Right heart pressure does not equal pericardial pressure in the potassium chloride–arrested canine heart in situ

BRYAN K. SLINKER, D.V.M., PH.D., ROY V. DITCHEY, M.D., STEPHEN P. BELL, B.A., AND MARTIN M. LEWINTER, M.D.

ABSTRACT Recently proposed concepts of pericardial surface pressure, as opposed to liquid pressure, have advanced our understanding of the relationship between pericardial and heart chamber pressures. However, the subsequent suggestion that right heart intracavitary pressure equals, or nearly equals, pericardial surface pressure is not strictly consistent with the physiology of pericardial constraint. If right heart pressure equals pericardial surface pressure, then transmural right heart pressure equals zero. Because of the difficulty in measuring pericardial pressure directly in the beating heart we designed an experiment in the recently arrested canine heart in situ to measure pericardial pressure indirectly and to test the hypothesis that right heart transmural pressure is zero under reasonably physiologic, static equilibrium conditions. According to a static equilibrium analysis of the pressures acting across the walls of the heart, at a given volume the change in right heart pressure caused by removing the pericardium is equal to the pericardial pressure when the pericardium is intact. We found that this drop in pressure caused by pericardiectomy did not equal right heart pressure and therefore that right heart transmural pressure does not equal zero.


RECENTLY PROPOSED CONCEPTS of pericardial surface pressure, as opposed to liquid pressure, have enhanced our understanding of the influence of the pericardium on ventricular function and the relationship between intrapericardial and cardiac chamber pressures.1, 2 This work established that open-ended, fluid-filled catheters cannot be used to measure pericardial surface pressure. Based on these concepts and direct measurement of pericardial surface pressure with flat balloons, it has been variously suggested that right heart pressure equals3, 4 or nearly equals intrapericardial surface pressure.5, 6

However, if right heart pressure equals the pericardial surface pressure, then the right heart transmural pressure must be zero. From results presented in several earlier reports, one can infer that this is not the case; that is, there seems to be a nonzero transmural right heart pressure.5–10 Unfortunately, none of these experiments was designed specifically to test the equality of right heart and pericardial pressures and all of them can be faulted in some way when interpreted in this manner. Nonetheless, this discrepancy suggests the need for a well-controlled experiment designed specifically to test the hypothesis that right heart and pericardial surface pressures are equal.

One possible explanation for this discrepancy is the difficulty in measuring pericardial surface pressure directly in situ. Because the pressures measured are relatively small and the beating heart is moving, there could be considerable random error in direct measurements of pericardial pressure. Another possible explanation is that the flat balloons used to measure pericardial pressure could distort the pericardium and lead to systematic errors in direct measurements of pericardial pressure.

We designed this experiment to test the hypothesis that right heart pressure was equal to pericardial surface pressure by measuring right and left heart pressure-volume relationships in the recently arrested canine heart in situ with and then without the pericardium. Compared with data obtained in the beating heart, these data are relatively noise free, reproducible, and
precise. In addition to equal volumes in the right and left hearts, this analysis also assumes an equal total cardiac volume before and after pericardiectomy. According to a static equilibrium analysis of the pressures acting across the wall of the heart, at any given chamber volume the drop in chamber pressure after pericardiectomy equals the pericardial pressure present before pericardiectomy. If it is true that the right heart transmural pressure is zero, then the right heart pressure-volume curve after pericardiectomy should be flat (over the ranges of volume that have been studied before pericardiectomy in vivo), with pressure always equal to zero; that is, the pressure drop caused by pericardiectomy at any right heart volume should be equal to the right heart chamber pressure at that volume when the pericardium was intact. We found that this was not the case.

We also used this preparation to test a speculation arising from a static equilibrium analysis of diastolic chamber and pericardial pressures; i.e., that the global pericardial surface pressures overlaying both sides of the heart are equal.2 We found that this was the case.

