Left ventricular pressure-volume relations shift to the left after long-term loss of pericardial restraint

JEFFREY D. HOSENPUDD, M.D., NAM N. YUNG, AND MARK J. MORTON, M.D.

ABSTRACT The short-term effect of pericardiectomy is to shift the in vivo left ventricular (LV) pressure-volume curve to the right. We studied nine weight-matched pairs of male guinea pigs 28 to 39 days (mean 35) after complete pericardiectomy or sham thoracotomy to determine the long-term effects of pericardiectomy on LV pressure-volume relations. Hemodynamic and in vitro LV pressure-volume data were collected in matched pairs on the same day, 2 to 3 hr after catheter placement and recovery from anesthesia. Cardiac output was measured by the microsphere reference sample method. Postsurgical weight gain was similar in both groups: 823 ± 6 (mean ± SD) to 925 ± 6 g in the pericardiectomy group and 829 ± 7 to 927 ± 7 g in the sham thoracotomy group. We found no difference in LV weight: 1.555 ± 0.145 g in the pericardiectomy group vs 1.564 ± 0.148 g in the sham thoracotomy group, nor any difference in heart rate, mean arterial, right atrial, or left ventricular end-diastolic pressures, cardiac outputs, or stroke volumes (p = NS). LV pressure-volume relations, however, were shifted to the left in the pericardiectomy group (p < 0.005). At 10 mm Hg, LV volume in the pericardiectomy group (0.85 ± 0.22 cc) was less than that in the sham thoracotomy group (1.02 ± 0.15 cc; p < 0.025). The LV stress-elastic modulus relationship was not different between groups (p > 0.30). One month after pericardiectomy, LV pressure-volume relations in vitro were shifted to the left without a change in LV weight, LV elastic modulus, or hemodynamics. We speculate that this shift compensates for the lack of pericardial restraint and returns LV volume and hemodynamics to normal in vivo. Circulation 68, No. 1, 155–163, 1983.

MORE than 50 years ago, Wilson and Meek1 demonstrated a constraining effect on the heart by the pericardium at physiologic filling pressures. Although several investigations have suggested that pericardial constraint of the heart is important only at elevated filling pressures,2-4 others have demonstrated that the pericardium affects the diastolic pressure-volume relationship of the left ventricle both at normal and elevated filling pressures.5-8 One concludes from these data that short-term pericardial removal shifts the left ventricular pressure-volume relationship to the right. Glantz and Parmley9 stressed the importance of pericardial restraint to left ventricular performance, and Misbach and Glantz10 later demonstrated this importance. Short-term pericardial removal shifts the left ventricular function curve upwards; stroke volume is increased at any given filling pressure. Nevertheless, Holt11 observed that “human beings with congenital absence of the pericardium and pericardectomy animals survive indefinitely and appear to carry out their gross physiologic functions in a more or less normal manner.” Evaluation of heart size by x-ray in these same groups has produced conflicting results.12-15

Studies of myocardial or circulatory adjustments to long-term pericardial removal have not been reported. Accordingly, we investigated the effect of 1 month of pericardial removal on left ventricular pressure-volume relations and hemodynamics in the guinea pig.

Methods

Animal selection and preparation. Male guinea pigs (Dunker-Hartley or English shorthair strain) were paired by age and weight. Pairs were housed together and cared for in an identical manner before and after surgery. Pair mates received surgical preparation and 1 month later hemodynamic and pressure-volume studies within 24 hr of each other.

After administration of 20 mg/kg ketamine hydrochloride and 0.5 mg/kg im xylazine for anesthesia in the pericardiectomy animals (group 1), a transverse thoracotomy without intubation was performed in the fourth intercostal space. After the pleural space was entered, the pericardium was gently lifted at the obtuse margin of the left ventricle with a pair of forceps and then torn from base to apex, resulting in retraction of the pericardium.
to the level of the great vessels anteriorly and the left atrial reflection posteriorly. Hereafter, this procedure will be termed "pericardiectomy," although no pericardium was actually removed from the animal. The thoracic cavity was closed by means of a purse-string suture in the intercostal muscle layer, and negative pressure was restored by manual thoracic compression and a chest tube. The overlying muscle was approximated, the skin was closed, and the chest tube was removed. Total apneic time (from opening of pleural space to purse-string closure) was less than 30 sec. Sham-operated animals (group 2) underwent the identical procedure except that the pericardium was touched with the forceps but not torn. Weight gain over the subsequent days was monitored to assess animal recovery to good health.

