The hemodynamic effects of cardiac tamponade: mainly the result of atrial, not ventricular, compression

Noble O. Fowler, M.D., and Marjorie Gabel

ABSTRACT We studied the hemodynamic effects of surgically induced regional cardiac tamponade in anesthetized dogs. Tamponade restricted to either the right or the left ventricle was compared with tamponade of either ventricle and both atria. Intrapericardial pressures were elevated to approximately 20 mm Hg. With tamponade of the right ventricle alone, aortic pressure rose from 161 ± 3.8 to 164 ± 3.4 mm Hg (p > .05) and cardiac output fell from 149.4 ± 16.1 to 134.9 ± 11.9 ml/kg/min (p > .05). However, tamponade of the right ventricle plus both atria decreased mean aortic pressure from 152.5 ± 3.6 to 115.9 ± 8.7 mm Hg (p < .01) and cardiac output fell from 118 ± 14.8 to 38.9 ± 4.8 ml/kg/min (p < .01). With tamponade of the left ventricle alone, aortic mean pressure changed significantly from 158.5 ± 6.1 (control) to 148.9 ± 5.0 mm Hg (tamponade) (p < .05) and cardiac output was 135.5 ± 28.3 (control) and 111 ± 24.7 ml/kg/min (tamponade) (p > .05). However, when the atria were included, mean aortic pressure fell significantly more from 155.5 ± 5.4 to 105.5 ± 10.4 mm Hg (p < .01) and cardiac output fell from 142.2 ± 16 to 47.8 ± 6.4 ml/kg/min (p < .01). Atrial pressure rose when the atria were included, but not with tamponade of the left ventricle alone. Right but not left atrial pressure rose slightly with isolated right ventricular tamponade. We conclude that the principal hemodynamic effects of cardiac tamponade are not the result of compression of either the right or the left ventricle, but are the consequence of compression of the atria and/or the venae cavae and the pulmonary veins.


THE HEMODYNAMIC effects of cardiac tamponade are generally believed to result from compression of the cardiac ventricles, which limits diastolic cardiac filling.1-3 However, an earlier theory stated that tamponade limited the circulation by compressing the systemic veins within the pericardial sac, thus restricting venous return to the right atrium.4 This question was studied by Isaacs et al.1 and they found no diastolic pressure gradient between the venae cavae and right atrium or pulmonary veins and left atrium. They thus concluded that compression of the great veins did not contribute to the reduced diastolic filling of the heart during tamponade.

However, despite the above-mentioned findings, the hemodynamic effects of regional cardiac tamponade have not been investigated. Accordingly, we stud-
and allowed compromise of the left circumflex artery and the left anterior descending artery to be avoided. The sutures were placed just below the circumflex branch of the left coronary artery and the great coronary vein, and then just a few millimeters to the left of the anterior descending branch of the left coronary artery. Suturing then followed the diaphragmatic margin of the left ventricle over to its apex and thus just to the left ventricular side of the dorsal interventricular branch of the left coronary artery.

The isolated left ventricular pericardial space excluded the atria and the right ventricular space included the atria. The catheters were tunneled to a left lateral subcutaneous pocket. The left atrial catheters and pericardial catheters were filled with heparinized saline (0.9%) and the ends were closed. The thoracotomy was repaired in four layers and the pneumothorax was reduced. Soluble Berocca-C (6 ml) and one million units penicillin G were given during the operation and this was followed by daily intramuscular injections of 2 ml Azimycin Combiotic.

Isolated right ventricular tamponade. The preparation was the same as above except that a right thoracotomy was performed and the right ventricle was isolated by attaching the pericardium with superficial interrupted 3.0 silk sutures on atraumatic needles. Sutures were placed in such a way that they outlined the right ventricle along the right coronary artery to the apex, excluding the atrium. The right margin for suture placement was a few millimeters inside the right coronary artery, with the suture points heading downward to the right ventricular side of the middle cardiac vein. The sutures were then carried down to the apex of the heart and then upward along the right ventricular side of the vertical interventricular branch of the left coronary artery. The left ventricular space included the atria.

Placement of catheters was as above, but the catheters were tunneled to a right lateral subcutaneous pocket.

All dogs were allowed to recover at least 10 days to ensure complete adhesions at the suture lines between pericardium and myocardium before tamponade experiments were performed. Cardiac tamponade restricted to the two atria was attempted in six additional animals, but was unsuccessful owing to leakage at the suture line.

