The Role of the Right Heart in Acute Cardiac Tamponade in Dogs

ROY DITCHET, ROBERT ENGLER, MARTIN LEWINTER, RICHARD PAVELEC,
VALMIK BHARGAVA, JAMES COVELL, WILLIAM MOORES, AND RALPH SHABETAI

SUMMARY Because of anatomic differences between the right and left heart, we hypothesized that the reduction in left ventricular stroke volume during acute cardiac tamponade is the passive result of decreased pulmonary venous return due primarily to right heart compression. To test this hypothesis, two types of experiments were performed in dogs. Group 1: in fresh, potassium-arrested hearts, right- and left-sided cardiac chambers were filled to previously determined physiological end-diastolic pressures. When saline then was introduced into the pericardial space, right heart volumes were reduced more rapidly than left heart volumes in all cases. These data indicate that the right heart is compressed more easily than the left heart by pericardial fluid when initial filling pressures are in the physiological range. Group 2: in intact animals, abrupt cardiac tamponade was created by rapid injection of saline into the pericardial space. Measurement of ventricular pressures, and descending output characteristics of this condition. It is uncertain whether decreased left ventricular stroke volume is primarily responsible for the systemic hypotension and low cardiac output characteristics of this condition. It is uncertain, however, whether decreased left ventricular volume results primarily from direct left ventricular compression by pericardial fluid, or from underfilling due to impaired pulmonary venous return.

BOTH left and right ventricular volumes are decreased during cardiac tamponade (Merlich, 1951; Isaacs et al., 1954; Sharp et al., 1960; Craig et al., 1968; DeCristofaro and Liu, 1969; D'Cruz et al., 1975; Pegram et al., 1975; Schiller and Botvinick, 1977; Settle et al., 1977). Since left ventricular systolic function is not severely compromised in most cases of tamponade (Isaacs et al., 1954; Sharp et al., 1960; DeCristofaro and Liu, 1969), reduced left ventricular end-diastolic volume is primarily responsible for the systemic hypotension and low cardiac output characteristics of this condition. It is uncertain, however, whether decreased left ventricular volume results primarily from direct left ventricular compression by pericardial fluid, or from underfilling due to impaired pulmonary venous return.

The importance of underfilling due to right heart compression in the pathophysiology of tamponade is suggested by anatomic differences between right- and left-sided cardiac structures. The right ventricle is thinner walled than the left ventricle and may therefore be more easily compressed by pericardial fluid under pressure. Based on this consideration, we hypothesized that acute cardiac tamponade results in a selective decrease in right heart volume because initially the right heart is more easily compressed, and that reduced left ventricular filling is largely a passive event resulting from decreased pulmonary venous return.

To test this hypothesis we designed two types of experiments. First, we sought to determine directly the relative susceptibility of the right- and left-sided cardiac chambers to compression by pericardial fluid under pressure. Second, we predicted that there would be a phase lag between the effects of abrupt tamponade on right and left ventricular stroke volumes, and we sought to verify this experimentally.

Methods

Group 1

Nine mongrel dogs were studied using pentobarbital anesthesia (25 mg/kg, iv) and positive pressure ventilation. A midline sternotomy was performed and the heart within the intact pericardium was exposed. BD vinyl tubing #6209 with multiple side-holes was placed through a small (approximately 1 cm) incision in the anterior pericardium and the tip located away from the free wall of the right ventricle in a dependent position within the pericardial space. The pericardium then was sealed around the catheter with a silk purse string tie. Angiographic catheters (#8 French, 30 cm in length) were placed via the superior vena cava into the right ventricle and...
via the carotid artery into the left ventricle. Right ventricular, left ventricular, and intrapericardial pressures were measured using Statham P23Db transducers calibrated with a mercury manometer. Zero reference level was at the mid-right atrium. Data were recorded on a Brush Clevite 8-channel forced ink recorder and on FM magnetic tape using a Honeywell model 7600 recorder. All pressure measurements were made at 200 mm/sec paper speed with ventilation suspended at end-expiration.

Left ventricular end-diastolic pressure then was raised to between 5 and 15 mm Hg by infusing 20% dextran solution intravenously. This was done to ensure conditions approximating normal cardiac filling. The right ventricular end-diastolic pressure at this level of left ventricular end-diastolic pressure was noted in each animal. The angiographic catheters then were removed and BD vinyl tubing #6209 (0.057" i.d., 0.107" o.d.) with multiple side-holes was introduced into the right ventricle via the superior vena cava and the left ventricle via a left pulmonary vein. The catheters were positioned so that side-holes were located in both atrial and ventricular chambers. Tubing position was confirmed by injection of Renografin 76 during fluoroscopy.

