Right ventricular and right atrial collapse in patients with cardiac tamponade — a combined echocardiographic and hemodynamic study

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ABSTRACT To determine the hemodynamic derangement associated with right ventricular diastolic collapse and to assess the value of right ventricular and right atrial collapse in identifying cardiac tamponade, two-dimensional echocardiograms were recorded simultaneously with measurement of right atrial, pulmonary capillary wedge, intrapericardial, and systemic arterial pressures and cardiac output in 16 patients as they underwent pericardiocentesis. Twelve patients (group I) had evidence of right ventricular or right atrial collapse or both on their echocardiograms and hemodynamic evidence of cardiac tamponade before pericardiocentesis. All hemodynamic parameters improved after pericardiocentesis (p ≤ .05). Continuous monitoring during pericardiocentesis in three of these patients showed significant improvement in all parameters except heart rate (p ≤ .02) at the point of disappearance of right ventricular diastolic collapse, with further improvement in cardiac output as pericardiocentesis continued (p < .01). Right atrial collapse persisted after right ventricular collapse disappeared but was no longer present when pericardiocentesis was completed. Three patients (group II) had no right ventricular or right atrial collapse, no hemodynamic evidence of cardiac tamponade, and no improvement in hemodynamic parameters after pericardiocentesis. A single patient (group III) was found to have elevated right heart pressures and right ventricular hypertrophy before pericardiocentesis. Although there was hemodynamic evidence of cardiac tamponade in this patient, there was no evidence of right ventricular or right atrial collapse. In this study, the sensitivity of right ventricular collapse as a marker for cardiac tamponade was 92%, its specificity 100%, its accuracy 94%, and its predictive value 100%. The sensitivity of right atrial collapse was 64%, its specificity 100%, its accuracy 75%, and its predictive value 100%. Right atrial collapse occurs very early in the course of hemodynamic deterioration due to cardiac tamponade. Right ventricular collapse appears somewhat later, at a point at which cardiac output is significantly depressed but before decline in systemic arterial blood pressure. These echocardiographic signs should be useful in detecting hemodynamically important pericardial effusions and assisting in their management.

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only a few patients and the precise hemodynamic correlates of cardiac tamponade and right ventricular and right atrial collapse were not established. We recently completed a long-term study in an animal preparation of cardiac tamponade that showed right ventricular diastolic collapse to occur early in the course of hemodynamic deterioration and, with some exceptions, to be a specific and sensitive sign of cardiac tamponade.11 Collapse of the right ventricle occurred when intrapericardial pressure exceeded right ventricular intracavitary pressure. To further elucidate the hemodynamic importance and validate the clinical usefulness of right ventricular and right atrial collapse as diagnostic signs of cardiac tamponade, we obtained echocardiograms and simultaneous hemodynamic measurements from patients with pericardial effusions as they underwent pericardiocentesis.

Methods

Patients. Sixteen consecutive patients referred to the Cardiology Division at the Medical College of Wisconsin for diagnostic and/or therapeutic pericardiocentesis were studied. All gave informed consent to a protocol approved by the Human Research Review Committee of the Medical College of Wisconsin.

The patients ranged in age from 26 to 79 years. Eight were women, eight were men; 11 were white, five were black. The cause of pericardial effusion was metastatic carcinoma in seven patients, congestive heart failure in one, uremia in three, progressive systemic sclerosis in one, viral pericarditis in one, Hodgkin's lymphoma in one, trauma in one, and postmyocardial infarction syndrome in one.

Fourteen of the 16 patients (88%) had jugular venous distention (greater than 3 cm above the sternal angle at 45 degrees). Twelve (75%) had pulsus paradoxus (greater than 10 mm inspiratory decline in systolic arterial pressure as measured with an arm sphygmomanometer) and three (19%) had a pericardial friction rub. Eight (50%) had low voltage on their 12-lead electrocardiograms and 15 (94%) had enlarged cardiac silhouettes on their chest roentgenograms.

