Can Cardiac Tamponade be Diagnosed by Echocardiography?

Experimental Studies

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SUMMARY The purpose of this study was to determine if respiratory variation and/or absolute size of echocardiographically measured right or left ventricular internal dimension at end-diastole (RVIDd or LVIDd) are accurate indicators of the presence or severity of cardiac tamponade. We measured RVIDd or LVIDd by echocardiography in nine closed-chest, spontaneously breathing dogs in control and during hypotensive tamponade. With tamponade, the end-expiratory RVIDd and LVIDd were significantly smaller than control. Inspiratory increases in RVIDd and decreases in LVIDd were exaggerated during tamponade. Because of the wide range and overlap of RVIDd and LVIDd, no single expiratory value or amount of respiratory change indicated the presence or severity of tamponade. We conclude that if serial echocardiograms show a pericardial effusion, a decreasing end-expiratory RVIDd and LVIDd and an increasing percentage change in ventricular diameter with inspiration, a progressive degree of tamponade should be suspected. However, a single echocardiogram cannot accurately predict the presence or severity of tamponade.

THE ECHOCARDIOGRAM is a sensitive and accurate tool for diagnosing pericardial effusion. Recent studies in patients with pericardial effusion have suggested that changes in ventricular diameter with respiration or right ventricular compression are echocardiographic findings that may indicate pericardial tamponade. However, such changes may not be specific for or predictive of cardiac tamponade. The purpose of this study was to determine if echocardiography could be used to predict the presence and/or severity of cardiac tamponade in an experimental model.

Methods

Animal Preparation

Nine mongrel dogs that weighed 15–35 kg were lightly anesthetized with a mixture of chloralose and urethane, 100 mg/kg and 1000 mg/kg, respectively. The left chest was entered through the fourth intercostal space and the pericardium was cannulated with a #8 polyethylene pigtail catheter through a stab incision. Two 00 silk ties were placed around the catheter using a pursestring suture and a cyanoacrylate ester glue was placed over the suture and surrounding areas to achieve a seal. The chest was closed, and a chest tube to an underwater seal was left in place to remove air and prevent atelectasis. Spontaneous respiration resumed after surgery was completed and the chest closed.

Hemodynamic Measurement

We measured right atrial, aortic arch and left ventricular pressures using a Statham P23Db transducer at the midchest level. Cardiac output was measured by the dye-dilution principle. Indocyanine green dye (2.5 mg) was injected into the right atrium, and dye concentrations in right brachial artery blood were recorded from duplicate curves with exponential decay.

Echocardiographic Studies

Echocardiographic studies were obtained using a commercially available ultrasonoscope and a 2.25-MHz transducer focused at 7.5 cm. The dogs were examined during spontaneous respiration in the right lateral decubitus position. The hand-held transducer was placed in the fourth or fifth right intercostal space approximately 5 cm from the midsternal line and angled slightly inferiorly and anteriorly. An M-mode sweep was initially performed to identify the septum and posterior wall of the left ventricle just below the mitral valve for left ventricular diameter measurements. In addition, a minor sweep was initially done in order to identify the largest end-diastolic dimension measured at the R wave of the QRS complex of the simultaneous electrocardiographic tracing. After the optimal transducer position and beam angulation were initially identified, the transducer was subsequently moved as little as possible to minimize artifacts caused by changing transducer position or beam direction. Measurements were taken during expiration and inspiration in the control state, and during various levels of tamponade (figs. 1 and 2). The phases of the respiratory cycle were identified by visual inspection of the records; this was verified in some of the dogs by simultaneously recording intrapleural pressures as well. Measurements of the ven-
tricular internal diameter were taken from the left or right ventricular free wall endocardial echo to the left or right septal echo. Identification of these structures was verified by injections of saline into the right atrium and left ventricle to produce ultrasonic contrast echoes. In all dogs, the left ventricular borders were adequately visualized to permit measurement of the left ventricular internal dimension. However, in only five of the nine dogs was the anterior right ventricular endocardial echo sufficiently defined to permit measurement of the corresponding right ventricular internal dimension.