The animals then were given intravenous heparin (10,000 U) and injected with 50 ml of 20% KCl solution intravenously. The heart was isolated by means of vascular clamps or surgical ties placed across the aorta and main pulmonary artery, superior and inferior vena cava, and pulmonary veins. The right and left heart structures then were lavaged with saline and emptied as completely as possible through the indwelling catheters. Both catheters were connected to manometers positioned with zero levels at the mid-right atrium.

The right-sided cardiac chambers then were filled with a volume of normal saline sufficient to raise the intracavitary pressure to within 0.5 cm of water of the right ventricular end-diastolic pressure previously measured in vivo after dextran infusion. This volume was recorded and the manometer was lowered to a level at which the top of the fluid column reached the top of the manometer. The left-sided cardiac chambers then were filled with a volume of normal saline sufficient to raise the left heart pressure to within 0.5 cm of water of the left ventricular end-diastolic pressure measured in vivo. This maneuver invariably resulted in displacement of saline from the right heart. The right heart effluent was allowed to drain freely from the top of the manometer and was collected and measured. The volume of saline infused into the left heart then was recorded and the left heart manometer lowered to a position where the top of the fluid column reached the top of the manometer. Thus, the left- and right-sided cardiac chambers were filled with measured volumes of saline which resulted in intracardiac pressures approximating the right and left ventricular end-diastolic pressures recorded in vivo.

Normal saline then was introduced into the pericardial space in 20-ml increments, and fluid displaced from the left and right heart via the tops of their respective manometers was collected in separate burettes. Intrapericardial pressure and the total volume of fluid displaced through each manometer were recorded after each increment of pericardial fluid content. This procedure was continued until drainage from left and right chambers ceased or intrapericardial pressure exceeded 20 mm Hg. The pericardial space then was drained and any fluid remaining in either the left or right heart chambers was withdrawn by suction and measured. All volumes were corrected for dead space contributed by the catheter-manometer system and connecting tubing.

To test the effects of initial filling conditions on subsequent left and right heart emptying patterns during infusion of pericardial fluid, the above procedure was modified in two dogs (nos. 8 and 9). In dog 8, the right- and left-sided cardiac chambers were filled in the same manner as for dogs 1–7. However, the right heart manometer then was raised to the same level as the left heart manometer before infusion of pericardial fluid. When saline subsequently was introduced into the pericardial space, right heart pressure thus was forced to rise (and equal left heart pressure) before right heart emptying could occur. In dog 9, right- and left-sided chambers were filled with measured volumes of fluid sufficient to raise both sides to equal initial pressures (13.6 cm H2O). Fluid then was infused into the pericardial space and displaced volumes collected as described previously.

These studies were concluded within 45 minutes of potassium arrest. The possibility of leakage from the pericardial space or through any of the vascular clamps or ligatures was excluded in three dogs by filling each respective chamber under pressure with Evans blue dye and inspecting the appropriate areas for discoloration.

**Group 2**

Five dogs were prepared with right and left ventricular intrapericardial catheters in the same manner as during the instrumentation in Group 1. In addition, a second vinyl catheter was placed within the pericardial space to allow uninterrupted monitoring of the intrapericardial pressure. Biotronex Series 5000 cuffed electromagnetic flow probes then were placed around the descending aorta and a branch of the left pulmonary artery and connected to Biotronex model 620 sine wave electromagnetic flow meters. The location of the flow probes was selected to avoid the disruption of the pericardium which would have been necessary if the probes had been placed around the ascending aorta and main pulmonary artery. Care was taken to ensure good contact between the flow probe and vessel during control and subsequent tamponade recordings. Flow signals later were integrated electronically.
from FM magnetic tape playback on an EAI-590 analog computer.

