Optimal Right Ventricular Filling Pressures and the Role of Pericardial Constraint in Right Ventricular Infarction in Dogs

James E. Calvin, MD, FRCP(C)

**Background.** Previous studies have reported an important role for right ventricular function in the pathophysiology of the low cardiac output state that can accompany right ventricular infarction. Some studies have suggested that right ventricular distensibility impairs right ventricular filling and stroke output; others have demonstrated that the pericardium can mediate depressed left ventricular filling and stroke output.

**Methods and Results.** To determine the role of pericardial constraint and optimal volume loading in an experimental model of right ventricular wall infarction, six mongrel dogs were studied before and after right ventricular wall infarction and after volume loading. The pericardium was then opened in two phases. In the first phase, the pericardium was opened partially to allow the atria to distend freely, and in the second phase, the pericardium was opened completely. The animals were preinstrumented with two sets of piezoelectric crystals attached to the right ventricular free wall, one in the infarct and the other in the noninfarct territory. Left ventricular size was estimated by left ventricular crystals on the anterior wall of the left ventricle. Right ventricular and left ventricular Millar catheters were used to assess intracavitary pressure, and a flat balloon was used to assess intrapericardial pressure. Right ventricular infarction reduced cardiac output by 23% and stroke volume by 30%. End-diastolic segment length and transmural pressure of the left ventricle decreased. Volume loading restored cardiac output to baseline values and was mediated by a significant increase in end-diastolic length in the noninfarct territory. This was achieved by increasing right ventricular end-diastolic pressure from 9±2 to 16±3 mm Hg (p<0.01). Partial opening of the pericardium mediated significant increases in both end-diastolic segment lengths of the left ventricle and the noninfarct territory. Left ventricular end-diastolic pressure decreased slightly by 3 mm Hg (p=NS). Complete opening of the pericardium increased cardiac output and stroke volume and mediated a significant decrease in right and left ventricular end-diastolic pressures. Left ventricular transmural pressure and end-diastolic segment lengths of the left ventricle and the noninfarct territory increased. Left ventricular diastolic pressure–segment length relations were shifted upward by right ventricular infarction. A partial opening of the pericardium shifted this relation downward in all animals, and complete opening of the pericardium shifted the relation rightward and further downward.

**Conclusions.** Cardiac output is restored to baseline values by volume loading sufficient to increase the right ventricular diastolic pressure to 16±3 mm Hg. Evidence of pericardial constraint was observed and appears to be mediated by an atrioventricular interaction in addition to the direct ventricular interaction. (Circulation 1991;84:852–861)

Although early studies of right ventricular function suggested that the right ventricle’s contribution to circulatory homeostasis was minimal, more recent reports have highlighted an important role for right ventricular function in the pathophysiology of the low cardiac output state that can accompany right ventricular infarction. However, depressed right ventricular contractility is not the sole mechanism for this derangement. Some investigators have suggested that reduced right ventricular distensibility impairs right ventricular filling and...
stroke output. Others have established that the pericardium can mediate depressed left ventricular filling and stroke output; these investigators hypothesized that the pericardium mediates a direct interaction between both ventricles whereby a dilated right ventricle engages the pericardium and encroaches upon space available to the left ventricle. This reduces left ventricular distensibility, filling volume, and cardiac output (by the Starling mechanism). Under such conditions, experimental pericardiectomy has increased left ventricular filling volume and output. Also, right ventricular distensibility is probably improved by pericardiectomy.

It is conceivable that right atrial distension during right ventricular infarction could also engage the pericardium and influence left ventricular distensibility. Indeed, Linderer and co-workers demonstrated this possibility in a canine model. To compound matters, volume loading is recognized as an important treatment in right ventricular infarction. However, excessive right ventricular or atrial distension secondary to volume loading may offset any benefit that could be attributed to the Frank-Starling mechanism by engaging the pericardium and altering left ventricular distensibility.

The purpose of this study was twofold. First, I sought in a closed pericardium, open-chest canine model of right ventricular infarction to determine the optimal right ventricular filling pressure for the restoration of the cardiac output to baseline values. This information would provide a better guide for volume loading patients so that the net effects of the Frank-Starling mechanism and pericardial constraint would be a benefit.

