Experimental Cardiac Tamponade: A Hemodynamic and Doppler Echocardiographic Reexamination of the Relation of Right and Left Heart Ejection Dynamics to the Phase of Respiration

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A hallmark of cardiac tamponade is pulsus paradoxus. However, the exact mechanism of pulsus paradoxus and the relation of left and right ventricular ejection dynamics remain controversial, with some studies suggesting an inverse relation in ventricular filling and ejection and others citing a more important role for the effects of right heart ejection dynamics delayed by transit through the pulmonary artery bed. To specifically reexamine this issue, six sedated but spontaneously breathing dogs were studied during experimental cardiac tamponade with use of extensive hemodynamic instrumentation and Doppler methods.

During cardiac tamponade, left ventricular systolic pressure decreased from 125.8 ± 12.1 to 81.7 ± 26.7 mm Hg (p < 0.01) and cardiac output from 5.86 ± 1.48 to 2.34 ± 0.98 liters/min (p < 0.001); mean pericardial pressure increased from -1.2 ± 0.8 to 10.7 ± 1.2 mm Hg (p < 0.001) and pulsus paradoxus from 4.3 ± 1.6 to 10.7 ± 1.2 mm Hg (p < 0.001) compared with baseline values. An inverse relation in left and right ventricular ejection dynamics that was very close to 180° out of phase was seen throughout the respiratory cycle in multiple hemodynamic and Doppler variables including peak systolic pressures, aortic and pulmonary flow velocities and ventricular ejection times.

Simultaneous recording of the transmitral pressure gradient provided indirect evidence that the ventricular ejection dynamics were directly related to changes in ventricular filling. However, the magnitude of ventricular pressure or output flow velocity for each respiratory cycle was variable, depending on the exact timing of filling and ejection in relation to the phase of respiration. Variation in left ventricular output due to changes in right ventricular output delayed by transit through the pulmonary vasculature was not recognized in any animal.

It is concluded that in spontaneously breathing dogs with acute cardiac tamponade, peak ventricular pressures, ventricular ejection times and pulmonary and aortic flow velocities have an inverse relation that is very close to 180° out of phase.

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A hallmark of cardiac tamponade is pulsus paradoxus. However, despite years of research, the exact mechanism of pulsus paradoxus and the relation of left and right ventricular ejection dynamics remain controversial (1). One theory proposes that an inspiratory increase in right ventricular filling precedes a decrease in left ventricular filling and that left ventricular stroke volume increases only after the two or three cardiac cycles necessary for the increased right heart stroke volume to traverse the pulmonary circulation (2-7). If true, maximal pulmonary artery pressure and flow in cardiac tamponade would seldom be coincident with minimal aortic pressure and flow and right and left ventricular stroke volumes would not be 180° out of phase.

An alternative theory about pulsus paradoxus proposes that total pericardial volume is "fixed" in cardiac tamponade and that augmented filling in one ventricle results in an immediate and opposite change in the volume of the other ventricle (8-10). In this case, pulmonary artery pressure and flow would be nearly reciprocal to aortic pressure and flow (11,12). In patients with cardiac tamponade, M-mode echocardiography has demonstrated that an inspiratory increase in the right ventricular diastolic diameter is associated with a decrease in left ventricular diastolic diameter and that opposite changes occur with expiration (13-16). Similar reciprocal changes with respiration, for right and left ventricular stroke volumes (17) and peak early diastolic mitral and tricuspid flow velocities (18-20) have also been demonstrated in patients. In these clinical studies (13-20), the variables used to estimate filling and output of the right and left ventricles appear to have an inverse relation and to be very close to 180° out of phase.

To help resolve this controversy, our study was designed to carefully reexamine the relation of left and right ventricular ejection dynamics in experimental cardiac tamponade.
using hemodynamic and Doppler flow velocity data. A lightly sedated, spontaneously breathing canine model was chosen in an attempt to mimic clinical cardiac tamponade as closely as possible.

