Left Ventricular Pseudohypertrophy in Cardiac Tamponade: An Echocardiographic Study in a Canine Model

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Objectives. This study was designed to establish whether left ventricular pseudohypertrophy in cardiac tamponade can be reproducibly induced in an experimental canine model and to investigate the mechanism of its production.

Background. Past experimental and clinical studies have shown reduction of ventricular volumes resulting from cardiac tamponade. Left ventricular pseudohypertrophy, a transient thickening of myocardial walls, was recently described as a new echocardiographic sign of cardiac tamponade.

Methods. Cardiac tamponade was induced in seven anesthetized open chest dogs with serial bolus injections of 50 ml each of 0.9% saline solution into the pericardial sac. Under hemodynamic monitoring, M-mode and two-dimensional echocardiographic measurements were performed from a right parasternal window at each stage of graded cardiac tamponade.

Results. There was a progressive increase of interventricular septal and posterior wall diastolic thickness. Mean wall thickness (interventricular septal thickness + posterior wall thickness) was 9.8 ± 1.3 mm at baseline, 14.3 ± 0.9 mm at peak tamponade and 9.0 ± 1.5 mm after fluid withdrawal (p < 0.0001). Mean wall thickness correlated directly with the severity of cardiac tamponade, as estimated from the level of right atrial pressures (r = 0.75 and p < 0.001), and with the decrease of left ventricular cavity volume (r = -0.67 and p < 0.001). Left ventricular mass did not change significantly.

Conclusions. Left ventricular pseudohypertrophy is a constant manifestation of cardiac tamponade in a canine model. The degree of myocardial thickening correlates with the reduction of ventricular dimensions and with the severity of hemodynamic compromise, representing a constant facet of heart remodeling in cardiac tamponade.

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artery. Systemic arterial pressure was measured from the side port of an 8F introducer (Cook Inc.) placed in the femoral artery. A left thoracotomy was performed at the fourth intercostal space and a 7.3F pigtail angiographic catheter was inserted into the pericardial space and secured with a tabac purse string suture to avoid fluid leakage during the experiment. The margins of the thoracotomy were approached but not closed.

The dog was then transferred to a specially constructed table with a hole in the center allowing positioning of the echocardiographic transducer over the right chest wall from below while the dog was lying on its right side. Pressures were measured with two P23 dB Statham transducers and recorded on ink paper with a Nihon Kohden multichannel polygraphic recorder. Zero reference line was centered at midchest.

Echocardiographic studies were performed with a Hewlett-Packard echocardiographic system 77020A and recorded on 0.5-in. (1.27 cm) videotape cassette. Two-dimensional long- and short-axis parasternal views were obtained in each dog. Linear dimensions were measured from two-dimensionally guided M-mode tracings. The measurements were made according to the recommendations of the American Society of Echocardiography (14,15). All measurements were averaged from three to four beats. The cross-sectional area of the left ventricle was measured by planimetry at the papillary muscle tip level. Left ventricular volumes and derived variables were measured with a computer algorithm based on the bullet formula (16). Left ventricular (LV) mass was calculated from two-dimensional recordings, with the following formula (17,18):

\[
LV \text{ mass} = 1.055 \times \frac{56}{W} (A_t \times L_t - A_c \times L_c).
\]

where \(A_t\) = left ventricular end-diastolic short-axis area including the outer left ventricular wall boundaries; \(L_t\) = left ventricular end-diastolic cavity length measured in the long-axis view from the aortic-mitral junction to the left ventricular apex with mean wall thickness added; \(A_c\) = left ventricular end-diastolic short-axis cavity area; \(L_c\) = left ventricular end-diastolic cavity length.

