The effect of pericardium on the end-systolic pressure–segment length relationship in canine left ventricle in acute volume overload

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ABSTRACT The effect of the pericardium on the end-systolic pressure–segment length relationship in the left ventricle was examined with an ultrasonic miniature gauge in open-chest dogs. In 12 dogs, blood was infused until left ventricular (LV) end-diastolic pressure reached about 20 mm Hg, and then the pericardium was opened widely. In the other 12 dogs a pericardiectomy was performed without blood infusion. Stroke volume was measured in six dogs in the former group and in seven dogs in the latter group. After blood infusion, LV systolic, end-systolic, and end-diastolic pressures increased from 120 ± 14 to 162 ± 16 mm Hg (mean ± SD), from 106 ± 13 to 146 ± 17 mm Hg, and from 8 ± 2 to 19 ± 2 mm Hg, respectively (all p < .01). End-systolic and end-diastolic segment lengths increased from 8.9 ± 2.1 to 10.6 ± 2.2 mm and from 11.6 ± 2.5 to 14.9 ± 2.7 mm, respectively (both p < .01). After pericardiectomy, the segments were further lengthened by 8.9 ± 4.4% and by 10.0 ± 6.2%, respectively (both p < .01). Heart rate, LV systolic and end-systolic pressures, and peak positive dp/dt did not change, although end-diastolic pressure fell from 19 ± 2 to 18 ± 2 mm Hg (p < .01). Stroke volume rose from 13.1 ± 3.7 to 23.9 ± 5.0 ml due to volume loading and further increased by 26.7 ± 9.0% after pericardiectomy. Without blood infusion, pericardiectomy did not change any pressures, segment lengths, or stroke volumes except the end-diastolic pressure, which fell from 8 ± 2 to 7 ± 2 mm Hg (p < .01). The LV end-systolic pressure–segment length relationship was almost linear in each case (r = .88 to .99, all p < .01) during blood infusion with the pericardium intact, but shifted to the right after pericardiectomy. Without volume loading, the pericardiectomy did not change the relationship. Thus, the LV end-systolic pressure–segment length relationship was shifted upward by the presence of the pericardium under volume loading. These results suggest that, in short-term volume overload, the pericardium reduces the increase of LV stroke volume but shifts the LV end-systolic pressure-volume relationship upward due to the mechanical restraint of the pericardium on the heart and/or the enhancement of both ventricular interaction produced by the pericardium.

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IT HAS BEEN REPORTED that the pericardium prevents the heart from overdistension and contributes to the elevation of left ventricular (LV) diastolic pressure relative to any given volume through its restraint.1–8 It has also been suggested that the pericardium enhances the diastolic interaction between both ventricles.4,7–9 Several authors have investigated the effect of the pericardium on systolic function. Stockland et al.10 showed that pericardiectomy did not change the LV systolic pressure but increased cardiac output in open-chest dogs after moderate volume expansion, and Mangano11 also reported that the radial artery systolic pressure did not change after pericardiectomy during cardiac surgery in patients whose pulmonary capillary wedge pressures ranged from 4 to 16 mm Hg. Janicki and Weber7 found variable changes in the LV peak developed pressure after pericardiectomy when the ventricle contracted isovolumetrically with the right ventricular (RV) end-diastolic pressure at zero and both atria empty.

Recently, the LV end-systolic pressure-volume (or diameter) relationship has been offered as a new index for quantitating the contractile state of the left ventricle independent of preload and afterload.12–15 and this relationship or its modifications have been applied to clini-
cal studies. However, there has been no in situ study examining the effect of the pericardium on this relationship.

We hypothesized that the pericardium alters the LV pressure-volume relationship at end-systole, as at end-diastole, through its restraint due to the following mechanisms. The pericardial sac is filled with two atria and two ventricles, which determine the intrapericardial volume or pressure. Both atria increase in size at end-systole when the ventricular volumes decrease, which suggests that the atrial as well as ventricular volumes do play a significant role in determining the intrapericardial pressure at end-systole. Under volume-loading conditions the intrapericardial pressure at end-systole may be elevated compared with that under normal conditions, which would alter the LV end-systolic pressure-volume relationship through restraint of the pericardium.

