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# Influence of Acute Right Ventricular Dysfunction on Cardiac Tamponade

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**Echocardiographic and hemodynamic data were measured in nine closed chest dogs during graded cardiac tamponade (pericardial pressure 5, 10, 15 mm Hg) before and after production of diffuse acute ischemic right ventricular dysfunction. Right ventricular dysfunction was produced by intracoronary injection of nonradioactive microspheres (mean diameter ± SD 54 ± 4 μm) and caused a significant increase in right atrial pressure (7.6 ± 1.4 vs. 1.6 ± 1 mm Hg, $p < 0.001$) and cross-sectional areas of both the right atrium (8.3 ± 0.3 vs. 5.6 ± 0.2 $cm^2$, $p < 0.001$) and right ventricle (8.8 ± 0.4 vs. 5.7 ± 0.4 $cm^2$, $p < 0.001$).**

**Right atrial and ventricular collapse required a significantly larger pericardial effusion and pericardial pressure after right ventricular infarction than before. Mean aortic pressure had fallen 1.9 ± 2% and 6.5 ± 6.9% at the time of right atrial collapse (p = NS before vs. after right ventricular dysfunction) and 3 ± 4.1% and 20.1 ± 20.8% at the time of right ventricular collapse ($p < 0.03$) before and after right ventricular dysfunction, respectively.**

**In the presence of ischemic right ventricular dysfunction, echocardiographic signs of cardiac tamponade are less sensitive and occur later in the hemodynamic progression of cardiac tamponade. Pulsus paradoxus with cardiac tamponade was not prevented by coexisting ischemic right ventricular dysfunction.**

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Cardiac tamponade is a hemodynamic condition characterized by equal elevation of atrial and pericardial pressures, an exaggerated inspiratory decrease in arterial systolic pressure (pulsus paradoxus) and 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, cardiac tamponade complicating coexisting heart disease may be difficult to diagnose. For example, in an animal model of cardiac tamponade and left ventricular dysfunction we recently showed that pulsus paradoxus may be absent (1,6); conversely, in that study right-sided cardiac chamber collapse occurred with small, otherwise insignificant pericardial effusions (6). Although cardiac tamponade may also complicate right ventricular dysfunction, there are no available data on the hemodynamics of cardiac tamponade in this setting. In addition, although it has been reported that right ventricular diastolic collapse is sensitive to abrupt alterations in right and left ventricular loads (7,8), the effect of right ventricular dysfunction on echocardiographically determined chamber collapse during cardiac tamponade has not been systematically examined.

Accordingly, we studied cardiac tamponade in an animal model of ischemia-induced right ventricular dysfunction to test the hypothesis that right atrial and ventricular collapse occur with larger pericardial effusions and later in the hemodynamic course of tamponade when ischemic right ventricular dysfunction is present. We also compared the hemodynamics of cardiac tamponade with coexisting right ventricular dysfunction with those of cardiac tamponade alone.

## Methods

**Initial experimental preparation.** Studies were performed in nine heartworm-free mongrel dogs of either gender (mean weight ± SD 22.1 ± 5.7 kg) anesthetized with intravenously administered sodium pentobarbital (25 mg/kg body weight), intubated and ventilated with a positive pressure respirator. 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 subcutaneously 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), 2 ml intramuscularly, was administered daily.

**Early studies.** Five to 7 days later, the dogs were anesthetized with sodium pentobarbital (25 mg/kg body weight intravenously) and allowed to breathe spontaneously. Additional doses of anesthesia were administered as necessary. Prophylactic quinidine, 320 mg, was administered intramus-

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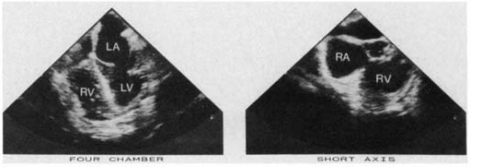


**Figure 1.** Transesophageal echocardiographic images illustrating cardiac chambers in the four-chamber (left) and short-axis (right) orientation. LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

cularly 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 $Po_2$ and acid-base balance. A flow-directed triple-lumen thermodilution catheter was advanced into the pulmonary artery through 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 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 MHz imaging and 2 MHz Doppler phased array transducer mounted on the distal tip of 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 0 pressure set at the level of the mid-right atrium. Temperature was monitored with 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 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 to 20 ml aliquots of physiologic saline solution at 38°C into the pericardial space. 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. In four of the dogs, cardiac output determinations were also performed at the time of right atrial and ventricular collapse. The pericardial fluid was withdrawn after observations at three levels of pericardial pressure and the dogs were allowed to recover until cardiac pressures stabilized for approximately 30 to 60 min.

