Cardiac Tamponade in Left Ventricular Dysfunction

Brian D. Hoit, MD, Marjorie Gabel, and Noble O. Fowler, MD

Echocardiographic and hemodynamic data were measured in closed-chest dogs during graded cardiac tamponade (pericardial pressure 5, 10, and 15 mm Hg) before and after production of diffuse ischemic left ventricular dysfunction. Left ventricular dysfunction was produced by intracoronary injection of nonradioactive microspheres (54±3.9 mm diameter). Changes in left atrial pressure with cardiac tamponade were influenced by coexisting left ventricular dysfunction. Left atrial pressure increased with tamponade and was equal to pericardial pressure before left ventricular dysfunction was produced. However, after left ventricular dysfunction was produced, left atrial pressure was significantly higher than pericardial pressure before tamponade, but it fell toward pericardial pressure when tamponade was produced. Pulsus paradoxus (>10 mm Hg) was present in all animals with cardiac tamponade before left ventricular dysfunction but in only one animal afterward. During each level of tamponade, the inspiratory fall of aortic systolic pressure was greater before than with left ventricular dysfunction. The slope of the linear regression between pericardial pressure and millimeters of mercury of inspiratory fall in aortic systolic pressure was significantly greater before than with left ventricular dysfunction (0.74±0.12 versus 0.32±0.12, p<0.05). Left ventricular dysfunction caused a leftward and upward shift of the pericardial pressure-volume relation. As a result, right atrial and ventricular collapse occurred with significantly smaller volumes of pericardial fluid after than before left ventricular dysfunction. We conclude that pulsus paradoxus may be absent in cardiac tamponade with coexisting left ventricular dysfunction and unequal filling pressures. Echocardiographic signs of cardiac tamponade may occur with small effusions in the presence of left ventricular dysfunction. (Circulation 1990;82:1370–1376)

Cardiac tamponade is a clinical syndrome characterized by elevated venous pressure, an exaggerated inspiratory fall in arterial systolic pressure (pulsus paradoxus), and, as a late event, by arterial hypotension. Pulsus paradoxus and echocardiographic evidence of right atrial and right ventricular collapse are useful signs because they may differentiate hemodynamically insignificant pericardial effusion from cardiac tamponade.1–5 However, clinical observations suggest that pulsus paradoxus may be absent in patients with left ventricular dysfunction and elevated left ventricular filling pressure.1,6 In addition, although it has been reported that right ventricular diastolic collapse is sensitive to alterations in ventricular load,7,8 little is known about the effects of left ventricular dysfunction on chamber collapse during cardiac tamponade. These gaps in our knowledge are particularly relevant in view of the increasing numbers of patients with cardiac tamponade and coexisting cardiovascular disease.9 Accordingly, we studied cardiac tamponade in an animal model of ischemia-induced left ventricular failure to test the hypothesis that pulsus paradoxus is not present when cardiac tamponade occurs with concomitant left ventricular dysfunction manifested by elevated left ventricular filling pressure. We also examined the influence of left ventricular dysfunction on atrial and right ventricular collapse during tamponade.

Methods

Initial Study

Studies were performed in eight nonconditioned, heartworm-free mongrel dogs (mean weight, 19.6±1.2 kg) anesthetized with pentobarbital sodium (25 mg/kg i.v.), intubated, and ventilated with a positive pressure respirator (Harvard Apparatus, Millis, Mass). The chest was opened through a left lateral thoracotomy at the fourth intercostal space and Tygon catheters were secured in the left atrial appendage and pericardial space. The pericardium was closed and the catheters were tunneled subcuta-
neously to exit at the neck. Catheters were filled with heparinized 0.9% saline solution and sealed. The thoracotomy was repaired and the pneumothorax reduced. Azymycin (Shering Kenilworth, N.J.; 2 ml i.m.) was administered daily.

