The hemodynamic derangement associated with right ventricular diastolic collapse in cardiac tamponade: an experimental echocardiographic study

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ABSTRACT  Clinical reports indicate that right ventricular diastolic collapse (RVDC) is associated with cardiac tamponade. To assess the hemodynamic abnormalities associated with RVDC, we studied six chronically instrumented conscious dogs (group A) with two-dimensional echocardiography during cardiac tamponade induced by continuous saline infusion into the pericardial space. We recorded cardiac output (electromagnetic flowmeter), heart rate, respiration, and aortic, intrapericardial, and right atrial pressures. In four additional animals (group B), we recorded right ventricular pressure and placed a hydraulic occluder around the pulmonary artery so that short-term reversible obstruction to right ventricular outflow could be produced. None of the dogs had RVDC when the pericardial space was empty, but all dogs showed RVDC during cardiac tamponade. The appearance of RVDC in group A was associated with a 21% reduction in cardiac output (p < .01) and no change in mean aortic pressure. Short-term partial pulmonary artery obstruction led to increased right ventricular pressures and a striking reduction in RVDC in early tamponade, suggesting that RVDC is caused by pericardial pressure exceeding right ventricular pressure in early diastole. An additional animal had right ventricular hypertrophy caused by a severe "heart worm" infestation and did not show RVDC during cardiac tamponade. These observations suggest that in the absence of increased resistance to right ventricular outflow or right ventricular hypertrophy, RVDC occurs early in the course of cardiac tamponade and is associated with a hemodynamically important decrease in cardiac output.


DETECTION of pericardial effusion was one of the earliest clinical uses of echocardiography,1 and the technique has proved to be accurate and extremely sensitive when used for that purpose.2 However, the assessment of the hemodynamic consequences of pericardial effusions by means of echocardiography has been disappointing. M mode echocardiographic findings, including phasic changes in the mitral EF slope,3 right ventricular epicardial systolic notching,4 right ventricular compression,5 and reciprocal alterations in right and left ventricular diameter,6,7 have been associated with cardiac tamponade but have not achieved widespread clinical use because of their lack of sensitivity or specificity. An abnormal inspiratory increase of right ventricular diastolic dimensions and decrease of left ventricular diastolic dimensions has been found to be present in patients with cardiac tamponade.8 Unfortunately, similar findings can be seen in other conditions associated with pulsus paradoxus and are not specific for cardiac tamponade.

Abnormal motion of the anterior right ventricular wall has been described in patients with impending cardiac tamponade.9 In a review of 21 M mode echocardiograms of patients with documented cardiac tamponade, a persistent posterior motion of the right ventricular endocardium after mitral opening has been reported.10 A retrospective study of 17 patients with proven cardiac tamponade showed that diastolic collapse of the right ventricle was a sensitive echocardiographic feature of this clinical problem.11 The specificity, extent of the associated hemodynamic derangement, and causes of right ventricular diastolic

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collapse (RVDC) in cardiac tamponade remain uncertain. The purpose of our study, therefore, was to define the hemodynamic correlates and to investigate possible mechanisms of RVDC in cardiac tamponade by means of a chronically instrumented, unanesthetized animal model.

Materials and methods

Two groups of chronically instrumented dogs were studied. The six animals in group A were used to study the extent of the hemodynamic abnormalities associated with RVDC, and the four animals in group B were used to further evaluate the mechanism that caused it.

In group A, six mongrel dogs weighing from 18 to 29 kg were studied. Each dog was brought to the laboratory after an overnight fast and was anesthetized (sodium pentobarbital 30 mg/kg iv), intubated, and ventilated by a volume respirator (Harvard Apparatus Co., South Natick, MA) with air enriched with oxygen (2 l/min). Each animal was then shaved and prepared in the usual manner, and a left thoracotomy was performed in the fifth intercostal space with sterile technique. The electrocardiogram was monitored throughout the procedure. A polyvinyl catheter was inserted into the left internal mammary artery and advanced to the aortic arch. After manual confirmation of the position of the arterial catheter, it was secured and used to monitor arterial blood pressure throughout the procedure. Another polyvinyl catheter was inserted into the right internal mammary vein, advanced to the right atrium, filled with a heparin solution, and sealed.