Twenty-eight to 39 days (mean 35) after thoracotomy, the matched pairs were again anesthetized with 20 mg/kg ketamine and 0.5 mg/kg im xylazine and the following vinyl catheters (0.90 mm od, 0.50 mm id) were placed: (1) a right atrial catheter through the jugular vein, (2) a left ventricular catheter through the carotid artery, and (3) a terminal aorta reference catheter (~2 cm below renal artery) through the superficial saphenous artery. Catheter position was verified by pressure monitoring and subsequently at autopsy. Catheters were tunneled subcutaneously to the back of the neck, filled with heparin, capped, and placed in a cloth pouch. Two animals, one group 1 animal and one group 2 animal, died shortly after catheter placement and no data on these were obtained. Because their pair mates had similar initial and prestudy weights, they were matched, resulting in a total of nine study pairs.

**Study protocol**

**In vivo hemodynamics.** Between 2 and 3 hr after anesthesia the animals were mildly restrained in a testing cage and allowed to acclimate for an additional 30 to 45 min. Right atrial, aortic, and left ventricular pressures were recorded with Statham P-23 Db transducers and a Beckman R611 dynograph recorder. Heart rate was obtained from the left ventricular pulse tracing and respiratory rate from the right atrial wave form.

Cardiac outputs were obtained by the radioactive microsphere indicator dilution method. A sample of approximately 1 million 15 pm single-labeled radioactive microspheres in a total volume of 1 ml of saline was injected over 15 sec into the left ventricle, followed by a 2 ml saline flush.

The reference sample was collected with a syringe pump (Harvard Apparatus) for at least 2 min. The withdrawal rate was determined from the quotient of the blood weight and withdrawal time and ranged from 1.28 to 1.51 ml/min. The radioactivity of the entire reference sample and six well-mixed, weighed aliquots of the ashed carcass were counted for 5 min (Packard Auto Gamma Scintillation Spectrometer 5230), and the total injected counts were determined by the quotient of the ashed carcass weight and total aliquot count divided by the total aliquot weight. The left and right kidneys were counted separately to verify adequate microsphere mixing.

After microsphere injection and reference sample collection, a blood sample for arterial blood gas and hematocrit was obtained.

**In vitro pressure-volume curves.** After hemodynamic data were obtained, the animals received 100 mg/kg iv pentobarbital to induce diastolic cardiac arrest, in addition to producing anesthesia. The chest was then rapidly opened, and the heart was examined for the presence or absence of the pericardium and then removed en bloc. The right ventricular outflow tract was incised and the right ventricle and papillary muscle were dissected from the septum. The atrioventricular groove on the right side was incised from the interatrial septum to the aorta. A snugly fitting double-lumen grooved plug 6 mm od was inserted through the left atrial appendage and the mitral valve. A suture was passed around the left atrioventricular groove, aortic valve, and interatrial septum, isolating the left ventricle without collapsing the mitral annulus. The ventricle was flushed with saline at room temperature. The heart was suspended in a shallow dish of normal saline at room temperature, and the inner lumen of the cannula was connected to a Statham P-23 Db strain gauge zeroed at the midventricular level. The outer lumen was connected to a stopcock manifold and a calibrated syringe infusion pump filled with room temperature saline (Harvard Apparatus). The syringe pump was driven at a constant rate, which filled the ventricle to a pressure of 25 mm Hg over 5 to 10 sec. The ventricle was manually emptied until no more fluid could be withdrawn, and intraventricular pressure was recorded continuously while the ventricle was filled. All fluid was then withdrawn and the process was repeated until at least three curves of volume and pressure were obtained.

Completion of pressure-volume curves was usually accomplished in less than 20 min from the time of anesthesia. We have noted rigor, as evidenced by a leftward shift of the pressure-volume curve, in ventricles studied in this manner in no less than 45 min. The stability of diastolic pressure-volume relations in isolated arrested ischemic ventricles has been confirmed in murine and canine hearts for 30 and 60 min, respectively. After satisfactory recordings were obtained, each ventricle was emptied and tested for leaks by the completeness of recovery of fluid injected into the ventricle to a pressure of 25 mm Hg; no leaks occurred. Pressure-time recordings were converted to pressure-volume curves with the known infusion rate. The syringe pump was calibrated after each experiment by duplicate 60 sec infusions into a graduated cylinder. The heart was disconnected from the plug and weighed after all extraneous tissue was removed, and the aorta was severed at the aortic valve. The incision of the atrioventricular groove was then completed, and the right ventricle, right atrium, and left atrium were removed from the left ventricle. The left ventricle was opened from the aortic annulus to the apex, blotted, and weighed.