Methods

Surgical preparation. The surgical methods used in this animal model of cardiac tamponade have been previously described (21). Briefly, six mongrel dogs (25 to 35 kg) underwent a left thoracotomy for placement of left atrial and pericardial catheters. After a small incision was made in the pericardium over the right ventricular free wall, an 8F Silastic catheter was placed in the pericardial space and secured with a pursestring suture. A second 8F Silastic catheter was placed in the body of the left atrium through the left superior pulmonary vein and was secured in a similar fashion. The two catheters were placed individually through the chest wall and tunneled subcutaneously to an area between the scapulae. The ribs, muscle layers, subcutaneous tissue and skin were closed in layers to provide an airtight seal. Afterwards, 60 ml of sterile saline solution was injected into the pericardial space to check for leaks and prevent the formation of adhesions. The two catheters were then flushed, filled with heparin solution and capped. Analgesics and antibiotics were administered as necessary and the dogs were allowed to recover for a minimum of 7 days before undergoing study.

Instrumentation and calibration protocol. On the day of the experiment, the dogs were sedated with diazepam (0.5 mg/kg body weight) and hydromorphone (0.3 mg/kg). After administration of halothane and nitrous oxide, they were intubated and mechanically ventilated with a Harvard respirator. Under fluoroscopic guidance, high fidelity micromanometer-tipped catheters (Millar Instruments) were placed in all four cardiac chambers, the ascending aorta and the pericardium for pressure monitoring. This instrumentation included a 7F catheter in the left ventricular apex inserted percutaneously through the left femoral artery, a 7F catheter in the ascending aorta through the right femoral artery, a dual sensor 7F catheter in the right atrium and right ventricle through the right external jugular vein and 5F catheters in the pericardium and left atrium through the surgically implanted Silastic tubing. In addition, a flow-directed pulmonary artery catheter was placed through the right external jugular vein for pulmonary artery pressure and cardiac output measurement. After instrumentation, anesthesia was discontinued and the dogs were allowed to recover for 30 min before the start of the protocol. An arterial blood gas measurement was performed before baseline data were obtained to ensure adequate oxygenation and normal acid-base status. Further sedation was provided as necessary by diazepam in incremental doses of 0.01 to 0.05 mg/kg.

Zero reference for all high fidelity catheters was obtained by immersing them in body temperature saline solution for 60 min before insertion at a height equal to 50% of the transthoracic diameter of the dog’s chest. The pulmonary artery catheter was attached to a strain gauge pressure transducer (Statham 231D, Gould, Inc.) and was referenced to zero in a similar fashion. Onset of “inspiration” and “expiration” was referred to the signal from a thermistor placed near the tip of the endotracheal tube. The timing of thermistor response to the onset of inspiration and expiration had been previously determined by recording respiration simultaneously with intrapleural pressure.

Data Gathering and Analysis

Hemodynamics. Hemodynamic data and phasic respiration were recorded on a 16 channel recorder (ES 2000, Gould) at paper speeds of 50 and 100 mm/s. Hemodynamic variables were measured for three consecutive cardiac cycles and averaged. Variables included heart rate, right and left ventricular systolic and end-diastolic pressures and mean pericardial, right atrial, left atrial and aortic pressures. All pressures were referenced to an atmospheric zero.

The maximal difference between inspiratory and expiratory systolic pressure in the ascending aorta (“pulsus paradoxus”) was measured at baseline and during cardiac tamponade. This value was “corrected” for changes in stroke volume and heart rate by dividing the absolute inspiratory systolic pressure decrease by the expiratory systolic blood pressure; a 10% change was considered significant (22). Cardiac output was determined in triplicate and averaged by thermodilution technique.