Hemodynamic measurements and pericardiocentesis. Right heart catheterization was performed by either the right brachial or right femoral approach with a No. 7 thermodilution Swan-Ganz catheter (Edwards Laboratories). Arterial pressure was recorded through a No. 7 sheath (Cordis, Inc.) inserted into the right femoral artery. Pressures were recorded with a fluid-filled system linked to pressure transducers (Gould-Statham, Inc.). A No. 18 gauge Sorensen needle was inserted into the pericardial space by the subxiphoid approach under electrocardiographic monitoring, a 0.035 inch guidewire was inserted through the needle, and the needle was removed and the guidewire exchanged for a subclavian hemodialysis catheter with multiple end and side holes (Sorensen SCS8). Intrapericardial, right ventricular, right atrial, pulmonary capillary wedge, and systemic arterial blood pressures and thermodilution cardiac output were recorded immediately before pericardiocentesis in all patients and repeated in 15 of the 16 at the completion of pericardiocentesis. In each case pericardiocentesis was performed until no more fluid could be withdrawn. Measurements could not be repeated in one patient because of malfunction of the recording system.

In three patients who appeared to be tolerating the procedure quite well, pressure and output measurements and the two-dimensional echocardiograms were monitored repeatedly during pericardiocentesis. To obtain information at the point of disappearance of right ventricular diastolic collapse, pericardial fluid was withdrawn in small increments in these three patients during frequent recording of pressure data and echocardiographic data.

Cardiac tamponade was considered to be present if there was equalization of right atrial, pulmonary capillary wedge, and intrapericardial pressures and elevation of these pressures to greater than 10 mm Hg.

No complications related to pericardiocentesis were encountered.

Echocardiography. All patients underwent standard M mode and two-dimensional echocardiographic examination with a commercially available instrument (Irex Medical Systems) before the hemodynamic study and pericardiocentesis. Recordings were made from all available acoustic windows. After insertion of the pericardial catheter, studies were performed from the cardiac apex or left parasternal border. Recordings were made immediately before pericardiocentesis and at the end of pericardiocentesis in all 16 patients. In three patients, the echocardiogram was also monitored continuously during pericardiocentesis, with recordings being made simultaneously with hemodynamic measurement after incremental withdrawals of pericardial fluid and at the point of disappearance of right ventricular diastolic collapse as well as at the end of pericardiocentesis.

Strip-chart and videotape recordings of the echocardiograms were later analyzed independently by two observers who were unaware of simultaneous hemodynamic findings. Real-time, slow-motion, and frame-by-frame analysis of the two-dimensional echocardiograms was carried out with a microcomputer-based analysis system (Microsonics, Inc.). Right ventricular diastolic collapse was defined as a persistent inward motion of the right ventricular endocardial surface after opening of the mitral valve.6 Right atrial collapse was considered to be present if the right atrial free wall appeared to be inverted at any point in the cardiac cycle.9 Quantitative analysis of wall curvature was not performed.

Statistical analysis. The sensitivity (positive echoes/total number of patients with tamponade), specificity (negative echoes/total number of patients without tamponade), accuracy (true positives + true negatives/total group), and the predictive value (true positives/true positives + false positives) of these markers of cardiac tamponade were calculated. Patients were also divided into three groups for comparison. Those in group I exhibited right ventricular or right atrial collapse or both and demonstrated hemodynamic evidence of cardiac tamponade before pericardiocentesis. Those in group II had no right ventricular or right atrial collapse and no hemodynamic evidence of cardiac tamponade before pericardiocentesis. The patient in group III had hemodynamic evidence of cardiac tamponade but no right ventricular or right atrial collapse. Mean hemodynamic data obtained before and after pericardiocentesis in the three groups were compared with Student's paired t test.

Results

Group I. Representative examples of the two-dimensional echocardiograms recorded in patients in group I before pericardiocentesis are illustrated in figures 1 and 2. Figure 1, top, shows right ventricular diastolic collapse early in diastole. Normal curvature of the right ventricle is seen during systole (bottom panel).

Figure 2 shows normal curvature of the right atrium
late in diastole before atrial systole (top panel). After atrial systole, early in ventricular systole, right atrial collapse occurred.

Both right ventricular and right atrial collapse disappeared after pericardiocentesis.