A 3 to 4 cm incision was made in the pericardium overlying the proximal pulmonary artery and left anterior descending coronary artery, and an electromagnetic flow probe was placed on the ascending aorta (Howell Instrument Co., Camarillo, CA, used with a Narcomatic electromagnetic flowmeter, Model RT-500, Narco Biosystems, Inc., Houston, TX). The electromagnetic flow measuring system was calibrated in vivo with the dye dilution cardiac output technique during a steady state at several different cardiac outputs. Cardiac output was varied by graded hemorrhage, pericardial tamponade, and isoproterenol infusion. After completion of the experiment, the aorta and heart were removed with the flow probe in place, and a second calibration was performed with timed collections of normal saline. Two catheters (Tygon Microbore Tubing, 0.050 inch id × 0.090 inch od; Norton Plastics and Synthesis, Akron, OH) were positioned in the pericardial space through separate purse-string sutures with their tips adjacent to the diaphragmatic surface of the left ventricle. The pericardium was carefully sealed with a continuous locking suture and three electrocardiogram leads were sutured to the parietal pleura. A chest tube was placed and all catheters and wires were passed individually through the chest wall and tunneled subcutaneously to an area between the scapulae. The ribs were approximated with two bands of umbilical tape, and the muscle, subcutaneous tissue, and skin were closed in layers to provide an airtight seal. All catheters were flushed, filled with a heparin solution, and capped. The chest was evacuated by gentle suction on the chest tube, and the dog was fitted with a vest having a small pocket into which all catheters and transducer leads were placed. The pericardial cavity was emptied, 30 to 40 ml of sterile saline was placed in it, and the pericardial catheters were sealed. The animal was extubated and allowed to recover for 3 to 4 days.

After recovery from surgery, the conscious dog was allowed to stand comfortably in a sling. The aortic, right atrial, and pericardial catheters were attached directly to Statham P23Db pressure transducers (Statham Instrument Co., Hato Rey, PR) with the zero-pressure reference point one-third of the distance between the sternum and spine. Respiration was measured by recording the change in electrical resistance in a small mercury-filled silastic tube (Whitney gauge) placed around the thorax. The pericardium and pleural space were drained, and control data were recorded when the animal was comfortable and steady-state situation had been achieved. When necessary, normal saline at body temperature was infused intravenously so that mean right atrial pressure in all animals was between 0 and 4 mm Hg during the control period. Pericardial tamponade was produced by continuous infusion of 0.9% saline at 37°C into the pericardial space at a rate of 10 ml/min with a Masterflex infusion pump (Cole Palmer Instrument Co., Chicago, IL). During the infusion, hemodynamic data were continuously recorded by an FM tape recorder (A. R. Vetter Co., Rebersburg, PA). The same data were also recorded every 2 min on a Gould strip chart recorder (Model 2800; Gould, Inc., Cleveland, OH). Short-axis two-dimensional echocardiograms were obtained with a handheld transducer in the right fourth or fifth intercostal space and a Varian V-3000 phased array echocardiograph (Varian Associates, Palo Alto, CA). The echocardiographic data were recorded on videotape by a Sony Betamax video-cassette recorder (Model SLO-323; Sony Corp., New York, NY). The echocardiograms were reviewed with a Microsonics Image Analyzer (Microsonics, Inc., Indianapolis, IN). End-systole was assumed to be associated with the smallest left ventricular cross-sectional area. Short-axis two-dimensional echocardiograms were studied in real time and slow motion, and RVDC was considered to be present if there was any indentation or abnormal posterior motion of the endocardium of the right ventricular free wall in diastole. Because of the location of the ultrasonic transducer in these animals, a derived M mode study often would not include the portion of the right ventricular wall showing RVDC, although it was clearly present on the two-dimensional echocardiogram. All echocardiograms were reviewed independently by two observers, and a consensus was reached regarding the presence or absence of RVDC. The appearance of RVDC was so striking and its onset frequently so abrupt that the volume of pericardial fluid causing it to occur could often be determined within 5 ml.