Second, I sought to determine whether pericardial constraint of the right atrium could contribute to depressed left ventricular distensibility during right ventricular infarction. I attempted to make this determination by opening the pericardium partially to allow release of the atria from pericardial constraint. By demonstrating a downward shift of the left ventricular pressure–segment length relation after this partial release, I attempted to confirm this effect and assess its magnitude. By opening the pericardia completely, the relative roles of right atrial and right ventricular distension upon left ventricular distensibility and filling could be assessed.

Methods

Six mongrel dogs were studied after premedication with benadryl (4 mg/kg) and morphine (2 mg/kg) and anesthetization with a combination of chloralose (50 mg/kg i.v.) and morphine (2 mg/kg) as previously described. The animals were ventilated with a volume-cycled animal respirator (model 607, Harvard Apparatus, South Natick, Mass.) using 100% oxygen, an initial respiratory rate of 18 breaths/min, and a tidal volume of 350 ml. The arterial oxygen saturation was always maintained above 85%, and the PCO₂ was adjusted by increasing the respirator rate to keep it in a physiological range. If mild metabolic acidosis was observed, bicarbonate was administered to maintain the pH above 7.35.

The animal’s heart was exposed through a median sternotomy. The pericardium was partially opened through a vertical incision extending from the great vessels to 1 cm below the atrioventricular groove. The heart was then removed from the remaining intact pericardial cavity for instrumentation. A 7F micropipette pressure transducer (model PC 480, Millar Instruments Co., Houston, Tex.) was advanced into the right and left ventricles from cutdowns on the right external jugular vein and carotid artery, respectively. These pressure transducers were attached to a solid-state bridge amplifier (model CD19, Validyne, Northridge, Calif.). The fluid-filled lumina of these catheters were attached to a transducer (Statham, Oxnard, Calif.). A 7F Swan-Ganz catheter was positioned in the pulmonary artery to determine cardiac output with the fluid-filled lumina attached to a Statham pressure transducer.

To assess right ventricular geometry, two sets of crystals were placed on the right ventricular surface (Figure 1). One pair was oriented along the septal free wall plane with one crystal embedded in the right ventricular myocardium near the septum at the level of the origin of the first diagonal branch of the left anterior descending coronary artery and the second crystal 1.5 cm away. This crystal measured regional wall motion of the outflow region (noninfarct territory). The second pair was imbedded in the inflow portion of the free wall of the right ventricle (infarct territory) midway between the acute margin of the right ventricle and the apex but oriented along the septal free wall plane (a principal axis of contraction). Left ventricular size was assessed by a pair of crystals directed along the septal free wall plane of the left ventricle with one of the pair imbedded near the septum below the origin of the first diagonal and its mate 1.5 cm away. The heart was returned to the pericardial sac, and a flat fluid-filled silicon balloon

FIGURE 1. Diagram of surgical instrumentation of experimental preparation. Pairs of dots correspond to segment length crystal placement. R.V.P.T., right ventricular pressure transducer.
approximately 3 by 3 cm was positioned over the anterior surface of the heart across the interventricular septum. The pericardial incision was loosely resutured to minimize any tendency to reduce pericardial surface area. According to our methods, this produces minimal artifact. We determined this in three pilot experiments in which loose resuturing of the pericardial incision increased intrapericardial pressure by only 1 mm Hg.

Correct positioning of crystals was determined by three methods. First, accurate triggering by the sonomicrometer was ensured, or the involved crystal was realigned. Second, systolic shortening and diastolic lengthening of the crystals was assessed from the computer monitor. Third, the crystal position was assessed during postmortem examination to ensure an intramyocardial placement.

Ventricular pressure (measured from the Millar pressure transducers) and segment length data were digitized simultaneously with an electrocardiographic signal directly at a rate of 200 samples/sec onto an Apple IIe with an eight-channel analog-to-digital converter (A 2 devices, Alameda, Calif.). Ventricular and aortic pulmonary artery pressures measured with fluid-filled lumina were recorded simultaneously with an electrocardiographic signal on a thermal array recorder (model TA-600, Gould, Cleveland, Ohio). Pressures from the pericardial balloon were also measured with a Statham transducer and recorded on the paper recorder.