Study protocol. The dogs were studied after an overnight fast. From the induction of anesthesia until the actual start of the experiment, each dog received intravenously 250 ml of 0.9% saline solution. At the beginning of the experiments, each dog was normovolemic (mean right atrial pressure 0 to 3 mm Hg) (19). During the experiments, each dog received 1,000 ml of fluid, including the amounts used for flushing the monitoring lines at regular intervals. Graded cardiac tamponade was produced with serial bolus injections into the pericardial sac of 50 ml of 0.9% saline solution at room temperature. A complete series of echocardiographic recording and hemodynamic measurements was made at baseline and at each stage of graded cardiac tamponade 5 min after each bolus injection to allow stabilization. Any increase in wall thickness from the baseline values was regarded as wall thickening. An increase in wall thickness above the maximal expected normal limit for dogs of this weight (20) was defined as left ventricular pseudohypertrophy. The severity of cardiac tamponade was estimated from the level of right atrial pressure. The point of equalization, within a range of 0.5 mm Hg, of mean right atrial and intrapericardial pressures was regarded as initial cardiac tamponade. The serial injections were continued until a 39% reduction of the systemic blood pressure was reached or the intrapericardial and right atrial pressures did not increase further with additional pericardial fluid. This stage was regarded as peak cardiac tamponade. The pericardial fluid was completely withdrawn and all the measurements were repeated. The animals were killed by injecting potassium chloride intravenously. The study protocol conformed with the "Position of the American Heart Association on Research Animal Use" adopted by the Association in November 1984 (21).

Statistical analysis. Statistical analysis was performed by using repeated measures analysis of variance. Analysis of staged cardiac tamponade was made after each bolus injection of pericardial fluid, at each level of right atrial pressure (baseline, 3.0 to 6.0 mm Hg, 7.0 to 9.0 mm Hg and >9.0 mm Hg) and at the stages of baseline, peak cardiac tamponade and after fluid withdrawal. Correlation of left ventricular wall thickness with severity of cardiac tamponade and with left ventricular dimensions was determined by regression analysis. Data are expressed as mean value ± 1 SD. Statistical calculations were performed with SAS software from SAS Institute Inc. (22).

Results

Hemodynamic measurements (Table 1). Heart rate was 142 ± 12 beats/min at baseline and did not change significantly at each stage of fluid injection. At peak cardiac tamponade, heart rate was 144 ± 7 beats/min. After withdrawal of the pericardial fluid, heart rate decreased to 119 ± 15 beats/min (p < 0.03).

Pressures (mm Hg) were as follows: mean arterial pressure, 113 ± 25 at baseline 89 ± 21 at peak tamponade (p < 0.01) and 128 ± 14 after fluid withdrawal; mean pulmonary artery pressure, 11.2 ± 2.0 at baseline, 13.3 ± 2.0 at peak tamponade and 13.3 ± 2.0 after fluid withdrawal (p = 0.4); left ventricular end-diastolic pressure, 4.6 ± 2.2 at baseline, 13.8 ± 3.8 at peak tamponade (p < 0.001) and 4.7 ± 2.2 after withdrawal of pericardial fluid.

Intrapericardial pressure increased from 0.8 ± 0.8 mm Hg after the 1st 50 ml of pericardial fluid injection to 10.0 ± 2.4 mm Hg at peak cardiac tamponade (p < 0.0001). Intrapercardial pressure, both at baseline and after complete fluid withdrawal, is not reported because the fluid-filled catheters we used accurately measure this pressure only if ≥30 ml of free fluid is present in the pericardial sac (23). Right atrial pressure increased similarly from 1.3 ± 1.3 mm Hg at baseline to 10.1 ± 2.7 mm Hg at peak tamponade (p < 0.0001). Initial cardiac tamponade was
1. Hemodynamic Measurements