**Methods**

**Preparation.** Dogs of both sexes (average weight 23 ± 3 kg [SD]) were anesthetized to effect with sodium pentobarbital (usually 25 mg/kg) after preanesthesia with 1 mg/kg morphine. The dogs were ventilated with room air at 12 breaths/minute and 15 ml/kg tidal volume with a Harvard respirator. Entry to the chest was made via a median sternotomy and bilateral fifth intercostal space thoracotomies, and the chest wall was retracted widely. The pericardium was cut on a 5 to 6 cm line extending from the mid left atrium to a point just cranial to the origin of the aorta. Double silk or umbilical tape ligatures were preplaced (but not tied) around the azygos vein, superior and inferior venae cavae, aortic root, main pulmonary artery, right coronary artery, left anterior descending coronary artery, circumflex coronary artery, and both pulmonary hilum. A large-bore cannula with multiple side holes and a separate pressure lumen (No. 20F left ventricular vent) was placed in the right heart via the superior vena cava. A smaller cannula (No. 14F oxygen catheter) and a separate catheter (1.35 mm id), both with multiple side holes, were placed into the left heart via pulmonary veins. All catheters were positioned so that side holes were present on both sides of the atrioventricular valves. Finally, the pericardial fat was gently removed by blunt dissection.

When these advance preparations were completed the heart was arrested by injecting 40 ml of saturated KCl into the left ventricle as the aorta was ligated, and the respirator was stopped. All preplaced ligatures were tied, and the heart was flushed repeatedly with normal saline (30° to 35°C), checked for leaks from the chambers, and finally emptied by suction. One or two stitches were used to close the pericardial incision loosely to prevent herniation of the left atrium. The large cannulas in each side of the heart were connected to a modified Edco syringe pump capable of delivering up to 135 ml into each side of the heart. The pressure lumina were connected to Statham P23Id transducers zeroed to a reference level at the base of the heart. Pressures during infusion of volume were recorded at slow chart speed on a Hewlett-Packard recorder.

**Protocol.** With the chambers empty, saline was infused slowly at equal rates into both sides of the heart until right heart pressure equaled 0 mm Hg. This volume, hereafter denoted right heart V₀, was measured directly from the syringe pump. (Left heart V₀ was invariably larger than right heart V₀ and was determined from the zero crossing of left heart pressure as more volume was infused.) Then, beginning at right heart V₀, volume was infused at a rate of 99 ml/min into both sides of the heart until left heart pressure reached 25 mm Hg. The pump was then reversed to empty the heart at the same rate. If the total volume removed from each side of the heart was less than the starting volume, we made one attempt to stop the leak and continued. If a leak was still present we abandoned the experiment.

The protocol was repeated two or three times for each condition of pericardiectomy intact and pericardium resected widely. We waited 1 min between each infusion/withdrawal run and rechecked zero pressure levels during this waiting period. We attempted to finish data collection within 40 minutes of KCl arrest to avoid the confounding effects of rigor mortis.11,13 We collected data in 16 dogs but, because leaks or inability to collect data within the 40 min period, report data from eight dogs. The time for data collection in these eight dogs ranged from 31 to 38 min (mean 34 ± 2.5 [SD]).

**Data analysis.** Data for analysis were selected by first identifying infusion runs for both pre- and postpericardiectomy pressure-time curves graphically. Second, we analyzed the relative contribution of the pericardium to chamber pressures in both sides of the heart by selecting prepericardiectomy chamber pressures of interest: 1.3, 5, 8, 11, and 15 mm Hg in the right heart and 2.5, 8, 11, 15, 20, and 25 mm Hg in the left heart. We then computed the pressure drop caused by pericardiectomy at the volumes obtained at these prepericardiectomy pressures. The relative contribution of the pericardium to chamber pressure was calculated as the ratio of the pressure drop to the prepericardiectomy pressure. Third, we analyzed the absolute contribution of the pericardium to left and right heart pressure by computing the pressure drop caused by pericardiectomy at total cardiac volumes of 25, 50, 75, 100, 125, 150, 175, 200, and 225 ml.

All data are summarized as mean ± SD. Details of statistical hypothesis tests are described as necessary in the Results.

**Results**

Typical pressure-volume curves for both sides of the heart and for both pre- and postpericardiectomy experimental conditions are shown in figure 1.

In general, pericardiectomy did not change right heart V₀ (.2 > p > .1 by paired t test) and produced small changes in left heart V₀ (p < .005 by paired t test; table 1).

