in Blood Volume**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Pericardial effusion 40 ml</th>
<th>Pericardial effusion 80 ml</th>
<th>Hemorrhage</th>
<th>Volume load + pericardial effusion 40 ml</th>
<th>Volume load + pericardial effusion 80 ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pericardial pressure (mm Hg)</td>
<td>2.3 ± 1.8</td>
<td>3.9 ± 1.8</td>
<td>4.9 ± 2.0</td>
<td>0.9 ± 0.6</td>
<td>12.6 ± 3.1</td>
<td>11.4 ± 3.6</td>
</tr>
<tr>
<td>LV volume (ml)</td>
<td>44 ± 11</td>
<td>33 ± 8</td>
<td>27 ± 8</td>
<td>32 ± 6</td>
<td>55 ± 12</td>
<td>41 ± 11</td>
</tr>
<tr>
<td>Mean aortic pressure (mm Hg)</td>
<td>110 ± 11</td>
<td>99 ± 19</td>
<td>88 ± 29</td>
<td>67 ± 23</td>
<td>126 ± 23</td>
<td>117 ± 21</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>2.3 ± 0.7</td>
<td>1.8 ± 0.4</td>
<td>1.4 ± 0.2</td>
<td>2.9 ± 0.2</td>
<td>2.3 ± 0.9</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>90 ± 14</td>
<td>94 ± 15</td>
<td>95 ± 12</td>
<td>96 ± 6</td>
<td>92 ± 12</td>
<td>90 ± 9</td>
</tr>
</tbody>
</table>

The effects of pericardial effusion of 40 and 80 ml, hemorrhage and volume load are compared with control. The effects of volume load plus pericardial effusions of 40 and 80 ml are compared with the effects of volume load alone. The values are mean ± SD.

For technical reasons, not all measurements were obtained in all 10 dogs studied. We excluded the pericardial pressure data from experiment 2 because excessive concern about leaks in the pericardium in this early experiment made us overestimate the incision, compromising pericardial volume and elevating pericardial pressure (figs. 7 and 8).

Abbreviations: LV = left ventricular.

**Discussion**

The mechanism of acute, large displacements in the LV diastolic pressure-volume curve is unclear. A central role of the pericardium has been suggested and considerable evidence has been adduced in its support. However, it remained for the current investigation to demonstrate that some shifts in the LV diastolic pressure-volume relation equal the changes in pericardial pressure. Furthermore, these changes in pericardial pressure correspond directly to changes in pericardial volume. We demonstrated this in anesthetized dogs whose cardiac volumes were manipulated by saline infusion, hemorrhage and pericardial effusion.

Our investigation demonstrates the complexity of the interaction between individual cardiac chambers and the inadequacy of the concept that LV diastolic pressure-volume relations can be approximated by a single curve. As expected from the hypothetical model, any intervention that increases the volume of the pericardium and thus increases pericardial pressure will shift the LV diastolic pressure-volume

**Figure 7. Left ventricular (LV) pressure-volume relations in six experiments. Open symbols represent LV mean diastolic pressure. Closed symbols represent LV transmural pressure (pressure

LV – pressure pericardium). Squares show control and effect of pericardial effusion of 40 and 80 ml before volume load. Triangles indicate hypovolemia effected by hemorrhage when the pericardium was empty. Circles show effect of volume load and volume load plus pericardial effusion of 40 and 80 ml.
curve upward. This conclusion is confirmed in the present study in which addition of fluid to the pericardium shifted the LV pressure-volume relation up and to the left (fig. 3). Weber and Janicki also concluded that there is no practical experimental intervention that will effect a continuous upward and rightward progression along a single LV pressure-volume curve if the pericardium is functionally intact. This does not deny that a line might be passed through the end-diastolic pressure-volume coordinates of such a series of curves. It also does not deny that such a progression may be seen after the pericardium has been removed.

Pressure-volume curves of the right ventricle have not been studied in the same detail as have those of the left ventricle, in part because of methodologic difficulties in measuring RV volume. We measured RV volume and showed that RV diastolic pressure-volume relations can be displaced similarly by alterations in pericardial volume and pressure (fig. 3).

The right ventricle also affects the LV pressure-volume curve. The magnitude of this effect in the intact animal is not clear, however. The LV transmural pressure-volume points recorded after volume load sometimes seemed to be shifted up or leftward (fig. 7). This could represent an effect of the right ventricle. If so, the contribution of the right ventricle is small relative to that of the pericardium. This is not surprising; RV pressure did not greatly exceed pericardial pressure in these experiments. This requires further investigation.

