The relationship of pericardial to pleural pressure is significant in studying distensibility as well as intrapericardial tamponade. Description of ventricular distensibility depends upon the net distending pressure, and since the immediate pressure to the heart is the pericardial pressure, assuming identity of the intrapericardial and intrathoracic pressures require validation. Holt stated that the assumption of equality of the two pressures was erroneous when ventricular end diastolic pressure was elevated "in the slightest," but failed to indicate the magnitude of error. Holt made no statement concerning the relation between respiration and the rhythmic changes of pressure in the pericardial sac and in the thorax. In acute experiments on animals, Kenner and Wood found general agreement between pleural and pericardial pressures, but did not report the nature or magnitude of cardiac or respiratory pressure fluctuations.

In pericardial effusion, marked blood pressure fluctuations with respiration, "pulsus paradoxus," have been ascribed to the failure of the pericardium to transmit the negative intrathoracic pressure to the left atrium and ventricle. Katz and Gauchat suggested that the pulmonary veins would be subject to negative inspiratory pressure and would early pool blood, causing a diminution into the left heart, and thereby a decreased stroke volume. Dock, from experiments with cadavers, inferred that pericardial pressure actually rose during inspiration in cardiac tamponade. Shabetai et al. discounted the theory of pooling in the pulmonary veins, but did not report observations on pericardial pressure swings with inspiration in their experiments.

To date, no reports exist of pericardial pressures in normal animals, nor in tamponade in chronic animal preparations. We are reporting our study of the respiratory fluctuations of pericardial pressure in chronic control animals, and under conditions of tamponade.

Methods

Fourteen dogs weighing 20 to 28 kg were prepared under aseptic conditions by suturing a silastic catheter into the pericardial sac and a...
TABLE 1
Pressure Relationships in Cardiac Tamponade

<table>
<thead>
<tr>
<th>Animal no.</th>
<th>Control pressures, mm Hg</th>
<th>Thoracic</th>
<th>Pericardial</th>
<th>Tamponade pressures, mm Hg</th>
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<th>Pericardial</th>
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<td></td>
<td>Insp.*</td>
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</tbody>
</table>

*Insp.: inspiratory.
†Exp.: expiratory.
‡ΔP: difference between pressures during maximal inspiration and expiration.

*See footnote on page 493.

weeks postoperatively under light anesthesia produced by morphine, 1.5 to 2 mg/kg, and pentobarbital, 12 to 15 mg/kg. Arterial and venous pressures were measured by catheterizing appropriate vessels. Pressures were recorded by means of Statham P23Db (arterial), P23bb (venous), and Sanborn 276 (pericardial and pleural) transducers, with zero level at the mid right atrium. All animals appeared to be in excellent health at the time of study, and at autopsy the pericardium was translucent and appeared normal. Tamponade was produced by infusion into the pericardial sac of 100 to 330 ml of sterile saline at 37.5°C.

FIGURE 1
Silastic pressure canula (from top to bottom): a) arterial pressure catheter; b) pleural pressure balloon 20 × 25 mm; c) pericardial pressure cannula, detail of tip shown in insert at lower left, cannula tip in pericardium emerges parallel to pericardial and epicardial surfaces preventing occlusion of tip with low pericardial pressure; d) pleural pressure balloon as (b) but distended with fluid.

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tion, was 3 mm Hg with agreement generally excellent (correlation coefficient 0.78, significant at the 1% level). The close correlation of pleural and pericardial pressures persisted, even during positive pressure ventilation.

The cardiac cycles were easily identifiable, being superimposed upon the respiratory fluctuations of pericardial pressure. The details of the cardiac pressure changes varied from animal to animal, and even in the same animal from one day to the next. In general, atrial contraction produced either an increase of pericardial pressure or a biphasic pulse (fig. 2). A considerable reduction of pericardial pressure occurred during ventricular systole, and again during the rapid filling phase of ventricular diastole. Pericardial pressure rose briefly during late systole, presumably because of atrial filling, and more gradually during the slow phase of ventricular filling. The pressure changes during the cardiac cycle amounted to 12 to 50% of the respiratory fluctuations in pericardial pressure, with an average of 30%.

Adding warm saline to the pericardial sac always increased pericardial pressure, but the slope of the pressure-volume curves varied considerably (fig. 3). Tamponade, with marked pulsus paradoxus and reduction of blood pressure, required volumes of 100 to 330 ml of saline. The pressure-volume curves in some dogs rose sharply with initial infusions, became level in all dogs for intermediate volumes, and finally rose steeply with the later increments. Hysteresis was present, without exception, in pressure-volume curves produced by withdrawal of the saline from the pericardial sac (fig. 4). However, the differences in pressure between infusion and withdrawal curves were relatively small, rarely exceeding 3 mm Hg.