*Acute right ventricular infarction was then produced in the following manner.* A Judkins right coronary artery catheter was advanced through a carotid artery to the right coronary artery under fluoroscopic control. Catheter tip placement was verified by test injections of contrast medium. Nonradioactive tracer microspheres (3M) 54 ± 4 μm in diameter, suspended in 20% dextran, were prepared, as previously described (6). One milliliter of microspheres (approximately $6 \times 10^6$ spheres) was mixed in a syringe with 1 ml of angiographic contrast medium (Angiovist 370, Berlex Labs) and injected into the right coronary artery. Boluses of microsphere solution were followed by a 1 ml flush of angiographic contrast medium. Intracoronary injections were repeated until the mean right atrial pressure was elevated to at least twice baseline. After 15 min 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/s) on a Grass 7D multichannel recorder. Two-dimensional echocardiographic studies were performed in the spontaneously breathing dogs with a Hewlett-Packard 7750C ultrasonograph, with data recorded on 0.5 in. (1.27 cm) VHS videotape. The experimental protocol was approved by the Institutional Animal Care and Use Committee at the University of Cincinnati. The study conforms to the "Position of the American Heart Association on Research Animal Use."

**Two-dimensional echocardiographic measurements.** Images were analyzed with use of an off-line image analysis system (GTI, Freeland Medical). The right ventricular chamber area was measured from the transesophageal four-chamber view and that of the right atrium from the transesophageal short-axis view (Fig. 1). End-diastolic atrial chamber areas were determined by planimetry from the video frame with the largest atrial area just before atrial systole and ventricular areas were determined by planimetry from the video frame containing the largest ventricular area. Three to five area determinations were made for each cardiac chamber and averaged. In addition, at baseline (before instillation of saline solution into the pericardial space) and before and after production of ischemic right ventricular dysfunction, end-systolic right ventricular areas were determined by planimetry from the video frame containing the smallest ventricular area. The right ventricular area shortening fraction was calculated as:

**Table 1.** Hemodynamic Values in Nine Dogs

| | No RV Dysfunction | | | | RV Dysfunction | | | |
|---|---|---|---|---|---|---|---|---|
| | Baseline | Tamp 1 | Tamp 2 | Tamp 3 | Baseline | Tamp 1 | Tamp 2 | Tamp 3 |
| Peri P (mm Hg) | −0.8 ± 2.1 | 5.0 ± 0* | 10.2 ± 0.4*† | 14.8 ± 0.4*†‡ | 0.2 ± 1.5 | 5.2 ± 0.3* | 9.9 ± 0.3*† | 14.6 ± 1.6*†‡ |
| RAP (mm Hg) | 1.6 ± 1 | 5.3 ± 1.2* | 10.2 ± 1.2*† | 14.6 ± 0.9*†‡ | 7.6 ± 1.48 | 8.5 ± 1.5*§ | 11.6 ± 1.9*†§ | 15.1 ± 2*†‡ |
| AoP (mm Hg) | 127.2 ± 14.4 | 125.6 ± 15.1 | 115.6 ± 18.1*† | 88.3 ± 2.3*†‡ | 128.1 ± 15.3 | 123.3 ± 15 | 114.8 ± 15* | 90.6 ± 15.7*†‡ |
| Ao insp (mm Hg) | 5.8 ± 1.1 | 10.9 ± 3.6* | 16 ± 4.8*† | 17 ± 3.8*‡ | 8.8 ± 38 | 10.4 ± 2.6 | 14 + 3.9*† | 15.3 ± 4.4*† |
| LAP (mm Hg) | 4.7 ± 1.6 | 5.9 ± 1.1* | 10.3 ± 1.4*† | 14.1 ± 1.8*†‡ | 8.3 ± 28 | 9.3 ± 2*§ | 11.3 ± 1.8*† | 13.7 ± 2.5*†‡ |
| HR (beats/min) | 169.4 ± 26.6 | 172.8 ± 18.6 | 193.9 ± 30.1 | 185 ± 33.8 | 138.9 ± 34.2§ | 143.4 ± 30.9§ | 150.1 ± 39.2§ | 148.1 ± 33.9§ |
| CO (liters/min) | 1.57 ± 0.47 | 1.41 ± 0.47* | 1 ± 0.36*† | 0.53 ± 0.16*†‡ | 0.98 ± 0.38§ | 0.89 ± 0.28§ | 0.72 ± 0.28*†§ | 0.47 ± 0.97*†‡ |
| SV (cc/beat) | 9.2 ± 2.1 | 8 ± 1.8* | 5.5 ± 1.9*† | 2.9 ± 0.8*†‡ | 7.4 ± 1.8§ | 6.6 ± 1.4*‡ | 4.9 ± 1.2*† | 3.3 ± 0.9*†‡ |