**Relative contribution of the pericardium to chamber pressure.** Removal of the pericardium resulted in nonparallel downward and rightward shifts of the pressure-volume curves of both sides of the heart (figure 1 and tables 2 and 3). The average pericardial contribu-
tion to right heart pressure ranged from a low of 20% at a prepericardiectomy pressure of 1 mm Hg to a high of 67% at a prepericardiectomy pressure of 15 mm Hg. In contrast, the average pericardial contribution to left heart pressure was smaller, ranging from about 22% for prepericardiectomy pressures of 5 to 11 mm Hg to a high of 37% at a prepericardiectomy pressure of 25 mm Hg. In no case was the right ventricular pressure-volume curve flat and equal to 0 mm Hg.

Absolute contribution of the pericardium to chamber pressures in both sides of the heart. At selected total cardiac volumes, the absolute drop in pressure caused by pericardiectomy was not different for the two sides of the heart (p > .50 by two-way analysis of variance; see tables 4 and 5) and the relationship between change in left heart pressure and change in right heart pressure was linear with a slope not different from 1 (.5 > p > .2) and an intercept not different from 0 (.5 > p > .2; figure 2).

Discussion

These data do not support the hypothesis that right heart transmural pressure is zero. Although the pericardium makes a substantial contribution to chamber pressure in the right heart, even at low right and left heart volumes, there is still a right heart transmural pressure when the pericardium is removed. In fact, the contribution of the pericardium to chamber pressure dominates only at right heart pressures of greater than 10 mm Hg.

### TABLE 2

Absolute and relative decrease in right heart pressure caused by pericardiectomy at selected prepericardiectomy pressures

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>Decrease in right heart pressure (mm Hg) at prepericardiectomy pressure (mm Hg) of:</th>
<th>1</th>
<th>3</th>
<th>5</th>
<th>8</th>
<th>11</th>
<th>15</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>0</td>
<td>1.0</td>
<td>2.0</td>
<td>3.8</td>
<td>6.2</td>
<td>9.8</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>0.5</td>
<td>1.8</td>
<td>4.0</td>
<td>6.8</td>
<td>11.2</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>0.6</td>
<td>1.4</td>
<td>3.1</td>
<td>4.9</td>
<td>A</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>0</td>
<td>1.0</td>
<td>2.2</td>
<td>4.6</td>
<td>7.2</td>
<td>10.9</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>0.4</td>
<td>0.8</td>
<td>1.9</td>
<td>3.9</td>
<td>6.1</td>
<td>9.6</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>0.6</td>
<td>1.6</td>
<td>2.5</td>
<td>4.2</td>
<td>6.8</td>
<td>10.0</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>0</td>
<td>0.4</td>
<td>1.0</td>
<td>3.0</td>
<td>5.2</td>
<td>9.0</td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>0.2</td>
<td>0.4</td>
<td>1.4</td>
<td>2.8</td>
<td>5.0</td>
<td>A</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.2</td>
<td>0.4</td>
<td>1.8</td>
<td>3.7</td>
<td>6.0</td>
<td>10.1</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>0.2</td>
<td>0.4</td>
<td>0.5</td>
<td>0.6</td>
<td>0.9</td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>% decrease</td>
<td>20</td>
<td>27</td>
<td>36</td>
<td>46</td>
<td>55</td>
<td>67</td>
<td></td>
</tr>
</tbody>
</table>

*Data not obtained at that prepericardiectomy pressure.

### TABLE 1

Right and left heart chamber volumes at zero pressure before and after pericardiectomy

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>Right heart V_e (ml)</th>
<th>Left heart V_e (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>3</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
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<tr>
<td>6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>8</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>21</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>23</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

\( .2 > p > .1 \) \( p < .005 \)

P values are from paired t test.
Tyberg et al.\textsuperscript{4} reported good linear correlations between right heart pressure and pericardial pressure measured with a flat balloon. However, close examination of those results reveals that, in spite of good correlations, the 95\% confidence bands for the associated linear regressions are quite wide. Confidence bands are not reported for individual subjects, but we estimate them to be about 4 mm Hg wide. For data pooled from all subjects, the confidence band is about 8 mm Hg wide. Thus, considering these data in the aggregate, for any given pericardial pressure we would predict with 95\% confidence that the corresponding chamber pressure is the pericardial pressure ± 2 to 4 mm Hg. Given the magnitude of normal right heart pressures, this is considerable scatter. This at least partially explains why these data obtained in “noisier” living subjects suggest that, on the average, right heart pressure closely tracks pericardial pressure, whereas our relatively noise-free study clearly indicates that these two pressures are not equal.