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>HR (beats/min)</th>
<th>IPP (mm Hg)</th>
<th>RAP (mm Hg)</th>
<th>LV EDP (mm Hg)</th>
<th>MAP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B Peak W</td>
<td>B Peak W</td>
<td>B Peak W</td>
<td>B Peak W</td>
<td>B Peak W</td>
</tr>
<tr>
<td>1</td>
<td>150 150 150</td>
<td>— 8.0 —</td>
<td>0.5 8.0 1.0</td>
<td>5.0 13.0 5.0</td>
<td>143 110 159</td>
</tr>
<tr>
<td>2</td>
<td>158 150 136</td>
<td>— 7.0 —</td>
<td>2.0 6.5 2.0</td>
<td>7.0 9.0 3.0</td>
<td>70 57 100</td>
</tr>
<tr>
<td>3</td>
<td>150 150 105</td>
<td>— 9.0 —</td>
<td>6.0 9.0 4.0</td>
<td>2.0 11.0 2.0</td>
<td>95 90 130</td>
</tr>
<tr>
<td>4</td>
<td>152 148 122</td>
<td>— 10.0 —</td>
<td>3.0 10.0 0.0</td>
<td>4.0 17.0 2.0</td>
<td>135 100 140</td>
</tr>
<tr>
<td>5</td>
<td>125 136 110</td>
<td>— 12.0 —</td>
<td>0.5 13.0 2.0</td>
<td>3.0 18.0 2.0</td>
<td>110 75 120</td>
</tr>
<tr>
<td>6</td>
<td>145 138 138</td>
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<td>8.0 19.0 8.0</td>
<td>120 115 130</td>
</tr>
<tr>
<td>7</td>
<td>122 134 96</td>
<td>— 10.0 —</td>
<td>0.0 10.0 6.0</td>
<td>3.0 10.0 5.0</td>
<td>120 80 130</td>
</tr>
<tr>
<td>Mean</td>
<td>142 144 119</td>
<td>— 10.0 —</td>
<td>1.3 10.1 2.7</td>
<td>4.6 13.8 4.7</td>
<td>113 89 128</td>
</tr>
<tr>
<td>SD</td>
<td>12 7 15</td>
<td>— 2.4 —</td>
<td>1.3 2.7 1.9</td>
<td>2.2 3.8 2.2</td>
<td>25 21 14</td>
</tr>
</tbody>
</table>

*Not significantly different from baseline. \( p < 0.0001 \) versus baseline and withdrawal. \( p < 0.01 \) versus baseline and withdrawal. \( p < 0.01 \) versus baseline and withdrawal. B = baseline; EDP = end-diastolic pressure; HR = heart rate; IPP = mean intrapericardial pressure; LV = left ventricular; MAP = mean arterial pressure; Peak = peak cardiac tamponade; RAP = mean right atrial pressure; W = fluid withdrawal. Values for intrapericardial pressure at baseline and after complete fluid withdrawal are not reported (see text).

attained in individual dogs with intrapericardial injection of 100 to 150 ml of fluid and right atrial pressure of 3.0 to 6.0 mm Hg. Peak cardiac tamponade was achieved in individual dogs with intrapericardial injections of 200 to 300 ml of fluid and mean right atrial pressure \( \geq 6.5 \) mm Hg.

Echocardiographic measurements (Tables 2 and 3). M-mode linear dimensions. Left ventricular end-diastolic diameter (cm) was 3.4 \( \pm 0.3 \) at baseline, 2.4 \( \pm 0.4 \) at peak tamponade and 3.4 \( \pm 0.3 \) after fluid withdrawal (\( p < 0.0003 \) for both changes). Left ventricular end-systolic diameter (cm) was 1.9 \( \pm 0.2 \) at baseline, 1.6 \( \pm 0.3 \) at peak tamponade and 2.3 \( \pm 0.4 \) after fluid withdrawal (\( p < 0.01 \)). There was a striking and progressively increase in left ventricular wall thickness during tamponade in each of the seven dogs. Interventricular septal diastolic thickness (mm) was 9.5 \( \pm 1.9 \) at baseline, increased to 14.2 \( \pm 1.2 \) at peak tamponade and decreased to 9.8 \( \pm 1.5 \) after withdrawal of pericardial fluid (\( p < 0.0001 \)). Posterior wall diastolic thickness (mm) was 10.2 \( \pm 2.0 \) at baseline, 14.4 \( \pm 1.0 \) at peak tamponade and 9.8 \( \pm 1.7 \) after fluid withdrawal (\( p < 0.0001 \)) (Fig. 1 to 3). Mean wall thickness (Interventricular septal + posterior wall thickness \( \div 2 \) (mm) was 9.8 \( \pm 1.3 \) at baseline, 14.3 \( \pm 0.9 \) at peak tamponade and 9.8 \( \pm 1.5 \) after fluid withdrawal (\( p < 0.0001 \)).

Two-dimensional measurements. As expected, the left ventricular cavity dimensions were reduced during tamponade. Left ventricular end-diastolic cavity area (cm\(^2\)) decreased from 8.9 \( \pm 2.9 \) at baseline to 5.5 \( \pm 1.9 \) at peak tamponade and increased to 9.7 \( \pm 3.3 \) after fluid withdrawal (\( p = 0.02 \)). Left ventricular end-systolic cavity area (cm\(^2\)) was 4.0 \( \pm 0.7 \) at baseline, 2.7 \( \pm 0.8 \) at peak tamponade, 4.9 \( \pm 1.6 \) after fluid withdrawal (\( p < 0.01 \)). The total cross-sectional left ventricular diastolic area (cm\(^2\)) including the outer edge of the left ventricular myocardium remained unchanged (25.3 \( \pm 1.6 \) at baseline, 25.0 \( \pm 2.4 \) at peak tamponade and 25.9 \( \pm 1.7 \) after fluid withdrawal).