Echocardiography. The dogs were placed in the left lateral position for the echocardiographic examination. Two-dimensional and Doppler echocardiographic data were obtained from parasternal short-axis and apical four-chamber views with a commercially available ultrasonograph (CPM 700, Interspec) with a dual-function transducer (3 MHz imaging and 2.5 MHz Doppler studies). Right atrial collapse was defined as inversion of the right atrial free wall that started in late diastole and persisted for >35% of the duration of the cardiac cycle (23). Right ventricular diastolic collapse was defined as inversion and abnormal posterior motion of the right ventricular free wall in early diastole (24–26). Aortic flow velocity was obtained from the apical transducer position with use of the pulsed wave Doppler technique and simultaneous recording of respiration. The sample volume was placed at the leaflet tips to record maximal flow velocity together with valve opening and closure clicks. Pulmonary flow velocity was obtained in a similar fashion with use of a left or right parasternal short-axis view. The Doppler tracings were recorded on a strip chart recorder at a paper speed of 50 mm/s. In addition, both the imaging and Doppler echocardiographic data were recorded on videotape with a Panasonic AG 7300 super VHS video cassette recorder. One of us (C.P.A.) analyzed the hemodynamic data and another (M.S.G.) performed the
Fig. 1. Hemodynamic recording of left (LV) and right (RV) ventricular pressures together with respiration (resp), illustrating how the relation of ventricular ejection dynamics to respiration was determined. The onset of inspiration (insp) and expiration (exp) is labeled. With one method, peak systolic ventricular pressures were measured and referenced to peak inspiration, defined as the maximal upward deflection of the respiratory recording. With the second method, peak left and right ventricular systolic pressures were determined for the first ejection beat of inspiration [I] and expiration [E] that followed the first diastolic filling period of each phase of respiration (I = first diastolic filling beat of inspiration; E = first diastolic filling beat of expiration). Right (RVPP) and left (LVPP) ventricular pulse pressures were calculated as the difference between peak systolic ventricular pressure and ventricular end-diastolic pressure (EDP); the reference for end-diastolic pressure is the maximal QRS amplitude. See Methods for more complete explanation. ECG = electrocardiogram.

Doppler measurements; each investigator was unaware of the results of the other.

Relation of ventricular pressures to phase of respiration. From the hemodynamic recordings, the relation of peak right and left ventricular systolic pressures throughout the respiratory cycle was determined in two ways. With the first method, peak pressure in each ventricle was measured on all beats over five consecutive respiratory cycles and referenced in time to peak inspiration (Fig. 1). To correct for differences in pressure between individual dogs, ventricular pressures were expressed as a percent of the minimal and maximal values recorded, 0% being equal to the lowest systolic pressure recorded and 100% being equal to the highest value recorded. Beats during inspiration were assigned negative time values and beats during expiration were assigned positive time values. Depending on the heart rate and rate of respiration, the number of cardiac beats and time intervals measured from peak inspiration varied for each dog.

With the second method, peak systolic ventricular pressures were measured on specific beats of the respiratory cycle as defined by their preceding diastolic filling period. The first ejection beat of inspiration was defined as the beat that followed the first inspiratory diastolic filling period. Similarly, the first expiratory ejection beat was the beat after the first expiratory diastolic filling period (Fig. 1).

During some respiratory cycles, a change in the phase of respiration occurred between the time of ventricular filling and ejection. To account for this occurrence, peak ventricular pressures were also measured on these “split” beats (ventricular filling during inspiration and ejection during expiration) occurring during the transition from inspiration to expiration.

Doppler variables. Peak pulmonary artery and aortic flow velocities and right and left ventricular ejection times were measured for three consecutive respiratory cycles, with inspiratory and expiratory beats being defined in the same way used for the hemodynamic measurements. The ejection time was determined from the opening and closure clicks of the pulmonary and aortic valves that were recorded with the flow velocity signals. To aid in identification of the diastolic filling period, the transmural pressure gradient was displayed simultaneously with the flow velocity on the Doppler hard copy recording.