Decompensated cardiac tamponade was defined as a drop in mean aortic pressure to 70% of the control level. Stroke volume was calculated from cardiac output and heart rate. All hemodynamic data were measured by averaging two full respiratory cycles, and each dog underwent between two and five experiments (average 3.5). A maximum of two experiments were performed in 1 day on a single animal, with sufficient time for recovery between experiments. The first experiment was performed in group A animals between 4 and 13 days (mean 5.8 days) and the last between 4 and 19 days (mean 8.7 days) after surgery.

To more clearly understand the mechanism causing RVDC in cardiac tamponade, further experiments were performed in four additional chronically prepared dogs (group B) weighing from 23 to 31 kg. These studies tested the hypothesis that RVDC was caused by pericardial pressure exceeding right ventricular pressure during diastole. If this hypothesis were true, it would suggest that RVDC might occur later in the course of cardiac tamponade or may not be found at all in the presence of increased resistance to right ventricular outflow. In these animals a hydraulic occluder (In Vivo Metro Systems, Healdsburg, CA) was positioned around the main pulmonary artery. When inflated, the device caused partial occlusion of that vessel. A No. 6F catheter with both a fluid-filled lumen and a high-fidelity pressure transducer near its tip (Mikrotip Pressure Transducer Model PC-460; Millar Instruments, Inc., Houston, TX) was inserted into the left internal mammary vein and advanced to the right
ventricle. A second high-fidelity pressure transducer was positioned in the pericardial space through a purse-string suture in the pericardium, with the tip at the level of the middle right ventricle. The surfaces of both catheter tip pressure transducers were protected from contact with adjacent structures but were allowed to remain in free contact with the surrounding liquid. In the pericardial space this was accomplished with a small open-ended cylinder 5 mm in diameter, which was just long enough to shield the transducer face. In the right ventricle this was done by careful adjustment of the unshielded catheter tip transducer. Fluid-filled pericardial and right ventricular catheters were referenced to the zero-pressure level, and the solid-state transducers in those two locations were adjusted accordingly.

The protocol for these additional experiments was identical to that mentioned previously, except that as soon as RVDC appeared, the pulmonary artery occluder was manually inflated. The amount of pulmonary artery occlusion produced by the device was determined in the control state with the pericardial space empty and was sufficient to increase the right ventricular systolic pressure between 10 to 20 mm Hg without causing irreversible hemodynamic compromise. Partial occlusion was maintained for 30 sec, and after release, the infusion into the pericardial space continued. This procedure was repeated with the same degree of occlusion at progressively greater pericardial volumes. The entire protocol was repeated at least once in each animal, with ample time for recovery between infusions. Two animals were studied on the first and second postoperative days and two animals were studied on the fifth postoperative day. Right ventricular diastolic pressure in Group B animals was measured at the lowest diastolic pressure, and intrapericardial diastolic pressure was taken at the same time. Early diastolic pressures were used because RVDC occurred during that part of the cardiac cycle.

The positions of all catheters were carefully checked at autopsy. Statistical analysis of the data was by analysis of variance and least significant difference test. All results are expressed as mean \( \pm 1 \) SD.