There was a trend, although of borderline statistical significance, toward shortening of the left ventricular cavity length during tamponade. Left ventricular diastolic length (cm) was 5.8 \( \pm 0.7 \) at baseline, 5.2 \( \pm 0.8 \) at peak tamponade and 6.1 \( \pm 0.8 \) after fluid withdrawal (\( p = 0.07 \)). Systolic length (cm) was 4.3 \( \pm 0.6 \) at baseline, 3.9 \( \pm 0.4 \) at peak tamponade and 4.6 \( \pm 0.6 \) after fluid withdrawal (\( p = 0.1 \)). Total left ventricular length (cm) was 6.9 \( \pm 0.6 \) at baseline,

Table 2. Echocardiographic Data of Individual Dogs

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>LV Mean Wall Thickness (mm)</th>
<th>LV ED Area (cm(^2))</th>
<th>LV ED Volume (cm(^3))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B Peak W</td>
<td>B Peak W</td>
<td>B Peak W</td>
</tr>
<tr>
<td>1</td>
<td>7.5 14.6 8.0</td>
<td>9.2 4.1 7.7</td>
<td>46 16 39</td>
</tr>
<tr>
<td>2</td>
<td>11.0 14.8 12.0</td>
<td>6.5 4.4 6.3</td>
<td>29 18 27</td>
</tr>
<tr>
<td>3</td>
<td>10.5 15.5 8.5</td>
<td>7.1 4.6 13.3</td>
<td>34 20 72</td>
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<td>4</td>
<td>10.5 13.5 10.5</td>
<td>6.4 3.5 6.6</td>
<td>29 21 31</td>
</tr>
<tr>
<td>5</td>
<td>10.5 14.0 10.0</td>
<td>8.3 4.3 8.3</td>
<td>32 14 38</td>
</tr>
<tr>
<td>6</td>
<td>10.0 13.5 11.5</td>
<td>14.6 9.8 14.3</td>
<td>81 50 90</td>
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<td>7</td>
<td>8.7 12.8 8.4</td>
<td>10.3 6.3 11.0</td>
<td>56 31 60</td>
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<td>Mean</td>
<td>9.8 14.3 9.8</td>
<td>8.9 5.5 9.7</td>
<td>44 24 51</td>
</tr>
<tr>
<td>SD</td>
<td>1.3 0.9 1.5</td>
<td>2.9 1.9 3.3</td>
<td>19 12 23</td>
</tr>
</tbody>
</table>

*\( p < 0.0001 \) versus baseline and fluid withdrawal. \( p < 0.02 \) versus baseline and fluid withdrawal. \( p < 0.05 \) versus baseline and fluid withdrawal. ED = end-diastolic; other abbreviations as in Table 1.
Table 3. Echocardiographic Measurements