To test this hypothesis we measured LV pressure and segment length with a catheter-tip micromanometer and a pair of ultrasonic miniature gauges, respectively, in open-chest dogs, and compared the end-systolic pressure–segment length relationship before and after pericardiectomy at the control state in 12 dogs and at the volume-loading state in another 12.

Methods

Animal preparations. Twenty-four mongrel dogs of both sexes weighing from 11 to 31 kg were anesthetized with intravenous pentobarbital sodium (30 mg/kg). An endotracheal tube was placed, and respiration was maintained mechanically by a constant tidal volume with a positive end-expiratory pressure of 5 cm H2O. A bilateral thoracotomy was performed in the fifth intercostal space with the sternum transected. Two small incisions less than 10 mm in length were made in the pericardium, and a pair of 5 MHz piezoelectric crystals (2 mm in diameter) were implanted through them into the subendocardium of the LV anterior wall for the measurement of segment length (Model 401; Schuessler). The initial distance between both crystals was about 12 mm and the positions were almost in the minor equator plane. Implantation of the crystals permitted us to see the correct pattern of the myocardial segment length on an oscilloscope. After we confirmed that no bleeding had occurred from the wounds on the surface of the myocardium through which the crystals were implanted, each incision in the pericardium was sutured by a single stitch with care being taken not to diminish the original pericardial space.

In 13 dogs an electromagnetic flow probe (MF-46; Nihon Kohden) was set around the ascending aorta through another incision in the surrounding pericardium and was used to measure stroke volume. The additional incision (about 25 mm in length) was also carefully sutured by a few stitches as described above.

A catheter-tip micromanometer with a fluid-filled lumen (No. 8F; Millar Instruments) was inserted into the left ventricle via the right carotid artery to measure its pressure. The fluid-filled lumen was connected directly to a Statham P23Db pressure transducer that was calibrated by a mercury manometer with zero reference set at midchest level. The pressure signal from the micromanometer was corrected by the fluid-filled system. The second lead from a standard electrocardiogram was obtained from subcutaneous needles inserted into the extremities. The respiratory phase was monitored by a thermistor placed inside the endotracheal tube. These measurements were recorded on an eight-channel recorder (8K 10; San-ei) and were stored on magnetic tape (EF 3097W; Sony) for subsequent analysis.

Protocol. In 12 dogs, six of which had flow probes attached, measurement of the initial values was followed by infusion of heparinized blood (37° C) into the femoral vein through a polyethylene cannula with an infusion pump. The infusion was continued at a constant rate (4 to 5 ml/kg/min) with the total volume ranging from 400 to 1200 ml until LV end-diastolic pressure reached about 20 mm Hg. Then, a rate of infusion sufficient to maintain the pressure and the segment constant for more than 3 min was used. During such a steady state of volume loading, the pericardium was opened widely enough to expose the entire heart. Care was taken not to induce arrhythmias and not to pull out the wire leads of the implanted crystals. Less than 2 min were necessary to accomplish this pericardiectomy. All parameter values before and after pericardiectomy were recorded, and then the blood infusion was stopped. The cessation of infusion produced small changes in segment length and pressure in eight dogs in which hemodynamics were continuously monitored. That is, the segment length was shortened and pressure was lowered without significant change in heart rate.

In the other 12 dogs pericardiectomy was performed after the control values were recorded.

After the experiment was finished, the positions of the crystals in the myocardium were verified. Data from any dogs with malpositioned crystals or hemorrhagic changes in the myocardium around the crystals were excluded from this study.

Data analysis. The pressure and segment length recordings were replayed at a paper speed of 100 mm/sec. An electrocardiogram and respiration were also recorded, and the first derivative of LV pressure (dp/dt) was obtained through an electric differentiation circuit (time constant = 3 msec). All the values reported here were obtained at the end-expiratory phase. The point of end-diastole was identified as the onset of upstroke of the LV pressure from the micromanometer, and the timing of end-systole was determined as the termination of segmental shortening that preceded peak negative dp/dt by approximately 20 msec. Stroke volume was calculated by planimetry of aortic flow referring to a known calibration and was expressed as the average value of 3 beats.