**Results**

**Hemodynamics.** Figure 1 graphically displays the protocol and the hemodynamic changes seen in dog 4 (group A) from the control state with empty pericardium to the point of decompensated cardiac tamponade. RVDC was first observed at a pericardial volume of 100 ml in this animal and persisted to the end of the experiment, at which time mean aortic pressure was less than 70\% of the control value. Table 1 presents the tabulated hemodynamic data recorded from all group A animals during the control state, when RVDC was first seen, and at the time of decompensated cardiac tamponade. Figure 2 summarizes these hemodynamic changes. When RVDC was first seen on the two-dimensional echocardiogram, the cardiac output had decreased significantly from a control level of 3.09 ± 0.55 to 2.44 ± 0.6 l/min (p < .01). The mean aortic pressure, however, had not changed significantly from the control value of 103 ± 13 to 100 ± 15 mm Hg (NS). Heart rate was significantly increased from 116 ± 18 to 159 ± 34 beats/min (p < .01), and mean intrapericardial pressure increased from 0 ± 2 to 10 ± 4 mm Hg (p < .01). Stroke volume had also changed substantially from 27 ± 6 to 16 ± 6 ml (p < .01). Although not measured in all dogs, right ventricular end-diastolic pressure and right atrial pressures, when recorded, showed an increase similar to the increase in intrapericardial pressure. Dog 1 became agitated during the progression of cardiac tamponade, and the experiment could not be continued to the point of decompensated cardiac tamponade. Except for dog 6, none of the dogs showed a significant change in mean aortic pressure from the control state to the point of earliest occurrence of RVDC. In contrast, each animal showed a significant reduction in cardiac output at the time RVDC was first seen (p < .01 to p < .05). With progression of cardiac tamponade to the decompensated state, all the hemodynamic variables changed significantly. Mean intrapericardial pressure increased to 21 ± 6 mm Hg (p < .01), heart rate increased to 209 ± 17 beats/min (p < .01), mean aortic pressure de-

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**Table 1**

**Hemodynamic variables (mean ± SD) of all six dogs in group A**

<table>
<thead>
<tr>
<th>Dog</th>
<th>IPP (mm Hg)</th>
<th>HR (bpm)</th>
<th>MAP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>C</td>
<td>RVDC</td>
<td>DCT</td>
</tr>
<tr>
<td>1^</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>0 ± 2</td>
<td>7 ± 2</td>
<td>26 ± 11</td>
</tr>
<tr>
<td>4</td>
<td>−1 ± 1</td>
<td>7 ± 1</td>
<td>18 ± 2</td>
</tr>
<tr>
<td>5</td>
<td>0 ± 1</td>
<td>12 ± 3</td>
<td>22 ± 1</td>
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<tr>
<td>6</td>
<td>3 ± 2</td>
<td>14 ± 0</td>
<td>15 ± 1</td>
</tr>
<tr>
<td>Mean</td>
<td>0 ± 2</td>
<td>10 ± 4^b</td>
<td>21 ± 6^c</td>
</tr>
</tbody>
</table>

C = empty pericardial space (control); RVDC = first occurrence of RVDC; DCT = decompensated cardiac tamponade; IPP = mean intrapericardial pressure; HR = heart rate; MAP = mean aortic pressure; CO = cardiac output; SV = stroke volume.

Every dog had three experiments except dog 6, which had two.

Statistical comparisons: ^bp .01 vs C; ^p < .01 vs C and RVDC.
TABLE 1
(Continued)

<table>
<thead>
<tr>
<th></th>
<th>CO (l/min)</th>
<th>SV (ml)</th>
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<tbody>
<tr>
<td></td>
<td>C</td>
<td>RVDC</td>
</tr>
<tr>
<td>C</td>
<td>RVDC</td>
<td>DCT</td>
</tr>
<tr>
<td>2.77± .08</td>
<td>2.38± .13</td>
<td>—</td>
</tr>
<tr>
<td>3.69± .53</td>
<td>3.34± .42</td>
<td>1.04± .36</td>
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<tr>
<td>2.45± .14</td>
<td>1.89± .33</td>
<td>.95± .12</td>
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<tr>
<td>3.80± .21</td>
<td>3.04± .16</td>
<td>1.47± .32</td>
</tr>
<tr>
<td>2.92± .18</td>
<td>2.12± .33</td>
<td>.47± .08</td>
</tr>
<tr>
<td>3.16± .06</td>
<td>2.03± .12</td>
<td>1.68± .12</td>
</tr>
<tr>
<td>3.90± .55</td>
<td>2.44± .68</td>
<td>1.03± .47</td>
</tr>
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</table>