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Peak Cardiac Tamponade</th>
<th>Fluid Withdrawal</th>
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</thead>
<tbody>
<tr>
<td><strong>M-mode echo</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Septum (mm)</td>
<td>9.5 ± 1.9</td>
<td>14.2 ± 1.2*</td>
<td>9.8 ± 1.5</td>
</tr>
<tr>
<td>PW (mm)</td>
<td>10.2 ± 2.0</td>
<td>14.4 ± 1.0*</td>
<td>9.8 ± 1.7</td>
</tr>
<tr>
<td>LV EDD (cm)</td>
<td>3.4 ± 0.3</td>
<td>2.4 ± 0.4†</td>
<td>3.4 ± 0.3</td>
</tr>
<tr>
<td>LV ESD (cm)</td>
<td>1.9 ± 0.2</td>
<td>1.6 ± 0.3‡</td>
<td>2.3 ± 0.4</td>
</tr>
<tr>
<td><strong>2-D echo</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV ED area (cm²)</td>
<td>8.9 ± 2.9</td>
<td>5.5 ± 1.9‡</td>
<td>7.7 ± 3.3</td>
</tr>
<tr>
<td>LV ES area (cm²)</td>
<td>4.0 ± 0.7</td>
<td>2.7 ± 0.8†</td>
<td>4.9 ± 1.6</td>
</tr>
<tr>
<td>LV total area (cm²)</td>
<td>25.3 ± 1.6</td>
<td>25.0 ± 2.4 NS</td>
<td>27.9 ± 3.7</td>
</tr>
<tr>
<td>LV cavity length (cm)</td>
<td>5.8 ± 0.7</td>
<td>5.2 ± 0.8‡</td>
<td>6.1 ± 0.8</td>
</tr>
<tr>
<td>LV S cavity length (cm)</td>
<td>5.3 ± 0.6</td>
<td>3.9 ± 0.4 NS</td>
<td>4.6 ± 0.6</td>
</tr>
<tr>
<td>LV total length (cm)</td>
<td>6.9 ± 0.6</td>
<td>6.6 ± 0.7 NS</td>
<td>7.3 ± 0.6</td>
</tr>
<tr>
<td>LV ED volume (cm³)</td>
<td>44 ± 19</td>
<td>24 ± 12†</td>
<td>51 ± 23</td>
</tr>
<tr>
<td>LV ES volume (cm³)</td>
<td>14.0 ± 5.0</td>
<td>8.6 ± 3.8#</td>
<td>17.7 ± 8.3</td>
</tr>
<tr>
<td>CO (liter/min)</td>
<td>4.0 ± 2.1</td>
<td>2.7 ± 1.3‡</td>
<td>3.8 ± 3.0</td>
</tr>
<tr>
<td>LV EF</td>
<td>0.67 ± 0.06</td>
<td>0.66 ± 0.03 NS</td>
<td>0.61 ± 0.12</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>108 ± 9</td>
<td>110 ± 16 NS</td>
<td>112 ± 15</td>
</tr>
</tbody>
</table>

*Significantly different (p < 0.0001); †Significantly different (p < 0.0003); ‡Significantly different (p < 0.01).
§Significantly different (p = 0.02); #Borderline different (p < 0.05); ‡Significantly different (p < 0.03). All p values compare values at peak cardiac tamponade with values at baseline and after fluid withdrawal. CO = cardiac output; D = diastolic; echo = echocardiography; EDD = end-diastolic diameter; EF = ejection fraction; ES = end-systolic; ESD = end-systolic diameter; PW = posterior wall; S = systolic; 2-D = two-dimensional; other abbreviation as in Table 1.

6.6 ± 0.7 at peak tamponade and 7.3 ± 0.6 after fluid withdrawal (p = 0.1).

End-diastolic left ventricular volume (cm³) was 44 ± 19 at baseline, 21 ± 12 at peak tamponade (p < 0.05), increasing to 51 ± 23 after fluid withdrawal (p = NS compared with baseline). End-systolic left ventricular volume (cm³) decreased from a baseline value of 14.0 ± 5.0 to 8.6 ± 3.8 at peak tamponade and increased to 17.7 ± 8.3 after fluid withdrawal (p < 0.03). Cardiac output (liters/min) was 4.0 ± 2.1 at baseline and 2.7 ± 1.3 at peak tamponade (p < 0.01), returning to 3.8 ± 2.7 after fluid withdrawal. Left ventricular ejection fraction was 0.67 ± 0.06 at baseline and did not change significantly either at peak tamponade (0.66 ± 0.08) or after fluid withdrawal (0.61 ± 0.12).

Right atrial and right ventricular diastolic collapse during cardiac tamponade were observed on two-dimensional echocardiography in each of the seven dogs.

Correlation between wall thickening and severity of cardiac tamponade. Mean wall thickness increased steadily, paralleling the decrease in left ventricular cavity dimensions. For end-diastolic diameter, the correlation coefficient (r) was -0.67, y = -2.8x +20; for end-diastolic area, r = -0.71, y = -1.1x +19; for end-diastolic volume, r = -0.67, y = -0.17x +17 (p < 0.0001) (Fig. 4).

Mean wall thickness correlated directly with the level of right atrial pressure (r = 0.75, y = 0.4x +9.5, p < 0.0001) (Fig. 5). Mean wall thickness above the conventional upper limit of 11 mm for dogs of this weight (20)—that is, left ventricular pseudohypertrophy—was not found in any of the dogs at baseline but was present in five dogs at initial cardiac tamponade and in each of the seven dogs at peak cardiac tamponade. Accordingly, for initial cardiac tamponade, this sig...
tive value. For peak cardiac tamponade, sensitivity, specificity and predictive value were 100% each.