To examine the pressure–segment length relationships, a pressure–segment length loop during each cardiac cycle was constructed on a storage oscilloscope (5103N; Tektronix), with the pressure signal from the micromanometer on the y axis and the segment length on the x axis. In 12 dogs subjected to volume overload several loops were superimposed on the storage oscilloscope, and photographs were taken with a Polaroid camera at control, at subsequent levels during volume expansion with the pericardium intact, and after the pericardiectomy.

Since, by visual inspection, the end-systolic point of the loop seemed to run upward and to the right in an almost linear fashion during volume loading with the pericardium intact, the data points of the end-systolic pressure–segment length relationship were fitted to a linear line with the least square method.

In the 12 dogs not receiving blood infusion the loops before and after pericardiectomy were compared.

The values of hemodynamics and segment length after the pericardiectomy were compared with those just before the pericardiectomy with paired t tests. A p value less than .05 was regarded as statistically significant.

Supplemental experiment. We designed a supplemental ex-
perimenter to determine whether a segment length of the left ventricle could be used to estimate the diameter of the entire ventricle whether or not the pericardium was present. In two dogs instrumented with catheter-tip manometers and a pair of ultrasonic crystals, as described above, the second pair of crystals was implanted through two additional incisions in the pericardium to measure an anterior-to-posterior internal diameter of the left ventricle.

Blood from a donor dog was infused once with the pericardium intact and again with pericardium removed. Electrocardiograms, respiration, LV pressure, segment length, and diameter were recorded and stored. The timing of the end-diastolic or end-systolic diameter was regarded as the timing of the segment. The end points of systolic shortening of both segment and diameter coincided with each other.

Results

Figure 1 shows the tracings from one dog subjected to volume loading. When the pericardium was intact, an infusion of blood gradually raised both systolic and end-diastolic pressures and lengthened segment length. During a steady state of volume overload the pericardectomy was performed. After the pericardectomy both end-systolic and end-diastolic segments further lengthened. LV systolic pressure slightly increased but end-systolic pressure did not change, and end-diastolic pressure decreased.

Since there was much noise on the flow pattern when the flow probe was used simultaneously with the ultrasonic dimension gauges, stroke volume was recorded at the steady state together with other parameters except for segment length. Figure 2 shows two examples of aortic flow patterns. When the pericardium was opened in dogs that did not undergo blood infusion, no remarkable change was observed (figure 2, left). In dogs in which stroke volume increased by blood infusion when the pericardium was intact, the stroke volume further increased after pericardectomy (right).

The changes in LV pressure, stroke volume, and segment length response to volume expansion, and changes after pericardiectomy are listed in table 1. Heart rate did not change when the pericardium was opened at either steady-state condition (with or without volume loading). Pericardiectomy at control did not change LV systolic pressure, peak dp/dt, or stroke volume. Under volume-loading conditions, LV systolic pressure slightly but insignificantly increased, and stroke volume also rose after pericardiectomy. Pericardiectomy did not significantly lengthen the LV end-diastolic segment under control conditions, but the segment lengthened under volume-loading conditions despite the decreases in LV end-diastolic pressures. End-systolic segment length did not change under control conditions but did significantly increase during volume overload and the LV end-systolic pressure did not change with either condition.

Figure 3 shows two loops before and after pericardiectomy in a dog not subjected to volume loading. No shift of the end-systolic point (figure 3, upper left corner) was observed. Although the end-diastolic segment seemed to be lengthened after pericardiectomy in this case, statistical significance was not found (table 1).

Figure 4 shows several pressure–segment length loops from one dog subjected to volume overload. When the pericardium was intact, an initial loop located on the left was followed by several different loops during blood infusion; the LV pressure and segment length at end-systole and end-diastole increased, and each end-systolic point of the individual loops shifted upward and to the right in a linear fashion, as shown by a white line. However, when the pericardium was widely opened the loop was displaced to the right and accompanied further increases in the end-diastolic and end-systolic segment lengths without a significant change in the end-systolic pressure. Thus, the end-systolic point of the loop shifted to the right of the linear (white) line obtained during acute volume loading when the pericardium was intact.