**Acute Studies**

Five to 7 days later, the dogs were anesthetized with pentobarbital (25 mg/kg i.v.) and allowed to breathe spontaneously. Additional doses of anesthesia were administered as necessary. Propylactic quinidine, 320 mg i.m., was administered before surgery. Arterial blood gases were monitored throughout the experiment, and supplemental oxygen and bicarbonate were administered as necessary to maintain a normal arterial blood Po2 and acid-base balance. A flow-directed triple lumen thermodilution catheter was advanced into the pulmonary artery via a jugular vein for measurement of cardiac output and right atrial pressure. A 7F Goodale-Lubin catheter was advanced into the ascending aorta for recording central aortic pressure. A Tygon catheter was placed in the femoral vein for infusion of intravenous fluids. All catheters were inserted under fluoroscopic guidance. A tungsten wire resistance gauge in series with the endotracheal tube was used to monitor respiration.  

Two animals were studied during supported ventilation with an Emerson respirator. This device consists of a molded hardshell chest plate that is fitted snugly to the chest with straps creating a relatively air-tight seal. The chest plate is connected to a variable power vacuum that expands the thorax by producing negative pleural pressure as suction is applied. Respiratory paralysis was produced with Anectine (0.05 mg/kg i.v.). The ventilator rate was set at 32 breaths/min to simulate the respiratory rate we observed during tamponade in animals with left ventricular dysfunction. The amount of suction was adjusted at the beginning of the experiment to maintain physiological arterial blood gases.

A Hewlett-Packard 21362A transesophageal imaging transducer was covered with a disposable sheath, lubricated, and advanced into the esophagus behind the left atrium (approximately 45 cm from the incisors). This instrument consists of a 5.0-MHz imaging and 2.0-MHz Doppler phased-array transducer mounted on the distal tip a 100-cm gastroscope and permits imaging of short- and long-axis views of the heart.

Fluid-filled catheters were connected to Statham 23Db pressure transducers with zero pressure set at the level of the midnight atrium. Temperature was monitored by the thermistor at the tip of the pulmonary artery catheter and a table warmer was used to maintain body temperature at 38° C.

**Experimental Protocol**

Hemodynamic and two-dimensional echocardiographic data and color flow mapping Doppler interrogation of the left atrium were recorded before and during cardiac tamponade at three steady-state levels of pericardial pressure (approximately 5, 10, and 15 mm Hg). Cardiac tamponade was created by the stepwise infusion of 10–20-cc aliquots of physiological saline solution at 38° C into the pericardial space. In addition, two-dimensional echocardiographic images were recorded after each aliquot to determine the relation between the volume of pericardial fluid and the appearance of cardiac chamber collapse. The pericardial fluid was withdrawn after observations at three levels of pericardial pressure, and the animals were allowed to recover until cardiac pressures stabilized for approximately 30–60 minutes. Acute left ventricular dysfunction was then produced in the following manner. A Judkins left coronary artery catheter was advanced through a femoral artery to the left main coronary artery under fluoroscopic control. Catheter tip placement was verified by test injections of contrast material. Nonradioactive tracer microspheres (3M, St. Paul, Minn.) 54±3.9 μm in diameter, suspended in 20% dextran were first separated by sonication for 30 minutes (Cole Palmer, Chicago, Ill.) and then agitated using a Vortex-Genie Mixer (Scientific Products, McGaw Park, Ill.). One milliliter of microspheres (approximately 6.0×10^4 spheres) was mixed in a syringe with 1 ml of angiographic contrast dye (Angiovisst 370, Berlex Labs, Wayne, N.J.) and injected into the left main coronary artery. Boluses of microsphere solution were followed by a 1-ml flush of angiographic contrast. Intracoronary injections were repeated until the mean left atrial pressure was elevated to at least 20 mm Hg. This procedure produces diffuse microinfarction of the left ventricle. After 15 minutes of hemodynamic stability, echocardiographic and hemodynamic measurements were repeated during staged cardiac tamponade at pericardial pressures of approximately 5, 10, and 15 mm Hg.