*$p < 0.05$ versus Baseline, †$p < 0.05$ versus Tamp 1, ‡$p < 0.05$ versus Tamp 2, §$p < 0.05$ versus no right ventricular dysfunction. Ao insp = inspiratory fall in aortic systolic pressure; AoP = mean aortic pressure; CO = cardiac output; HR = heart rate; LAP = mean left atrial pressure; Peri P = pericardial pressure; RAP = mean right atrial pressure; RV = right ventricular; SV = stroke volume; Tamp = tamponade level.

$$\frac{\text{End-diastolic area} - \text{End-systolic area}}{\text{End-diastolic area}}$$

***Right atrial collapse was identified*** by inversion of the respective atrial free wall at any point of the cardiac cycle (3). Right ventricular diastolic collapse was identified by a persistent inward motion of any portion of the endocardial surface during diastole after tricuspid valve opening (4). Real time, slow motion and frame by frame analysis were used to identify these events. Videotape replay was also used to confirm chamber collapse in the four dogs studied with cardiac output determinations at the time of collapse.

**Interobserver differences.** Thirty right atrial and right ventricular images were randomly selected and digitized by two independent observers. Interobserver differences were calculated as the difference between two observations divided by the mean of the two observations. The onset of cardiac chamber collapse was determined by a consensus of two observers.

**Hemodynamic data.** Pulsus paradoxus was defined by an absolute decrease in aortic systolic pressure of ≥10 mm Hg during inspiration (expiratory minus inspiratory systolic pressure). 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 a repeated measures analysis of variance with use of a commercially available statistical program (SuperANOVA, Abacus Concepts). When a significant interaction between severity of tamponade and right ventricular dysfunction was found, contrasts were used to determine where those differences were significant. 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 right ventricular dysfunction were compared with Student's paired *t* tests. Pericardial pressure and cardiac output were compared with linear regression both before and after left ventricular dysfunction. The slopes of the linear regression were compared with a *t* test. Data are expressed as mean values ± SD. In all comparisons, a p value <0.05 was considered statistically significant.

## Results

**Hemodynamic measurements (Table 1).** Microsphere injections into the right main coronary artery resulted in significant increases in mean right and left atrial pressures and significant decreases in cardiac output, stroke volume and heart rate without significant changes in mean aortic or pericardial pressure.