Right ventricular diastolic collapse was present in 13 patients before pericardiocentesis; right atrial collapse was noted in nine of these 13. In the remaining four patients the right atrial free wall was inadequately imaged and the presence or absence of right atrial inversion could not be accurately judged. Neither right ventricular nor right atrial collapse was present after pericardiocentesis in any of the patients in group I.

The hemodynamic data obtained in group I are listed in table 1. Right atrial, pulmonary capillary wedge, and intrapericardial pressures were all elevated and showed “equalization” characteristic of cardiac tamponade. The right atrial pressure waveforms showed obliteration of the Y descent. Cardiac output was not as low as might have been expected, perhaps because many of these patients were severely anemic.

Cardiac output and stroke volume both rose significantly after pericardiocentesis as heart rate and right atrial, pulmonary capillary wedge, and intrapericardial pressures all fell. Measurements made in three patients during pericardiocentesis (table 2) showed significant increases in cardiac output and stroke volume and significant decreases in pulmonary capillary wedge pressure and intrapericardial pressure at the point when right ventricular diastolic collapse disappeared. Heart rate and right atrial pressure were not significantly different at that time. Further improvement occurred in cardiac output as pericardiocentesis was continued, after right ventricular diastolic collapse had disappeared. Although the precise point of disappearance was not noted, right atrial collapse disappeared after the disappearance of right ventricular diastolic collapse in all three patients.

No interobserver variability was noted in the detection of either right ventricular or right atrial diastolic collapse. Right ventricular diastolic collapse was especially striking and easy to detect. The average volume of pericardial fluid drained in group I was 802 ml (range 190 to 2100).

Group II. The three patients in group II did not have cardiac tamponade. Hemodynamic measurements in this group, listed in table 1, showed no evidence of cardiac tamponade before pericardiocentesis and no significant change after pericardiocentesis. Neither right ventricular nor right atrial collapse were seen in these patients. The average volume of pericardial fluid drained in these patients was 427 ml (range 200 to 850).

Group III. One patient had severe pulmonary hypertension resulting from sclerodermic lung disease (pulmonary arterial pressure 110/60 before pericardiocentesis) with right ventricular and right atrial hypertrophy. No right ventricular or right atrial collapse was present before or after pericardiocentesis. The volume of pericardial fluid drained in this patient was 850 ml. Hemodynamic measurements were not repeated after pericardiocentesis, but the condition of the patient clinically appeared to be improved.

Sensitivity and specificity. The sensitivity of right ventricular collapse as a marker of cardiac tamponade was 92%, its specificity was 100%, its accuracy 94%, and its predictive value 100%. The sensitivity of right atrial collapse was 64%, its specificity 100%, its accuracy
TABLE 1
Hemodynamic characteristics of groups I and II

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pretap</th>
<th>Posttap</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I (pretap n = 12; posttap n = 11)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>4.0 ± 1.4</td>
<td>7.9 ± 2.0</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>112 ± 19.1</td>
<td>103 ± 14.25</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>SV (ml/beat)</td>
<td>37.1 ± 14.2</td>
<td>78.2 ± 18.4</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>RAP (mm Hg)</td>
<td>17.8 ± 9.2</td>
<td>8.5 ± 5.9</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PCW (mm Hg)</td>
<td>22.8 ± 7.7</td>
<td>15.5 ± 7.5</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>IPP (mm Hg)</td>
<td>20.8 ± 7.3</td>
<td>2.1 ± 3.8</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>98 ± 6.2</td>
<td>128 ± 7.9</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Group II (n = 3 pretap and posttap)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>5.39 ± 1.06</td>
<td>5.45 ± 1.4</td>
<td>NS</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>111 ± 15.5</td>
<td>94.7 ± 19.9</td>
<td>NS</td>
</tr>
<tr>
<td>SV (ml/beat)</td>
<td>48.85 ± 7.33</td>
<td>59.2 ± 19.8</td>
<td>NS</td>
</tr>
<tr>
<td>RAP (mm Hg)</td>
<td>8.88 ± 4.4</td>
<td>7.78 ± 5.05</td>
<td>NS</td>
</tr>
<tr>
<td>PCW (mm Hg)</td>
<td>11.5 ± 1.0</td>
<td>10.25 ± 4.9</td>
<td>NS</td>
</tr>
<tr>
<td>IPP (mm Hg)</td>
<td>5 ± 4.24</td>
<td>2.0 ± 0</td>
<td>NS</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>111 ± 11.9</td>
<td>116 ± 17.2</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean ± SE. Group I consists of patients who had tamponade and either right ventricular or right atrial collapse or both. Group II patients had no evidence of tamponade and no right ventricular or right atrial collapse.