Protocol. Before the start of the experiment, filling pressures were standardized in each dog by infusing normal saline solution (if necessary) to increase mean left atrial pressure to 5 to 7 mm Hg; 30 ml of warmed (37°C) saline solution was infused into the pericardium before the start of the protocol. This amount of fluid does not significantly alter intracardiac pressures and has previously been shown (27) to aid accurate pericardial pressure measurement with micromanometer-tipped catheters. After a 10 min stabilization period, baseline hemodynamic and Doppler data were obtained. Cardiac tamponade was produced by infusion of 0.9% saline solution at body temperature into the pericardium at a rate of 10 ml/min (26). Cardiac tamponade was defined by 1) elevation of mean right atrial pressure, 2) near equalization (<5 mm Hg) of diastolic intracardiac pressures, 3) two-dimensional echocardiographic evidence of both right atrial and right ventricular diastolic collapse, and 4) a ≥25% decrease in mean aortic pressure and cardiac output. During the protocol, the dogs were breathing spontaneously.

This protocol was approved by the Animal Research Committees of the Tucson Veterans Affairs Hospital and the University of Arizona. Specific attention was given to the appropriateness and welfare of the animal model, the adequacy of anesthesia and the methods of instrumentation. This protocol is also in accordance with the “Position of the American Heart Association on Research Animal Use.”

Statistical analysis. This was performed using a statistical package (Version 3.1, SPSS) for a personal computer (IBM PC AT). Differences in mean values for each hemodynamic variable between baseline study and cardiac tamponade were compared by a Student’s t test for paired data. Doppler ultrasound variables at each phase of respiration during baseline study and during cardiac tamponade were compared with use of an analysis of variance. To determine the relation of systolic ventricular pressures to the time of peak inspiration, the raw data were plotted and then fit by using a
Results

Echocardiography. Adequate two-dimensional imaging for data analysis was obtained in all dogs. The effusion was circumferential in all cases. As the size of the pericardial effusion increased, cardiac size and volume qualitatively decreased. Right atrial collapse appeared before right ventricular collapse in all dogs.

Hemodynamics (Table 1). The mean volume of saline solution infused to produce well developed cardiac tamponade was 185 ± 38 ml. The hemodynamic variables at baseline study and during cardiac tamponade are compared in Table 1. With cardiac tamponade, heart rate increased and ventricular systolic pressures and cardiac output decreased. Intra-ventricular diastolic pressures and pericardial pressure became "equalized" in all dogs, with the mean pericardial pressure increasing from -1.2 ± 0.8 to 10.5 ± 3 mm Hg (p < 0.001).

The parabolic equation \( y = a + b(x + c)^2 \), where \( y \) = systolic pressure and \( x \) = ms from peak inspiration. The statistical program then estimated \( a, b \) and \( c \) and \( r \) values with use of a nonlinear regression technique (Levenberg-Marquardt algorithm). Because the most important variable for analyzing the phase relation of ventricular pressures is \( c \) (maximal or minimal point of each parabola), the 95% confidence intervals for right and left ventricular \( c \) values were computed in each dog and in the entire group. All results are expressed as mean values ± 1 SD.

<table>
<thead>
<tr>
<th>Heart Rate and Hemodynamic Data in Six Dogs Before and After Creation of Cardiac Tamponade</th>
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<tbody>
<tr>
<td>Before Tamponade</td>
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<tr>
<td>------------------</td>
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<tr>
<td>Heart rate (beats/min)</td>
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<tr>
<td>LV systolic pressureapnea (mm Hg)</td>
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<tr>
<td>Pulsus paradoxus (mm Hg)</td>
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<tr>
<td>Corrected pulsus paradoxus* (%)</td>
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<tr>
<td>RV systolic pressureapnea (mm Hg)</td>
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<tr>
<td>RV systolic pressureexp, ap (mm Hg)</td>
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<tr>
<td>LV end-diastolic pressure (mm Hg)</td>
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<tr>
<td>RV end-diastolic pressure (mm Hg)</td>
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<tr>
<td>Mean left atrial pressure (mm Hg)</td>
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<tr>
<td>Mean right atrial pressure (mm Hg)</td>
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<tr>
<td>Mean pericardial pressure (mm Hg)</td>
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<tr>
<td>Cardiac output (liters/min)</td>
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See Methods for definition of corrected pulsus paradoxus. LV = left ventricular; n = number of animals; RV = right ventricular.