Hemodynamic Data to Be Analyzed

Routine hemodynamic variables were measured at each phase. These included mean end-diastolic segment lengths; peak systolic, end-systolic, and end-diastolic pressures; and cardiac output. End diastole was measured at the time of the onset of the R wave on the electrocardiogram. Cardiac output was measured by thermodilution. Four determinations were taken during each condition and averaged. Left ventricular transmural end-diastolic pressure was calculated by subtraction of the pericardial pressure measured by the balloon from the intracavitary left ventricular pressure at the onset of the R wave from the electrocardiogram (end diastole).

All reported end-diastolic lengths were normalized by the following formula: Length is equal to (observed length divided by observed length at baseline) multiplied by 10 mm. This results in all baseline lengths being equal to 10 mm and reduces interdog variability.

Production of a Right Ventricular Infarction

An isolated right ventricular infarction was produced using the method previously described by Goldstein et al.\textsuperscript{9} Briefly, after completing the baseline measurements in each experiment, the right coronary artery was dissected through a small pericardial incision from midway between its origin and the acute right ventricular margin. A ligature was placed around the right coronary artery at this point to prevent mercury reflux. Then, a Teflon catheter was inserted into the proximal right coronary artery. Mercury was then injected in a dose of 0.15 ml over several seconds. This method produces an isolated right ventricular infarct (largely by impairing collateral filling of the distal right coronary artery bed) involving the inflow portion of the right ventricular free wall and the posterior right ventricular wall. It spares the right ventricular outflow tract because this region is supplied by a right ventricular branch arising proximally to the ligature. After a stabilization period of 10 minutes, a second set of measurements was obtained.

Protocol

All hemodynamic measurements were made at end expiration. Two sets of baseline measurements (B1 and B2) were taken 10 minutes apart after the animal had been allowed to stabilize for 30 minutes after the surgical instrumentation. The measurements were then repeated after the right ventricular wall infarction was produced. Dextran was then infused in sufficient amounts to restore the cardiac output to within 10% of the baseline values, and repeated measurements were then obtained. At this point, the pericardium was opened partially along its superior aspect using a horizontal incision at the level of the atrioventricular groove and resecting the superior portion of the pericardium to allow the atria to freely distend. Repeated measurements were again obtained at end expiration. At this point, the remainder of the pericardium was opened to allow free distension of both ventricles. Repeated measurements were again obtained.

Statistical Analysis

All variables were analyzed with one-way analysis of variance with repeated measures. Where significant differences were observed, a post-hoc analysis with a Newman-Keuls test was then performed.

Results

Hemodynamic Findings

Hemodynamic results are reported in Table 1 expressed as mean±SD. In general, hemodynamic measurements did not vary significantly between both baseline requirements.

Effects of right ventricular wall infarction. The creation of a right ventricular infarction reduced cardiac output and stroke volume by approximately 30% (Table 1). Heart rate increased but not significantly, largely because of significant heart rate increases (>30 beats/min) in two animals. Left and right ventricular end-diastolic pressures (Figure 2) did not change. Right ventricular infarction increased the end-diastolic length of the infarct territory in five of six animals (overall mean change was not significant). In the animal in which the end-diastolic length actually decreased, a large increase in heart rate (>30 beats/min) was noted. Right ventricular infarction did not change the end-diastolic length of the
TABLE 1. Hemodynamic Variables (n=6)

