CARDIAC TAMPOONADE was recognized in the nineteenth century as a cause of impaired cardiac function.1, 2 Pericardial disease has become much more common in recent years. Longer survival of patients with malignant disease, growing numbers of cardiac surgical operations, the treatment of chronic renal disease with dialysis procedures, the common use of anticoagulant drugs and the use of new drugs and irradiation in tumor therapy are largely responsible. Patients with acute pericarditis may have cardiac tamponade as the presenting feature. Medical patients with acute or subacute tamponade commonly differ from those with acute tamponade due to penetrating cardiac injury, because the fluid accumulation is often more gradual. Hence, the classic small quiet heart with increasing venous pressure and decreasing blood pressure may not be found in the medical patient with tamponade. We reviewed 56 cases of cardiac tamponade seen since 1963 by members of the Division of Cardiology of the University of Cincinnati. In this report, we emphasize the etiologic background, the clinical diagnosis, and the electrocardiographic, echocardiographic and radiologic features of cardiac tamponade in these medical patients.

Methods

Clinical and laboratory findings of patients with cardiac tamponade due to pericardial effusion who were evaluated between 1963 and 1980 were determined from review of notes of Cardiology Division staff members and from patient charts at five hospitals affiliated with the University of Cincinnati Medical Center. Most of these patients had been examined by one or more of our Cardiology Division staff. Cardiac tamponade was defined by the following criteria: increased jugular venous pressure as determined by inspection of the cervical veins, a paradoxical arterial pulse and the documentation of pericardial effusion by echocardiography, radioisotope scanning, contrast angiography or pericardial drainage. In addition, if a drainage procedure was performed, then the elevated venous pressure and paradoxical pulse improved or resolved. Paradoxical pulse was defined as an inspiratory decrease in systolic blood pressure of 10 mm Hg or more, measured by cuff or arterial cannulation. One patient had cardiac tamponade without a paradoxical pulse.

Results

Between 1963 and 1980, cardiac tamponade was diagnosed in 56 patients — 30 men and 26 women, average age 48 years (range 14–79 years).

Etiologies

The etiologies are listed in table 1. In part, the distribution of etiologies reflects the population served by the Cincinnati General Hospital, which provided 37 of the 56 cases, and to a degree the special interests of the University of Cincinnati Medical Center. The Cincinnati General Hospital principally serves a low-income population and has a relatively low admission rate of patients with ischemic heart disease or those who are to have cardiac surgery. On the other hand, the oncology service and the hemodialysis program are very active.

Ten of the 56 patients were receiving anticoagulant drugs and an additional six patients were receiving chronic hemodialysis for treatment of uremia. During dialysis all six uremic patients received systemic anticoagulants. Thus, 16 of our 56 patients were receiving anticoagulants at the time of, or immediately before, the recognition of cardiac tamponade.

Of the five patients who were receiving anticoagulant therapy for the treatment of acute cardiac infarction, each had received heparin during the first several days after hospital admission. Heparin was given intravenously in bolus doses of 4000–9000 U every 4 hours. Three patients were still receiving heparin when cardiac tamponade was recognized; coagulation studies in these three patients at that time

SUMMARY We reviewed the cases of 56 medical patients with cardiac tamponade who were treated at the University of Cincinnati. A paradoxical arterial pulse was critical in the diagnosis because most patients did not have a small quiet heart, and blood pressure was often well maintained. Fifty-two of 55 patients had enlarged cardiac silhouette by chest radiogram; heart sounds were diminished in 19 patients; arterial systolic pressure was ≥ 100 mm Hg in 35, and arterial pulse pressure was ≥ 40 mm Hg in 27. Echocardiograms in 23 patients showed abnormally increased right ventricular dimensions and decreased left ventricular dimensions during inspiration, except in one patient with left ventricular dysfunction. The causes of cardiac tamponade were metastatic tumor in 18 patients, idiopathic pericarditis in eight and uremia in five; five cases of tamponade occurred after heparin administration in acute cardiac infarction. Myxedema and dissecting aneurysm each caused tamponade in two patients. Pericardiocentesis relieved tamponade initially in 40 of 46 patients; however, two suffered fatal complications. Pericardial resection was done in 18, including 12 of these 46.