The parabolic regression curves of right and left ventricular systolic pressures referenced to peak inspiration are shown for three dogs in Figure 4. A composite graph for the entire group showing 95% confidence intervals for the maximal and minimal point of each parabola (c value) generated by the regression analysis is also shown. The r values for the individual parabolic equations ranged from 0.52 to 0.81 and all individual confidence intervals of the c values overlapped each other. In all dogs, the maximal point of the fitted parabola for right ventricular systolic pressure slightly preceded the minimal point of the left ventricular parabola. For the entire group, the right ventricular c value was 115.9 ms after peak inspiration (range -4.2 to 236), whereas the left ventricular c value was -49.6 ms (range -171.8 to 72.6). This indicates that the peak right ventricular and minimal left ventricular systolic pressures were nearly reciprocal (within 170 ms), with peak right ventricular pressure occurring slightly after minimal left ventricular pressure.

Doppler variables. Table 2 shows mean values for the ventricular ejection times and peak aortic and pulmonary artery flow velocity for apnea, inspiration and expiration at baseline study and during cardiac tamponade. The mean differences from expiratory to inspiratory values for these variables are also shown. In contrast to the small changes observed with respiration at baseline study, right ventricular ejection time and pulmonary flow velocity increased markedly with inspiration during cardiac tamponade, whereas left ventricular ejection time and aortic velocity decreased. Opposite changes were seen on expiration. The changes in

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Figure 2. Two recordings of left (LV) and right (RV) ventricular pressures with simultaneous respiration (resp) recorded during cardiac tamponade in the same dog. The onset of inspiration (insp) and expiration (exp) is shown. A, A respiratory cycle in which ventricular filling and output occur during the same phase of respiration. In this case, right ventricular pressure increases and left ventricular pressure decreases on the first inspiratory ejection beat [2] that occurs after the first inspiratory diastolic filling period (Ii). Compare with beat [1], in which diastolic filling occurred during apnea. The opposite changes are seen on the next ejection beat [3], which in this respiratory cycle follows the first expiratory diastolic filling period (Ie). B, A respiratory cycle in which ventricular filling and ejection are separated ("split") by a change in the phase of respiration. In this case, the first ejection beat [2] after the first inspiratory diastolic filling period (Ii) is similar to that in A, with right ventricular pressure increasing and left ventricular pressure decreasing compared with a beat during apnea [1]. However, in the next beat, the diastolic filling period again occurs during inspiration (2i), but the ejection [3] occurs during expiration, resulting in right and left ventricular pressures that are intermediate between beats [1] and [2]. Left ventricular pressure is highest and right ventricular pressure lower on the next beat [4], which follows the first diastolic filling period of expiration (Ie).

right heart variables were larger than those seen in left heart variables.

The timing of the aortic and pulmonary flow velocity changes in relation to respiration and the transmitral pressure gradient are shown in Figure 5. The lowest aortic velocity in cardiac tamponade was seen during inspiration on the beat that followed the smallest transmitral pressure gradient. Conversely, the highest velocity is seen in expiration on the beat that followed the largest transmitral gradient. Opposite changes are seen for pulmonary flow velocity. Figure 6 shows the inverse relation of aortic and pulmonary flow velocities during cardiac tamponade.
Changes in ventricular pressures and flow velocities after periods of apnea. In a few dogs during cardiac tamponade, the respiratory rate was slow enough or there was a pause in respiration so that ventricular pressures and flow velocities could be observed after a period of apnea containing 5 to 10 cardiac cycles. In these instances, changes in ventricular pressures and flow velocities appeared identical to those seen during faster or more continuous respiration.