Normal saline was infused slowly into the pericardium until a slight (less than 5 mm Hg) fall in left ventricular systolic pressure was noted and the preparation allowed to stabilize. This maneuver was necessary because it was not possible to inject sufficient fluid rapidly enough in a single injection to produce abrupt, severe cardiac tamponade in less than five cardiac cycles. With ventilation suspended at end-expiration, left ventricular, right ventricular, and intrapericardial pressures, as well as descending aortic and pulmonary arterial flow signals, were recorded continuously at 200 mm/sec paper speed. After recording approximately 10 cardiac cycles, an additional 60 ml of normal saline were injected rapidly into the pericardial space. This was accomplished with either a Cordis angiographic power injector (three dogs) or by rapid hand injection (two dogs). Recording was continued until all pressures and flows stabilized at new levels. Ventilation then was resumed and the 60 ml of pericardial fluid withdrawn. Occasionally a repeat injection was necessary at a slightly slower rate to avoid arrhythmias. If major cardiac tamponade did not occur (fall in left ventricular systolic pressure of at least 30 mm Hg), an 80-ml rapid injection was performed. This was sufficient to cause major tamponade in all animals. The rapid injection was repeated a maximum of five times, and the response was always qualitatively similar. In one dog, this procedure was repeated after the pulmonary flow probe had been moved from a branch of the left to a branch of the right pulmonary artery.

Statistical Analysis

Statistical significance was tested using unpaired t-tests in group 1 and repeated measures analysis of variance (Winter, 1971) in group 2. All results are expressed as mean values ± 1 SD.

Results

Group 1

Initial postmortem right and left heart pressures were 6.2 ± 1.5 mm Hg and 9.0 ± 1.6 mm Hg, respectively, in dogs 1–7. Initial right heart volume was 53.3 ± 18.3 ml, and initial left heart volume was 44.6 ± 17.2 ml. The total volume of fluid infused into the pericardial space averaged 190 ± 38 ml. Final intrapericardial pressure was 18.6 ± 3.1 mm Hg.

The absolute amount of fluid displaced from the left- and right-sided cardiac chambers of each of these seven dogs following each increment in pericardial volume is shown in Figure 1. Right heart volume was reduced by a greater amount than left heart volume by the initial three increments in pericardial volume in all cases. In six of seven dogs, the cumulative volume of fluid displaced from the right heart structures was greater than that for the

![Figure 1](http://circres.ahajournals.org/)

**Figure 1** Volume displaced from left- and right-sided cardiac chambers by postmortem pericardial fluid in the first seven group 1 dogs. Fluid was infused into the pericardial space in 20-ml increments until right and left heart drainage ceased or intrapericardial pressure exceeded 20 mm Hg.

left heart structures at every level of infused pericardial volume. In dog 5, the volume displaced from the left heart began to exceed that from the right heart after right heart emptying was complete.

These data were "normalized" in two ways to correct for differences in initial right and left heart volumes. First, the calculated volumes remaining within the chambers on each side of the heart after each increment of pericardial fluid was expressed as a percentage of the initial volume. These results are shown in Figure 2. In six of seven dogs, the percent of initial volume remaining in the left-sided chambers exceeded that in the right at every level of infused pericardial volume. In dog 3, the percent of right heart volume remaining slightly exceeded the percent of left heart volume remaining when both sides were nearly empty. Mean data for these seven dogs, "normalized" for maximal volume of infused pericardial fluid, are illustrated in Figure 3. As an example of the difference between left and

![Figure 3](http://circres.ahajournals.org/)
right heart emptying, at approximately 50% of maximal pericardial volume, when pericardial pressure was 7.0 ± 1.7 mm Hg, right heart chambers were reduced to 15 ± 15% of initial volume and left heart chambers were reduced to 64 ± 16% of initial volume ($P < 0.001$).

As an alternate means of "normalization," calculated right and left heart volumes ($V$) were divided by the volume present at zero transmural pressure ($V_o$) (Fig. 4). When precisely zero transmural pressure did not occur during an experiment, $V_o$ was estimated by extrapolating between the volumes measured at the smallest positive and negative transmural pressures recorded. Dog 2 was excluded from this analysis because the estimated $V_o$ was a negative number. These results again clearly demonstrate the different effects of infused pericardial fluid on right and left heart volumes, with more rapid right heart emptying in five of six cases. In dog 4, rates of change in $V/V_o$ were similar for the right and left heart. However, left sided $V/V_o$ exceeded right-sided $V/V_o$ at all levels of infused pericardial fluid.