<table>
<thead>
<tr>
<th>CO (l/min)</th>
<th>SV (ml)</th>
<th>HR</th>
<th>LVP_d (mm Hg)</th>
<th>LVP_a (mm Hg)</th>
<th>L_aV (mm)</th>
<th>AP (mm Hg)</th>
<th>RVP_d (mm Hg)</th>
<th>L_4 (mm)</th>
<th>L_NI (mm)</th>
<th>PP (mm Hg)</th>
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</thead>
<tbody>
<tr>
<td>B1 3.1±0.4</td>
<td>33±8</td>
<td>98±20 12±2</td>
<td>5±2</td>
<td>10</td>
<td>110±11</td>
<td>7±1</td>
<td>10</td>
<td>10</td>
<td>7±1</td>
<td></td>
</tr>
<tr>
<td>B2 3.3±0.6</td>
<td>33±9</td>
<td>101±16 12±2</td>
<td>5±2</td>
<td>10.1±0.2</td>
<td>110±7</td>
<td>8±1</td>
<td>10.0±0.2</td>
<td>10.0±0.2</td>
<td>7±1</td>
<td></td>
</tr>
<tr>
<td>RVI 2.4±0.5†</td>
<td>22±11</td>
<td>122±29 12±2</td>
<td>2±3</td>
<td>9.3±0.5†</td>
<td>100±10</td>
<td>9±2</td>
<td>10.3±1.7</td>
<td>10.0±1.4</td>
<td>10±2</td>
<td></td>
</tr>
<tr>
<td>VL 3.1±0.3‡</td>
<td>27±5</td>
<td>115±15 21±3‡</td>
<td>4±2</td>
<td>9.5±0.5†</td>
<td>101±19</td>
<td>16±3‡</td>
<td>10.3±1.7</td>
<td>10.7±1.4‡</td>
<td>16±3‡</td>
<td></td>
</tr>
<tr>
<td>OP1 3.3±0.5‡</td>
<td>327‡</td>
<td>107±14 19±3‡</td>
<td>6±2‡</td>
<td>10.0±0.6‡</td>
<td>105±10</td>
<td>14±2‡</td>
<td>10.5±1.3</td>
<td>13.0±2.3‡†</td>
<td>15±4‡</td>
<td></td>
</tr>
<tr>
<td>OP2 4.3±0.5‡</td>
<td>41±6‡</td>
<td>106±7</td>
<td>16±3‡</td>
<td>15±4‡</td>
<td>11.7±0.5‡</td>
<td>116±13</td>
<td>10±3</td>
<td>11.4±0.8</td>
<td>13.2±2.1‡ †</td>
<td>0±0‡</td>
</tr>
</tbody>
</table>

CO, cardiac output; SV, stroke volume; HR, heart rate; LVP_d, left ventricular end-diastolic pressure; LVP_a, left ventricular end-diastolic transmural pressure; L_aV, left ventricular end-diastolic segment length; AP, mean arterial pressure; RVP_d, right ventricular end-diastolic pressure; L_4, end-diastolic segment length of infarct territory; L_NI, end-diastolic segment length of noninfarct territory; PP, pericardial pressure at end-diastole; B1, first baseline; B2, second baseline; RVI, right ventricular infarction; VL, volume loading; OP1, partial opening of pericardium (release of atria); OP2, complete opening of pericardium.

*p<0.05 vs B1, †p<0.05 vs B2, ‡p<0.05 vs RVI, §p<0.05 vs OP2, ††p<0.05 vs OP1, ‡‡p<0.05 vs VL.

noninfarct territory of the right ventricle. Overall, it reduced left ventricular end-diastolic segment length by 7% (Figure 3).

Effects of volume loading. On average, 500±185 ml dextran was used to restore the cardiac output to baseline values. The increase in cardiac output was mediated by a 7% increase in end-diastolic length of the noninfarct territory because neither the infarct territory nor left ventricular end-diastolic segment length increased significantly (Figure 3). Volume loading increased left and right ventricular end-diastolic pressures but not the left ventricular transmural pressure (Table 1). Cardiac output was restored to baseline values at a right ventricular end-diastolic pressure of 16±3 mm Hg.

Effects of opening the pericardium. Immediately after partially opening the pericardium, stroke volume, left ventricular end-diastolic transmural pressure, and left ventricular end-diastolic segment length increased. Cardiac output increased insignificantly as heart rate slightly decreased. Also, an increase in end-diastolic length of the noninfarct territory was observed. Left ventricular end-diastolic pressure decreased slightly, and pericardial pressure decreased slightly by 3 mm Hg (p=NS). After completely opening the pericardium, significant increases occurred in cardiac output, stroke volume, left ventricular end-diastolic segment length, and left ventricular end-diastolic transmural pressure. Right ventricular end-diastolic pressure decreased significantly. Left ventricular end-diastolic pressure decreased by 3 mm Hg (p=NS).