When cardiac tamponade was produced at three stages of intrapericardial pressure, pericardial and right atrial pressures increased significantly both before and after microsphere administration. Cardiac output decreased significantly with tamponade and, except at the third level of tamponade, was significantly lower at each stage after production of ischemic right ventricular dysfunction. There was a significant negative correlation between pericardial pressure and cardiac output both before and after ischemic right ventricular dysfunction (both $p < 0.001$) (Fig. 2). The difference in slopes (−0.067 ± 0.010 [SE] versus −0.033 ± 0.008) was significant ($p < 0.05$). Thus, despite a lower baseline cardiac output, a greater increase in pericardial

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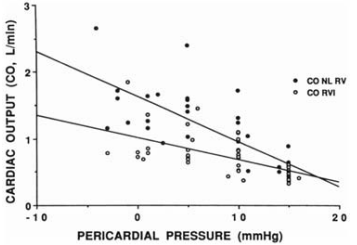


**Figure 2.** Plot of linear regression between cardiac output (CO) and pericardial pressure before (NL RV, **closed circles**) and after production of acute right ventricular dysfunction (RVI, **open circles**) in nine dogs. The slope of the two regression lines are significantly different. See text for details. NL RV = normal right ventricular function.

pressure (and larger effusion) was needed to effect a similar decrease in cardiac output after right ventricular dysfunction. Despite these differences, the mean arterial blood pressure decreased only in the latter stages of tamponade both before and after ischemic right ventricular dysfunction.

*After ischemic right ventricular dysfunction, pulsus paradoxus was seen in four dogs without tamponade.* The mean inspiratory fall in aortic systolic pressure was significantly greater after production of ischemic right ventricular dysfunction (8.8 ± 3 vs. 5.8 ± 1.1 mm Hg, $p < 0.05$). With cardiac tamponade, the magnitude of pulsus paradoxus was similar at each stage of tamponade before and after production of ischemic right ventricular dysfunction.

**Echocardiographic chamber areas and chamber collapse.** Microsphere injections resulted in significant increases in right atrial (8.3 ± 0.3 vs. 5.6 ± 0.2 $cm^2$, $p < 0.001$) and ventricular (8.8 ± 0.4 vs. 5.7 ± 0.4 $cm^2$, $p < 0.001$) areas and decreased right ventricular area shortening fraction (0.45 ± 0.09 vs. 0.16 ± 0.08, $p < 0.001$). Significant decreases in both right atrial and ventricular chamber areas occurred at each stage of cardiac tamponade before and after production of ischemic right ventricular dysfunction (Fig. 3).

*With cardiac tamponade*, right atrial collapse was identified in all dogs and right ventricular collapse was seen in eight of nine dogs both before and after production of ischemic right ventricular dysfunction. The volume of pericardial fluid at the onset of atrial and ventricular collapse is shown in Figure 4. Right atrial and ventricular collapse occurred with larger volumes of pericardial fluid after ischemic right ventricular dysfunction. Cardiac pressures at the time of chamber collapse are shown in Table 2. Pericardial and atrial pressures at right atrial and ventricular collapse were significantly higher after ischemic right ventricular dysfunction. Mean aortic pressure was significantly less at the time of right ventricular, but not atrial, collapse after

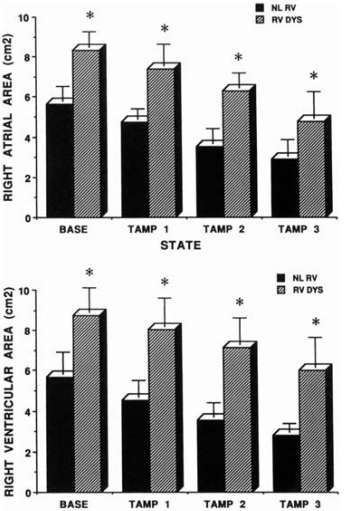


**Figure 3.** Effect of cardiac tamponade (TAMP) and right ventricular dysfunction (RV DYS) on right atrial (**top panel**) and ventricular (**bottom panel**) areas measured from transesophageal short-axis and long-axis views, respectively. With cardiac tamponade, right atrial and right ventricular areas decreased. At each level of tamponade, chamber areas were larger after right ventricular dysfunction. *$p < 0.05$ versus normal right ventricular function. Other abbreviation as in Figure 2.

right ventricular dysfunction was produced. Mean aortic pressure had fallen 1.9 ± 2% and 6.5 ± 6.9% at the time of right atrial collapse ($p$ = NS before vs. after right ventricular dysfunction) and 3 ± 4.1% and 20.1 ± 20.8% at the time of right ventricular collapse ($p < 0.03$) before and after ischemic right ventricular dysfunction, respectively.