In five of seven dogs (nos 1, 2, 5, 6, and 7) the total volume of fluid displaced from the right heart exceeded the measured initial volume by an average of 13% (range 8–20). If this excess were due to a left-to-right fluid shift during the course of the experiment (e.g., via the coronary arterial system), the volume apparently displaced from the right heart would be increased falsely, and that displaced from the left heart would be lowered falsely. To exclude the possibility that such a circumstance was solely responsible for the differences in volume displacement from the right- and left-sided cardiac structures, we reanalyzed our results in the following manner. The excess right heart drainage was added to the volume displaced from the left heart and subtracted from the volume displaced from the right heart by the first 20 ml increment in pericardial fluid. The percent of initial volume remaining in both right- and left-sided cardiac chambers following each increment in pericardial volume was then recalculated. The results of this “correction” are shown in Figure 5 and continue to demonstrate a clear separation between the effects of increasing pericardial volume on right- and left-sided structures. Thus, at 50% of the final pericardial fluid content, “corrected” right and left heart volumes were 20 ± 13% and 53 ± 12% of their initial volumes, respectively ($P < 0.001$).

The results of postmortem studies in dogs 8 and 9 when manometer heights were equal are shown in Figure 6. In contrast to the findings in the first
right heart in cardiac tamponade/Ditchey et al.

Figure 4 Volume (V) displaced from right and left heart chambers by postmortem pericardial fluid (group 1), "normalized" with respect to estimated volumes present at zero transmural pressure (Vo). Dog 2 was excluded since estimated Vo was negative.

seven experiments, right- and left-heart emptying patterns in these two dogs did not differ substantially. This was true at all levels of pericardial volume in dog 8. In dog 9, the percent initial left heart volume remaining began to exceed percent initial right heart volume remaining only at high levels of pericardial volume.

Group 2

Beat-to-beat changes in left and right ventricular pressures, intrapericardial pressure, descending aortic flow and branch pulmonary arterial flow immediately preceding and following abrupt tamponade are summarized in Table 1 and Figures 7 and 8. Beat numbers are referenced with respect to the onset of rapid pericardial fluid injection (beat number one was defined as the first systole following the onset of rapid injection). Mean control values were derived from the five cardiac cycles immediately preceding pericardial fluid injection.

The volume of initial intrapericardial fluid ranged from 40 to 200 ml. Subsequent rapid injections of 60 to 80 ml of additional fluid were completed within 3 to 5 cardiac cycles. Mean intrapericardial pressure rose from an initial value of 4.9 ± 1.1 mm Hg to a peak of 9.8 ± 1.7 mm Hg (P < 0.01 compared to control) by the end of injection (average beat number = 4), and then gradually fell to a new stable level of 8.7 ± 1.6 mm Hg (P < 0.01 compared to beat 4). Left ventricular systolic pressure fell progressively from 139 ± 6 to 91 ± 5 mm Hg (P < 0.01) during the first 14 beats after the onset of tamponade. Right ventricular systolic pressure fell from 28.9 ± 1.6 to 19.2 ± 1.7 mm Hg by beat 6 (P < 0.01, compared to control), then rose gradually to 22.7 ± 3.5 mm Hg by beat 14 (NS).

Left ventricular end-diastolic pressure rose from 12.4 ± 1.6 to 15.6 ± 2.9 mm Hg (P < 0.01) during injection of pericardial fluid (first 4 beats), then fell gradually to 13.0 ± 1.7 mm Hg by beat 14 (P < 0.05 compared to beat 4). Right ventricular end-diastolic pressure was 8.8 ± 0.1 mm Hg initially, 11.1 ± 3.6 mm Hg after fluid injection (beat 4), and 10.5 ± 4.1 mm Hg by beat 14 (all NS).

Right and left ventricular transmural end-diastolic pressures were calculated by subtracting the
pericardial pressure from the measured end-diastolic pressure (Table 1). Left ventricular transmural end-diastolic pressure declined steadily from 7.5 ± 3.6 mm Hg during control to 4.3 ± 3.3 mm Hg by beat 14 (P < 0.01). In contrast, right ventricular transmural end-diastolic pressure fell abruptly from a control value of 3.9 ± 4.9 mm Hg to 1.3 ± 2.8 mm Hg by beat 4 (P < 0.05). By beat 14, right ventricular transmural end-diastolic pressure was 1.8 ± 2.8 mm Hg (NS compared to control).