The end-systolic pressure–segment length relationships in 12 dogs subjected to volume loading are shown in figure 5. In each dog, the end-systolic points during blood infusion and when the pericardium was intact were positioned in a linear line with a strong correlation (r = .88 to .99; all p < .01), but after pericardiectomy the point shifted to the right and was positioned outside the limits of 95% confidence. In addition, the point after cessation of infusion was also positioned outside the limits.

Figure 6 shows the average effect of pericardiectomy on the pressure–segment length loop at each con-
tion. At control (figure 6, A) the end-systolic points of the loops before and after pericardiectomy did not differ from each other. During volume loading (figure 6, B) an infusion to a dog with an intact pericardium changed the position and the size of the loop from left to middle and shifted the end-systolic point upward and to the right linearly. After the pericardium was opened the middle loop was displaced to the right; its end-systolic point also shifted to the right without a significant change in the corresponding pressure.

In our supplemental experiment (figure 7) both segment length and anterior-to-posterior internal diameter of the left ventricle were recorded during two consecutive blood infusions to dogs with and without pericardia. As shown in figure 8, the LV segment length related linearly to its internal diameter not only at end-diastole but at end-diastole in either dog; relationship of the segment length to internal diameter is shown for one dog in figure 8, A, and for another dog in figure 8, B. The relationships before pericardiectomy did not differ from those after pericardiectomy.

Discussion

Critique of methods. We measured a segment length of the LV wall as an indicator of the volume because the accurate measurement of the volume or diameter is still difficult under these conditions. We realized from our supplemental experiment that the values of both end-systolic and end-diastolic segment length related linearly to the values of the internal diameter in dogs with or without pericardium. Previously Bishop et al. \(^{21}\) reported that the changes in the transverse dimension of the left ventricle were linearly related to the changes in ventricular volume. A more recent study\(^{22}\) also described this relationship for a wide physiologic range of LV volumes. It therefore seems likely that the LV segment length changes in proportion to the change in diameter or volume of the left ventricle when the ventricular volume is varied by volume expansion.

In this study, a pair of ultrasonic miniature dimension gauges was implanted into the myocardium through two small incisions in the pericardium. Each incision was less than 10 mm (ordinarily 5 mm) in length and was loosely sutured by a single stitch, with care being taken not to diminish the original pericardial space. If this procedure caused a reduction in the pericardial space, the effect of opening the pericardium on the present results may be overestimated. However, the observed lengthening of the end-diastolic segment after pericardiectomy at control was 2.3%, correlating well with the results found by Shirato et al. \(^{5}\) They used the same technique that we did to measure LV segment length, and their technique was evaluated later by Stockland et al. \(^{10}\) who found that it kept the pericard-
TABLE 1
Hemodynamics and left ventricular segment length before and after pericardiectomy (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 12)</th>
<th>Volume loading (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>136 ± 39</td>
<td>135 ± 39</td>
</tr>
<tr>
<td>LVSP (mm Hg)</td>
<td>121 ± 20</td>
<td>120 ± 23</td>
</tr>
<tr>
<td>LVESL (mm Hg)</td>
<td>110 ± 22</td>
<td>111 ± 25</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>8 ± 2</td>
<td>7 ± 2</td>
</tr>
<tr>
<td>Peak dp/dt (mm Hg/sec)</td>
<td>2880 ± 790</td>
<td>3070 ± 940</td>
</tr>
<tr>
<td>LVEDL (mm)</td>
<td>12.38 ± 2.13</td>
<td>12.68 ± 2.32</td>
</tr>
<tr>
<td>LVESL (mm)</td>
<td>9.26 ± 1.91</td>
<td>9.30 ± 1.97</td>
</tr>
<tr>
<td>LVSV (ml)</td>
<td>11.6 ± 5.6</td>
<td>12.6 ± 7.0</td>
</tr>
</tbody>
</table>

HR = heart rate; LVEDL = left ventricular end-diastolic segment length; LVEDP = left ventricular end-diastolic pressure; LVESL = left ventricular end-systolic segment length; LVESP = left ventricular end-systolic pressure; LVSV = left ventricular stroke volume.