During cardiac tamponade the pericardial pressure greatly exceeded the pleural pressure, which remained at control levels (fig. 5, table 1). Nevertheless the respiratory fluctuations of pleural and pericardial pressure were similar (table 1) although they showed much more scatter than in the control state ($r = 0.40$, not significant statistically). Also, the inspiratory decreases of pericardial pressure in the control state were close to those observed during cardiac tamponade (fig. 5, table 1; correlation coefficient 0.69, significant at 1% level). Without exception, a significant inspiratory decrease of pericardial pressure occurred during cardiac tamponade as well as during the control state. Cardiac pulsations in pericardial pressure

![FIGURE 2](http://www.ahajournals.org/doi/suppl/10.1161/01.RES.16.3.495.tfig2)

*Records obtained from a healthy animal (C-9) three weeks after surgical preparation, and during sedation. AS: atrial systole; VS: ventricular systole; VDRF: rapid filling phase of ventricular diastole; VDSF: slow filling phase of ventricular diastole.*

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were much more prominent than in the control state, with amplitudes of 36 to 67% of the respiratory pressure swings, and with an average of 50% (fig. 5).

Caval pressure was measured in 12 animals preceding and during cardiac tamponade. Inferior vena caval pressure was determined in eight, superior vena caval pressure in three, while in one dog simultaneous measurements were made in both cavae. In seven dogs there was an inspiratory fall in venous pressure of 1 to 5 mm Hg in the resting state while five animals showed a 1 to 4 mm Hg inspiratory increase. The inspiratory increase of pressure occurred only in the abdominal inferior vena cava. During cardiac tampon-

de, all twelve animals showed an inspiratory decrease of pressure in the inferior as well as the superior cava. However, the pressure fluctuation was considerably less than in the pleural or pericardial spaces.

Acute hypovolemia, produced by removing 30 ml of blood/kg body wt, produced no consistent change in the base line of pericardial pressure, although respiration was increased in rate and depth. Hypervolemia, induced by rapid infusion of 30 ml/kg of saline, produced a gradual rise in pericardial pressure of 2 to 5 mm Hg, as well as an increase of respiratory and cardiac pressure excursions in the pericardial sac. The effects of saline infusion into the pericardium in an animal during hypo- and hypervolemia are demonstrated in figure 4. The later portions of the respective pressure-volume curves show that the rise of pericardial pressure with added saline was roughly proportional to the blood volume of the animal.

Discussion

Our results indicate that there is a close relationship between pleural and pericardial pressures in the normal dog, and that the use of pleural pressure to estimate distending pressure for the heart in chronic experiments

FIGURE 3

Pressure-volume relationships from four representative experiments, to show effects of infusing warm saline into the pericardial sac.

FIGURE 4

Pressure-volume relationships in the same animal (C-23) in normovolemic state, after removal of 30 ml blood/kg body wt, and after replacement of blood plus 30 ml saline/kg. The two normovolemic curves represent infusion and withdrawal relationships, demonstrating mild hysteresis.

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is reasonable under normovolemic conditions. The error introduced by the use of pleural rather than pericardial pressure will rarely exceed 2 mm Hg, or 30% of the respiratory fluctuations of intrathoracic pressure. Although the absolute level of pressure in the pericardium during tamponade is considerably elevated, the transmission of the

![Diagram](image)

**FIGURE 5**

Representative experiment showing (A) control and (B) tamponade in same animal. Paper speeds fast (left) and slow (right).
inspiratory drop in pressure is very faithful. Considering the relative incompressibility of liquid, this is not surprising.

There is adequate evidence that inspiratory drop in pleural pressure is transmitted to the left heart, both in man\textsuperscript{9,10} and animals.\textsuperscript{11} There is no evidence that pulsus paradoxus is due to failure of the pericardial sac to transmit the inspiratory drop in pleural pressure.\textsuperscript{9} Certainly, the pericardial pressure does not rise with inspiration in cardiac tamponade.\textsuperscript{9}

**Summary**

Pericardial pressure was studied in chronic experiments on dogs with relation to intrathoracic pressure in the normal state and in cardiac tamponade. In the normal resting animal, the two pressures were in good agreement throughout the respiratory cycle. The cardiac cycle produced superimposed pressure fluctuations amounting to 30% of the respiratory swings.

Adding saline to the pericardial sac produced sigmoid pressure-volume curves; tamponade required 100 to 330 ml of fluid. Although during tamponade the pericardial pressure greatly exceeded the intrathoracic pressure, the pericardial pressure invariably fell with inspiration, generally by the same amount as the intrathoracic pressure. The cardiac cycle produced greater pressure fluctuations (50% of the respiratory pressure fluctuations). The superior vena caval pressure always fell with inspiration in the normal state and during tamponade.

Hypovolemia did not change the resting pericardial pressure, but decreased the slope of the pressure-volume curve measured during cardiac tamponade. Hypervolemia increased the resting pericardial pressure considerably, and increased the slope of the pericardial pressure-volume curve.

The results suggest that pleural pressure is a reasonable approximation of the pericardial pressure in normal dogs. There appears to be no substantial evidence that pulsus paradoxus is due to failure of the pericardial sac to transmit inspiratory reductions of pleural pressure.

**References**

Relationship of Pericardial to Pleural Pressure During Quiet Respiration and Cardiac Tamponade

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