**Discussion**

Ventricular ejection hemodynamics during cardiac tamponade. Despite years of study, the exact relation of ventricular ejection dynamics in cardiac tamponade has remained controversial. Using high-fidelity hemodynamic pressure recordings and Doppler echocardiographic techniques, this study was designed specifically to study the relation of left and right ventricular peak systolic pressures, output flow velocity and ejection time. In the model of acute cardiac tamponade, the major findings are 1) left and right ventricular systolic pressures, ejection times and output flow velocity exhibit an inverse relation during cardiac tamponade that appears to be very close to 180° out of phase; 2) the reciprocal changes in pressure and flow velocity are seen during every respiratory cycle, even if preceded by a period of apnea; and 3) the magnitude of change in ventricular pressures and output flow velocity for individual beats is determined by the precise timing of ventricular filling and flow output to the phase of respiration.

The inverse relation of left and right ventricular ejection dynamics in cardiac tamponade was seen in multiple ways, including both peak systolic pressures, aortic and pulmonary flow velocities and ventricular ejection times. These results were similar regardless if these variables were analyzed in reference to peak inspiration (ventricular pressures <170 ms out of phase, Fig. 4) or to specific filling beats of the respiratory cycle (Fig. 2, 3, 5 and 6). The simultaneous recording of the transmitral pressure gradient with pulmonary and aortic flow velocities (Fig. 5) also provided indirect evidence that the changes seen in these variables were directly related to variation in ventricular filling (15,16).

**Comparison with previous studies.** The findings in this study support theories that view cardiac tamponade as a state in which the total pericardial volume is fixed, with respiration-driven changes in volume in one ventricle resulting in an immediate and opposite effect in the other ventricle (8). Earlier experimental work (5,9,11) in acute cardiac tamponade also demonstrated peak right ventricular and systemic pressures that appear reciprocal. Recent noninvasive (13–16,18–20,28) and invasive (17) clinical studies have supported this concept. These studies have shown that in cardiac tamponade, respiration-driven changes occur in ventricular dimensions (13–16,28), transvalvular flow velocities (18–20) and ventricular stroke volume (17). In all cases, the changes in the two ventricles appeared to demonstrate an inverse relation very close to 180° out of phase. With use of multisensor catheters with electromagnetic flow velocity probes, peak aortic and pulmonary flow velocities were

**Table 2. Left Ventricular and Right Ventricular Ejection Times and Aortic and Pulmonary Artery Flow Velocities During Apnea, Expiration and Inspiration in Six Dogs Before and After Creation of Cardiac Tamponade**