Right heart pressure after pericardiectomy did not equal zero over the entire range of values. These results are qualitatively similar to those reported by other investigators.\textsuperscript{6, 8–10, 12} Quantitatively, in the normal

\begin{table}[ht]
\centering
\caption{Absolute and relative decrease in left heart pressure caused by pericardiectomy at selected prepericardiectomy pressures.}
\begin{tabular}{|c|c|c|c|c|c|c|c|}
\hline
Dog & 2 & 5 & 8 & 11 & 15 & 20 & 25 \\
\hline
3 & 0.5 & 0.8 & 1.2 & 2.0 & 3.6 & 6.8 & 9.6 \\
5 & 0.9 & 1.2 & 2.0 & 2.8 & 4.6 & 7.4 & 10.0 \\
6 & 0.9 & 1.8 & 2.6 & 4.0 & 6.1 & 9.5 & 13.0 \\
7 & 0.2 & 0.8 & 1.6 & 2.4 & 4.4 & 6.1 & 8.0 \\
8 & 0.4 & 0.6 & 1.0 & 1.3 & 2.8 & 4.5 & 7.0 \\
12 & 1.0 & 1.4 & 1.8 & 2.0 & 3.5 & 5.2 & 9.0 \\
21 & 1.4 & 0.9 & 1.2 & 1.6 & 3.2 & 5.9 & 8.5 \\
23 & 2.0 & 1.4 & 1.9 & 2.2 & 3.1 & 4.2 & 6.5 \\
\hline
Mean & 0.9 & 1.1 & 1.7 & 2.3 & 3.9 & 6.2 & 9.3 \\
SD & 0.6 & 0.4 & 0.5 & 0.8 & 1.1 & 1.7 & 1.9 \\
\% decrease & 45 & 22 & 21 & 21 & 26 & 31 & 37 \\
\hline
\end{tabular}
\footnote{Data not obtained at that prepericardiectomy pressure.}
\end{table}

\begin{table}[ht]
\centering
\caption{Absolute decrease in right heart pressure caused by pericardiectomy at selected total cardiac volumes.}
\begin{tabular}{|c|c|c|c|c|c|c|c|}
\hline
Dog no. & 25 & 50 & 75 & 100 & 125 & 150 & 200 \\
\hline
3 & 0.2 & 0.6 & 0.9 & 1.3 & 2.0 & 2.6 & 4.4 \\
5 & 0.1 & 0.2 & 0.4 & 2.0 & 4.5 & 13.8 & \textsuperscript{\textperiodcentered} \\
6 & 0.4 & 0.8 & 0.9 & 1.4 & 1.8 & 2.7 & 4.0 \\
7 & 0.2 & 0.5 & 1.1 & 2.6 & 7.0 & \textsuperscript{\textperiodcentered} & \textsuperscript{\textperiodcentered} \\
8 & 0.4 & 0.6 & 1.3 & 2.6 & 5.3 & \textsuperscript{\textperiodcentered} & \textsuperscript{\textperiodcentered} \\
12 & 0.7 & 0.7 & 1.2 & 1.5 & 2.3 & 2.8 & 6.1 \\
21 & 0.2 & 0.3 & 0.4 & 0.9 & 0.9 & 1.7 & 3.3 \\
23 & 0.8 & 0.8 & 0.8 & 1.3 & 1.5 & 2.1 & 2.8 \\
Mean & 0.4 & 0.6 & 0.9 & 1.7 & 3.2 & 4.3 & 6.7 \\
SD & 0.3 & 0.2 & 0.3 & 0.6 & 0.6 & 4.7 & 0.6 \\
\hline
\end{tabular}
\footnote{Data not obtained at that volume.}
\end{table}