Beat-to-beat changes in branch pulmonary arterial and descending aortic blood flow during abrupt tamponade are shown graphically in Figure 7. Flow is expressed as a percent of a control value calculated from the mean integrated systolic flow of the five cycles immediately preceding pericardial fluid injection. Pulmonary flow decreased immediately with the onset of tamponade, whereas systemic flow initially was unchanged. Thus, by beat 2, branch pulmonary arterial and descending aortic flows were 62 ± 26% and 99 ± 8% of their initial values, respectively. Aortic flow then declined, but still

Figure 8 Typical tracings obtained following the abrupt onset of cardiac tamponade. Rapid injection of pericardial fluid begins at arrow.
exceeded pulmonary flow during each of the first 10 beats following the onset of cardiac tamponade. Thus, on beat 4, branch pulmonary arterial and descending aortic flows were 24 ± 26% and 67 ± 12% (P < 0.01) of their initial values, respectively. Pulmonary flow then gradually increased until a new steady state was achieved in which right and left ventricular outputs were reduced to comparable fractions of their initial values (53 ± 23% and 51 ± 27%, respectively, by beat 13).

These changes in systemic and pulmonary blood flow paralleled simultaneous changes in left and right ventricular transmural end-diastolic pressures. Thus, both right ventricular transmural end-diastolic pressure and pulmonary arterial flow were reduced to comparable fractions of their initial values (53 ± 23% and 51 ± 27%, respectively, by beat 13). These changes in systemic and pulmonary blood flow were similar whether a right or left pulmonary arterial branch was used to measure pulmonary flow following left branch pulmonary arterial flow were compared.

The effects of abrupt tamponade on right and left branch pulmonary arterial flow were compared in one dog. Changes in pulmonary flow following tamponade were similar whether a right or left pulmonary arterial branch was used to measure flow.

**Critique of the Method**

**Group 1**

In five of the first seven dogs studied, the total volume of fluid displaced from the right heart chambers exceeded the calculated initial volume by an average of 13%. There are several possible explanations for this observation. First, although an attempt was made to empty both sides of the heart completely by suction prior to initial volume infusion, the possibility of trapping of small amounts of fluid in areas inaccessible to the drainage catheters under conditions of negative pressures cannot be excluded. These same areas might have been more completely drained by positive pressure resulting from extrinsic compression when the pericardial space was filled with saline. Second, since the aorta was routinely clamped or ligated above the level of the coronary ostia, and left-sided pressures uniformly exceeded right, some left to right shift of fluid may have resulted from flow through the coronary vascular bed. However, when the volumes displaced from the right and left heart chambers were "corrected" by assuming that this excess was due entirely to left to right fluid shifts, the response of right- and left-sided cardiac structures to progressive increments in pericardial volume remained clearly separable (Fig. 5). Therefore, left to right flow through the coronary arterial system or variability in the volume of fluid accessible to drainage from the right heart are not responsible for the observed differences in volume displaced from the right- and left-sided cardiac chambers.

These differences were no longer apparent, however, when the height of the drainage manometer of the right- and left-sided chambers were equal prior to infusion of pericardial fluid (dogs 8 and 9, Fig. 6). This indicates that relative right and left heart volume changes due to the accumulation of