Pretap = before pericardiocentesis; Posttap = after pericardiocentesis; CO = cardiac output; HR = heart rate; IPP = intrapericardial pressure; PCW = pulmonary capillary wedge pressure; RAP = right atrial pressure; RVDC = right ventricular diastolic collapse; SV = stroke volume.

DISCUSSION

The value of echocardiography in detecting pericardial effusion is undisputed.1,2 We believe echocardiography can also help establish the presence or absence of cardiac tamponade. We have documented a major derangement of hemodynamic function associated with right ventricular and right atrial collapse. Pericardiocentesis led to improvement in hemodynamic function and resulted in the disappearance of right ventricular and right atrial diastolic collapse.

Continuous recording in three patients during pericardiocentesis showed significant increases in cardiac output and decreases in intrapericardial and pulmonary capillary wedge pressures at the point at which right ventricular collapse disappeared. Further improvement occurred with continued pericardiocentesis. Although the precise moment when right atrial collapse disappeared was not established, right atrial collapse persisted after right ventricular collapse disappeared. Right atrial collapse, which ceased after pericardiocentesis, thus may exist with lesser hemodynamic change than right ventricular collapse and may be an earlier sign of cardiac tamponade. The smallest hemodynamic change necessary to produce right atrial collapse was not determined in this study.

Four patients undergoing pericardiocentesis had neither right ventricular nor right atrial collapse before the procedure. Three of the four did not have hemodynamic evidence of cardiac tamponade and showed no significant change after pericardiocentesis.

One patient, however, did have hemodynamic evidence of cardiac tamponade in the absence of right ventricular or right atrial collapse. This patient had severe pulmonary hypertension, elevation of right ventricular and right atrial pressures, and hypertrophy of both chambers, which presumably prevented their collapse. This false-negative finding was predicted by our observations in the dog that right ventricular hypertrophy or short-term elevation of right heart pressures can result in the absence of right ventricular diastolic collapse during cardiac tamponade.

TABLE 2
Hemodynamic data from three patients in group I in whom measurements were made at the point at which right ventricular collapse disappeared

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pretap</th>
<th>Point of RVDC disappearance</th>
<th>p value</th>
<th>Posttap</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO (l/min)</td>
<td>3.3 ± 1.1</td>
<td>5.6 ± 0.8</td>
<td>&lt;.02</td>
<td>6.7 ± 1.6</td>
<td>NS</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>89 ± 9</td>
<td>92 ± 10</td>
<td>NS</td>
<td>87.3 ± 11.6</td>
<td>NS</td>
</tr>
<tr>
<td>SV (ml/beat)</td>
<td>36.5 ± 11.8</td>
<td>61.2 ± 10.3</td>
<td>&lt;.001</td>
<td>78.1 ± 18.4</td>
<td>NS</td>
</tr>
<tr>
<td>RAP (mm Hg)</td>
<td>24 ± 6</td>
<td>16 ± 5</td>
<td>NS</td>
<td>12 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>PCW (mm Hg)</td>
<td>25.3 ± 0.6</td>
<td>19 ± 0</td>
<td>&lt;.01</td>
<td>16 ± 3.5</td>
<td>NS</td>
</tr>
<tr>
<td>IPP (mm Hg)</td>
<td>25.3 ± 0.6</td>
<td>3.7 ± 12.5</td>
<td>&lt;.01</td>
<td>3.3 ± 6.4</td>
<td>NS</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>100.3 ± 22.5</td>
<td>126.2 ± 26.5</td>
<td>&lt;.01</td>
<td>131.0 ± 26.9</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviations are as in table 1.
Significance values compare measurements made when RVDC disappeared with pretap values; posttap measurements were compared with measurements made at the point RVDC disappeared.