Influences of pericardial constraint. The creation of a right ventricular wall infarction alone shifted the left ventricular diastolic pressure segment length relation significantly in four of the six animals (Figure 4). Smaller upward shifts were noted in the other two animals.

Volume loading after the production of a right ventricular wall infarction shifted the left ventricular diastolic pressure–segment length relation further upward in all animals (Figure 5). Opening the pericardium partially to allow the atria to freely distend shifted this relation downward and to the right. Complete opening of the pericardium resulted in a further downward and rightward shift of the relation. However, when left ventricular end-diastolic transmural pressure was plotted against left ventricular end-diastolic length (Figure 6), the shifts observed in Figures 4 and 5 are less apparent, indicating these changes were related to pericardial pressure.

END-DIASTOLIC PRESSURES

![END-DIASTOLIC PRESSURES](image)

**FIGURE 2.** Plot showing that right ventricular infarction (RVI) had little influence on end-diastolic pressures by itself. VL increased all pressures. OP2 decreased left and right ventricular intracavitary pressures but increased left ventricular transmural pressure (LVPTM). B1, B2, baseline measurements; VL, measurements after dextran had been infused to restore cardiac output to within 10% of the baseline measurements; OP1, OP2, measurements after pericardium had been partially and completely opened, respectively; LVP, left ventricular pressure; RVP, right ventricular pressure.
Discussion

Right ventricular infarction is an important and treatable cause of cardiogenic shock. A complete understanding of its pathophysiology should lead to better treatment strategies. Because volume loading has been an accepted therapy,2,11 I believed it important that the first objective in this study be a determination of the level of right ventricular filling pressure at which cardiac output could be restored to baseline levels in a closed-pericardium canine model of right ventricular infarction. Indeed, I found that the baseline cardiac output was restored by volume loading sufficient to raise the right ventricular filling pressure from a baseline value of 7±1 to 16±3 mm Hg.

The second objective was to determine whether releasing pericardial constraint upon the right ventricular infarction and volume loading could affect left ventricular distensibility through a direct interaction mediated by the pericardium. It was confirmed that pericardial constraint of the atria did, indeed, impair left ventricular filling and distensibility but not sufficiently enough to influence cardiac output. Last, it was confirmed that a direct ventricular interaction mediated by the pericardium was an important mechanism in reducing left ventricular distensibility and cardiac output.

Although volume loading has been commonly used as a mainstay in the treatment of low cardiac output states accompanying right ventricular infarction, an optimal range of filling pressures for volume loading has not been identified as has been for left ventricular infarction.14 It is conceivable that optimal levels of right atrial pressure or right ventricular filling pressure may be different than those proposed for pulmonary capillary wedge pressure in left ventricular infarction.14 Furthermore, controversy now exists about the usefulness of volume loading in right ventricular infarction. Recently, Dell'Italia and co-workers15 reported in a group of 10 patients with right ventricular infarction that volume loading sufficient to raise right atrial pressure from 11±2 to 15±2 mm Hg did not increase cardiac output. However, in the same canine model of right ventricular infarction as used in this study, Goldstein et al11 reported an increase in cardiac output when right ventricular end-diastolic pressure was increased from 6.3±1.8 to 9.6±7.4 mm Hg. These results and ours are not inconsistent with each other because the sensitivity of the right ventricular Frank-Starling mechanism.
FIGURE 5. Plots of left ventricular (LV) diastolic pressure–segment length relations in all experiments. Volume loading after right ventricular (RV) infarction shifted the relation further upward in all experiments. Partial opening of the pericardium (OP1) shifted it downward, and complete opening of the pericardium (OP2) returned it to baseline levels of pressure although lengths increased.

mechanism likely changes at different operating pre-load points. For instance, in the study by Goldstein et al and the present study, right ventricular filling pressure after right ventricular infarction but before volume loading was relatively low compared with that in Dell'Italia et al's study. Hence, there was a greater likelihood of right ventricular recruitable stroke volume when the constraining effects of the pericardium were not completely engaged. Indeed, these constraining effects were obvious in the present study because cardiac output occasionally decreased when volume loading was excessive but then increased once the pericardium was completely opened.