\begin{table}[ht]
\centering
\caption{Absolute decrease in left heart pressure caused by pericardiectomy at selected total cardiac volumes.}
\begin{tabular}{|c|c|c|c|c|c|c|c|c|}
\hline
Dog no. & 25 & 50 & 75 & 100 & 125 & 150 & 200 & 225 \\
\hline
3 & 0.5 & 0.6 & 0.9 & 1.2 & 1.7 & 1.9 & 3.5 & 6.6 \\
5 & 0.4 & 2.4 & 4.2 & 6.6 & 9.8 & 15.5 & \textsuperscript{\textperiodcentered} & \textsuperscript{\textperiodcentered} \\
6 & 0.5 & 0.8 & 1.0 & 1.2 & 2.2 & 3.5 & 5.5 & 10.2 \\
7 & 0.7 & 0.1 & 0.3 & 0.9 & 3.8 & \textsuperscript{\textperiodcentered} & \textsuperscript{\textperiodcentered} & \textsuperscript{\textperiodcentered} \\
8 & 0.4 & 0.6 & 0.7 & 1.2 & 3.6 & \textsuperscript{\textperiodcentered} & \textsuperscript{\textperiodcentered} & \textsuperscript{\textperiodcentered} \\
12 & 1.1 & 1.0 & 1.4 & 1.4 & 2.0 & 2.3 & 3.4 & 5.2 \\
21 & 0.5 & 0.6 & 0.7 & 1.1 & 1.1 & 1.5 & 4.8 & 8.6 \\
23 & 3.1 & 1.5 & 1.2 & 2.0 & 2.0 & 2.4 & 3.2 & 4.2 \\
Mean & \textsuperscript{\textperiodcentered} & 1.0 & 1.3 & 2.0 & 3.2 & 4.3 & 3.7 & 6.0 \\
SD & \textsuperscript{\textperiodcentered} & 0.7 & 1.2 & 1.9 & 2.8 & 5.5 & 0.6 & 1.7 \\
\hline
\end{tabular}
\footnote{Data obtained at small volumes where buckling of the wall at or around \(V_v\) makes interpretation of the data difficult.}
\footnote{Data not obtained at that volume.}
\end{table}
physiologic range of right heart pressures (from 2 to 5 mm Hg), the pericardium contributes about one-third of the measured right heart pressure. At higher pressures, the pericardium contributes one-half or more of the measured right heart pressure. For comparison, we used data reported by others to estimate the relative contribution of the pericardium to right heart pressure. Two of these were reports of directly measured pericardial pressure where the pericardium contributed 42% at a right ventricular pressure of 9.6 mm Hg in the conscious dog and from 10% to 50% at mean right atrial pressures of 2 to 14 mm Hg in the anesthetized dog. From other reports we used the shift in the pressure-volume relation caused by pericardiectomy to estimate the relative contribution of the pericardium to right heart pressure in the isolated canine heart. This amounted to 35% to 56% at right ventricular pressures of 5 to 20 mm Hg with an empty left ventricle and 26% to 48% at right ventricular pressures of 7 to 29 mm Hg with left ventricular pressure held constant at 4 mm Hg. These estimates are similar to our findings of a 20% to 67% contribution over a range of prepericardiectomy right heart pressures of 1 to 15 mm Hg. Thus, our data, as well as our estimates from these earlier reports, indicate that although pericardial pressure is responsible for a substantial portion of right heart pressure, the two pressures are not equal and, in fact, that right heart transmural pressure is about 3 mm Hg at a “normal” intracavitary pressure of 5 mm Hg.

Our indirect estimates of pericardial pressure from the chamber pressure drop caused by pericardiectomy suggest that pericardial pressure is lower than indicated by the work of Smiseth et al. The pressure drops caused by pericardiectomy in our study are closer in magnitude to the pericardial pressures measured directly by Mann et al. in the beating heart. Because the profiles of the flat balloons used by both of these groups to measure pericardial pressures are similar, both could be expected to distort locally the very narrow pericardial space. However, it may be that the nondistensible fluid-filled balloons used by Smiseth et al. or the fluid-filled flat balloons used by Mann et al., producing greater distortion than do the distensible air-filled balloons used by Mann et al., resulting in an overestimation of pericardial pressure. Such an overestimation could also explain why others have found near equality between right heart and pericardial pressures.

Another possible explanation of this systematic error is the effect of closing the pericardium. If the tight reapproximation (including the use of glue that reacts with the tissue) used in the animal studies reported by Tyberg and co-workers is constrictive then pericardial pressure could be artificially elevated. On the other hand, we left a narrow gap to avoid constricting the pericardium. If our loose reapproximation of the incision reduced the normal restraint of the pericardium, then we may have underestimated pericardial pressure. However, because of the location (principally over the great vessels) and short length (5 to 6 cm) of our incision, we are confident that even if we had not closed the incision, pericardial restraint of the cardiac chambers would have been nearly normal.