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FIGURE 1. Representative experiment in dog 4 (group A), illustrating the hemodynamic changes characteristically seen during experimental cardiac tamponade. Pericardial volume (Peric. Vol.) was continuously increased at a rate of 10 ml/min. Mean intrapericardial pressure (IPP), heart rate (HR), mean arterial pressure (MAP), cardiac output (CO), and two-dimensional echocardiograms were recorded at 2 min intervals. RVDC was first observed at a pericardial volume of 100 ml and persisted to the end point of the experiment (MAP <70% of control). CO and SV declined consistently as tamponade progressed. MAP, however, was well maintained at the time RVDC was first observed and fell rapidly late in the course of cardiac tamponade.

FIGURE 2. Mean intrapericardial pressure (IPP), heart rate (HR), mean arterial pressure (MAP), cardiac output (CO), and stroke volume (SV) with empty pericardial space (C) at first occurrence of RVDC and in decompensated cardiac tamponade (DCT) of all six dogs in group A. At the time when RVDC is first seen, CO and SV are substantially reduced without a significant change in MAP. Data expressed as mean ± SD. NS = no significant change vs C. *p < .01 vs C; †p < .01 vs C and RVDC.

clined to 66 ± 11 mm Hg (p < .01), cardiac output further decreased to 1.03 ± 0.47 l/min (p < .01), and stroke volume decreased to 5 ± 2 ml (p < .01).

In one additional animal we were unable to observe RVDC despite pericardial infusion to the point of decompensated cardiac tamponade and circulatory collapse. At postmortem examination this animal was found to have marked right ventricular hypertrophy (right ventricular free wall thickness was 10 mm; left ventricular free wall thickness was 12 mm; total weight of the heart was 280 g) and a severe "heart worm" (Dirofilaria immitis) infestation was present. This animal was excluded from statistical analysis because of the presence of right ventricular hypertrophy. All other animals were free of "heart worm" disease.

In group B animals, RVDC was eliminated or mark-
TABLE 2
Hemodynamic variables (mean ± SD) of the four group B animals

<table>
<thead>
<tr>
<th>RVs (mm Hg)</th>
<th>RVd (mm Hg)</th>
<th>IPPd (mm Hg)</th>
<th>HR (bpm)</th>
<th>MAP (mm Hg)</th>
<th>CO (l/min)</th>
<th>SV (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVDC</td>
<td>31±13</td>
<td>6±3</td>
<td>6±3</td>
<td>170±22</td>
<td>81±13</td>
<td>1.82±0.77</td>
</tr>
<tr>
<td>PAO</td>
<td>47±16</td>
<td>10±3</td>
<td>8±5</td>
<td>201±26</td>
<td>61±10</td>
<td>0.81±0.66</td>
</tr>
<tr>
<td>p value</td>
<td>&lt;.01</td>
<td>&lt;.05</td>
<td>NS</td>
<td>&lt;.05</td>
<td>&lt;.01</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

PAO = pulmonary artery obstruction; RVs = right ventricular systolic pressure; RVd = right ventricular early diastolic pressure; IPPd = intrapericardial early diastolic pressure; HR = heart rate; MAP = mean aortic pressure; CO = cardiac output; SV = stroke volume.

*At the first occurrence of RVDC and at the same degree of cardiac tamponade during pulmonary artery obstruction with reversal of RVDC. Each dog had two experiments except one dog, which had four.

edly reduced by transient manual inflation of the pulmonary artery hydraulic occluder. The hemodynamic alterations associated with partial pulmonary artery obstruction are summarized in Table 2. At the first occurrence of RVDC, right ventricular and intrapericardial pressures were equalized in early diastole. We could not demonstrate that intrapericardial pressure actually exceeded right ventricular pressure during RVDC. Pulmonary artery obstruction, however, caused a significant increase (p < .05) in right ventricular early diastolic pressure and a slight but nonsignificant increase in intrapericardial early diastolic pressure. This positive right ventricular transmural pressure gradient was associated with the disappearance of RVDC. As expected, partial occlusion of the pulmonary artery caused a marked decrease in mean aortic pressure (p < .01), cardiac output (p < .01), and stroke volume (p < .01) and an increase in heart rate (p < .05). When the partial occlusion of the pulmonary artery was released, RVDC promptly reappeared. The experiments in group B animals were intended to investigate only the effect of intrapericardial and right ventricular pressure on right ventricular wall motion in diastole. Because of this limited goal, these animals were studied sooner after surgery. The hemodynamic situation at the time of RVDC therefore differs from that seen in group A animals and is generally characterized by a higher heart rate and a lower stroke volume, cardiac output, and mean aortic blood pressure.