<table>
<thead>
<tr>
<th></th>
<th>Heart Rate (beats/min)</th>
<th>Phase of Respiration</th>
<th>LVET (ms)</th>
<th>Aortic Flow Velocity (cm/s)</th>
<th>RVET (ms)</th>
<th>Pulmonary Artery Flow Velocity (cm/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before tamponade</td>
<td>110.8 ± 14.5</td>
<td>Aorta</td>
<td>199.7 ± 3.0</td>
<td>102.8 ± 18.8</td>
<td>203.3 ± 43.9</td>
<td>92.3 ± 18.1</td>
</tr>
<tr>
<td>(n = 6)</td>
<td></td>
<td>inspiration</td>
<td>198.3 ± 3.4</td>
<td>105.2 ± 18.3</td>
<td>204.5 ± 44.1</td>
<td>88.2 ± 18.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>expiration</td>
<td>196.3 ± 3.4</td>
<td>98.5 ± 15.5</td>
<td>218.5 ± 43.5</td>
<td>98.7 ± 16.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Δ(Exp – Inspr)</td>
<td>2.0 ± 2.7</td>
<td>5.4 ± 2.1</td>
<td>-14.0 ± 11.8</td>
<td>-8.7 ± 2.5</td>
</tr>
<tr>
<td>After tamponade</td>
<td>158.8 ± 16.7</td>
<td>Aorta</td>
<td>154.8 ± 3.0</td>
<td>75.6 ± 17.5</td>
<td>142.8 ± 35.3</td>
<td>58.8 ± 20.4</td>
</tr>
<tr>
<td>(n = 6)</td>
<td></td>
<td>inspiration</td>
<td>151.3 ± 3.3</td>
<td>81.5 ± 17.0</td>
<td>123.0 ± 52.9</td>
<td>61.5 ± 33.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>expiration</td>
<td>142.2 ± 4.9</td>
<td>63.3 ± 19.8</td>
<td>184.8 ± 22.4</td>
<td>118.3 ± 30.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Δ(Exp – Inspr)</td>
<td>19.1 ± 4.2</td>
<td>19.2 ± 12.2</td>
<td>-61.8 ± 43.8</td>
<td>-56.7 ± 27.9</td>
</tr>
</tbody>
</table>

Δ = differences; Exp – Inspr = expiration minus inspiration; n = number of dogs; LVET and RVET = left and right ventricular ejection time, respectively.
found to be inversely related in three patients with cardiac tamponade (17). A similar but much smaller degree of variation in flow velocity was seen in control subjects, suggesting that the reciprocal findings during tamponade were an exaggeration of normal physiology. An inverse relation between aortic and pulmonary flow velocities obtained with Doppler technique under normal conditions was also seen in this and a previous clinical study (18), but the changes are so small (approximately 5%) that a reciprocal relation in ventricular stroke volume during normal respiration is difficult to confirm with certainty. However, peak ventricular pressures do not show an inverse relation under control conditions, but rather parallel each other (Fig. 3). This suggests that peak ventricular pressures occur after changes in intrathoracic pressure under normal circumstances, but when cardiac tamponade is present, alterations

Figure 5. Aortic (A) and pulmonary (B) flow velocities during cardiac tamponade. The onset of inspiration (insp), expiration (exp), aortic valve opening (Ao), aortic valve closure (Ac), pulmonary valve opening (Po) and pulmonary valve closure (Pc) clicks are labeled. Left ventricular ejection time (LVET) is the interval from aortic valve opening to aortic valve closure and right ventricular ejection time (RVET) is the interval from pulmonary valve opening to pulmonary valve closure. A. Pulsed wave aortic flow velocity recording during cardiac tamponade with simultaneous left ventricular pressure (LV), left atrial pressure (LA) and respiration (resp). Note the decrease in aortic flow velocity during the first ejection beat [1] that occurs after the first inspiratory diastolic filling period (Ii). Conversely, aortic velocity and left ventricular ejection time increase on the first expiratory beat [2] that follows the first expiratory filling period (Ie). The lowest aortic velocity [1] occurs after the diastolic filling period with the smallest transmitral pressure gradient (Ii)* and the largest aortic velocity occurs after the diastolic filling period with the largest transmitral gradient (Ie). B. Pulsed wave pulmonary flow velocity recording during cardiac tamponade using the same format as in A. Note the opposite changes in pulmonary compared with aortic flow velocity, with the largest pulmonary velocity during the first beat of inspiration (I) that occurs after the first inspiratory diastolic filling period (Ii) and the lowest velocity and shortest right ventricular ejection times seen on the first beat of expiration (2) that follows the first expiratory diastolic filling period (Ie). The pulmonary velocity changes are exactly 180° out of phase with the transmitral pressure gradient, suggesting that reciprocal ventricular filling and ejection dynamics are present (see text for complete discussion).