Procedure. Animals were reanesthetized with intravenous pentobarbital sodium, 25 mg/kg body weight. The animals were breathing spontaneously. Under fluoroscopy, No. 7F Courmand catheters were placed in the right atrium, pulmonary artery, and aorta of each for pressure measurements; a thermistor-tipped catheter was placed in the pulmonary artery for thermodilution cardiac output determinations. Statham 23Db pressure transducers were used to measure right atrial, left atrial, intrapericardial, and systemic arterial pressures. These and an electrocardiogram were recorded with an eight-channel Grass 7D polygraph recorder. Electronically measured pressures were measured with the animal lying on its side; the center of the thorax was used for zero reference. Pressure transducers were calibrated with mercury manometers before each experiment. Control cardiac pressures and cardiac outputs were measured in duplicate. Cardiac tamponade was then induced by stepwise increases of intrapericardial pressure by injection of 0.9% NaCl 37°C solution into the pericardial sac. Intrapericardial pressure was raised to 20 mm Hg or to the highest value tolerated without a continued fall in systemic arterial pressure at that intrapericardial pressure level, whichever occurred first. Cardiac output and pressure measurements were then repeated in duplicate. At the termination of experiments, each animal was killed with an intravenous overdose of pentobarbital. Absence of leaks at the suture lines was demonstrated by subjecting the individual pericardial spaces to pressures of 50 mm Hg or more of saline solution colored with blue dye. The pericardial spaces themselves were demonstrated to be free of adhesions.

Results

Successful studies were carried out in 19 animals. Eleven underwent studies of tamponade of the right ventricle and both atria; six of these 11 also had tamponade of the left ventricle alone. Eight animals underwent tamponade of the left ventricle and both atria; six of these underwent tamponade of the right ventricle alone. We could not study the same number of animals in each group because in some animals catheter plugging or external leaking from one of the two pericardial catheters was noted. Seven additional animals could not be studied because of leaks between the two surgically separated pericardial spaces.

The hemodynamic data are summarized in table 1. Tamponade of the right or left ventricle alone (mean intrapericardial pressure in the space subjected to tamponade increased to 21.2 and 19.6 mm Hg, respectively) produced no significant change in left atrial pressure or cardiac output (p > .05). Aortic mean pressure fell slightly with left ventricular tamponade (p < .05). On the other hand, when tamponade of the right ventricle plus both atria was produced, aortic mean pressure fell more than with tamponade of either ventricle alone. With a mean intrapericardial pressure of 19 mm Hg, aortic pressure fell from 152.5 ± 3.6 to 115.9 ± 8.7 mm Hg. Right atrial pressure rose significantly more than with right ventricular tamponade alone from 2.9 ± 0.5 to 19.1 ± 0.6 mm Hg, and left atrial pressure rose significantly from 8.0 ± 1.1 to 17.3 ± 1.3 mm Hg. Likewise, mean cardiac output fell significantly from 118 ± 14.8 to 38.9 ± 4.8 ml/kg/min.

Similarly, tamponade of the left ventricle and both atria produced significant hemodynamic changes when mean intrapericardial pressure in the space subjected to tamponade was increased to 17.9 mm Hg. Mean aortic blood pressure fell from 155.5 ± 5.4 to 105.5 ± 10.4 mm Hg; this fall was significantly greater than that with isolated left ventricular tamponade. Right atrial pressure fell from 6.1 ± 1.2 to 16.5 ± 0.5 mm Hg and left atrial pressure rose from 7.7 ± 1.3 to 16.6 ± 0.9 mm Hg. In a like manner, cardiac output decreased significantly from 142.2 ± 16 to 47.8 ± 6.4 mm Hg.

Tamponade of the right ventricle plus both atria when compared with tamponade of the right ventricle alone produced significantly greater decreases in aortic pressure and cardiac output and significantly greater increases in right and left atrial pressures (table 1). Similarly, tamponade of the left ventricle plus both atria produced significantly greater changes when compared with tamponade of the left ventricle alone: there were significantly greater decreases in aortic...
pressure and cardiac output, together with greater increases in both right and left atrial pressures.

Discussion

Cohnheim suggested that tamponade of the heart might compromise the circulation by compressing the great veins within the pericardial sac, thus limiting diastolic filling of the heart. Issacs et al. measured pressures in the venae cavae, pulmonary veins, and right and left atria during experimental tamponade. These investigators found no diastolic gradient between venae cavae and right atrium or pulmonary veins and left atrium, and concluded that compression of the great veins did not limit cardiac filling in an important way. However, diastolic pressure differences between the atria and ventricles were not measured so that if the atria were compressed as well as the veins, the significance of compression of both atria and great veins might be overlooked. Furthermore, especially at low flows, significant pressure differences might be extremely small and difficult to measure. It is generally accepted that ventricular diastolic size is reduced in the presence of cardiac tamponade. Gated blood pool scintigraphy has been used to show a decrease in both right and left ventricular end-diastolic volumes in five patients with cardiac tamponade. It is generally assumed that this reduction in diastolic volume is the result of compression of the ventricles; however, a reduction in size might also be caused by atrial and/or intrapericardial venous compression that limits ventricular diastolic filling. Compression of the right atrium and superior vena cava has been demonstrated in our laboratory, and in man by Miller et al. It is known that caval compression and both right and left atrial compression can be demonstrated in man by echocardiography during cardiac tamponade, but the hemodynamic significance of the phenomenon has not been evaluated. Kronzon et al. studied nine patients with tamponade of the heart by means of M mode and two-dimensional echocardiography. All nine had diastolic right atrial compression; seven had right ventricular compression and five diastolic left atrial compression.