The electrocardiogram, pressures, flows, and respiration were recorded at slow and rapid paper speeds (10 and 100 mm/sec). Studies were recorded on a Grass 7D multichannel recorder. Two-dimensional echocardiographic studies were performed in the spontaneous breathing animals using a Hewlett-Packard 7750C ultrasonograph, with data recorded on 0.5 in. VHS videotape. The output from the respiratory gauge was monitored in the auxiliary channel of the ultrasonograph. The experimental protocol was approved by the Institutional Animal Care and Use Committee at the University of Cincinnati.

**Two-Dimensional Echocardiographic Measurements**

Images were analyzed off-line using the computational algorithms resident on the ultrasonograph. The left ventricle chamber area was determined from the transthoracic short-axis view at the level of the papillary muscles, obtained using a 5.0-MHz short-focus transducer placed on the chest wall. Enddiastolic left ventricular areas were planimetered from the videoframe containing the largest ventricular area, and five area determinations were averaged.
### TABLE 1. Hemodynamic Changes With Three Levels of Cardiac Tamponade Before and After Left Ventricular Dysfunction

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>TAMP 1</th>
<th>TAMP 2</th>
<th>TAMP 3</th>
<th>Control</th>
<th>TAMP 1</th>
<th>TAMP 2</th>
<th>TAMP 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAP (mm Hg)</td>
<td>1.4±0.8</td>
<td>5.8±0.9*</td>
<td>10.5±1.01*</td>
<td>14.2±1.8*</td>
<td>28.7±3.8</td>
<td>22.3±7.1*</td>
<td>14.3±2.1*</td>
<td>15.2±2.0*</td>
</tr>
<tr>
<td>RAP (mm Hg)</td>
<td>1.1±1.0</td>
<td>5.9±1.0*</td>
<td>10.4±1.1*</td>
<td>14.7±2.7*</td>
<td>4.0±1.9</td>
<td>5.7±1.6</td>
<td>8.8±1.5*</td>
<td>12.8±2.7*</td>
</tr>
<tr>
<td>PERI P (mm Hg)</td>
<td>−1.6±0.7</td>
<td>5.0±0.8*</td>
<td>9.9±0.2*</td>
<td>14.7±2.1*</td>
<td>0.8±1.4</td>
<td>5.3±0.5*</td>
<td>8.8±1.3*</td>
<td>12.8±2.3*</td>
</tr>
<tr>
<td>Aortic P (mm Hg)</td>
<td>130±19</td>
<td>124±18</td>
<td>109±19*</td>
<td>66±10*</td>
<td>119±17</td>
<td>114±17</td>
<td>102±23*</td>
<td>63±14*</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>2.2±0.6</td>
<td>2.0±0.5</td>
<td>1.3±0.4*</td>
<td>0.5±0.1*</td>
<td>1.2±0.3</td>
<td>0.9±0.2</td>
<td>0.7±0.2*</td>
<td>0.4±0.1*</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>170±34</td>
<td>182±39</td>
<td>185±39</td>
<td>149±32*</td>
<td>163±27</td>
<td>168±30</td>
<td>160±37</td>
<td>14±23*</td>
</tr>
</tbody>
</table>

LVD, left ventricular dysfunction; TAMP, tamponade level; LAP, mean atrial pressure; RAP, mean right atrial pressure; PERI P, pericardial pressure; Aortic P, mean aortic pressure; CO, cardiac output; HR, heart rate. All values mean±1 SD.

*p<0.05 vs. Control, ’p<0.05 vs. TAMP 1, ’p<0.05 vs. TAMP 2.

The maximal percent change in chamber area was computed as 100 multiplied by (chamber area before tamponade minus chamber area at the third stage of tamponade) divided by chamber area before tamponade. Left ventricular end-diastolic areas during inspiration and expiration were compared at control and at a pericardial pressure of 10 mm Hg before and after left ventricular dysfunction. Fractional shortening of the left ventricular area measured in the short-axis projection was calculated with empty pericardia before and after production of left ventricular dysfunction as 100 multiplied by (end-diastolic area minus end-systolic area) divided by end-diastolic area.