The mean of three pressure-volume curves during infusion was obtained for each left ventricle by averaging pressure at 0.05 cc increments of volume. The volume at each even value of pressure was obtained for analysis by interpolation from the pressure-volume curve over a range from 2 to 24 mm Hg.

Ventricular dimensions, wall stress, and stress-elastic modulus relationships were determined by a thin-wall spherical model. The pressure-volume curves were approximated by a fifth order polynomial and left ventricular stiffness (dP/dV) was determined by differentiation. Ventricular radius and wall thickness were calculated from instantaneous volume and the mass of the ventricle:

\[ V = \frac{4}{3} \pi \left( r^3 - h^2 / 2 \right) \]  
\[ h = W / (4 \pi r^2) \]

Wall stress was determined as follows:

\[ \sigma = \frac{Pr}{2h} \]

and elastic modulus was calculated, assuming that h/2 was small compared with r and that V = 4/3 π r³:

\[ E = 30[(V/P) dP/dV + 1] \]

(In the above equations, V = volume contained in ventricle, r = mean radius of ventricle, h = wall thickness, W = weight of ventricle, p = density of heart [1.05 g/cc], σ = wall stress, and E = elastic modulus.)

By simultaneous inspection of the pressure-volume relationships and equation 3 and first-order differentiation of P with respect to V for the polynomial, elastic modulus was calculated at values of wall stress from 4 to 24 mm Hg with a Data Control Nova Computer.)
TABLE 1
Hematocrit, arterial blood gas tensions, and pH 1 month after thoracotomy (mean ± SD)

<table>
<thead>
<tr>
<th>Study group</th>
<th>Hct (%)</th>
<th>Po2 (torr)</th>
<th>Pco2 (torr)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 (n = 9)</td>
<td>35 ± 6</td>
<td>91 ± 7</td>
<td>41 ± 4</td>
<td>7.37 ± 0.06</td>
</tr>
<tr>
<td>Group 2 (n = 8)</td>
<td>35 ± 4</td>
<td>86 ± 7</td>
<td>44 ± 6</td>
<td>7.34 ± 0.06</td>
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<tr>
<td>p value</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Hct = hematocrit; Po2 = partial pressure of oxygen; Pco2 = partial pressure of carbon dioxide.

AArterial sample not obtained because of catheter failure.

Short-term pericardiectomy. Although short-term pericardial removal shifts left ventricular pressure-volume relationships to the right in the dog,6,7 we are unaware of published reports of pericardial physiology in the guinea pig. Accordingly, we investigated the effects of short-term pericardial removal on left ventricular pressure-volume relationship in five male guinea pigs. With ketamine and xylazine anesthesia, as previously described, a small vinyl catheter was placed in the jugular vein. The animals underwent anticoagulation with 500 units of heparin and were then given 100 mg/kg iv pentobarbital to induce diastolic cardiac arrest. The chest was rapidly opened. A balloon with infinite compliance over the volumes of interest, attached to a catheter on both ends, was inserted deflated into the left ventricle through a small apical stab wound. The catheter at the distal end of the balloon was exited through the aorta. A circumferential suture proximal to the exit site just above the aortic valve was placed to secure the balloon catheter and to isolate the left ventricle from the aorta. The left ventricle was manually emptied of blood through the apical vent. Care was taken to leave the pericardium intact except for the small apical slit through which the balloon was inserted. The catheter at one end of the balloon was connected to a syringe pump (Harvard Apparatus) filled with saline at room temperature; the other catheter was connected to a Statham P-23 Db strain gauge zeroed at midventricular level. Pressure-volume curves were generated and analyzed as previously described with the pericardium intact and immediately after pericardial removal. The entire procedure was performed in less than 20 min for each animal. The left ventricle was then completely opened and inspected to ensure the absence of blood and was removed from around the balloon. Pressure-volume curves were then generated for the isolated balloon to confirm the previously noted compliance characteristics.