\*p < .01 when compared with initial value.

\#p < .01 when compared with that before pericardiectomy.

ium intact. Although an additional incision in the pericardium that encloses the ascending aorta was made to set flow probes in seven dogs in which the pericardium was opened at control, the lengthening of the end-diastolic segment after pericardiectomy in these dogs was less than that in the remaining five. Accordingly, the method used in this study for the implantation of crystals and the setting of flow probes should not induce erroneous results.

FIGURE 4. Several pressure–segment length loops during volume loading and a loop after pericardiectomy (indicated by arrow) from one dog. During blood infusion with pericardiectomy intact, the end-systolic point shifted upward and to the right in a linear fashion (white line). After pericardiectomy the point shifted to the right side of the line. See figure 1 for abbreviations.

FIGURE 5. Effect of pericardiectomy at volume overload on the left ventricular end-systolic pressure–segment length relationship in all cases. Each solid line was derived from the data points of end-systolic pressure–segment length relationships during volume loading with pericardium intact (open circles). Arrow indicates the shift of the point due to pericardiectomy. Note that the data point after pericardiectomy (closed circle) shifted rightward and is positioned outside of the limits (broken line) of 95% confidence. The additional closed circle indicates the relationship obtained after cessation of blood infusion. For abbreviations see table 1.
FIGURE 6. Schematic representation of the pressure–segment length loops. The solid loop represents the pericardium intact and the broken loop the pericardium removed. A. The average effects of pericardectomy at control. The end-systolic point did not change after pericardectomy. B. An initial (left), the loops just before (middle), and after (right one) pericardectomy at volume loading state. The end-systolic point shifted to the right from the point just before pericardectomy. Values are mean ± SEM. For abbreviations see figure 1.

Effect of pericardium. The present study showed that the end-diastolic segment length of the left ventricle increased after pericardectomy at volume loading, but did not at control, with significant decreases in LV end-diastolic pressure occurring under both conditions. These results indicate that at end-diastole the LV wall in a dog without pericardium was more compliant than in a dog with intact pericardium during volume overload, whereas the effect of the pericardium was of less significance without volume loading. It is well known that the pericardium prevents the heart from overdistension and contributes to the increase in LV end-diastolic pressure relative to any given volume through its restraint.1–8 Our results agree with those of previous reports. When pericardial restraint was eliminated by removal of the pericardium at volume loading the stroke volume increased by 26.7%. This increase did not reflect an alteration in the inotropic state or a reduction in afterload since heart rate, peak dp/dt, and LV peak pressure were unchanged. Considering the fact that pericardiectomy lengthened the end-diastolic segment and increased systolic excursion (end-diastolic segment length minus end-systolic segment length), the observed increase in stroke volume probably resulted from the enhancement of Starling’s mechanism. A recent study10 also reported that the stroke volume increased by 13% when the pericardium was opened after blood volume expansion to about 13 mm Hg of the LV end-diastolic pressure. In contrast, the stroke volume was unchanged after pericardectomy at control (LV end-diastolic pressure, 8 mm Hg) in the present study. Mangano11 also found similar results; pericardiectomy at 10 mm Hg of pulmonary artery diastolic pressure resulted in insignificant changes in cardiac index, heart rate, and thus stroke volume in patients with coronary artery disease. Therefore, it is likely that the restrictive effect of the pericardium on LV filling becomes greater as the cardiac volume is enlarged.

Furthermore, the present study demonstrated that in volume overload the pericardium altered the LV pressure–segment length relationship at end-systole as it did at end-diastole. After pericardiectomy the endsystolic point of the loop shifted to the right of the linear line that was derived from the data points of the loops observed during volume loading in dogs with intact pericardia, although the points before and after pericardiectomy at control did not differ from each other. These results also suggest that the pericardium altered the LV end-systolic pressure-volume (or diameter) relationship at volume overload, but did not do so at control. Recently, on the basis of animal experiments,12–15 the end-systolic pressure-volume (or diameter) relationship was offered as a new index for quantitating the contractile state of the left ventricle, and several investigators have been trying to apply this index to clinical studies.16–20 The index is considered to be sensitive to change in the contractile state but insensitive to changes in preload and afterload for the ventri-
systolic pressure–segment length relationship are complex, but there are several possible explanations.