Right and left atrial collapse were identified by inversion of the respective atrial free wall at any point of the cardiac cycle. Right ventricular diastolic collapse was identified by a persistent inward motion of any portion of the endocardial surface during diastole after tricuspid valve opening. Real-time, slow motion, and frame-by-frame analyses were used to identify these events.

**Hemodynamic Data**

Pulsus paradoxus was defined by an absolute decrease in aortic systolic pressure of at least 10 mm Hg during inspiration (expiratory minus inspiratory systolic pressure). The percentage decrease in arterial systolic pressure (mm Hg pulsus paradoxus divided by expiratory systolic pressure multiplied by 100) was also calculated. Thermodilution cardiac output determinations were made in triplicate and averaged.

**Statistical Analysis**

Changes in hemodynamic and echocardiographic measurements at each stage of cardiac tamponade, before and after production of ventricular dysfunction, were compared by repeated-measures analysis of variance. When a significant interaction between severity of tamponade and left ventricular dysfunction was found, Tukey’s test was used to determine where those differences were significant. Pericardial pressure and millimeters of mercury inspiratory fall in aortic systolic pressure were compared with linear regression analysis both before and after left ventricular dysfunction. The slopes of the linear regressions were compared using Student’s t test. The volume of infused pericardial fluid and cardiac and pericardial pressures at the time of atrial and ventricular chamber collapse before and after production of left ventricular dysfunction, respiratory changes in left ventricular end-diastolic areas and maximal percent chamber area changes were compared with paired Student’s t tests. Data are expressed as mean±SD. In all comparisons, a probability value less than 0.05 was considered statistically significant.

**Results**

**Hemodynamic Measurements**

Microsphere injections into the left main coronary artery resulted in large increases in mean left atrial pressure, small, but significant, increases in mean pericardial and right atrial pressures, and decreased cardiac output without significant changes in mean aortic pressure or heart rate. The percent fractional area shortening of the left ventricular area in the short-axis projection (area ejection fraction) decreased from 45.1±5.2% to 14.8±1.4% (p<0.05).

When cardiac tamponade was produced at three stages of intrapericardial pressure, pericardial and right atrial pressures increased significantly both before and after microsphere administration. With tamponade, left atrial pressure increased before left ventricular dysfunction was produced but fell significantly afterward. The fall in left atrial pressure began early during the infusion of pericardial fluid. Cardiac output decreased significantly with tamponade, and at each stage, it was significantly lower after production of left ventricular dysfunction than before. Mean arterial blood pressures fell only in the latter stages of tamponade both before and after left ventricular dysfunction (Table 1).

Pericardial pressures before and after left ventricular dysfunction were matched at the first two stages but were significantly different at the third stage of tamponade. Although cardiac output fell significantly with tamponade both before and after left ventricular dysfunction, the percent fall in cardiac output before and after left ventricular dysfunction at each stage was similar. Thus, despite the small difference in
pressure) +6, \( r=0.791, p<0.0001 \) and after left ventricular dysfunction [mm Hg inspiratory fall in pressure=0.32 (pericardial pressure) +1.74, \( r=0.41, p<0.05 \). The difference in slopes (0.74±0.12 versus 0.32±0.12) was significant (\( p<0.05 \)).

In the two animals studied on the Emerson ventilator, the inspiratory fall of systolic pressure during tamponade was greater before than after production of left ventricular dysfunction (10 and 18 mm Hg, and 7 and 7 mm Hg, respectively, at the third level of tamponade). Although inspiration caused a fall in cardiac and pericardial pressures, the magnitude of respiratory variation was less than during spontaneous ventilation.