Statistics. The significance of differences between means of measurements for group 1 and group 2 animals was determined by Student’s two-tailed t test for unpaired data. The significance of differences between pressure-volume coordinates for group 1 and group 2 animals was determined by two-way analysis of variance, by analysis of covariance for the linear relationship defined by pressure as a monoeponential function of volume, and by Student’s t test for individual filling pressure. The approximation of pressure-volume curves by polynomials was accomplished by computing F values from the reduction of the sum of squares for successive Nth order polynomials. We used the Nth order polynomial with an F value indicating a probability of difference of less than 5%.20 The significance of differences in the stress-elastic modulus relationship between group 1 and group 2 animals was determined by both two-way analysis of variance at individual levels of left ventricular stress as well as by comparison of slopes of the linear relationship with Student’s two-tailed t test for unpaired data. The significance of differences after short-term pericardiectomy were determined with a two-way analysis of variance (treatment by treatment by subject), for all data points, and Student’s t test for paired data for volumes at individual pressures. Each animal served as its own control.

Results

Animal recovery. Both groups were well matched by similar body weights at the time of pairing (0.823 ± 0.061 and 0.829 ± 0.069 kg, respectively). Weight gain occurred in both groups, and at the end of the study, group 1 weight (0.925 ± 0.060 kg) and group 2 weight (0.927 ± 0.068 kg) were not different. Arterial blood gas tensions and hematocrit values obtained at the end of the study are shown in table 1. These values are similar to those previously reported in control animals21,22 and suggest that the animals recovered successfully from thoracotomy.

Hemodynamic data. Hemodynamic data are presented in table 2. Left ventricular end-diastolic pressures are reported; however, the natural frequency response of the left ventricular catheters used (approximately 10 cycles/sec) was less than optimal. Figure 1 demonstrates a representative example of pressure recordings...

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obtained in the guinea pigs. Cardiac outputs and stroke volumes were successfully obtained from eight group 1 and six group 2 animals. There was no statistically significant difference in heart and respiratory rates, in aortic, right atrial, or left ventricular end-diastolic pressures, in cardiac output, or in stroke volumes between both groups.

**Autopsy data.** One month after pericardiectomy, visual inspection of the mediastinum of all animals revealed absence of the pericardium (figure 2). The pericardium was intact in all group 2 animals (figure 3). There was no evidence of adhesions, scar, or infection in any of the animals in either group, and whole heart weights were not different between groups (group 1, 2.328 ± 0.214 g; group 2, 2.344 ± 0.235 g), nor were left ventricular weights (group 1, 1.555 ± 0.145 g; group 2, 1.564 ± 0.148 g).

**Pressure-volume data.** All curves demonstrated the characteristic shape present when the left ventricle is filled from zero volume. Negative pressure was re-

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**TABLE 3**

Left ventricular pressure-volume measurements for individual animals

<table>
<thead>
<tr>
<th>Pressure (mm Hg)</th>
<th>Sham-operated (group 2)</th>
</tr>
</thead>
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<tr>
<td></td>
<td>1</td>
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<tr>
<td>2.0</td>
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<td>4.0</td>
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<tr>
<td>12.0</td>
<td>1.023</td>
</tr>
<tr>
<td>14.0</td>
<td>1.071</td>
</tr>
<tr>
<td>16.0</td>
<td>1.111</td>
</tr>
<tr>
<td>18.0</td>
<td>1.145</td>
</tr>
<tr>
<td>20.0</td>
<td>1.172</td>
</tr>
<tr>
<td>22.0</td>
<td>1.195</td>
</tr>
<tr>
<td>24.0</td>
<td>1.217</td>
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required to deform the ventricle and achieve complete emptying. Negative pressure and puckering of the ventricle were sought in all studies to ensure a reproducible zero volume. The onset of filling is identified by the rapid increase in ventricular pressure, which marks zero ventricular volume on the record. Ventricular pressure-volume relations for the two groups are presented in Figure 4 and Table 3. For any given left ventricular pressure, mean left ventricular volume was smaller for the group 1 (pericardiectomy) animals compared with the group 2 (sham-operated) animals. This was highly statistically significant whether absolute volumes ($p < .001$), volumes corrected for body weight ($p < .005$), or volumes corrected for left ventricular weight ($p < .005$) were compared.