First, the pericardial restraint at end-systole should be discussed. The pericardial space contains the heart and great vessels. When both ventricular volumes decrease at end-systole because of ejecting blood, both atria are enlarged due to the concomitant increase in venous return in situ. Hence, at end-systole during volume loading compared with that during control, the atrial and the ventricular volumes become larger due to the augmented venous return, possibly causing an elevation in intrapericardial pressure. As a result, the increased intrapericardial pressure (pericardial restraint on the heart) might contribute to the rise in LV end-systolic pressure relative to any given volume. However, intrapericardial pressure was not actually measured in our experiment for various reasons. We were afraid not only of exaggerating the pericardial restrictive effect but also of causing a deformity in cardiac shape, since the balloon device used for measurement occupies some intrapericardial space. Furthermore, we found it necessary to make the pericardial incision as small as possible to exclude the potential for error in our results. Holt et al. reported that although the intrapericardial pressure is usually subatmospheric under normal conditions, the absolute LV pressure at end-systole is higher than its transmural pressure at volume loading because of the positive intrapericardial pressure. They also presented a formula for estimating the intrapericardial pressure at end-systole from the LV end-diastolic pressure. The mean value of the LV end-diastolic pressure achieved by blood infusion in our study was 19.4 mm Hg just before the pericardiectomy, and the end-systolic intrapericardial pressure was calculated as 9.2 mm Hg according to the formula of Holt et al. Since opening the pericardium eliminated this positive pericardial pressure, the pericardiectomy caused no increase in the LV end-systolic pressure despite the concomitant increase in end-systolic length. For these reasons, the elimination of pericardial restraint on the heart might be one of the mechanisms responsible for the rightward shift of the end-systolic point after pericardiectomy. Janicki and Weber found variable shifts of the LV peak developed isovolumetric pressure-volume curves after pericardiectomy in isolated hearts, and suggested that it might be possible to extrapolate their results to the end-systolic pressure-volume relationship of the ejecting ventricle. However, we cannot agree with their conclusions completely because the RV end-diastolic pressure was zero and both atria were empty when the influence of the pericardium was examined. The pericardium

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**FIGURE 8.** The results of supplemental experiments were given. Both end-diastolic and end-systolic values of LV segment lengths were plotted against their corresponding internal diameters in two dogs. A. The relationship for one dog. B. The relationship for another dog. For each dog the end-systolic segment length correlated linearly with the diameter at either condition in which the pericardium was intact or removed. The two relationships did not seem to be different from each other. Open circle = end-diastole with pericardium intact; closed circle = end-diastole with pericardium removed; open triangle = end-systole with pericardium intact; closed triangle = end-systole with pericardium removed.

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... That is, the ratio of LV end-systolic pressure to its volume is changed when the contractile state changes, but the ratio is kept constant when the contractile state is constant. However, no one has examined whether or not the end-systolic pressure-volume relationship is altered by the pericardium. The present results indicate that there is a possibility that the pericardium modifies the end-systolic pressure-volume relationship during volume expansion. Therefore, we should take into consideration the effect of the pericardium on this index when we evaluate the contractile state of the volume-overloaded heart.

The mechanisms for the alteration in the LV end-
cardial restraint was probably underestimated in their study compared with that in situ.