From the Division of Cardiology, Department of Medicine, University of Cincinnati College of Medicine, Cincinnati, Ohio.

Dr. Gueron, Visiting Professor of Medicine, 1976–1979, was on sabbatical leave from the Division of Cardiology, Ben Gurion University School of Medicine, Beershava, Israel.

Address for correspondence: Noble O. Fowler, M.D., Cardiology Division, University of Cincinnati Medical Center, 231 Bethesda Avenue, Cincinnati, Ohio 45267.

Received August 22, 1980; revision accepted December 29, 1980.

Circulation 64, No. 3, 1981.
TABLE 1.  

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malignant idiopathic pericarditis</td>
<td>18</td>
<td>32</td>
</tr>
<tr>
<td>Definite</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Probable</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Idiopathic pericarditis</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>Uremia</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>Acute cardiac infarction (receiving heparin)</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>Diagnostic procedures</td>
<td>4</td>
<td>7.5</td>
</tr>
<tr>
<td>Bacterial</td>
<td>4</td>
<td>7.5</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Radiation</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Myxedema</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Dissecting aortic aneurysm</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Postpericardiotomy syndrome</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Cardiomyopathy (receiving Coumadin)</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>56</td>
<td>100</td>
</tr>
</tbody>
</table>

showed the following: three-tube Lee-White clotting times were 16 minutes in one and 26 minutes in one (normal, 15 minutes) and one-tube Lee-White clotting time was 12 minutes in one (normal 4–6 minutes). In two patients, heparin had been stopped 6 days or more previously and the patients were receiving warfarin (Coumadin) when cardiac tamponade was recognized. On the day that tamponade was recognized in one of these two patients, the prothrombin time was 34 seconds (control 13 seconds) and in the other the prothrombin time was 15%.

In addition to the five patients with cardiac infarction and the six uremic patients undergoing hemodialysis, five other patients were receiving anticoagulant drugs when cardiac tamponade developed. Two patients with carcinoma of the lung were receiving anticoagulants because of thrombophlebitis. One patient had a prosthetic aortic valve, one patient had cardiomyopathy without a history of thromboembolism, and in one patient with idiopathic pericarditis, pulmonary embolism had been erroneously diagnosed. Four patients were receiving Coumadin and one was receiving heparin (5000 U i.v. every 4 hours). On the day that tamponade developed, prothrombin times in three of the four patients receiving Coumadin were 26.9 seconds, 85.2 seconds and 40.9 seconds (11.2 seconds control); in one patient, prothrombin time was less than 10% (time in seconds not given). In the patient receiving heparin, the partial thromboplastin time was 29 seconds (control 28 seconds) on the day before fatal tamponade was recognized. Thus, three of the five patients had evidence of excessive anticoagulant effect when tamponade occurred.

Physical Findings

Elevated systemic venous pressure was present in all patients, and paradoxical pulse was present in all patients except RM. Only one patient was believed to have ascites. Other physical findings are listed in table 2.

Systemic systolic blood pressure declined progressively to values below 90 mm Hg in 11 patients. Of these, two had bleeding without relation to anticoagulants, one had dissecting aneurysm and the other had perforation of the right ventricle by a pacing catheter. Four were receiving anticoagulants: two had recent cardiac infarction, one had idiopathic pericarditis and one had cardiomyopathy. Of the remaining three, one had metastatic carcinoma involving the pericardium, one had β-hemolytic streptococcal pericarditis and one had probable uremic pericarditis.

Fifty-three patients had a careful study of blood pressure variation with respiration. In three other patients a palpable paradox pulse was recorded in the chart but the decrease in systolic pressure was not recorded. One had no paradox pulse. Although 10 mm Hg was the minimum inspiratory decrease in blood pressure (pulsus paradoxus), most patients had a much larger paradox pulse (mean 49 mm Hg). In these 53 patients, the paradox pulse was 20 mm Hg or more in 41 (77%), 30 mm Hg or more in 26 (49%), 40 mm Hg or more in 20 (38%) and 50 mm Hg or more in 17 (32%). Total paradox (inspiratory disappearance of the brachial and radial pulses with total disappearance of Korotkoff sounds) was present in 12 patients, 11 of whom had a systolic blood pressure of 95 mm Hg or less.