Left ventricular mechanical and material properties were investigated. $dP/dV$ was determined by differentiation of the polynomial used to describe pressure-volume relations. Values calculated at $P = 10$ mm Hg for group 2 animals (41 ± 10 mm Hg/cc) and group 1

Figure 2. The chest opened for removal of the heart of a group 1 animal that underwent a pericardiectomy 1 month earlier. The pericardium is absent and there is no evidence of scar or adhesions. LV = left ventricle; LAA = left atrial appendage; RV = right ventricle; RAA = right atrial appendage; L = lung.

**Table 3**

(Continued)

<table>
<thead>
<tr>
<th>Left ventricular volume (ml)</th>
<th>Pericardiectomy (group 1)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>1</td>
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<tr>
<td>-----------------------------</td>
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</tr>
<tr>
<td>0.694</td>
<td>0.381</td>
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<tr>
<td>0.969</td>
<td>0.512</td>
</tr>
<tr>
<td>1.111</td>
<td>0.596</td>
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<tr>
<td>1.184</td>
<td>0.656</td>
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<tr>
<td>1.230</td>
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</tr>
<tr>
<td>1.271</td>
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<tr>
<td>1.313</td>
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<td>1.414</td>
<td>0.900</td>
</tr>
<tr>
<td>1.410</td>
<td>0.912</td>
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</table>
animals (42 ± 7 mm Hg/cc) were not different. Figure 5 demonstrates that there was no significant difference in the stress-elastic modulus relationship for left ventricles of the group 1 and 2 animals for any given level of left ventricular stress (p > .25). In addition, the slopes of the linear relationship (18.5 ± 4.6 and 16.6 ± 3.3, respectively; p > .30) were not different.

Pressure-volume relations were also assessed, assuming that pressure was a monoeXponential function of volume. Figure 6 shows that the log pressure-volume relationship is shifted to the left after pericardiectomy. Analysis of covariance of the linear regression of the log pressure-volume relationship showed no difference in slopes (group 1, 3.60 ± 1.22; group 2, 3.40 ± 1.26; p = NS), but the x-intercept was shifted to the left (group 1, .263 ± .169; group 2, .374 ± .134; p < .001) after pericardiectomy. Ventricular volume at 0 pressure (V₀), determined from individual pressure-volume curves, was also lower after pericardiectomy (0.171 ± 0.067 vs 0.197 ± 0.056) but not statistically significant. Therefore, when pressure-volume relations were normalized for V₀ (figure 7), the curves for both groups were identical.

Calculated ventricular dimensions at a filling pressure of 10 mm Hg are shown in table 4. Left ventricular radius was decreased after pericardiectomy with borderline significance. Left ventricular wall thickness after pericardiectomy was increased (p < .025) and the radius-to-wall thickness ratio after pericardiectomy was decreased (p < .01).

**Short-term pericardiectomy.** Figure 8 shows that the left ventricular pressure-volume relations are shifted to the right after short-term pericardiectomy in the five animals studied (p < .025). At 6 mm Hg the left ventricular balloon volume was 0.35 ± 0.10 cc with the pericardium intact and 0.40 ± 0.10 cc with the pericardium removed (p < .05).

**Discussion**

Like that in other species, the pericardium in the guinea pig restrains left ventricular filling at physiologic pressure. The procedure we used to test the effect of the pericardium on left ventricular pressure-volume relations produced insignificant damage to the pericardium and did not involve suturing or closing the pericardium. Pericardial repair has been shown to importantly alter pericardial function. The ventricular volume measurement, although reproducible in each animal, did not measure actual left ventricular volume. Because the mitral valve was intact, important displacement of the ventricular balloon by chordae and papillary muscles was unavoidable. Our data demonstrate that short-term pericardial removal in the guinea pig shifts the left ventricular pressure-volume relationship to the right. Precise quantitation of this shift must await studies with different methods.
FIGURE 4. The in vitro left ventricular (LV) pressure-volume curve is shifted to the left 1 month after loss of pericardial restraint. This finding is present whether absolute LV volumes (A, p < .001), LV volumes normalized to body weight (B, p < .005), or LV volumes normalized to LV weight (C, p < .005) are used. This left-shifted pressure-volume relationship is what might be expected with the added influence of the pericardium. Pericardiectomy = group 1; sham = group 2.