*The apparent negative transmitral gradient during the first beat of inspiration is probably due to slight drift in one of the catheters or a small change in catheter position during the experiment. ECG = electrocardiogram.
in ventricular filling and ejection may become the dominant factors affecting peak pressures.

As shown in previous experimental work (29), our study also demonstrates a reduced left atrial to left ventricular pressure gradient during inspiration in cardiac tamponade (Fig. 5), which immediately precedes a marked decrease in aortic flow velocity. Similar observations using pulmonary wedge pressure have been made in clinical studies (18,22,30), illustrating the tight coupling of ventricular filling and ejection dynamics that occurs in cardiac tamponade.

The results of this study do not support theories regarding cardiac tamponade that propose a dominant role for left heart ejection dynamics reflecting changes in right heart output delayed by transit through the pulmonary bed (2,3,5). Our observations that ventricular ejection variables after periods of apnea were similar to those during continuous respiration are not compatible with this theory. This immediate effect of respiration on left ventricular ejection dynamics has been made previously in patients under normal conditions (31) and with cardiac tamponade (18).

Nevertheless, clinical pressure recordings of right ventricular and systemic arterial pressures do not appear to have an inverse relation to respiration (32,33). Similar results were reported (9) during an experimental study using right heart bypass and simulated increases in right heart filling. The reason for the differences in these results in our study and those previously cited are unknown, but may be related to the effects of anesthesia, the use of fluid-filled catheter systems for pressure recordings or concomitant disease processes. Alternatively, it may be that the depth and pattern of respiration could influence the relative effect of "reciprocal" versus "series" ventricular output changes that are observed. Further research will be necessary to clarify these issues and establish whether there are circumstances in which changes in right heart output delayed by pulmonary transit play a more important role in left heart ejection dynamics.

Relation of heart rate and respiratory rate. In this and previous clinical studies of cardiac tamponade (18,20), the most common relation of heart rate and respiratory rate is one or two cardiac cycles during each phase of respiration (inspiration and expiration). Therefore, depending on the precise timing of ventricular filling, variation in the magnitude of ventricular pressure and output flow velocities was frequently observed between different respiratory cycles. The largest changes in output were seen when filling occurred in early inspiration or expiration at the time when changes in intrathoracic pressure were maximal. Beats in which filling occurred later during inspiration or expiration showed less change. There were also many "split" beats in which filling occurred during one respiratory phase and output occurred during another. Although not emphasized, this cycle to cycle variation in ventricular output has been shown previously in clinical cardiac tamponade for both ventricular dimensions (13,28) and output flow velocities (17).

Study limitations. Because left and right ventricular outflow tract diameters were not measured during the study, the flow velocities in these vessels cannot be used as a direct measure of stroke volume. Furthermore, the compliance characteristics of the two vessels are probably markedly different at baseline study and during cardiac tamponade. Thus, a linear relation between the Doppler variables and stroke volume would not be expected and, indeed, was not observed in this study.

The dogs in this study were heavily sedated to permit the use of extensive instrumentation. This may have resulted in a reduced respiratory effort in some dogs and more hemodynamic compromise during cardiac tamponade than that seen in fully conscious animals (26,32). It is a possibility that greater changes in intrathoracic pressure and right heart filling with respiration could have altered the results. Although this study demonstrates that a respiration-driven reciprocal relation in ventricular filling and ejection exists in cardiac tamponade, it does not provide data on the exact pathophysiology by which this occurs. This also requires further investigation.

Conclusions. Our results show that in spontaneously breathing dogs with acute cardiac tamponade, peak left and right ventricular pressures and ejection times and pulmonary and aortic flow velocities have an inverse relation that is very close to 180° out of phase. These results support
previous work suggesting that total cardiac volume is fixed in cardiac tamponade and that respiration-induced changes in filling in one ventricle result in an immediate and opposite change in filling in the other. Further work is necessary to elucidate the exact pathophysiology by which this reciprocal filling occurs and whether altering the depth and pattern of respiration influences these results.

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References