Finally, there was also a close correlation between mean left ventricular wall thickness and the amount of fluid injected ($r = 0.79, y = 0.01x + 9.6, p < 0.0001$) (Fig. 5).

Left ventricular mass (Table 3, Fig. 6). Two-dimensional echocardiographic left ventricular mass (g) was $108 \pm 9$ at baseline, $115 \pm 16$ at peak tamponade and $112 \pm 15$ after fluid withdrawal ($p = 0.2$ for all differences).

**Discussion**

This study demonstrates that a transient left ventricular wall thickening can consistently be induced in a canine model of cardiac tamponade. This phenomenon occurred in each dog, progressively increasing during the development of cardiac tamponade and immediately subsiding on evacuation of the pericardial fluid. It appeared to be directly related to the reduction in left ventricular cavity size and to the severity of cardiac tamponade.

Review of published studies. Many experimental and clinical studies have been devoted to morphologic aspects of cardiac tamponade (3–11). Reduction of heart dimensions and of ventricular volumes has been demonstrated in experimental studies of cardiac tamponade (5–7), but to our knowledge, none of these studies specifically investigated wall thickness. Echocardiographic studies have focused on the signs of right heart compression—right atrial and right ventricular diastolic collapse (1–3). These findings proved to be reliable and sensitive signs of cardiac tamponade that can be detected before hemodynamic embarrassment becomes clinically evident (24, 25). Shrinking of the heart and reduction of left ventricular size as a consequence of heart compression have long been recognized in echocardiographic studies (8–11), but concomitant thickening of ventricular walls has not been stressed. There are sporadic reports on the occurrence of left ventricular outflow tract obstruction in cardiac tamponade with systolic anterior...
Figure 4. Plots of the inverse correlation of mean left ventricular (LV) wall diastolic thickness with left ventricular variables: end-diastolic diameter (left), end-diastolic area (middle) and end-diastolic volume (right). For data see text.

motion of the mitral valve, such as that occurring in hypertrophic cardiomyopathy, but without notable left ventricular hypertrophy (26,27). We (4) have recently shown that left ventricular pseudohypertrophy appears in the clinical course of cardiac tamponade in humans.

Pathophysiology. In our present study, the main change associated with ventricular pseudohypertrophy was the decrease in left ventricular cavity dimensions. Thus, ventricular wall thickening is one of the facets of cardiac remodeling in cardiac tamponade, together with the decrease in left ventricular volumes and shortened left ventricular length, giving the heart a more globular appearance. Wall thickening secondary to a decrease in ventricular volumes should clearly be expected in cardiac tamponade (18), as with other conditions (hypovolemia after hemodialysis, cardiac surgery or thoracotomy) in which an increase in wall thickness associated with a reduction in left ventricular cavity size was shown (28–31). The relation between left ventricular volume and thickness, determined in the formula of left ventricular mass (15,17–18), establishes that if left ventricular mass remains constant, any change in left ventricular volume should be followed by an opposite change in wall thickness. In the present experiment, indeed, left ventricular mass did not change. Thus, left ventricular wall thickening was the inevitable consequence of reduction of left ventricular volume. Assuming that left ventricular mass and the related

Figure 5. Plots of the correlation of mean left ventricular diastolic wall thickness with the right atrial pressure and with the amount of pericardial fluid. Top, Linear correlation between mean left ventricular diastolic wall thickness and right atrial pressure (r = 0.75, p < 0.0001). Bottom, Linear correlation of left ventricular diastolic wall thickness with the amount of intrapericardial fluid (r = 0.79, p < 0.0001).

Figure 6. Bar graph of mean left ventricular mass (g) at baseline, at peak cardiac tamponade and after withdrawal of pericardial fluid. The mild increase in mass at peak cardiac tamponade is not statistically significant (p = 0.2, NS).
total cross-sectional area of the left ventricle remain constant during the development of cardiac tamponade, as actually occurred in our study, the expected wall thickness for each value of cavity area can be predicted from the formula of Schiller et al. (18):

\[
\text{Wall thickness} = \sqrt{\text{Total cross-sectional area}/\pi} - \sqrt{\text{Cavity area}/\pi}.
\]

It can be shown with this calculation that the degree of wall thickening observed in our study, although striking, corresponds to the theoretically expected value.