Electrocardiograms

Conventional 12-lead ECGs from 53 of the 56 patients were reviewed. Forty-eight patients (91%) were in sinus rhythm during the episode of tamponade. Two patients had atrial flutter, two had atrial fibrillation and one patient had chaotic atrial tachycardia. Four of the five patients with atrial tachyarrhythmias had pulmonary disease or underlying cardiac disease.

ST-segment elevation was present in one or more leads in 16 of the 53 ECGs. In six, ST-segment eleva-

*One patient was intubated.
tion was present in all standard and precordial leads except aVR and V1. ST-segment elevation was not associated with ST-segment depression except in leads aVR and V1. Low voltage (total QRS deflection < 5 mm in each standard lead) was present in 21 of 53 ECGs.

Total electrical alternans (P, QRS and T) was found in only four patients; alternans of the QRS complex only was found in an additional seven patients. Six of 11 patients with either total alternans or only QRS alternans had malignant pericardial effusion. The average amount of fluid obtained from the initial drainage procedure was 970 ml in patients with alternans, 515 ml in those without alternans and 621 ml in the entire patient group.

Three of 53 ECGs were normal, and five were normal except for low QRS voltage.

Radiologic Investigations

Chest radiograms were obtained in 55 of the 56 cases. Fifty-two (95%) had an enlarged cardiopericardial silhouette (cardiothoracic ratio greater than 50%) and three had normal heart size by radiogram. One of the three patients with normal heart size had acute traumatic tamponade from a perforated right ventricular wall during placement of an endocardial pacing electrode. One had metastatic carcinoma of the lung and bloody pericardial fluid. The third patient had an acute dissecting aortic aneurysm that ruptured into the pericardial sac. Pulmonary congestion on radiogram was absent in all but two patients. These two patients had pericardial effusion associated with myxedema and each had evidence of congestive heart failure before tamponade.

Cardiac fluoroscopy was performed in only 10 cases. All 10 had decreased pulsations of the cardiopericardial shadow, but in only two of the 10 was the epicardial fat pad seen within the pericardial shadow, demonstrating the presence of pericardial effusion. Cardiac fluoroscopy was not done in most instances because of its limited value in separating pericardial effusion from congestive cardiomyopathy and because of the risk to the patient incurred by absence from the critical care unit.

In six of the seven patients who underwent radioisotope blood pool scanning, the study was positive for pericardial effusion. In one patient the scan was interpreted as equivocal, even though 300 ml of fluid were removed by pericardial resection within 1 week of the scan.

By angiography, eight of nine patients had concavity to the right of the right atrial border after contrast injection. Similar observations were reported by Spitz and Holmes* in six of nine patients with cardiac tamponade, of whom seven are included in this report. The right atrial border was convex to the right in four patients with pericardial effusion and no tamponade in that study.

Hemodynamic Investigations

Twelve patients underwent right-heart catheterization during cardiac tamponade. Right atrial pressure during forward angiograms was measured in two others. The average pulmonary artery systolic pressure was 42 mm Hg (range 26–85 mm Hg). The average pulmonary artery diastolic pressure was 23 mm Hg (range 15–37 mm Hg). The average pulmonary artery mean pressure was 30 mm Hg (range 20–55 mm Hg). The average pulmonary artery wedge pressure was 23 mm Hg (range 17–28 mm Hg). The average right atrial mean pressure was 21 mm Hg (range 13–32 mm Hg). In 11 patients, mean right atrial and pulmonary capillary wedge or pulmonary artery diastolic pressures were measured simultaneously. In 10 patients these three pressures were within 4 mm Hg of one another.

Echocardiograms

Of the 56 patients, 23 had standard M-mode echocardiographic examination before relief of tamponade and 21 after tamponade was relieved by pericardiocentesis or pericardectomy. Seventeen patients had technically adequate studies both before and after relief of tamponade. Tables 3 and 4 present a summary of the echocardiographic findings during cardiac tamponade and after pericardial drainage. Twenty of 21 patients had evidence of both anterior and posterior pericardial effusion, and five of 24 had an echo-free space posterior to the left atrium (table 3).4

Other than evidence of pericardial effusion, the most consistent findings on M-mode echocardiographic examination in patients with cardiac tamponade were an abnormal inspiratory increase of right ventricular diastolic dimension and an abnormal inspiratory decrease of left ventricular diastolic dimension, each exceeding 10% of the expiratory diastolic