Unlike short-term pericardial removal, long-term absence of the pericardium appears to shift the left ventricular pressure-volume relationship to the left of the sham-operated controls. The animals in both groups achieved their preoperative weight within 10 days after thoracotomy. By the end of the study, there was a weight increase of 12% and all animals had normal hematocrit values and arterial oxygen tensions. The hemodynamic measurements and arterial blood samples were obtained in the postanesthetic state, which may explain the mild acidosis, Pco2 elevation, and the slightly lower heart rates and cardiac outputs when compared with previously published data.21-23

This study investigated in vitro pressure-volume re-

FIGURE 5. The stress-elastic modulus relationship for pericardiectomy (group 1) and sham-operated animals (group 2) is linear. There is no statistical difference between groups in the elastic modulus for a given level of left ventricular (LV) stress (p > .25), nor was there statistical difference in the slopes of the linear relationship between groups (p > .30).

FIGURE 6. The monoexponential fits of the left ventricular pressure-volume relationships of pericardiectomy (group 1) and sham-operated animals (group 2) are demonstrated. There is a parallel and leftward shift of the pressure-volume relationship in the pericardiectomy animals (p < .001).
FIGURE 7. When volume is normalized to $V_0$ (volume at 0 pressure), the pressure-volume curve of the pericardiectomy (group 1) and sham-operated animals (group 2) are superimposed. This is consistent with a change in left ventricular geometry in the group 1 animals.

relationships of the arrested isolated guinea pig left ventricle. The in vitro passive pressure-volume curve only partially determines in vivo pressure-volume relations. One important determinant of in vivo pressure-volume relations is the pericardium. Because this study was performed in vitro, the pressure-volume curves in group 2 animals were obtained in the absence of the pericardium. One would speculate that in vivo with the pericardium intact, the intracavitary pressure-volume relationship would be somewhere to the left of the in vitro pressure-volume relationship. For example, in figure 4, A, a restraining pressure of 4 to 8 mm Hg would be required at physiologic filling pressures to reduce the volume of the group 2 ventricle to the volume of the group 1 ventricle. Holt et al. demonstrated that the pericardium is responsible for a similar degree of left ventricular restraint. Therefore the apparent smaller ventricle in the pericardiectomy animal in vitro could be construed as an attempt to return the ventricle in vivo to an intracavitary pressure-volume relationship that is present when both ventricle and pericardium are present.

Other factors that are important in determining in vivo left ventricular pressure-volume relations include right ventricular interactions, coronary artery pressure, early diastolic relaxation, and changes in loading conditions which allow stress relaxation or creep. In addition, the viscoelastic properties of the left ventricle are dependent on the rate of filling. This may be particularly relevant in this animal model with heart rates above 200 beats/min. We did not account for these important variables in our study. Nevertheless, passive pressure-volume relations of the left ventricle are a major determinant of in vivo diastolic pressure-volume relations.

To determine whether changes seen between group 1 and group 2 animals were a result of a change in myocardial stiffness or a change in left ventricular geometry, a geometric model was used to investigate left ventricular dimension, wall stress, and elastic modulus. The thin-wall sphere model was used because current techniques for direct stress measurement are unreliable and the best model, which is the ellipsoid, cannot be applied because of the lack of knowledge of left ventricular major and minor axes from this study. Although the left ventricle is neither a thin-walled sphere nor composed of isotropic material, errors produced by these assumptions are consistent and this model has been found to provide valid comparisons among ventricles. No significant difference in muscle elasticity was present between groups. A change in geometry rather than material properties is further supported by the similar slopes of the monoexponential

<table>
<thead>
<tr>
<th>Study group</th>
<th>$r$ (mm)</th>
<th>$h$ (mm)</th>
<th>$r/h$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 (n = 9)</td>
<td>7.001 ± 0.421</td>
<td>2.358 ± 0.292</td>
<td>3.02 ± 0.49</td>
</tr>
<tr>
<td>Group 2 (n = 9)</td>
<td>7.315 ± 0.264</td>
<td>2.162 ± 0.217</td>
<td>3.42 ± 0.39</td>
</tr>
<tr>
<td>p value</td>
<td>&lt; .05</td>
<td>&lt; .05</td>
<td>&lt; .025</td>
</tr>
</tbody>
</table>

$r =$ radius; $h =$ wall thickness.
fits to the pressure-volume curves and the correction of the shift by normalizing volume to $V_o$.

The short-term effect of pericardial removal is to shift the ventricular function curve to the left. Some form of circulatory or cardiac adjustment must therefore occur to compensate for this shift and return hemodynamics to baseline. We propose that the leftward shift in the left ventricular pressure-volume relationship 1 month after pericardectomy may be the compensation by which somatic needs and normal filling pressures are maintained.

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References

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