Echocardiography. Two-dimensional echocardiograms of good quality were obtained in all dogs. In none of the animals was RVDC present when the pericardial space was empty, and all dogs in groups A and B showed RVDC during cardiac tamponade. Once RVDC was present it persisted and became more pronounced as cardiac tamponade progressed. In severe cardiac tamponade the right ventricular cavity was almost completely obliterated and RVDC persisted throughout diastole.

In group A, four dogs had RVDC that began in early diastole. In two dogs, RVDC was more pronounced in middiastole. The collapsing motion of the right ventricle continued until the beginning of the R wave, when atrial systole expanded the right ventricle. In five of six dogs the collapse was most severe in the body of the right ventricle and the outflow tract. In dog 3 the collapse was more pronounced at the apex of the right ventricle. In dogs 3, 4, and 5 the right ventricular size was considerably reduced during cardiac tamponade, and special attention had to be paid to wall motion to detect RVDC. The echocardiogram of dog 6, group A, is shown in Figure 3 as still frames obtained in early diastole and end-systole, with a small pericardial effusion (Figure 3, A), and with a larger effusion at the first appearance of RVDC (Figure 3, B).

Figure 4 illustrates diastolic still frames from dog 3, group B, in the control state with empty pericardium (Figure 4, A) and at a pericardial volume of 100 ml (Figure 4, B) when RVDC was evident. At the same pericardial volume as in Figure 4, B, inflation of the pulmonary artery occluder resulted in disappearance of RVDC (Figure 4, C). We were able to reverse RVDC or markedly diminish it at nearly all degrees of cardiac tamponade except severe cardiac tamponade.

Figure 5 shows the changes in right ventricular pressure and pericardial pressure seen in dog 2, group B, at the first appearance of RVDC (Figure 5, A) and at the same pericardial volume just after partial inflation of the pulmonary artery occluder had led to the abrupt disappearance of RVDC (Figure 5, B). The small early diastolic pressure differential across the right ventricular free wall seen in Figure 5, A, was quickly increased by occluder inflation as right ventricular pressures increased.

Discussion

Pericardial tamponade should be viewed clinically as a continuous spectrum ranging from pericardial ef-
fusion with minimal hemodynamic impairment, which may be asymptomatic to an effusion with severe cardiac compression and circulatory collapse.\textsuperscript{15} The diagnosis and early detection of a hemodynamically important pericardial effusion remains a challenge to the clinician. The original description of RVDC emphasized its clinical value in detecting impending cardiac tamponade,\textsuperscript{9} and our experimental findings corroborate these clinical observations. We have shown in a normally hydrated animal model that the earliest appearance of RVDC is associated with a 21% reduction in cardiac output at a time when mean aortic pressure is unchanged.

Several mechanisms could contribute to the RVDC observed in cardiac tamponade. A negative transmural pressure gradient in diastole between the right ventricular cavity and the pericardial space in open-chested dogs with cardiac tamponade has been demonstrated.\textsuperscript{16} Similarly, in our canine model, infusion of saline into the pericardial space produced a progressive increase in intrapericardial pressure but a lesser increase in right ventricular early diastolic pressure. In group B animals, manual inflation of the pulmonary artery pneumatic occluder in the presence of RVDC promptly increased right ventricular pressure in early diastole and was associated with a marked reduction of RVDC. These observations are consistent with the hypothesis that RVDC is a pressure-related phenomenon and suggests that it may occur later in the course of cardiac tamponade (or not at all) in the presence of an increased right ventricular diastolic pressure. The absence of RVDC during cardiac tamponade in the animal with right ventricular hypertrophy suggests that RVDC may not be a reliable clinical sign in this situation and in others associated with decreased right ventricular compliance.