<table>
<thead>
<tr>
<th>Table 3. Echocardiographic Findings During Tamponade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior echo-free space</td>
</tr>
<tr>
<td>Posterior echo-free space &gt; 1 cm</td>
</tr>
<tr>
<td>Echo-free space posterior to left atrium</td>
</tr>
<tr>
<td>Mitral EF slope &gt; 70 mm/sec</td>
</tr>
<tr>
<td>Inspiratory decrease in mitral EF slope</td>
</tr>
<tr>
<td>Inspiratory decrease in left ventricular diastolic dimension</td>
</tr>
<tr>
<td>Inspiratory increase in right ventricular diastolic dimension</td>
</tr>
<tr>
<td>Inspiratory decrease in left ventricular fractional shortening</td>
</tr>
<tr>
<td>Right ventricular diastolic end-diastolic dimension &lt; 8 mm</td>
</tr>
<tr>
<td>Right ventricular systolic notch</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 4. Echocardiographic Changes After Relief of Tamponade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased total cardiac dimension</td>
</tr>
<tr>
<td>Increased right ventricular diastolic dimension</td>
</tr>
<tr>
<td>Increased mitral EF slope</td>
</tr>
<tr>
<td>Persistent inspiratory decrease in left ventricular diastolic dimension</td>
</tr>
<tr>
<td>Persistent inspiratory increase in right ventricular diastolic dimension</td>
</tr>
</tbody>
</table>
value (table 3). In normal adults, respiratory variations in right or left ventricular dimensions do not exceed 3 mm. The diagnostic value of this observation is illustrated by the fact that among 14 of our patients with measurable inspiratory and expiratory right and left ventricular dimensions during tamponade, 12 had abnormal respiratory variation in dimensions of both ventricles. The one patient who had neither finding (figs. 1 and 2) had left ventricular dysfunction with pulmonary wedge pressure of 28 mm Hg and intrapericardial pressure of 24 mm Hg during tamponade (figs. 3–5). An inspiratory decrease in minor-axis fractional shortening (a measure of myocardial function) has been observed during tamponade, which would suggest an inspiratory alteration of left ventricular function. We observed persistent respiratory variations in ventricular dimensions in some patients after relief of cardiac tamponade by pericardiocentesis or pericardectomy, but initial relief of tamponade may have been incomplete in some instances. Although Schiller and Botvinick consistently found an end-expiratory right ventricular dimension of less than 8 mm in cardiac tamponade, this was not confirmed by our observations; however, the consistent increase in right ventricular dimension after relief of tamponade in eight of nine patients in whom these data were available suggests that right ventricular compression is, in fact, present during tamponade (table 4). Unfortunately, this may not be apparent until after the hemodynamic abnormality has been relieved. Similarly, our observation of an increase in the total cardiac dimension after pericardial drainage is not useful in the original diagnosis, but confirms the clinical impression of cardiac compression due to pericardial effusion (table 4).

**Treatment**

Pericardiocentesis was attempted in 46 patients for relief of tamponade. Fluid was obtained and tamponade was relieved in 40 of the 46 patients. In three patients fluid could not be obtained. One of these three had infectious pericarditis (microaerophilic β-hemolytic streptococcus); pericardial resection was successful and 500 ml of purulent pericardial fluid were removed. Of the other two patients, one died of tamponade from metastatic lung carcinoma involving the pericardium and the other responded to oral adrenal steroid therapy for nonspecific pericarditis with no recurrence.

The right ventricle was lacerated during the three other unsuccessful pericardiocenteses. One patient died during the procedure. The second patient suffered a cardiorespiratory arrest, from which she was resuscitated and then underwent emergency open pericardial drainage and repair of the lacerated right ventricle. Although this patient remained hemodynamically stable thereafter, hypoxic brain damage occurred during arrest and led to death from aspiration pneumonia 2 weeks later. The third patient did well after emergency open pericardial drainage and repair of the right ventricle. Thus, there were three major complications from 46 attempts at pericardiocentesis, yielding a major complication rate of 7%.