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The volume of pericardial fluid drained in groups I and II did not differ. Patients in both groups had large effusions, but those in group II must have had more compliant pericardia since there was no evidence of significant compression. Intrapercardial pressure before pericardiocentesis was 20.8 ± 7.3 mm Hg in group I but only 5.0 ± 4.2 mm Hg in group II patients.

These observations extend those of Shina et al., who found that the right ventricular free wall continued to move in a posterior direction through early and mid-diastole in patients with "impending tamponade." Armstrong et al. and Engel et al. confirmed the association of right ventricular diastolic collapse and cardiac tamponade, but since invasive hemodynamic measurements were often not made in their studies, some patients presented confusing clinical pictures and unequivocal assessment was not possible.

The association between right atrial collapse and cardiac tamponade has previously been noted in studies in which echocardiography and angiography were used. The appearance of right atrial collapse on the echocardiogram seemed to be a highly sensitive indication of cardiac tamponade. Gillam et al. demonstrated that the specificity of right atrial collapse in diagnosing cardiac tamponade was also quite high, especially if collapse lasted through greater than 34% of the cardiac cycle. However, invasive hemodynamic measurements were made in a minority of the patients in these reports. While we did not determine the precise hemodynamic correlates of right atrial collapse in cardiac tamponade, we have confirmed that it is a highly sensitive feature.

This study is a natural extension of our previous work investigating the hemodynamic changes associated with right ventricular collapse in an anesthetized animal preparation of cardiac tamponade studied over the long term. In both animals and humans, right ventricular diastolic collapse appears to occur early in the course of hemodynamic deterioration resulting from cardiac tamponade but at a time when significant changes of potential clinical importance have occurred. Right ventricular collapse was noted to occur in the animal preparation when intrapericardial pressure exceeded right ventricular intracavitary pressure. Conditions altering transmural pressure or wall stiffness, such as hypertension or hypertrophy, may prevent collapse from occurring and cause false-negative diagnostic findings. We have not encountered false-positive findings of right ventricular diastolic collapse.

The genesis of right ventricular and right atrial collapse in cardiac tamponade is of interest. As cardiac tamponade develops, pericardial fluid accumulates under increasing pressure as the distensibility of the pericardial sac is exceeded. Right atrial collapse presumably occurs when intrapericardial pressure transiently exceeds right atrial pressure. Right atrial pressure and volume are at a minimum at end-diastole, when right ventricular diastolic pressure and volume are at their maximum. As intrapericardial pressure rises further, right atrial collapse occurs in pericardium. Collapse of the right ventricle occurs early in diastole (during and after isovolumetric relaxation), at a time when right ventricular pressure and volume are at their lowest. Right ventricular and right atrial collapse thus appear at opposing times during the cardiac cycle, in association with alternating changes in right ventricular and right atrial intracavitary pressures. Right atrial collapse may occur earlier in the progression of cardiac tamponade than does right ventricular collapse, perhaps because the right atrial wall is more compliant.

Right ventricular and right atrial collapse are thus very sensitive and accurate markers of cardiac tamponade. The sensitivity and specificity of these findings are impaired under circumstances in which the hemodynamic status and properties of the ventricular or atrial walls are altered by other processes. Collapse may be absent during cardiac tamponade in some patients due to lung disease or other conditions leading to a marked elevation in right heart pressures and hypertrophy. Tricuspid regurgitation and ventricular pacing may mask these signs.

In summary, right atrial collapse occurs very early in the course of cardiac tamponade. Right ventricular collapse occurs somewhat later, but still at a relatively early point when cardiac output is significantly depressed but before systemic arterial pressure has changed. These echocardiographic signs should provide an important new clinical tool enabling noninvasive discrimination of pericardial effusions causing cardiac tamponade from those that are hemodynamically benign. Cardiac tamponade thus may be detected noninvasively early in its course when therapy can be more safely provided.

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