Another important reason that volume loading is not always successful in patients with right ventricular infarction is because of concomitant left ventricular infarction. The associated left ventricular infarction not only depresses left ventricular contractility and directly reduces left ventricular compliance (making it more difficult to fill the left ventricle) but also increases right ventricular afterload, which further impairs right ventricular systolic function. The present model produces primarily an isolated right ventricular infarction. Hence, the range of right ventricular filling pressures associated with restoring cardiac output to baseline values may be different from clinical practice. One may anticipate that with an associated left ventricular infarction, right ventricular systolic function would be worse (because of elevated right ventricular afterload); left ventricular diastolic function would be worse, and filling pressures would necessarily be higher for an adequate cardiac output.

Opening the pericardium partially to release any constraining influence upon the atria increased left ventricular end-diastolic segment length and left ventricular end-diastolic transmural pressure without a significant effect on intracavitary left ventricular end-diastolic pressure. Furthermore, left ventricular diastolic pressure segment lengths shifted downward. All of these observations suggest that pericardial constraint of the atria influenced left ventricular distensibility by a direct interaction. This has been suggested by others. Linderer et al demonstrated in a group of nine anesthetized open-chest, closed-pericardium dogs that loss of atrial systole (atrioventricular asynchrony) shifted the left ventricular end-diastolic pressure diameter relation upward largely by increasing intrapericardial pressure (because the atria remained full at end diastole). Therefore, evidence already exists for an atrioventricular interaction mediated by the pericardium. Maruyama et al carefully looked at the mechanical interaction between all four chambers in excised canine hearts. Significant atrial ventricular interactions were ob-
served and were of similar magnitude to those observed in this study. Last, in a group of six patients with atrial septal defect and elevated left ventricular end-diastolic pressure, normalized left ventricular compliance was less than that of a control group, again suggesting the possibility of an atrioventricular interaction. Results of the present study clearly demonstrate the existence of such an interaction, but this interaction was not of sufficient magnitude to influence cardiac output.

Complete opening of the pericardium increased left ventricular end-diastolic segment length and transmural pressure, reduced intracavitary left ventricular end-diastolic pressure, and shifted the left ventricular diastolic pressure–segment length relations down and to the right in all animals. This was of sufficient magnitude to increase cardiac output, thereby confirming the significant role that the pericardium plays in the pathophysiology of the low cardiac output state accompanying right ventricular infarction. These results confirm the importance of the direct ventricular interaction mediated by the pericardium.

One limitation of this study was the use of a single pair of left ventricular segment length crystals. Goto et al. recently reported in a canine model of acute right ventricular pressure overload that left ventricular wall stresses are nonuniform. However, the anterior location of the segment length crystals used in this study was found by Goto’s group to be the most sensitive to a decrease in left ventricular preload produced by acute pulmonary artery constriction. Other locations (free wall and posterior wall) also showed decreases in left ventricular dimensions during right ventricular pressure overload although not to the same magnitude. A shift upward in the normalized anterior end-diastolic pressure–segment length relation was also reported. This was not observed in the other regions.
In Goto's study, the nonuniformity was less when the pericardium was opened. This observation, and those of other studies, which determined that intrapericardial pressure did not increase during pulmonary artery constriction or actually decrease over the left ventricular surface, was attributed to the pericardium enhancing leftward septal shift. However, this abnormality was clearly demonstrated to be a result of pericardial constraint. Septal shift may contribute to the upward shift in the left ventricular diastolic pressure–segment length relation observed with both the production of a right ventricular infarction and volume loading in the present study. However, the present results show little evidence for either a decrease or reversal of transseptal pressure during a right ventricular infarction or after volume loading (Figure 2), which is usually observed when the septum has shifted leftward. Furthermore, the shifts in the left ventricular diastolic pressure–segment length relation appear to be diminished substantially when left ventricular transmural pressure (as defined in "Methods") is used.

Our results are also similar to another report in which the effect of right ventricular ischemia on left ventricular three-dimensional geometry and end-diastolic pressure–volume relations were studied before and after pericardectomy. In this study, closed-pericardial animals had leftward and upward shifts in left ventricular end-diastolic pressure–volume relations after transient right coronary occlusion and volume loading, the extents of which were reduced after pericardectomy when right coronary occlusion was repeated. The major contributor to change in left ventricular volume and in the left ventricular pressure–volume relation appeared to be secondary to changes in the septal–lateral wall dimensions. Indeed, right ventricular infarction independent of volume loading was shown to reduce left ventricular distensibility in most animals although these effects appeared to be magnified by volume loading.