Compression of the inferior vena cava and superior vena cava as they enter the pericardial space has been demonstrated in cardiac tamponade.\textsuperscript{17} Such compression would be expected to occur if intrapericardial pressure exceeded venous pressure in diastole. This situation could lead to impaired filling of the right atrium and right ventricle causing lower pressures in those chambers than in the pericardial space and result in RVDC. Similar observations suggest that in early cardiac tamponade (before in vivo diastolic pressure

\textbf{FIGURE 3}. Two-dimensional echocardiogram (short axis) of dog 6 (group A) with a small pericardial effusion (A) and with a larger effusion at the first occurrence of RVDC (arrow) (B). Still frames were taken in early diastole and at end-systole. RVDC starts in early diastole and is reversed by atrial contraction. Once RVDC is seen, it persists, and the severity of collapse increases with progression of cardiac tamponade. PE = pericardial effusion; RV = right ventricle; LV = left ventricle.
FIGURE 4. Two-dimensional echocardiograms of dog 3 (group B) with empty pericardial space (A), with RVDC (B), and at the same pericardial volume as in B but with partial pulmonary artery occlusion (C). Pulmonary artery obstruction was sufficient to raise the right ventricular systolic pressure by 26 mm Hg and the right ventricular diastolic pressure by 5 mm Hg, and was associated with the disappearance of RVDC. See legend to figure 3 for abbreviations.

equalization), direct compression of right heart structures is greater than of left heart structures. Collapse of the right atrium during late diastole and/or isovolumic systole has been reported. As we observed, atrial systole appears to generate enough pressure to reverse RVDC. One could speculate that when RVDC is seen, it may indicate an impairment of right ventricular filling caused by a hemodynamically significant pericardial effusion.

Since we did not see RVDC in our study without a significantly diminished cardiac output and stroke volume, we are unable to comment on the specificity of RVDC. Perhaps events that increase the compliance of the right ventricular free wall may lead to RVDC with hemodynamically unimportant pericardial effusions. The left ventricle, perhaps because of its increased thickness and symmetric shape, does not show any discrete collapse during diastole in cardiac tamponade in spite of a negative transmural pressure.

The sensitivity of RVDC has previously been emphasized. Armstrong et al. concluded that “the presence of normal right ventricular free wall motion in patients with moderate to large pericardial effusions is a strong indicator that the effusion is not hemodynamically significant.” However, our study suggests that if resistance to right ventricular outflow is increased or right ventricular compliance is decreased, RVDC may be absent even in the presence of severe cardiac tamponade. It has been reported that RVDC was absent in a patient who had constrictive pericarditis and cardiac tamponade, suggesting that the decreased right ventricular compliance seen in this condition may have prevented RVDC. In preliminary experiments we documented the time course of changes in pericardial compliance in our animal model. We selected a postoperative period for study in which the pericardium is still compliant (i.e., from 3 to 20 days postoperatively). There is a remarkable decrease in pericardial com-
other change in pericardial compliance throughout the period of data collection. Since this animal model does not permit multiple two-dimensional echocardiographic views, no attempt was made to calculate right or left ventricular volume. Although respiratory variation in the degree of RVDC was observed in some animals, no systematic analysis of the influence of respiration on RVDC was made in this study.

We conclude that RVDC seen in this canine model of cardiac tamponade occurs before a fall in systemic arterial pressure but is associated with a reduction in cardiac output of at least 20%. RVDC is probably caused by right ventricular pressure falling below pericardial pressure in early diastole. Although the sensitivity and specificity of RVDC in cardiac tamponade remain to be established in humans, these data suggest that RVDC may occur later in the course of cardiac tamponade or not at all in the presence of increased resistance to right ventricular outflow or decreased right ventricular compliance.

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