Another possible mechanism for the alteration might be the attenuation of systolic interaction between both ventricles due to the removal of the pericardium. Because of an anatomic association established by the interventricular septum and the common muscle bundles surrounding both ventricles, one ventricular contraction would modify the performance of the opposite ventricle. Santamore et al.24 found that without the pericardium small RV volumes augmented LV isovolumetric developed pressure and large RV volumes reduced it. However, they concluded that the effect of RV volume on LV developed pressure might be small and probably insignificant under normal physiologic conditions. In contrast, Janicki and Weber7 found that when the RV end-diastolic pressure was increased the elevation of the LV developed isovolumetric pressure with the pericardium intact was larger than that with the pericardium removed at several levels of LV volume. Thus, they suggested that there was a tendency for systolic interaction between both ventricles to be less after the removal of the pericardium. More recently, Maughan et al.25 showed that the effect of RV filling on the LV systolic pressure-volume relationship in isolated cross-circulated ejecting hearts was little when the pericardium was removed and small when it was present over a normal range of volumes. As demonstrated by the last two studies, it is likely that during volume overload the LV end-systolic pressure was elevated to some extent as a result of the enhancement of systolic ventricular coupling produced by the presence of pericardium, but at control the systolic interaction was very small. The attenuation of the systolic interaction also accounts in part for the phenomenon of no change in the LV end-systolic pressure despite the further lengthening of the end-systolic segment after pericardectomy during volume loading.

Since the end-systolic pressure-volume relationship has been accepted as an index sensitive to a change in inotropic characteristics of the left ventricle but insensitive to changes in preload and afterload for the ventricle, it is also possible to interpret the rightward shift of the end-systolic pressure–segment length relationship after pericardectomy as a depression of the LV contractile state. Since the removal of the pericardium during volume loading caused acute cardiac distension, there was a possibility that cardiac reflexes were elicited by afferent stimuli from the mechanoreceptors in the atria or ventricles. However, the lack of changes in heart rate, LV systolic pressure, or peak positive dp/dt indicates that the contribution of the reflex mech-

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isms was minimal in the present study. In addition, myocardial damage due to acute overdistension did not occur because LV end-diastolic pressure was not overly elevated in the present study. For these reasons the alteration of the end-systolic pressure–segment length relationship after pericardectomy probably did not result from inotropic changes or myocardial dysfunction.

If the segment length in the LV free wall increased disproportionately to the increase in the LV volume, it would lead us to misinterpret the alteration of the end-systolic pressure–segment length relationship. However, we consider the geometric factor to be almost nil when the left ventricle is distended by blood infusion. This premise can be supported by the result of our supplemental experiment (figure 8). Another counterpart of geometric change is the displacement of the interventricular septum. When the pericardium is removed, encroachment of the right ventricle on the left ventricle may be so weak that distortion of the left ventricle is very small. On the other hand, when cardiac dilatation is limited by the presence of the pericardium, septal bulging toward the left ventricle may be more prominent. Nevertheless, we do not consider that the degree of the septal bulging gives an adequate explanation for the phenomenon at end-systole. This view is supported by Stool et al.26 who investigated dimensional changes in the left ventricle during a progressive increase in mean pulmonary arterial pressure with the pericardium loosely reapproximated. In their study, the end-systolic septal-lateral axis length of the left ventricle (determined by an endocardial marker technique) decreased only 3% from control even at a RV pressure loading of 45 mm Hg and the anterior-posterior axis did not change. Thus, the distortion of the left ventricle at end-systole caused by volume expansion is negligible.

As opposed to our findings that blood volume expansion caused an insignificant but small decrease in heart rate, a rapid infusion of Tyrode’s solution27 or saline28 into conscious dogs has been reported to produce a substantial increase in heart rate. Therefore, the ventricular size during volume-loading may be somewhat small in awake dogs, slightly reducing the pericardial effect. However, when lactated Ringer’s solution was infused at a rate slower than that in our study, the small increase in heart rate was not significant.29 Accordingly, we consider our results to be applicable to conscious, intact animals.

We demonstrated that the pericardium had a restrictive effect on the LV pressure–segment length relationship at end-systole as well as end-diastole in acute
volume overload, although its effect was little without volume loading. These results suggest that the LV end-systolic pressure-volume relationship is changed by the presence of the pericardium without reflecting a change in inotropic state. When the end-systolic pressure-volume relationship is used to evaluate the contractile state of the left ventricle in a clinical setting, it is suggested that the effect of the pericardium on this relationship should be taken into consideration. Of course, our experiments were done during acute volume loading. Therefore, the present results should be extrapolated with care to patients with heart disease because of the possible change in the characteristics of the pericardium induced by long-standing volume overload.

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