Pericardial resection was required in 12 of these 46 patients. In nine this was done because fluid reaccumulated rapidly after one or more pericardiocenteses, in two to repair a laceration of the right ventricle, and in one because no fluid was obtained by pericardiocentesis. Pericardiocentesis was not attempted in 10 patients; open-chest pericardial resection was carried out in six and no drainage procedure...
was done in four. Of these four, one was terminally ill with metastatic lung cancer, one was successfully treated with adrenal steroids for Dressler's post–myocardial infarction syndrome, and one was successfully treated with parenteral antibiotics for staphylococcus aureus endocarditis and pericarditis. A fourth patient died of ruptured aortic dissecting aneurysm with cardiac tamponade; the diagnosis was not suspected in time for surgical treatment.

Pericardial fluid was obtained in 50 patients and its quantity was measured in 48. The volume was 65–2000 ml (average 634 ml) and exceeded 1000 ml in 11 patients. Pericardial fluid was described as bloody in 44 of 50 patients; the fluid hematocrits were from 8–47% in these 44 patients. Pericardial fluid hematocrits were 40% or more in five patients; one of these had Hodgkin's disease, one had dissecting aortic aneurysm, one had catheter-induced perforation of the right ventricle, one had idiopathic pericarditis and was receiving Coumadin, and one had cardiomyopathy and was also receiving Coumadin. Five others had pericardial fluid with hematocrit of 30% or more; of these, one had metastatic lung cancer, one had non-specific pericarditis, and three were receiving anti-coagulant therapy. Pericardial fluid cultures were positive for bacteria other than M. tuberculosis in three patients (staphylococcus aureus, microaerophilic β-hemolytic streptococcus and pneumococcus). One patient had staphylococcus aureus endocarditis without a positive culture of the pericardial fluid.

Cytologic study of pericardial fluid was done in 12 patients who had metastatic cancer. Studies for tumor cells were positive in six and unrevealing in six.

**Clinical Course**

Thirty-eight of the 56 patients survived the episode of cardiac tamponade and were discharged from the hospital. Of 18 patients with metastatic malignant disease, eight survived the hospitalization for cardiac tamponade. Thirty of 38 patients without malignant effusion survived hospitalization. Seven of the eight patients with malignant effusion died within 13 months of the initial hospital discharge. Of the 30 patients without malignant effusion, 26 were alive at the last follow-up examination, after an observation period of 1 month to 11 years (average 3.2 years).

**Discussion**

In 1935, Beck described two triads for the diagnosis of cardiac compression. The triad of acute cardiac compression consisted of a decreasing arterial pressure, an increasing venous pressure and a small quiet heart. This triad occurred most commonly.
because of intrapericardial hemorrhage caused by penetrating wounds of the heart, rupture of a myocardial infarct, rupture of a coronary or aortic aneurysm, or rupture of the base of a sclerotic aorta. The second chronic cardiac compression triad consisted of a high venous pressure, ascites and a small quiet heart. This triad occurred most often in the setting of tuberculosis, idiopathic pericarditis or constrictive pericarditis without effusion.\(^5\)\(^,\)\(^6\)

Our experience with cardiac tamponade at the University of Cincinnati Medical Center reveals a clinical pattern difference from either of Beck's two triads. Our patients were usually acutely but not critically ill. Dyspnea was the most frequent complaint and tachypnea or frank respiratory distress was generally present. Most patients were not in a shock-like state: 64% had an average systolic arterial blood pressure greater than 100 mm Hg, and 55% had an arterial pulse pressure of 40 mm Hg or more. Most patients were alert, oriented and able to provide a meaningful history. Urinary output was maintained and their extremities were usually warm.

The recognition of pericardial effusion and cardiac tamponade was often difficult in this series of 56 medical patients. Elevated systemic venous pressure, dyspnea and tachycardia were present in most patients; thus, myocardial failure was commonly the first diagnosis and pericardial disease was often not recognized in the beginning. The presence of a paradoxical arterial pulse was often the first clue to the diagnosis of tamponade. Only two of our patients had evidence of pulmonary congestion on radiogram, and each of them had had myocardial failure. Thus, clear lung fields despite an enlarged cardiopericardial silhouette suggested the possibility of pericardial disease rather than myocardial failure.