**Protocol**

After the dogs had resumed spontaneous respiration, they were allowed to stabilize for 30 minutes. Arterial blood gases and pH were: $\text{PCO}_2 = 40 \pm 16$ torr (SD), $\text{PO}_2 = 130 \pm 18$ torr and $\text{pH} = 7.35 \pm 0.10$. Pressures, duplicate cardiac output determinations, and echocardiograms were performed. Then, 150–250 ml of normal saline at $37^\circ\text{C}$ was injected into the pericardial space to achieve a fall of 10–30 mm Hg in mean arterial pressure. In all nine dogs, recordings were repeated after stabilization for 10–20 minutes. In

**Figure 1.** Echocardiographic recording from a dog before tamponade. Right ventricular internal diameter at end-diastole (RVIDd) is larger during inspiration than during expiration. Left ventricular internal diameter at end-diastole (LVIDd) is smaller during inspiration. Ao = aortic.

**Figure 2.** Echocardiographic recording from the same dog as in figure 1, after cardiac tamponade was produced by injecting 150 ml of fluid intrapericardially. The dimensions of both ventricles were reduced, and the phasic variation with respiration was exaggerated. RVID = right ventricular internal diameter; LVID = left ventricular internal diameter; Ao = aortic.
three dogs, recordings were done during serial, 30-ml saline injections. After the study, the fluid was removed from the pericardium and measured. The fluid volume withdrawn always equaled that administered, indicating that an adequate pericardial seal was present.

**Statistical Methods**

All data in the tables are expressed as mean ± SD. Hemodynamic and echocardiographic data were evaluated by the paired *t* test, comparing measurements taken during control periods with those taken during tamponade. Linear regression analysis was performed on the data comparing echocardiographic measurements with hemodynamic variables.

**Results**

**Hemodynamics (table 1)**

Injection of body-temperature saline into the pericardium caused a significant rise in right atrial pressure and a significant fall in mean aortic pressure. The inspiratory fall in aortic systolic pressure increased from a control of 5 ± 0 mm Hg to 15 ± 2 mm Hg (pulsus paradoxicus). The heart rate increased in six of the nine dogs; in the remaining three, the heart rate decreased 3–35 beats/min during the induction of tamponade, probably due to activation of vagal efferents.9

The control mean aortic pressure varied widely between dogs, and there was considerable overlap between control and tamponade states. Thus, no absolute value of mean aortic pressure indicated pericardial tamponade in a particular dog. There was no overlap in the values for cardiac output, right atrial pressure or pulsus paradoxicus (table 1).

**Echocardiographic Measurements (table 2)**

In these spontaneously breathing dogs, control measurements showed a significant inspiratory increase in end-diastolic right ventricular internal dimension (RVIDd) and a significant inspiratory decrease in end-diastolic left ventricular internal dimension (LVIDd). During cardiac tamponade, the respiratory changes were exaggerated (fig. 2).

In tamponade, the RVIDd in expiration was smaller than it was in the control state (table 2). During both expiration and inspiration, LVIDd was significantly smaller in tamponade than control. Decreases in RVID and LVID during tamponade occurred in all dogs, including the three whose heart rate slowed with tamponade, proving that these decreases in ventricular size were not simply caused by tamponade-induced tachycardia. The range of absolute values of RVIDd and LVIDd in dogs varied widely, with considerable overlap between control and tamponade values (fig. 3).

To evaluate the possibility that the magnitude of respiratory change of RVIDd or LVIDd could separate tamponade from control hemodynamics, we expressed the inspiratory value of RVIDd or LVIDd as a percentage of the expiratory value (%RVID insp/exp and %LVID insp/exp) (fig. 4). Tamponade was associated with exaggeration of the respiratory changes. Again, however, there was a wide range and overlap of control and tamponade values.

Since various degrees of tamponade were produced in the study, we also correlated changes in ventricular dimensions with indices of the severity of tamponade in two ways. First, in three dogs undergoing repeated measurements during progressive tamponade, serial measurement of mean aortic pressure and the amount of pulsus paradoxicus were correlated with %RVIDd insp/exp or %LVIDd insp/exp. The only significant

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**Table 1. Hemodynamic Effects of Pericardial Saline**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th></th>
<th>Tamponade</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>Mean</td>
<td>sd</td>
<td>Range</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>124–220</td>
<td>164</td>
<td>32</td>
<td>90–273</td>
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<tr>
<td>Mean aortic pressure (mm Hg)</td>
<td>80–128</td>
<td>99</td>
<td>16</td>
<td>51–120</td>
</tr>
<tr>
<td>Mean right atrial pressure (mm Hg)</td>
<td>0–3</td>
<td>1</td>
<td>1</td>
<td>7–18</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>2.9–7.2</td>
<td>4.7</td>
<td>1.5</td>
<td>1.6–2.6</td>
</tr>
<tr>
<td>Pulsus paradoxicus</td>
<td>3–6</td>
<td>5</td>
<td>1</td>
<td>7–23</td>
</tr>
</tbody>
</table>

* *p <0.01, tamponade vs control.
‡ ‡p <0.01 vs expiration.
† †p <0.05 vs control.
§.§p <0.05 vs expiration.