### Table 1 Mean Beat-to-Beat Data in Group 2 Animals before and after Abrupt Tamponade

<table>
<thead>
<tr>
<th>Beat no.</th>
<th>PP (mmHg)</th>
<th>LVSP (mmHg)</th>
<th>RVSP (mmHg)</th>
<th>LVEDP (mmHg)</th>
<th>RVEDP (mmHg)</th>
<th>LVTMP (mmHg)</th>
<th>RVTMP (mmHg)</th>
<th>%AF</th>
<th>%PF</th>
</tr>
</thead>
<tbody>
<tr>
<td>-4</td>
<td>4.9</td>
<td>139</td>
<td>28.9</td>
<td>12.4</td>
<td>8.9</td>
<td>7.5</td>
<td>4.0</td>
<td>100</td>
<td>98</td>
</tr>
<tr>
<td>-3</td>
<td>4.9</td>
<td>139</td>
<td>28.8</td>
<td>12.4</td>
<td>8.7</td>
<td>7.5</td>
<td>3.8</td>
<td>98</td>
<td>97</td>
</tr>
<tr>
<td>-2</td>
<td>4.9</td>
<td>139</td>
<td>28.8</td>
<td>12.2</td>
<td>8.7</td>
<td>7.3</td>
<td>3.8</td>
<td>102</td>
<td>100</td>
</tr>
<tr>
<td>-1</td>
<td>4.9</td>
<td>139</td>
<td>29.2</td>
<td>12.7</td>
<td>8.7</td>
<td>7.8</td>
<td>3.8</td>
<td>103</td>
<td>104</td>
</tr>
<tr>
<td>1*</td>
<td>6.3</td>
<td>139</td>
<td>29.7</td>
<td>12.6</td>
<td>9.3</td>
<td>6.3</td>
<td>3.0</td>
<td>105</td>
<td>86</td>
</tr>
<tr>
<td>2</td>
<td>7.7</td>
<td>130</td>
<td>27.1</td>
<td>14.2</td>
<td>10.3</td>
<td>6.5</td>
<td>2.6</td>
<td>92</td>
<td>62</td>
</tr>
<tr>
<td>3</td>
<td>9.2</td>
<td>134</td>
<td>23.1</td>
<td>15.2</td>
<td>10.2</td>
<td>6.0</td>
<td>1.0</td>
<td>75</td>
<td>37</td>
</tr>
<tr>
<td>4</td>
<td>9.8</td>
<td>128</td>
<td>21.1</td>
<td>15.6</td>
<td>11.1</td>
<td>5.8</td>
<td>1.3</td>
<td>67</td>
<td>24</td>
</tr>
<tr>
<td>5</td>
<td>9.7</td>
<td>122</td>
<td>19.6</td>
<td>15.1</td>
<td>10.7</td>
<td>5.4</td>
<td>1.0</td>
<td>59</td>
<td>17</td>
</tr>
<tr>
<td>6</td>
<td>9.4</td>
<td>117</td>
<td>19.2</td>
<td>14.4</td>
<td>10.5</td>
<td>5.0</td>
<td>1.1</td>
<td>59</td>
<td>19</td>
</tr>
<tr>
<td>7</td>
<td>9.2</td>
<td>111</td>
<td>19.8</td>
<td>14.0</td>
<td>10.0</td>
<td>4.8</td>
<td>0.8</td>
<td>50</td>
<td>25</td>
</tr>
<tr>
<td>8</td>
<td>9.1</td>
<td>108</td>
<td>20.7</td>
<td>13.8</td>
<td>10.0</td>
<td>4.7</td>
<td>0.9</td>
<td>55</td>
<td>33</td>
</tr>
<tr>
<td>9</td>
<td>8.9</td>
<td>105</td>
<td>21.1</td>
<td>13.9</td>
<td>10.0</td>
<td>5.0</td>
<td>1.1</td>
<td>50</td>
<td>42</td>
</tr>
<tr>
<td>10</td>
<td>8.8</td>
<td>103</td>
<td>21.7</td>
<td>13.7</td>
<td>10.3</td>
<td>4.9</td>
<td>1.5</td>
<td>55</td>
<td>44</td>
</tr>
<tr>
<td>11</td>
<td>8.8</td>
<td>96</td>
<td>22.0</td>
<td>13.3</td>
<td>10.4</td>
<td>4.5</td>
<td>1.6</td>
<td>46</td>
<td>49</td>
</tr>
<tr>
<td>12</td>
<td>8.8</td>
<td>97</td>
<td>22.4</td>
<td>13.7</td>
<td>10.5</td>
<td>4.9</td>
<td>1.7</td>
<td>61</td>
<td>51</td>
</tr>
<tr>
<td>13</td>
<td>8.7</td>
<td>92</td>
<td>22.1</td>
<td>13.0</td>
<td>10.6</td>
<td>4.3</td>
<td>1.9</td>
<td>51</td>
<td>53</td>
</tr>
<tr>
<td>14</td>
<td>8.7</td>
<td>91</td>
<td>22.7</td>
<td>13.0</td>
<td>10.5</td>
<td>4.3</td>
<td>1.8</td>
<td>65</td>
<td>52</td>
</tr>
<tr>
<td>15</td>
<td>8.7</td>
<td>88</td>
<td>22.4</td>
<td>12.7</td>
<td>10.6</td>
<td>4.0</td>
<td>1.9</td>
<td>56</td>
<td>53</td>
</tr>
</tbody>
</table>

PP = Intrapericardial pressure; LVSP = left ventricular peak systolic pressure; RVSP = right ventricular peak systolic pressure; LVEDP = left ventricular end-diastolic pressure; RVEDP = right ventricular end-diastolic pressure; LVTMP = left ventricular transmural end-diastolic pressure; %AF = percent of control aortic flow; %PF = percent of control pulmonary artery flow.