Although a paradoxical pulse is a valuable sign, it is not specific for cardiac tamponade and may be absent. A paradoxical pulse may occur in patients with acute or chronic obstructive airway disease,\(^11\)\(^,\)\(^12\) shock,\(^18\) pulmonary embolism,\(^14\) constrictive pericarditis,\(^15\) right ventricular infarction,\(^16\) restrictive cardiomyopathy,\(^17\) and extreme obesity or tense ascites.\(^18\) Inspiratory decline of systolic blood pressure of 10 mm Hg may occur in normal persons.\(^19\) Pulsus paradoxus may be misleading in patients with obstructive airway disease and cor pulmonale if taken as evidence of cardiac tamponade. Such patients may be separated from those with tamponade when there is no right ventricular failure, as the systemic venous pressure is then elevated with tamponade only. However, when there is cor pulmonale with right-heart failure, not only may there be pulsus paradoxus, but systemic venous pressure is elevated as well. In some such instances pericardial effusion is present, and there may be abnormal inspiratory increase of right ventricular dimension on the echocardiogram without tamponade.\(^5\)

Pulsus paradoxus may be absent during cardiac tamponade under some conditions. Atrial septal defect,\(^20\) aortic regurgitation, severe aortic stenosis,\(^21\) or uremia with left ventricular dysfunction may be complicated by cardiac tamponade without pulsus paradoxus. In addition, with severe hypotension accompanying advanced tamponade, the paradoxical pulse may be difficult or impossible to detect with a blood pressure cuff. Despite these exceptions, pulsus paradoxus is present in nearly every patient with tamponade, and cardiac tamponade is to be strongly suspected in every patient who has symptoms and findings suggestive of myocardial failure, but who also has a paradoxical arterial pulse.

Reddy et al.\(^22\) found that four patients with uremia and left ventricular dysfunction failed to have a significant paradoxical pulse during tamponade. They reasoned that in tamponade, right- and left-heart filling usually occur against a common ventricular diastolic stiffness (equal ventricular diastolic pressures). However, when left ventricular filling pressure exceeds intrapericardial pressure, there is less or no respiratory variation in left ventricular filling, because filling is then controlled by the higher left ventricular diastolic pressure. Our observations in patient RM were consistent with those of Reddy et al.\(^22\) In RM, the initial pulmonary wedge pressure (28 mm Hg) exceeded right atrial pressure (25 mm Hg) and intrapericardial pressure by 3 mm Hg (fig. 3). After removal of 1200 ml of pericardial fluid, the wedge pressure remained elevated at 25 mm Hg, right atrial pressure decreased to 10 mm Hg, and intrapericardial pressure was —3 mm Hg (figs. 4 and 5). Thus, there was an elevation of both right and left ventricular pressures during tamponade, but the right atrial pressure was controlled by the intrapericardial pressure, whereas the left ventricular filling pressure was not. Hence, systemic systolic pressure decreased only 6—8 mm Hg during inspiration with tamponade. There was no inspiratory decline of systemic systolic blood pressure after relief of tamponade.

Cardiac tamponade was frequently the initial clue to pericardial disease. In 81% of the patients in the present series, cardiac tamponade was the presenting manifestation of pericarditis, but a history of chest pain was obtained in many of these patients.

In contrast to Beck’s description, few of our cases of tamponade were associated with rapid intrapericardial bleeding due to trauma. Our series contained two cases of tamponade after diagnostic pericardiocentesis, one case of perforation of the right ventricle with an endocardial pacemaker and one case of tamponade after left ventricular puncture as a Brock procedure.

Pericardial effusion is frequently present in myxedema and was found in 30% of 33 patients studied by echocardiography.\(^28\) Nevertheless, cardiac tamponade as a result of myxedema was not reported until 1965, by Martin and Spathis\(^44\) and by Ivy.\(^24\) Four earlier reports of patients with myxedema and pericardial effusion included hemodynamic data consistent with cardiac tamponade,\(^26\)\(^—\)\(^28\) and nine other reports have since appeared.\(^30\)\(^—\)\(^32\) In the present series, two patients had cardiac tamponade as a result of myxedema.