In addition, left ventricular wall thickening correlated also with the severity of cardiac tamponade, as estimated from right atrial pressures. This observation may become of potential practical importance as an additional echocardiographic sign that may help in estimating the severity of cardiac tamponade. In our study, mean wall thickness above the upper normal limits (left ventricular pseudohypertrophy) occurred in five of seven experiments at initial cardiac tamponade but in each of the dogs during severe cardiac tamponade. In our study, mean wall thickness above the upper normal limits (left ventricular pseudohypertrophy) did not exclude the presence of tamponade but may indicate relatively mild hemodynamic compromise.

Whether the reduction of left ventricular volume and the resultant increase in wall thickness were due to underfilling of the left ventricle or to compression was not directly addressed in this study. However, by comparing our data with those of other reports using similar animal models (12,13), an interpretation of the hemodynamic mechanism involved can be attempted. The relation between volume and pressure changes in cardiac tamponade is defined by the exponential pressure-volume curve. For any given level of intravascular volume and cardiac contractility, any decrease in transmural pressure will cause a decrease in ventricular volume (6,32). Negative transmural pressure of only 0.05 to 0.1 mm Hg represents the critical buckling pressure causing collapse of the thin-walled normal right atrium and right ventricle (33), whereas the thick-walled left ventricle is less compliant and naturally more resistant to compression. We observed zero or negative transmural pressures, impairing right heart filling and indicating direct compression, in the right heart chambers of each dog. In six of the seven dogs, left ventricular end-diastolic transmural pressure remained positive, possibly indicating that decreased venous return was the primary cause of the reduction of left ventricular volume. Transmural and intracavitary pressures observed in our experiment were close to those reported in the classic paper of Ditchey et al. (13), who demonstrated, in an open chest canine model comparable to ours, that reduced left ventricular volume occurs passively during the development of cardiac tamponade because of reduced right heart output and reduced pulmonary venous return.

The reduction of volume was uniformly distributed all over the left ventricle in our model of circumferential pericardial effusion. In regional (experimental and human) cardiac tamponade, localized negative transmural pressure over the left ventricle may occur, leading to left ventricular diastolic collapse and asymmetric left ventricular wall motion (34,35).

Clinical implications. In the present study, left ventricular wall thickening was a constant feature of cardiac tamponade, yet until recently it has not been reported clinically (4). The question can thus be raised whether reliable wall thickness measurements are feasible in the clinical setting of cardiac tamponade. The reliability of two-dimensionally guided M-mode wall thickness measurement is well established (14,36). This measurement in the clinical setting usually does not represent a problem. Increased distance of the heart from the transducer does not influence resolution as it is caused by an echo-free space. Edge definition may even be improved by separation of the parietal pericardium from the epicardium. Nevertheless, a "swinging heart" may negatively affect wall thickness measurements especially if only two-dimensional echocardiography is used. The theoretic concern of confusing apposition of trabeculations with true wall thickening also should not represent a major problem. It has been shown that trabeculations may be misinterpreted as mural thrombi but not as a thickened wall (37).

To establish the diagnosis of left ventricular pseudohypertrophy, one should have a previous reference echocardiogram with normal wall thickness. Very often, the first echocardiogram performed on these patients is the one obtained during cardiac tamponade. In these cases, the diagnosis of left ventricular pseudohypertrophy can be made only retrospectively, after a subsequent echocardiogram performed after resolution of cardiac tamponade.

We believe that the most important clinical application of this new echocardiographic sign of left ventricular pseudohypertrophy would be in patients with acute or subacute cardiac tamponade with rapid accumulation of pericardial fluid. In these cases, echocardiographic images may demonstrate a relatively small amount of pericardial fluid and thickened ventricular walls, mimicking other conditions with left ventricular hypertrophy, such as hypertrophic cardiomyopathy. Accordingly, in an appropriate clinical situation, the possibility of cardiac tamponade should be kept in mind and considered in the differential diagnosis when unexplained left ventricular hypertrophy is observed, even if only a relatively small amount of pericardial fluid is present (Fig. 7).