* First beat following abrupt tamponade.
pericardial fluid are dependent upon initial filling conditions in these in vitro studies. Since right- and left-sided filling pressures progress from their normal physiological relation to near equalization as tamponade evolves in vivo, it is apparent that no single set of postmortem conditions can represent accurately the full spectrum of tamponade physiology. Studies in dogs 1–7 were performed under conditions approximating the usual (non-tamponade) relationship between right and left heart pressures and volumes. These conditions were chosen so that the initial response of right- and left-sided cardiac chambers to pericardial fluid accumulation could be assessed. The greater susceptibility of the right heart to changes in volume imposed by small volumes of pericardial fluid is clearly demonstrated by these studies. It should be appreciated, however, that this model becomes progressively less relevant to tamponade as pericardial pressure and volume increase.

The initial conditions used in dogs 1–7 are relevant to early acute tamponade when the relationship of left- and right-sided filling pressures is physiological, whereas the initial conditions in dogs 8–9 are relevant to late in tamponade when nearly equal filling pressures exist. Group 2 studies were designed to test the applicability of the group 1 results in a physiological model. The fact that right ventricular diastolic pressure remained significantly less than left ventricular diastolic pressure during the initial phase of acute tamponade in vivo (Table 1) supports the applicability of data for dogs 1–7 to the initial phase of acute tamponade.

**Group 2**

In these studies, flow was measured in the descending aorta and a branch of the left pulmonary artery. We assumed that flow at these sites represented a constant fraction of left and right ventricular stroke volumes, respectively. It is unlikely that reflex changes in blood flow distribution occurred during the first 15 beats (approximately 8 seconds) of cardiac tamponade (Guyton et al., 1954; Guyton et al., 1957). This is supported by our observation that heart rate did not change during this period. Furthermore, when flow was measured in both a right and left pulmonary arterial branch in the same animal, the percent changes in branch pulmonary flow were comparable. Although it would have been preferable to measure total stroke volume for both right and left ventricles, this is very difficult without significant disruption of the pericardium. The method employed allowed the pericardium to remain intact for studies during tamponade.

These studies were performed in open-chest dogs. In cardiac tamponade in closed-chest animals, respiratory changes in intrathoracic pressure are fully transmitted to the pericardial space (Gunteroth et al., 1967). Under these conditions, respiratory fluctuations in transmural left and right ventricular pressures may contribute to phasic changes in ventricular stroke volumes. However, in the presence of freely communicating pericardial fluid, any change in intrapericardial pressure would be uniform over both ventricles, and the phasic respiratory changes would be superimposed on the more basic mechanism. It is therefore likely that our hypothesis regarding the primary mechanism of reduced left ventricular volume during cardiac tamponade is tested adequately in the open-chest preparation.

**Discussion**

Based on anatomic differences between right- and left-sided cardiac chambers, we hypothesized that cardiac tamponade results in selective right heart compression, and that reduced left ventricular volume during tamponade occurs passively due to decreased right heart output and reduced pulmonary venous return. The results of the two types of experiments in this study support this hypothesis.

Our findings in fresh, potassium-arrested canine hearts demonstrate that right-sided cardiac chambers are compressed much more easily by pericardial fluid under pressure than are left-sided structures when a normal relationship between right and left heart filling pressures is maintained. This was manifested both by a greater cumulative decrease in right heart volume following each increment of pericardial fluid, and by a marked decrease in right-sided volume when left-sided volume was reduced only moderately. These differences suggest that early tamponade (prior to in vivo pressure equalization) causes greater direct compression of right heart structures than of left heart structures.

The importance of these differences in the pathophysiology of cardiac tamponade is supported further by our findings in the beating heart. Abrupt tamponade in group 2 animals caused an immediate rise in both right and left ventricular end-diastolic pressures; transmural end-diastolic pressure fell abruptly in the right but not in the left ventricle (Table 1). This indicates a much lesser degree of immediate and direct interference with left ventricular filling as compared to right ventricular filling. These findings imply that reduced left ventricular volume during cardiac tamponade results primarily from underfilling due to right heart compression and decreased pulmonary venous return. This concept is supported further by previous angiographic studies which document severe distortion and flattening of the free wall of the right ventricle against the intraventricular septum in dogs with tamponade (Shabetai et al., 1979). However, it must be recognized that our findings do not separate the relative importance of right atrial and right ventricular compression in reducing right ventricular volume. Furthermore, the possibility that right ventricular filling is reduced in part by interference with blood return proximal to the right atrium.
cannot be excluded by our studies. Apparent infringement on the intrapericardial portions of the venae cavae during tamponade has been observed angiographically (Shabetai et al., 1979). Such venae caval constriction theoretically could impair right heart filling, although attempts to document a pressure gradient between the superior or inferior vena cava and the right atrium during tamponade have been unsuccessful (Shabetai et al., 1965).