We used a balloon similar to that described by Holt et al. to measure pericardial pressure. The balloon was free from air bubbles and contained a volume of saline (2 ml) that was much less than the maximal unstressed volume (8 ml). The pressure measured by such a balloon may equal the average force exerted over the surface area of the balloon. In some experiments, we compared this pressure to that measured with an open, multiple-side-hole catheter left within the pericardium. Agreement was very good when the pericardium was sealed and contained variable volumes of fluid.

CT is particularly useful for measuring total pericardial volume if a steady state can be achieved for approximately 30 seconds while the scans are recorded. The accuracy of the method is supported by a comparison of diluted contrast medium added to the pericardium and the amount measured by CT (fig. 6). We have described pericardial transmural pressure vs volume curves similar to those of Holt et al.. We have shown that CT can be used to measure human LV cast volumes more accurately and precisely than biplane angiography. Furthermore, in contrast to angiographic methods, CT measurements are largely independent of chamber orientation.

We used ungated images and related them to mean diastolic pressures. Our instrument is capable of constructing an image from information gathered from a particular interval during the cardiac cycle. For instance, an image can be formed from exposures made only during the periods of isovolumic contraction and relaxation, thus defining end-diastolic and end-systolic volumes, respectively. However, approximately 20 seconds of continuous scanning are needed to acquire sufficient information to make such a gated image for each section. A steady state cannot be maintained for the several minutes required to measure the whole heart in this way. One could presume that a single section could be chosen to represent the volume change of the left ventricle. However, analysis has shown that motion of the whole heart makes it difficult to determine ejection fraction from a single section. (The conical apex moves cephalad during systole, exaggerating the calculated ejection fraction.) We have compared the area of the LV lumen in a particular ungated section to the area of that section gated at end-diastole. The area measured on the ungated image was always 80–90% of the area on the gated end-diastolic image. This fact supports our plotting mean diastolic pressures as functions of ungated volumes. Our measured LV volumes were of the order of 50 ml (fig.

**Figure 8.** Right ventricular (RV) pressure-volume relations for the experiments shown in figure 7. Open symbols represent RV mean diastolic pressure. Closed symbols represent RV transmural pressure (pressure RV — pressure pericardium). Symbols are the same as in figure 7. RV pressures were measured in experiments 3 and 7; in experiments 2, 4, 8 and 9, right atrial pressures were plotted.
7). This then would correspond to an end-diastolic volume of 59 ml (assuming ungated volume = 0.85 × gated diastolic volume). Cardiac output was about 2 l/min and heart rates were approximately 100 beats/min, so stroke volumes were about 20 ml. The end-diastolic volume minus one-half the stroke volume approximately equaled the ungated volume. Thus, using mean diastolic pressures seemed appropriate.

We studied the mechanism of acute shifts in the LV diastolic pressure-volume relation using volume load, hemorrhage, and pericardial effusion to change cardiac and pericardial volumes. Acute volume expansion and pericardial effusions of 40 and 80 ml shifted the LV diastolic pressure-volume relation upward. However, when transmural LV pressure (pressureLV − pressure pericardium) was plotted against LV volume, the points approximated a single curve. Similar phenomena were described for the right ventricle. Shifts in LV and RV diastolic pressure-volume relations corresponded to changes in pericardial pressure. Increases in pericardial pressure were caused by progression along a pericardial pressure-volume relation.

Thus, under the conditions of our experiment, shifts in the LV diastolic pressure-volume curve are due to changes in pericardial pressure, in turn due to changes in pericardial volume. This mechanism also seems to prevail in shifts due to increased or decreased afterload.10,11 In contrast, Serizawa et al.21 studied open-chest dogs and removed the pericardium and showed apparent rightward shifts after ischemia and dramatic upward shifts in the immediate postpacing interval. Further work of this group indicates that these postpacing upward shifts are accentuated by caffeine and diminished by nifedipine, suggesting an important role for calcium in this mechanism.

We conclude that some shifts in the LV diastolic pressure-volume relation are mediated to a major degree by changes in pericardial pressure, e.g., pericardial effusion and tamponade, changes in blood volume, changes in LV afterload, and some postoperative cardiac failure. Other shifts are due to changes in the material properties of the myocardium, e.g. the transient effect of pacing in the presence of ischemia. Ischemia alone may shift the pressure-volume relation rightward as it shifts the pressure-length relation rightward in experimental animals. Mechanisms for other shifts in the pressure-volume curve may be combinations of these effects or other mechanisms not yet elucidated.

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