Ditchey et al. studied experimental cardiac tamponade in dogs. They found that decreases in left ventricular stroke volume followed changes in right ventricular stroke volume by an average of 13 cardiac cycles when cardiac tamponade was produced abruptly. These investigators concluded that the hemodynamic effects of tamponade were primarily the result of right heart compression; the decrease in left heart output observed was postulated to result from a decrease in right ventricular stroke volume and therefore in pulmonary blood volume.

The present study further defines the site of cardiac compression in cardiac tamponade. The results demonstrate that neither right or left ventricular compression is responsible for the hemodynamic changes associated with elevation of intrapericardial pressure.

<table>
<thead>
<tr>
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<th>Ao (mean pres. in mm Hg)</th>
<th>Pericardium (mean pres. in mm Hg)</th>
<th>Right atrium (mean pres. in mm Hg)</th>
<th>Left atrium (mean pres. in mm Hg)</th>
<th>Cardiac output (ml/kg/min)</th>
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<tbody>
<tr>
<td>RV alone</td>
<td></td>
<td></td>
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<tr>
<td>Control</td>
<td>161 ± 3.8</td>
<td>0.92 ± 0.9</td>
<td>5.3 ± 1.2</td>
<td>7.5 ± 1.4</td>
<td>149.4 ± 16.1</td>
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<td>Tamponade</td>
<td>164 ± 3.4</td>
<td>21.2 ± 0.7</td>
<td>7.7 ± 1.4</td>
<td>8.5 ± 1.4</td>
<td>134.9 ± 11.9</td>
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<tr>
<td>LV alone</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Control</td>
<td>158.5 ± 6.1</td>
<td>1.2 ± 1</td>
<td>3.4 ± 1.4</td>
<td>8.8 ± 1.4</td>
<td>135.5 ± 28.3</td>
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<tr>
<td>Tamponade</td>
<td>148.9 ± 5.0</td>
<td>19.6 ± 0.4</td>
<td>3.5 ± 0.4</td>
<td>10.6 ± 1.4</td>
<td>111 ± 24.7</td>
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<td>RV plus both atria</td>
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<td>Control</td>
<td>152.5 ± 3.6</td>
<td>1.41 ± 0.7</td>
<td>2.9 ± 0.5</td>
<td>8.0 ± 1.1</td>
<td>118 ± 14.8</td>
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<tr>
<td>Tamponade</td>
<td>115.9 ± 8.7</td>
<td>19.0 ± 0.6</td>
<td>19.1 ± 0.6</td>
<td>17.3 ± 1.3</td>
<td>38.9 ± 4.8</td>
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<tr>
<td>LV plus both atria</td>
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<tr>
<td>Control</td>
<td>155.5 ± 5.4</td>
<td>1.9 ± 0.8</td>
<td>6.1 ± 1.2</td>
<td>7.7 ± 1.3</td>
<td>142.2 ± 16</td>
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<tr>
<td>Tamponade</td>
<td>105.5 ± 10.4</td>
<td>17.9 ± 0.7</td>
<td>16.5 ± 0.5</td>
<td>16.6 ± 0.9</td>
<td>47.8 ± 6.4</td>
</tr>
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</table>

Pressures are expressed as mean ± SE and in mm Hg.

RV = right ventricle; LV = left ventricle; Ao = aorta.

*Significant difference from control value (p < .01, Student paired t test).

**Significant difference from control value (p < .05, Student paired t test).

'Significant difference from tamponade of right ventricle or left ventricle alone (p < .01, Student unpaired t test).
Thus, the compression must be exerted primarily on the atria and great veins. The present study does not separate the effects of atrial compression from those of compression of the venae cavae or pulmonary veins, nor does it separate the effects of right atrial (or vena caval) compression from those of left atrial (or pulmonary venous) compression. However, the studies of Ditchey et al. suggest that the significant compressive effects of tamponade are on the right heart, implying that the right atrium and/or vena cava are the important sites of compression. This hypothesis is in keeping with the echocardiographic observation that right atrial compression is more consistent than left atrial compression in human cardiac tamponade, as well as with the fact that pericardial fluid often cannot be demonstrated behind the left atrium in echocardiographic studies of patients with cardiac tamponade. The present study shows that there is some hemodynamic impairment with isolated right or left ventricular tamponade; it is possible that compression of both ventricles would have a greater hemodynamic effect. Further studies are needed to establish the effects of tamponade of one or both atria alone and effects of tamponade of both ventricles alone.

References
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