*In the four dogs in which cardiac output determinations were made at the time of chamber collapse*, cardiac output had fallen 3.2 ± 5.5% and 19.2 ± 18.7% at the time of right atrial collapse ($p$ = NS) and 6.3 ± 5% and 38.2 ± 11.2% at the time of right ventricular collapse ($p < 0.05$) before and after ischemic right ventricular dysfunction, respectively (Fig. 5).

**Interobserver differences.** The percent interobserver error for measurements of right atrial and ventricular areas was 16.3 ± 9.4% and 3.5 ± 15.6%, respectively. Observers agreement on the onset of cardiac chamber collapse was within 20 ml of pericardial fluid in each dog.

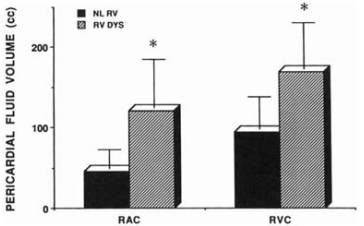


**Figure 4.** Amount of fluid added to the pericardial space to produce right atrial collapse (RAC) and right ventricular collapse (RVC). Larger pericardial fluid volumes were needed for right atrial and right ventricular collapse after production of right ventricular dysfunction (RV DYS). *$p < 0.05$ versus normal right ventricular function. Other abbreviation as in Figure 2.

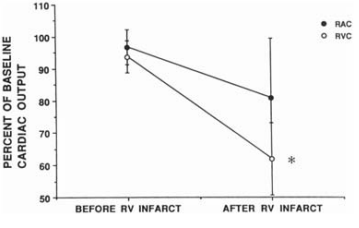


**Figure 5.** The decrease in cardiac output expressed as a percent of baseline output at the time of right atrial collapse (RAC) and right ventricular collapse (RVC) before and after production of acute right ventricular dysfunction (RV INFARCT). *$p < 0.05$ versus before right ventricular dysfunction. Values are expressed as mean values ± SD.

## Discussion

The study results indicate that in the presence of acute ischemic right ventricular dysfunction cardiac chamber collapse occurs later in the hemodynamic progression of acute cardiac tamponade in the anesthetized dog; that is, right atrial and ventricular collapse required larger pericardial effusion and higher pericardial and atrial pressures. At the time of right ventricular collapse, the percent decrease in mean aortic pressure and cardiac output from baseline value was greater in dogs after production of ischemic right ventricular dysfunction.

**Relation between intracardiac pressures and volume and chamber collapse.** Previous studies (8,9) have shown that in normovolemic conscious animals the onset of right ventricular diastolic collapse occurs when cardiac output has fallen 11% to 20% from control levels but before a significant decrease in mean arterial pressure. We found that both right atrial and ventricular collapse occurred early in the acute anesthetized dog; cardiac output had fallen only 3% and 6% from baseline at the time of right atrial and ventricular collapse, respectively. Expansion of the intravascular volume delays the onset of right ventricular diastolic collapse (8), which occurs at higher aortic, atrial and pericardial pressures. In contrast, hypovolemia hastens the onset of right ventricular diastolic collapse, which occurs at lower aortic, atrial and pericardial pressures (8). Thus, the early appearance of right heart chamber collapse in our dogs might be explained by relative hypovolemia in our preparation.

**Role of ischemic right ventricular dysfunction in cardiac tamponade.** In our study, ischemic right ventricular dysfunction changed the relation between pericardial pressure and cardiac output, so that a greater change in pericardial pressure was necessary to effect a similar decrease in cardiac output. Similar results were found with cardiac tamponade in animals with volume expansion (8). These data suggest that resistance to right heart compression, whether from volume loading or right ventricular dysfunction, attenuates the decrease in cardiac output with tamponade. Furthermore, these data support the contention (10) that right heart compression is an important mechanism of this decrease. Because of this "protective effect," echographic signs of cardiac tamponade occur later and, at least for right ventricular diastolic collapse, they occur when there has been a significant deterioration of mean aortic pressure and cardiac output. We cannot exclude the possibility that elevated left atrial pressure influenced the compression effects of cardiac tamponade on cardiac output.