**Echocardiographic Measurements**

Left ventricular short-axis areas decreased significantly with cardiac tamponade. When expressed as a percent decrease in chamber area with tamponade, the area of the left ventricle decreased more before than after production of left ventricular dysfunction (38.3±9.1\% versus 19.1±9.1\%, \( p=0.031 \)).

Before production of left ventricular dysfunction, the maximum percent change in left ventricular diastolic area during respiration (expiratory minus inspiratory left ventricular end-diastolic area divided by expiratory left ventricular diastolic area multiplied by 100) was 6.0±0.8\% before and 27.1±8.2\% after cardiac tamponade (\( p<0.05 \)). In contrast, after left ventricular dysfunction, the maximum percent change in left ventricular diastolic area during respiration was 3.1±2.4\% before and 7.3±2.4\% after cardiac tamponade (NS). Thus, with cardiac tamponade, there was a significant inspiratory decrease in left ventricular end-diastolic volume before, but not after, production of left ventricular dysfunction.

**Echocardiographic Chamber Collapse**

With cardiac tamponade, right atrial collapse was identified in all animals both before and after left ventricular dysfunction (Table 2). Right ventricular collapse was seen in five of six dogs before left ventricular dysfunction. The right ventricle could not be imaged adequately in the short axis in one of the animals after left ventricular dysfunction. As a result, right ventricular collapse was seen in only four of the dogs after left ventricular dysfunction. Left atrial collapse was observed in all animals before, but in only one animal after production of left ventricular dysfunction. The volume of pericardial fluid at the onset of atrial and ventricular collapse is shown in Figure 2. Right atrial and ventricular collapse occurred with smaller volumes of pericardial fluid than left atrial collapse both before and after left ventricular dysfunction. Significantly less pericardial fluid was needed to collapse the right atrium and ventricle, but more fluid was needed to collapse the left atrium, after microsphere injections than before. Despite smaller pericardial fluid volumes, the pericardial and atrial pressures at right atrial and ven-

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**Figure 1.** Bar graph of absolute (top panel) and percent (bottom panel) inspiratory fall in aortic systolic pressure during staged tamponade (TAMP) before and after left ventricular dysfunction (LV DYS). \( ^{*}<0.05 \) vs. control, \( ^{a}<0.05 \) vs. TAMP 1, \( ^{b}<0.05 \) vs. TAMP 2. See text for details.

**Pulsus Paradoxus**

Pulsus paradoxus was present at each stage of tamponade in five of six dogs before production of left ventricular dysfunction. In contrast, with left ventricular dysfunction, pulsus paradoxus was seen in only one animal. This occurred at the third level of tamponade, at which time right and left atrial pressures had equilibrated with pericardial pressure. For the six animals combined, an inspiratory systolic pressure decline of at least 10 mm Hg was observed at each stage of tamponade before microsphere injection (Figure 1A). At each stage of tamponade, the mean inspiratory fall in aortic systolic pressure was significantly greater before than after production of left ventricular dysfunction. When expressed as a percent of the maximal expiratory systolic arterial pressure, significantly greater inspiratory changes in systolic arterial pressure were present at each stage of tamponade before production of left ventricular dysfunction than afterward (Figure 1B). There was a significant correlation between pericardial pressure and inspiratory fall in aortic systolic pressure before [mm Hg inspiratory fall in pressure=0.74 (pericardial pressure) +6, \( r=0.791, p<0.0001 \) and after left ventricular dysfunction [mm Hg inspiratory fall in pressure=0.32 (pericardial pressure) +1.74, \( r=0.41, p<0.05 \). The difference in slopes (0.74±0.12 versus 0.32±0.12) was significant (\( p<0.05 \)).

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tricular collapse were slightly, but significantly, higher after left ventricular dysfunction than before.

**Pericardial Pressure-Volume Infused Relation**

Ischemia-induced left ventricular dysfunction caused a significant upward shift of the pericardial pressure-volume infused relation. As seen in Figure 3, less pericardial fluid was needed to reach each stage of pericardial pressure after than before production of left ventricular dysfunction.