The use of an intrapericardial balloon to measure intrapericardial pressure is well supported by previous studies. In the present study, the method of resurfacing the pericardial incision was shown to increase the pericardial pressure by 1 mm Hg (in three separate experiments). Furthermore, the resting left and right ventricular end-diastolic pressures were 12±2 and 8±1 mm Hg, respectively. These observations are identical to the results of Stokland et al22 for an intact pericardial preparation, and these represent minimal reduction in pericardial surface area.

The location of the pericardial balloon along the anterior surface was chosen to minimize discrepancies in pericardial pressure measured from either the right or left ventricular free wall. Pericardial pressure measured from this location accurately reflects pressure measured over the left ventricular lateral wall and can be used to estimate left ventricular transmural pressure in this model (see Appendix).

The results show that intrapericardial pressure did not increase significantly after right ventricular infarction. This probably occurred because of either the small number of experiments or the fact that the balloon position underestimated intrapericardial pressure over the right ventricular free wall. The results also show that right ventricular end-diastolic pressure did not decrease to 0 mm Hg after pericardectomy. This observation is in contrast to that of Tyberg et al23 and Smiseth et al24 who demonstrated that right atrial pressure was equivalent to pericardial pressure. However, it is consistent with that in the study by Slinker et al.25 The difference between the results of this study and others24,25 may be a result of abnormal right ventricular distensibility secondary to the right ventricular infarction (i.e., right-sided pressure is influenced by diastolic properties as well as distension).

In summary, a canine model of right ventricular infarction was used to determine an optimal right ventricular filling pressure to guide volume loading; this should help identify patients who may respond to
volume and who should be considered for inotropic therapy. Also, an atrial contribution to reduced left ventricular distensibility during right ventricular infarction was identified. Last, an important role of pericardial constraint upon left ventricular filling and output was identified.

**Appendix**

*Validation of an Anterior Positioned Pericardial Balloon to Estimate Left Ventricular End-Diastolic Transmural Pressure*

Smiseth et al. previously determined that nonuniformity of pericardial surface pressure measured by a flat pericardial balloon exists in dogs. Specifically, significant differences in pericardial pressure measured over the right and left ventricular free walls were found during pulmonary and aortic constrictions with pulmonary artery constrictions producing greater increases in pericardial pressure measured over the right ventricular free wall and aortic constriction producing greater increases over the left ventricular free wall. However, volume loading produced proportional increases in pericardial pressure from these sites with the slope of the calculated regression similar to unity.

In this experiment, pericardial pressure was measured with a flat balloon positioned over the anterior ventricular surface straddling the intraventricular septum.

To determine whether an anterior-positioned balloon was suitable for measurement of left ventricular end-diastolic transmural pressure, end-diastolic pericardial pressure was measured from both an anterior-positioned balloon and one positioned over the left ventricular free wall similar to the location described by Smiseth et al. A pericardial incision was fashioned as described in “Methods” and then loosely resutured after the balloons were positioned. Baseline measurements were obtained, and then a right ventricular infarction was produced (see “Methods”). Repeat measurements were obtained. Volume loading with dextran was performed in 100-ml aliquots until pericardial pressure exceeded 15 mm Hg, and then, serial phlebotomies were performed. Repeat measurements were obtained at each stage.

The results are described in Table 2. The relation between pericardial pressure measured over the left ventricular lateral surface and the anterior ventricular surface was excellent with a slope of 0.95 (r=0.95, p<0.001) and a y intercept of −0.48 in dog 1 and a slope of 0.83 (r=0.97, p<0.0001) and a y intercept of 1.06 in dog 2. The slope in each case is within the 95% confidence interval for the data. Combined results are shown in Figure 7.

These results are similar to those of volume loading as demonstrated by Smiseth et al., suggesting that any disproportionate right ventricular volume changes produced by right ventricular infarction are not of the same magnitude as a pulmonary artery constriction. Inasmuch as volume loading was the major intervention of this study, these results are predictable.

**References**

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