**Right atrial versus right ventricular collapse.** Right atrial collapse is a more sensitive but less specific marker for

**Table 2.** Pressures at the Time of Chamber Collapse in Nine Dogs

| | Right Atrial Collapse | | Right Ventricular Collapse | |
|---|---|---|---|---|
| | Before RVD | After RVD | Before RVD | After RVD |
| Peri P | 2.5 ± 1 | 8.1 ± 1.8* | 5.3 ± 1.8 | 11.6 ± 1.9* |
| RAP (mm Hg) | 2.9 ± 1.4 | 9.8 ± 2.8* | 5.1 ± 2 | 12.2 ± 2* |
| AoP (mm Hg) | 125 ± 14.6 | 117.9 ± 12.3* | 123.8 ± 16.6 | 98.8 ± 22.8* |
| Ao insp (mm Hg) | 7.1 ± 2.4 | 11.8 ± 4* | 10.2 ± 4.6 | 15 ± 3.5* |
| LAP (mm Hg) | 4.2 ± 1.1 | 10.2 ± 1.8* | 5.5 ± 1.2 | 11.8 ± 2* |

*$p < 0.05$ versus before right ventricular dysfunction (RVD). Other abbreviations as in Table 1.

cardiac tamponade compared with right ventricular collapse (3,5). Both phenomena have been explained by a transient negative transmural pressure gradient across the chamber wall and both indicate increased pericardial pressure relative to pressure in the respective cardiac chamber. The earlier appearance of right atrial collapse is probably due to greater atrial than ventricular compliance. At the time of right atrial collapse, mean aortic pressure and cardiac output were not changed significantly from baseline, either before or after production of ischemic right ventricular dysfunction.

An interesting observation is that right heart chamber collapse occurred at a time when mean right atrial pressure exceeded mean pericardial pressure. This is probably due to the phasic nature of chamber collapse; thus, right atrial collapse occurs during late diastole and early systole and right ventricular collapse occurs during early diastole when the instantaneous pericardial pressure exceeds the pressure in the respective chamber. However, regional hydrostatic differences in intrapericardial pressure and ventricular diastolic suction (11) cannot be excluded.

**Role of elevated right heart pressures.** Isolated reports in animal studies (9) and humans (5,12) have suggested that right-sided chamber collapse may be absent when right heart pressures are elevated. Right ventricular collapse was absent in a single dog with right ventricular hypertrophy (9). In a patient with severe pulmonary hypertension and normal left heart pressures, cardiac tamponade was accompanied by left, but not right, ventricular diastolic collapse (12). Although not specifically examined in our study, left atrial collapse did not occur before right heart collapse; left ventricular diastolic collapse was not observed. This finding may be a consequence of elevated left heart diastolic pressures, reflected in the increase in mean left atrial pressure measured after production of ischemic right ventricular dysfunction.

**Hemodynamics of right ventricular dysfunction and cardiac tamponade.** Right coronary microembolization caused an increase in right atrial and right ventricular size, increased right atrial pressures and a decrease in cardiac output due to decreased stroke volume and heart rate. Pericardial restraint of the dilated right heart chambers and ventricular interaction could explain the equalization of elevated right and left atrial pressures. Another possible explanation for the elevated left atrial pressure after right coronary embolization is that portions of the interventricular septum were infarcted. Some degree of septal involvement was present in nearly 60% of right ventricular infarctions created in dogs by injections of metallic mercury into the right coronary artery (13). In that study there was no evidence of septal involvement in 6 of 14 dogs; in 6 other dogs <5% of the septum was necrotic. In the remaining two dogs, infarction involved 15% and 20% of the septum. Thus, it is unlikely that septal infarction contributed significantly to our findings. Motion of the interventricular septum was not evaluated in the present study.