**Table 2. Echocardiographic Ventricular Dimensions**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th></th>
<th>Tamponade</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>sd</td>
<td>Mean</td>
<td>sd</td>
</tr>
<tr>
<td>RVIDd (mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expiration</td>
<td>8</td>
<td>3</td>
<td>6†</td>
<td>2</td>
</tr>
<tr>
<td>Inspiration</td>
<td>11‡</td>
<td>3</td>
<td>10‡</td>
<td>3</td>
</tr>
<tr>
<td>LVIDd (mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expiration</td>
<td>32</td>
<td>7</td>
<td>25*</td>
<td>5</td>
</tr>
<tr>
<td>Inspiration</td>
<td>25†</td>
<td>10</td>
<td>21†</td>
<td>6</td>
</tr>
<tr>
<td>LVIDs (mm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expiration</td>
<td>19</td>
<td>7</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td>Inspiration</td>
<td>17</td>
<td>7</td>
<td>14§</td>
<td>8</td>
</tr>
</tbody>
</table>

* *p <0.01 vs control.
‡ ‡p <0.01 vs expiration.
† †p <0.05 vs control.
§.§p <0.05 vs expiration.

Abbreviations: RVIDd = right ventricular internal diameter at end-diastole; LVIDd = left ventricular internal diameter at end-diastole; LVIDs = left ventricular internal diameter at end-systole.
correlation was between the absolute pulsus paradoxicus and %LVIDd insp/exp (r = 0.69) (fig. 5). While this relationship in a single dog showed a fair correlation, the changes in %LVIDd insp/exp were so small that accurate prediction of a specific level of pulsus paradoxicus would be very difficult.

Second, we compared the echocardiographic data for all dogs with indices of severity of tamponade, including the percent fall in cardiac output, the percent fall in the mean aortic pressure, and the absolute value of pulsus paradoxicus. A significant but poor 

\( r = 0.48 \) correlation was found between the %RVIDd insp/exp related to the pulsus paradoxicus. No single echo value of %RVIDd insp/exp could predict the presence or absence of pulsus paradoxicus.

**Discussion**

The main points of this experimental study are: 1) Cardiac tamponade exaggerates the normal phasic respiratory change in the diameter of both ventricles. 2) There is considerable overlap between the control and tamponade values of individual dogs with regard to both absolute chamber size and the percentage change with respiration. Therefore, no single value for any of these parameters is a useful predictor of the presence of tamponade. 3) Echocardiographic parameters correlate poorly with the hemodynamic severity of tamponade.

Phasic variations in left and right ventricular diameters in tamponade were initially described by D'Cruz et al.\(^6\) and Warren et al.\(^7\) and confirmed by Settle et al.\(^8\) The relationship of this echocardiographic finding to the pathophysiology of cardiac tamponade has been noted.\(^9,10\) in tamponade, right ventricular expansion during inspiration is associated with a reciprocal decrease in left ventricular filling and stroke volume and hence a paradoxical pulse.

Phasic changes in ventricular diameters have occurred in patients with pericardial effusion without tamponade\(^6\) (fig. 6), and even in the absence of effusion.\(^6\) Brenner et al.\(^7\) established that respiratory variation of the echocardiographic left ventricular dimension occurs in normal subjects who have no evidence of pericardial disease. Nonechocardiographic techniques have shown similar respiratory changes in normal man and in animals.\(^11,12\) Our study supports that of Brenner et al., because we showed that respiratory variation of ventricular diameters occurred in the spontaneously breathing dog even before pericardial tamponade was created. During tamponade, these respiratory variations were exaggerated (fig. 4), but the wide range and overlap of the individual control and tamponade values precluded the use of phasic variation as a reliable separator of the control from the tamponade condition.