**Discussion**

The hemodynamics and echocardiographic findings during cardiac tamponade in experimental animals and in humans have been studied extensively. The present study demonstrates distinctive hemodynamic features and echocardiographic manifestations of experimental cardiac tamponade in the presence of ischemic left ventricular dysfunction. The absence of pulsus paradoxus and the altered manner in which the cardiac chambers are compressed aid our understanding of the pathophysiology of cardiac tamponade and its clinical signs.

Linear regression analysis indicated that as the severity of tamponade increased, the inspiratory fall in aortic systolic pressure increased significantly both before and after production of left ventricular dysfunction. However, the slope of this relation was significantly smaller after than before left ventricular dysfunction. Thus, in the presence of left ventricular dysfunction, increasing severity of cardiac tamponade causes smaller inspiratory decreases in aortic systolic pressure. The lower correlation after left ventricular dysfunction suggests that factors other than pericardial pressure are responsible for the smaller respiratory changes in aortic pressure.

There are several possible explanations for the absence of pulsus paradoxus during cardiac tamponade in dogs with left ventricular dysfunction. First, pulsus paradoxus may not develop in the presence of systemic hypotension. However, significant differences in the magnitude of inspiratory fall in systolic arterial pressure remained even after correction for the expiratory systolic aortic pressure. Second, the inspiratory fall in aortic systolic pressure may have been less after production of left ventricular dysfunction because of unequal hemodynamic effects of cardiac tamponade at the same pericardial pressures.

However, the percent fall in cardiac output at each stage of cardiac tamponade was similar before and after creation of left ventricular dysfunction, indicating reasonably matched hemodynamic levels of tamponade. Third, differences in the frequency and depth of respiration may have affected the development of pulsus paradoxus. Increased impedance to left ventricular emptying has been suggested as a mechanism for respiratory changes in systolic aortic pressure, and the pattern and rate of respiration changed after production of left ventricular dysfunction. We studied two additional dogs with depth and rate of respiration controlled by an external chest respirator. The results in these animals were similar to those from the larger study, although the magnitude of the inspiratory fall in systolic pressure was less. Thus, changes in the rate and depth of respiration after left ventricular dysfunction cannot entirely explain our results. Finally, potential artifacts of the experimental design must be considered, but it is unlikely that cardiac and pericardial adaptation or deterioration of the preparation due to either the effects of serial tamponade or time alone were responsible for our results because tamponade states were well matched before and after left ventricular dysfunction.

**TABLE 2. Cardiac and Pericardial Pressures at the Time of Chamber Collapse Before and After Left Ventricular Dysfunction**

<table>
<thead>
<tr>
<th></th>
<th>Before LVD</th>
<th>After LVD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RAC (n=6)</td>
<td>RVC (n=5)</td>
</tr>
<tr>
<td>PERI P (mm Hg)</td>
<td>2±1</td>
<td>3±1</td>
</tr>
<tr>
<td>RAP (mm Hg)</td>
<td>3±1</td>
<td>3±1</td>
</tr>
<tr>
<td>LAP (mm Hg)</td>
<td>4±2</td>
<td>4±2</td>
</tr>
</tbody>
</table>

LVD, Left ventricular dysfunction; RAC, right atrial collapse; RVC, right ventricular collapse; LAC, left atrial collapse; PERI P, pericardial pressure; RAP, mean right atrial pressure; LAP, mean atrial pressure. All values mean±1 SD.

*p<0.05, †p=0.06, after vs. before LVD.