If the only source of error in assessing pericardial surface pressure in vivo from right heart pressure is random noise, then right heart pressure may be clinically useful for estimating left heart transmural pressure. One must remember, however, that such a correction is only an average effect and the true left heart transmural pressure could differ by as much as ± 2 to 4 mm Hg because of random error. Depending on the circumstances, this could be a large error. In addition, if the fluid-filled flat balloons used by Tyberg and co-workers systematically overestimate pericardial pressure, there will be a systematic underestimate of left heart transmural pressure when right heart pressure is used to estimate pericardial surface pressure.

Again, whether this error is small enough to ignore for practical purposes depends on the use to be made of left heart transmural pressure. The combined effect of the possible systematic error and the random error could be quite large. For example, if at an intracavitary right heart pressure of 5 mm Hg, pericardial pressure is actually 2 mm Hg, there will be a 3 mm Hg underestimate of left transmural pressure when right heart pressure is substituted for pericardial pressure. Add to this the largest probable random error of ± 4 mm Hg and the error in estimating transmural left heart pressure will be somewhere between +1 and -7 mm Hg.

Unlike the previous reports referred to above, our experiment was designed specifically to test the hypothesis that pericardial pressure equals right heart pressure. Compared with these earlier studies, our experimental design has several advantages. First, we used an arrested heart (in which we truly have a static equilibrium) to measure absolute pressures and volumes in both sides of the heart, which are more precise and are affected by less noise than measurements in the beating heart. Second, by measuring pericardial pressure indirectly we avoided potential problems arising from distortion of the pericardial space by the flat balloons used to measure pericardial pressure. Third, we studied the heart in situ (with the lungs and the chest wall retracted widely) rather than removing it. Thus the relationship between the heart and pericardial...
ium is unaltered. Fourth, we studied the right heart pressure-volume relationship at physiologic relative right and left heart volumes. Because our experimental design required the arbitrary selection of both left and right heart volumes, we chose to keep the volumes of both sides of the heart equal because this state most closely represents the normal physiologic steady state. Finally, we measured lumped (i.e., common atrial and ventricular) pressures on each side of the heart, in contrast to studies in which the ventricle is separate from the atrium, because these chambers normally communicate freely during diastasis and because Tyberg and his co-workers have noted the near equality of pericardial and both mean right atrial and right ventricular diastolic pressures. Hence, although we chose to study an arrested heart to obtain precise and repeatable measurements of pressure and volume in a true static equilibrium state, we designed our experiment to closely resemble physiologic steady-state conditions in a beating heart.

Finally, our data support Tyberg’s speculation, arising from his static equilibrium analysis of intra-chamber and intrapericardial pressures, that the global surface pressures caused by the pericardium are the same over the right and left hearts. Although there is scatter in the data, there was no statistically significant difference in the drops in absolute right and left heart pressures caused by pericardiectomy at any given total cardiac volume (tables 4 and 5 and figure 2).

In summary, we sought to examine recently reported findings regarding the nature of pericardial surface pressure and its relation to chamber pressure in the heart. We started from the basic assumption that the surface pressure and static equilibrium concepts of Tyberg and co-workers are true but that the suggestion that right heart pressure equals or nearly equals pericardial pressure is not strictly true. We showed that the implied zero transmural right heart pressure in the absence of the pericardium does not exist. The basic concepts underlying Tyberg’s static equilibrium analysis of pressures acting across the wall of the heart are not invalidated — in fact our approach assumes that analysis to apply — but the noise in data reported in living subjects (both humans and dogs) may obscure the fact that there is a significant transmural right heart pressure. Thus we believe that any statement of equality between right heart and pericardial pressures is inaccurate. However, we do not disagree with the notion that right heart pressure may be a useful empirical estimate of pericardial pressure when calculating left heart transmural pressure. We do urge some caution, however, because the errors may be on the order of ±2 to 4 mm Hg, and because our results also suggest that the fluid-filled, flat balloons used by Smiseth et al. overestimate true pericardial surface pressure on the heart. Finally, the suggestion that surface pressures over both sides of the heart are equal appears to be correct, since we showed equal absolute decrease in right and left heart pressures caused by pericardiectomy.

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References
Right heart pressure does not equal pericardial pressure in the potassium chloride-arrested canine heart in situ.

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