*Pulsus paradoxus may be seen in right ventricular infarction (14).* An inspiratory decrease in aortic systolic pressure sufficient to constitute pulsus paradoxus was present in several dogs after production of right ventricular dysfunction but before infusion of pericardial fluid. Thus, the hemodynamics in our model resemble clinical right ventricular infarction in many respects.

*The hemodynamics of cardiac tamponade were similar before and after production of acute right ventricular dysfunction.* Pericardial and atrial pressures increased and cardiac output and stroke volume decreased at each stage of cardiac tamponade; mean aortic pressure did not fall until late in the course of tamponade. Although pulsus paradoxus may be absent during tamponade in patients with chronic right ventricular hypertension (12,15), we noted a similar mean inspiratory decrease in aortic systolic pressure before and after production of acute right ventricular dysfunction at each stage of cardiac tamponade. We have shown previously (6) in animals with ischemic left ventricular dysfunction and cardiac tamponade that pulsus paradoxus may be absent when atrial pressures are unequal. Thus, the presence of pulsus paradoxus in the present study may be explained by the nearly equal right and left atrial pressures with cardiac tamponade in dogs with right ventricular dysfunction. However, it should be emphasized that our study was not designed to examine the mechanisms of pulsus paradoxus in ischemic right ventricular dysfunction.

**Methodologic limitations.** Our results should be interpreted in the context of the experimental model and extrapolated cautiously to cardiac tamponade in humans. Acute cardiac tamponade in anesthetized dogs differs from cardiac tamponade in patients on a general medical ward (16). In addition, although right coronary artery embolization produces a hemodynamic picture of right ventricular infarction with elevated right heart pressures and volumes, most patients with right ventricular dysfunction have chronic elevation of pressures and associated changes in the compliance of the pulmonary vasculature and right ventricle. These changes are likely to have an influence on the development of cardiac chamber collapse and pulsus paradoxus. Thus, our findings may be most relevant to the acute right ventricular dysfunction that may occur with right ventricular infarction and may not necessarily apply to other and more chronic causes of right ventricular dysfunction.

Chamber collapse, like cardiac tamponade, represents a spectrum. In this study we examined hemodynamics at the onset of chamber collapse, a period that is relatively easy to recognize and is reproducible. However, the clinical utility of chamber collapse is enhanced by measuring its duration relative to the cardiac cycle (3,17). Although the rapid heart rates in our study precluded accurate quantitation of the percent duration of collapse, we observed progressive cardiac compression as pericardial pressure increased, as has been noted by others (17).

Finally, cross-sectional area measurements of the right ventricle in a single plane do not accurately reflect volume changes in a complex three-dimensional structure, such as

the right ventricle. However, we were interested primarily in an estimate of the magnitude of right heart chamber size and relative changes with cardiac tamponade. Because of the dramatic changes in right ventricular areas with right ventricular dysfunction and cardiac tamponade, it is likely that the right ventricular areas we measured represent relative changes in volume.

**Conclusions.** In this canine model of ischemic right ventricular dysfunction, characterized by equal and elevated right and left atrial pressures, decreased cardiac output and normal mean aortic pressures, incremental cardiac tamponade caused progressive increases in pericardial and atrial pressures, pulsus paradoxus and, as a late event, decreased mean arterial pressure. Although cardiac output decreased, right ventricular dysfunction attenuated the decrease in cardiac output with cardiac tamponade. Right atrial and ventricular diastolic collapse occurred at higher pericardial and atrial pressures and with larger effusions after production of ischemic right ventricular dysfunction.

In a recent study (6) we showed that right heart chamber collapse occurs with small, hemodynamically insignificant effusions in dogs with left ventricular dysfunction. Taken together, these studies demonstrate that the presence of ischemic right or left ventricular dysfunction alters the sensitivity of right atrial and ventricular collapse as signs of cardiac tamponade and suggest that these echocardiographic signs should be used cautiously in patients with coexisting heart disease. In this short-term animal study, ischemic right ventricular dysfunction, unlike left ventricular dysfunction, did not prevent the development of pulsus paradoxus during cardiac tamponade.

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