![Figure 2](http://circ.ahajournals.org/DownloadedFrom)

**FIGURE 2. Bar graph of volume of fluid instilled into the pericardial space producing collapse of the right atrium (RAC), right ventricle (RVC), and left atrium (LAC) before and after left ventricular dysfunction (LV DYS). LAC was observed in only one dog after LV DYS. †p<0.05 vs. no LV dysfunction. See text for details.**
Thus, the changes we observed with cardiac tamponade after production of left ventricular dysfunction probably relate to fundamental pathophysiological differences due to abnormal left ventricular function and elevated left heart volumes and pressures that characterize this model. When respiratory-induced changes in right and left ventricular filling are no longer competitive, such as may occur in atrial septal defect and pulmonary arterial obstruction, pulsus paradoxus may be absent. Unequal diastolic pressures, either due to aortic insufficiency or ventricular dysfunction, may be an additional means of reducing competitive ventricular filling. In our study, after production of left ventricular dysfunction, left atrial pressure was significantly greater than the equilibrated right atrial and pericardial pressures at the first two stages of tamponade. At the third stage of tamponade, left atrial pressure had fallen and equilibrated (within 2 mm Hg) with right atrial and pericardial pressures in two of the dogs. In these two dogs, there was an inspiratory decrease in aortic systolic pressure of 16 and 8 mm Hg, respectively. It is also possible that a small end-diastolic volume is necessary for producing a paradoxical pulse. With left ventricular dysfunction, left ventricular end-diastolic volumes were significantly larger, and tamponade produced a smaller percent change in left ventricular chamber area. Although the precise mechanism by which the inspiratory fall in aortic pressure is less after than before production of left ventricular dysfunction is uncertain, we have shown that the difference is associated with decreased respiratory variation in left ventricular end-diastolic volume.

Echocardiographic identification of a hemodynamically significant pericardial effusion is facilitated by the presence of right ventricular, and to a lesser extent, right atrial collapse. Although regarded as a sensitive and specific sign of hemodynamically significant pericardial effusion, right ventricular diastolic collapse has recently been shown to be sensitive to loading conditions in dogs with normal left ventricular function. Our results are consistent with these studies, although it is possible that the results were mediated by different pathophysiological mechanisms.

In interpreting the data in this paper, one should extrapolate the findings from this acute, anesthetized animal model of cardiac tamponade to the clinical setting with caution because this model differs in several important ways from cardiac tamponade in patients. In experimental tamponade, pericardial fluid accumulates in minutes, whereas effusions usually accumulate over days to months in humans. A related concern is that pericardial effusions are usually larger and pericardial pressures usually higher in clinical than experimental tamponade. The effects of anesthesia in experimental tamponade, which are usually not a concern in clinical tamponade, also need to be considered. Furthermore, although the experimental model most closely resembles ischemic left ventricular systolic dysfunction, whether similar findings will be found in hearts with predominantly diastolic dysfunction remains to be determined. Finally, it is not certain whether similar results would be found in left ventricular dysfunction with lesser elevation of left atrial pressure or in the absence of mitral regurgitation.

In conclusion, the inspiratory decline of systolic arterial pressure is significantly less during cardiac tamponade in animals with left ventricular dysfunction than with normal left ventricular function. Our results are consistent with the hypothesis that filling against common right and left ventricular diastolic pressures set by the pericardial pressure-volume relation is necessary for the production of a paradoxical pulse, as suggested by Reddy et al, because this condition was met before, but not after, the creation of left ventricular dysfunction. In this animal model, left ventricular dysfunction altered the manner in which pericardial fluid compressed the cardiac chambers. In the presence of left ventricular dysfunction, there is a shift in the pericardial pressure-volume relation, and right atrial and ventricular collapse occur with smaller pericardial fluid volumes. As a result of elevated left atrial pressures, left atrial collapse may not be seen at all.

The practical importance of these findings is that increasing numbers of patients with pericardial effusion have significant preexisting heart disease manifested by elevated left ventricular end-diastolic pressure such as occurs with hypertensive heart disease, coronary artery disease, cardiomyopathy, and uremia. In these patients, pulsus paradoxus may not appear and echocardiographic and hemodynamic evidence of cardiac tamponade may be seen with a small pericardial effusion.

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References

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