Direct proof of the primary importance of compromised left ventricular filling in the pathogenesis of cardiac tamponade is provided by comparison of the instantaneous effects of rapidly produced tamponade on left- and right-sided cardiac output. We have shown that abrupt tamponade causes an immediate fall in pulmonary arterial flow, while aortic flow initially is unchanged. Left heart output then falls progressively to a lower level, but continues to exceed right heart output during each of the first 10 beats following the onset of tamponade. In contrast, right heart output rapidly reaches its nadir and then rises gradually until left and right heart outputs are again equal (Figures 7 and 8). Thus, there is a significant delay between the onset of tamponade as manifested by right-sided events and the completed fall in left-sided cardiac output.

This initial phase lag, during which systemic and pulmonary blood flows are unequal, is of central importance in the pathophysiology of abrupt cardiac tamponade. Since left heart output transiently exceeds right heart output, blood volume is shifted from the pulmonary to the systemic circulation. This reduction in pulmonary blood volume would be expected to lower mean pulmonary pressure (Guyton et al., 1954; Guyton et al., 1957), leading to a subsequent decrease in pulmonary venous return. Elevated left atrial pressure during tamponade potentiates this reduction by further decreasing the pressure gradient for blood flow into the left atrium. In contrast, increased systemic blood volume would be expected to raise mean systemic pressure (Guyton et al., 1954; Guyton et al., 1957) and increase venous return to the right atrium. In addition, as left ventricular volume gradually declines (due to underfilling), intrapericardial pressure is reduced (Table 1) and the severity of right heart compression is decreased. This effect further facilitates recovery of right heart filling (Bemis et al., 1974; Alderman and Glanty, 1976; Shirato et al., 1978). As a result, right ventricular end-diastolic volume and transmural end-diastolic pressure begin to increase and right ventricular stroke volume and pulmonary arterial blood flow improve until a new steady state is reached in which right and left heart outputs are again equal (Table 1 and Figure 7).

Although reflex sympathetic nervous system responses to reduced cardiac output eventually would modify the precise level of the new equilibrium reached during cardiac tamponade, the time frame of our abrupt tamponade studies suggests that basic hemodynamic adjustments occur which are independent of such responses (Friedman et al., 1977). These adjustments are attributable directly to the different effects of pericardial fluid on the left and right heart. It is reasonable to expect that a similar sequence evolves when tamponade develops more slowly. Under these circumstances, however, neurohumoral alterations in peripheral vascular resistance, venous capacitance, heart rate, myocardial contractility, and blood volume, as well as other time-dependent phenomena (for instance, stretching of the pericardium) also would influence the ultimate effects of accumulated pericardial fluid on ventricular volume, blood pressure, and cardiac output.

We conclude that major cardiac tamponade can occur without significant direct compression of the left heart. Although localized left ventricular tamponade can be produced experimentally (Carey et al., 1967), when freely communicating pericardial fluid accumulates under pressure, right heart filling and pulmonary blood flow are critically compromised before significant left heart compression occurs. Decreased left ventricular diastolic volumes and reduced systemic output during tamponade are largely passive phenomena which reflect decreased pulmonary venous return and a shift of blood volume from the central to the systemic circulation.

Acknowledgments

We gratefully acknowledge the secretarial skills of Susan Connolly.

References

Guyton AC, Lindsay AW, Abernathy B, Richardson T (1957)
Venous return at various right atrial pressures and the normal venous return curve. Am J Physiol 189: 609-615
Merlich ME (1951) Determinants of impairment of cardiac filling during progressive pericardial effusion. Circulation 3: 377-383
Schiller NB, Botvinick EH (1977) Right ventricular compression as a sign of cardiac tamponade. An analysis of echocardiographic ventricular dimensions and their clinical implications. Circulation 56: 774-779

Effects of respiration and pericardiocentesis. Circulation 56: 951-959
The role of the right heart in acute cardiac tamponade in dogs.
R Ditchey, R Engler, M LeWinter, R Pavelec, V Bhargava, J Covell, W Moores and R Shabetai

Circ Res. 1981;48:701-710
doi: 10.1161/01.RES.48.5.701

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/48/5/701.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation Research can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation Research is online at:
http://circes.ahajournals.org/subscriptions/