Five patients had tamponade related to the peri-
carditis associated with acute myocardial infarction. Each patient was receiving anticoagulants and had received them early during the course of the infarction. This experience illustrates the possible risk of causing tamponade with anticoagulant therapy in patients with known or suspected pericardial disease.39-41 Patients with acute myocardial infarction who are receiving anticoagulants should be observed closely for a pericardial friction rub and signs of tamponade. Chalmers et al.42 concluded that anticoagulant therapy does not increase the risk of intrapericardial bleeding after myocardial infarction, as they found an incidence of only 0.1% of hemopericardium in patients with cardiac infarction whether anticoagulants were given or not. However, our experience with five patients who had hemopericardium and were receiving anticoagulants suggests a cause-and-effect relationship.

This series yields further information on the place of the echocardiogram in the diagnosis of cardiac tamponade.43,44 Of 22 patients with adequate echocardiograms, an echo-free space posterior to the left ventricle was found in 21. In patient ER, the echocardiogram was technically inadequate to recognize pericardial effusion, and angiography was needed to make the diagnosis. The most consistent M-mode echocardiographic findings in our patients with tamponade were an abnormal inspiratory increase of right ventricular diastolic dimension and an abnormal inspiratory decrease of left ventricular diastolic dimension, each of which exceeded 10% of the end-diastolic diastolic value. Similar respiratory variations in ventricular dimensions may occur in patients with pulsus paradoxus due to obstructive airway disease45 and acute pulmonary embolism.46 Thus, abnormal respiratory variations of ventricular dimensions46 are not specific for tamponade, but are suggestive when a large pericardial effusion is present. Tumor encasing the heart may suggest cardiac tamponade by echocardiogram.47,48 On the other hand, as shown by patient RM, both pulsus paradoxus and abnormal respiratory changes in ventricular dimensions may be absent when left ventricular dysfunction has increased left ventricular end-diastolic pressure above intrapericardial pressure.

Until now, abnormal respiratory variation in ventricular dimensions by echocardiogram has been observed in all reported patients with cardiac tamponade who have a paradoxical pulse.49,50,51,52,53,54 The electrocardiographic findings were nonspecific in the majority of our patients with tamponade. Sinus rhythm was present in 48 of 54 patients, and only 17 had ST-segment changes suggestive of acute pericarditis. Electrical alternans, especially P, QRS and T-wave alternans, may be associated with pericardial effusion, especially when there is tamponade.50,51 However, only 11 of our patients had alternans of the QRS complex, and only four had alternans involving P, QRS and T complexes.

Our study does not deal with the question as to whether pericardiocentesis by needle is preferred to open surgical drainage as initial treatment of pericardial effusion with or without tamponade.52 Needle aspiration is clearly indicated when there is immediate danger of death from tamponade; surgical drainage is clearly indicated when there is failure to relieve acute tamponade by needle aspiration, when tamponade continues to recur after needle aspiration, when surgical repair of a cardiac injury is required, or when diagnostic pericardial biopsy is essential. Cytologic study of pericardial fluid failed to show malignant cells in four of 10 of our patients with verified metastatic tumor. This experience was unlike that of Krikorian and Hancock,44 but resembles that of King and Kieberg.45 We had three instances of right ventricular laceration with two deaths among 46 pericardiocenteses, and there was one death among 19 pericardial resections. Our policy is to require that all pericardiocentesis be done by cardiology staff members.

References
1. Chevers N: Diseases of the orifice and valves of the aorta. Guy's Hospital Report (1st series) 7: 387, 1842
8. Schiller NB, Botvinick EH: Right ventricular compression as a sign of cardiac tamponade. Circulation 56: 774, 1977
15. Spodick D: Chronic and Constrictive Pericarditis. New York, Grune & Stratton, 1964, pp 244
20. Winer HE, Kronzon I: Absence of paradoxical pulse in patients
with cardiac tamponade and atrial septal defects. Am J Cardiol 44: 378, 1979
33. Sharma SK, Bordia A: Cardiac tamponade due to pericardial effusion in myxedema. Indian Heart J 21: 210, 1969
45. Winer H, Kronzon I, Glassman E: Echocardiographic findings in severe paradoxical pulse due to pulmonary embolism. Am J Cardiol 40: 808, 1977
Cardiac tamponade in medical patients.
B A Guberman, N O Fowler, P J Engel, M Gueron and J M Allen

Circulation. 1981;64:633-640
doi: 10.1161/01.CIR.64.3.633
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/64/3/633

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation 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 is online at:
http://circ.ahajournals.org//subscriptions/