Schiller and Botvinick\(^4\) found right ventricular compression in 16 of 17 tamponade patients and proposed this as a relatively specific echocardiographic sign for cardiac tamponade. Tamponade caused significant reductions of the mean expiratory right and left ven-

**FIGURE 3.** Tamponade caused decreases in end-expiratory right ventricular internal diameter at end-diastole (RVIDd) and left ventricular internal diameter at end-diastole (LVIDd). The large range and overlap of individual values precludes separation of control and tamponade states by either parameter on a single echocardiogram.

**FIGURE 4.** Tamponade (Tamp) caused increases in inspiratory right ventricular internal diameter at end-diastole (RVIDd) expressed as a percentage of expiratory RVIDd (RVIDd ins/exp) and decreases in left ventricular internal diameter at end-diastole (LVIDd ins/exp). The large range and overlap of individual values precludes separation of control and tamponade states by either parameter on a single echocardiogram.
tricular diameters in our dogs. But in our study, the degree of overlap between the control and tamponade ranges for both ventricles was so extensive that no absolute value of ventricular diameter predicted tamponade. However, Schiller’s patients all had systolic blood pressures < 100 mm Hg, indicating a greater degree of hemodynamic embarrassment than our dogs showed. More severe tamponade in our dogs might have caused a better separation between control and tamponade ventricular diameters.

Schiller and Botvinick also suggest that directional change of ventricular size might be an effective indicator of hemodynamic compromise, since they were able to show increases of right ventricular diameter after pericardial drainage. Our study supports this suggestion: We found a reduction in all of the individual left and right ventricular diameters as tamponade was induced.

Settle et al. suggest that cardiac tamponade is unlikely in the absence of respiratory change in ventricular dimension. We agree with this, since all the dogs in our study showed phasic increases of RVID during tamponade, so that the inspiratory RVID ranged from 134-200% of the expiratory RVID. Similar inspiratory decreases in LVID were present. Thus, in a patient with a pericardial friction rub and/or a large cardiac silhouette on x-ray, serial echocardiograms showing a pericardial effusion and an increasing respiratory variation in ventricular diameter coupled with a progressive decrease in end-expiratory ventricular size should suggest that cardiac tamponade is developing.

Phasic changes in mitral valve DE amplitude and EF velocity have also been described in tamponade. In this study, we attempted to reduce artifacts of changing transducer positions by moving the transducer as little as possible during the induction of tamponade, and hence we did not attempt to obtain mitral valve echoes. We would predict (on the basis of

![Graph](https://example.com/graph.png)

**Figure 5.** This graph, from a single dog, shows a correlation between inspiratory (INS) change of the left ventricular internal diameter at end-diastole (LVIDd) (%LVIDd ins/exp) and pulsus paradoxus measured during progressive inflation of the pericardium with saline. EXP = expiratory.

![Echocardiogram](https://example.com/echocardiogram.png)

**Figure 6.** Echocardiogram of a 65-year-old man with chronic renal failure. Pericardial effusion and pronounced respiratory changes in right ventricular internal diameter (RVID) and left ventricular internal diameter (LVID) are present. Clinically, this patient was asymptomatic and had no findings suggestive of tamponade. RV = right ventricular; LV = left ventricular; Insp = inspiratory; Exp = expiratory.
Vignola et al. described a systolic notch in the right ventricular motion in four tamponade patients, but we did not encounter this in our dogs.

Our experimental study has several limitations. We were able to produce tamponade by injecting only 150–250 ml of saline acutely into the dog's pericardial sac. In most patients, tamponade develops more slowly and usually involves considerably more fluid; the pericardial sac is more distended. It is conceivable that with larger amounts of fluid or more severe hypotension or both there might be larger changes of echocardiographic dimensions.

Another problem concerns the effect of pericardial fluid on cardiac position within the thorax. If the position of the entire heart in the pericardial sac were to change as tamponade developed, the ultrasound beam would traverse the cardiac chambers somewhat differently, possibly contributing to the dimensional changes we noted.

In summary, our results suggest that although serial echocardiograms may be useful in demonstrating dimension changes as tamponade develops, a single echocardiogram cannot be used to predict the presence or severity of tamponade. In evaluating a suspected tamponade patient, the time-honored clinical signs of elevated jugular venous pressure and paradoxical pulse should suggest the diagnosis, with the primary application of the echocardiographic technique being the confirmation of the presence of pericardial effusion.

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