Limitations of the study. A direct application of the results of this study to human cardiac tamponade is limited because the current study was conducted in open chest dogs and not in chronically instrumented closed chest dogs (38). It has been shown (30) that abolishing the negative intrathoracic pressure that occurs after opening the chest reduces the venous return and the heart dimensions. Also, in the open chest preparation, the normal respiratory augmentation of venous return is lost (12) and the positive pressure
Figure 7. Dog 5. Two-dimensional echocardiogram, long-axis view, at peak cardiac tamponade. Severe pseudohypertrophy of the cardiac walls is clearly shown. Both the interventricular septum and the posterior wall are 15 mm thick. Significant right ventricular wall thickening is also shown. Pericardial effusion (PE) is moderate. The echocardiographic image is reminiscent of other conditions associated with ventricular hypertrophy, such as hypertrophic cardiomyopathy, rather than resembling the usual echocardiographic appearances of cardiac tamponade. The distance between the dots is 10 mm. LA = left atrium; LV = left ventricle; RV = right ventricle.

respiration additionally impairs the venous return. A major difference lies in the time element. In experimental tamponade, the pericardial fluid is accumulated in minutes; whereas in most clinical situations, it takes much longer for a sufficient amount of fluid to accumulate and to produce hemodynamic impairment (32). Clinical pericardial effusions are usually larger and pressure may be higher than those in experimental tamponade (39). Thus, experimental tamponade resembles more a sudden tamponade rather than the other common forms of clinical cardiac tamponade. Another difference between the experimental model and clinical tamponade is the heart rate response. Barbiturate-induced anesthesia caused rapid heart rates with little change during the different stages of the experiment, a finding obviously different from that in clinical situations (13,32). For the purpose of our study, however, this effect was of some value, avoiding the interference of different levels of heart rate on diastolic ventricular filling, ventricular volume and related wall thickness. The loss of the respiratory pump and of related phasic respiratory changes of cardiac dimensions also stabilized ventricular volumes in our preparation.

Limitations in echocardiographic measurements should also be mentioned. 1) Our measurement of left ventricular length from the long-axis view might have caused some underestimation of this variable. We had to use this method of measurement because in our experimental setting the parasternal window was the best echocardiographic window available. However, others (40) have used the same approach and have found it to be reliable in calculating two-dimensional echocardiographic left ventricular mass. 2) Because of the unavailability of the apical four-chamber view, we calculated ventricular volume with the bullet formula as opposed to the Simpson formula, which is recommended for volumetric calculation in humans (15).

For hemodynamic measurements during the production of cardiac tamponade, we used fluid-filled catheters and not high fidelity pressure systems. We believe that in our experimental echocardiographic study, primarily designed to investigate morphologic changes of the left ventricle and not to reinvestigate well established hemodynamic and physiologic changes occurring during cardiac tamponade, the standard fluid-filled pressure systems were sufficiently accurate to allow precise monitoring of the hemodynamic evolution of cardiac tamponade and to correlate them with the observed wall thickening.

Nevertheless, the current study clearly demonstrated that left ventricular pseudohypertrophy indeed occurs in cardiac tamponade. The morphologic left ventricular changes observed during the experiments were so striking that it seems highly unlikely that the preceding study limitations might have influenced the results. The striking correlation of wall thickening with left ventricular volumetric changes, however, indicates that ventricular pseudohypertrophy should not be considered specific for cardiac tamponade. Other conditions with reduced ventricular volume should lead to similar findings. Shift in blood volume as seen at transesophageal echocardiographic monitoring during cardiac surgery is probably a common cause of impressive wall thickness changes that should not be confused with cardiac tamponade.

Conclusions. We demonstrated that left ventricular pseudohypertrophy, a transient and reversible thickening of the left ventricular walls, is a constant manifestation in an experimental model of cardiac tamponade. We think that it should no longer be regarded as an uncommon phenomenon but rather as the other facet of the reduction of ventricular volumes caused by cardiac tamponade. Recognition of this phenomenon may be important clinically because in certain circumstances it may be the most impressive echocardiographic manifestation of cardiac tamponade, mimicking the echocardiographic appearance of other conditions with left ventricular hypertrophy. However, care should be taken in the appropriate use of this new finding, and in assessing whether it is due to external cardiac compression or to reduction of ventricular volume in hypovolemic states.

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References


