Subacute Effusive-Constrictive Pericarditis

By E. W. Hancock, M.D.

SUMMARY
Clinical and hemodynamic observations are reported in 13 patients who demonstrated a distinct pathophysiologic form of compressive pericardial disease characterized by effusion into a free pericardial space associated with constriction of the heart by the visceral pericardium. The pericardial disease was idiopathic (nine patients) or subsequent to radiotherapy (four patients), with a maximal duration of symptoms of 16 months. Enlarged heart shadow, prominent paradoxical pulse, predominant systolic dip in the venous pressure pulse, and absence of atrial fibrillation were features that tended to distinguish the effusive-constrictive cases from those with noneffusive chronic constrictive pericarditis. The value of pressure measurements during combined cardiac catheterization and pericardiocentesis is emphasized in the differentiation of effusive-constrictive pericarditis from cardiac tamponade without constriction. Four patients were observed to progress through an effusive-constrictive phase to noneffusive constrictive pericarditis. Corticosteroids, pericardiocentesis, and other medical therapy produced some benefit but did not relieve constriction. The results of surgery were generally satisfactory.

Additional Indexing Words:
Pericardiocentesis  Pericardiectomy  Radiation heart disease
Venous pulse

Pericardial disease that results in compression of the heart is usually classified as chronic constrictive pericarditis or as cardiac tamponade, the former consisting of constriction by a fibrotic fusion of visceral and parietal pericardial layers, and the latter consisting of tense effusion into a free pericardial space. A mixed “effusive-constrictive” condition in which there is constriction of the heart by the visceral pericardium in the presence of tense effusion in a free pericardial space has been less often described, and clinical and hemodynamic features that distinguish these cases from other forms of pericardial disease have not been clearly delineated. For this reason, 13 patients with subacute effusive-constrictive pericarditis seen in the past decade are described in this report.

Methods
Twenty-three patients had surgery for constrictive pericarditis at the Stanford University Hospital during 1960–69, and one additional patient had surgery elsewhere after clinical study and cardiac catheterization at Stanford. On the basis of preoperative studies and the findings at the time of operation, this group of 24 proven constrictive cases could be classified according to the pathophysiologic nature of the constrictive disease into four groups (table 1). The effusive-constrictive condition was found at the time of operation in nine patients. In six of them a combined pericardiocentesis and cardiac catheterization had been performed preoperatively, and in each instance it was demonstrated that the right atrial pressure remained elevated after the intrapericardial pressure was reduced to normal levels by removal of pericardial fluid (fig. 1). In one other patient preoperative pericardiocentesis failed to relieve signs and symptoms, but pressure measurements were not made; the two remaining patients did not have pericardiocentesis. Effusive-constrictive pericarditis leading to constrictive disease was demonstrated in four patients. Each had pericardiocentesis with cardiac catheterization, showing persistent elevation of right atrial pressure despite reduction of intrapericardial pressure to normal, but at the time of operation, at 11 days, 35 days, and (in two patients) 15 months later, effusion was no longer present, being replaced by a complete fibrotic fusion.

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of visceral and parietal pericardial layers in the manner of classical chronic constrictive pericarditis. Thus, 13 patients with the effusive-constrictive condition were studied clinically, two with cardiac catheterization and 11 with simultaneous cardiac catheterization and pericardiocentesis.

Noneffusive chronic constrictive pericarditis was found at operation in three additional patients who had had previous operations, at which time pericardial effusion had been present and various amounts of the parietal pericardium had been removed for the relief of cardiac tamponade. However, pressure measurements had not been made, and none had been considered to have visceral constrictive pericarditis at that time. These patients are termed constrictive pericarditis with previous effusion, since it could not be determined whether the effusive-constrictive condition had been truly present. Finally, eight patients had classic noneffusive chronic constrictive pericarditis with no history of past effusion. Thus, 15 patients had noneffusive constrictive at the time of operation, and 12 of these had cardiac catheterization performed in this stage of the disease just before surgery.

For the purpose of comparison of certain hemodynamic features in pericardial effusion and pericardial constriction, an additional group of seven patients with pericardial tamponade without constriction was assembled. These were patients who had combined pericardiocentesis and cardiac catheterization, at which time it was shown that elevated right atrial pressure did fall to normal when the intrapericardial pressure was lowered to normal by removal of pericardial fluid. Four of these patients subsequently had pericardiectomy for the relief of recurrent tamponade, and the nonconstrictive character of the visceral pericardium was confirmed by direct observation.

The intravascular and pericardial pressure measurements in this study were all recorded in the cardiac catheterization laboratory, by standard strain gauge electromanometric techniques and phasic photographic recording. A reference level halfway between the anterior and posterior chest surfaces at the fourth intercostal space was used. The upper limit for normal right atrial or central venous pressure was considered to be 6 mm Hg, and pericardial pressure was considered to be normal if it reached zero or negative values in inspiration and did not exceed 7 mm Hg in expiration. Pericardiocentesis was performed by the subxiphoid route with a 16 or 18 gauge needle.

Table 1
The Etiology and Duration of Disease in 24 Patients Operated upon for Constrictive Pericarditis, Classified According to the Pathologic Type of Constrictive Disease

<table>
<thead>
<tr>
<th>Pathologic type of constriction</th>
<th>Etiology</th>
<th>Effusion-constrictive</th>
<th>Effusion-constrictive progressing to constriction</th>
<th>Constriction with previous effusion</th>
<th>Constriction without previous effusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etiology</td>
<td>Idiopathic</td>
<td>7</td>
<td>2</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Radiation</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Tuberculosis</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Duration of pericardial disease</td>
<td>Less than 1 yr</td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>1-2 yr</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>More than 2 yr</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>5</td>
</tr>
</tbody>
</table>

Results
Clinical and Hemodynamic Observations
The effusive-constrictive group was composed of generally young patients (Table 2)

Figure 1
Right atrial and intrapericardial pressures before and after removal of pericardial fluid in a patient with idiopathic subacute effusive-constrictive pericarditis. Right atrial pressure remains elevated after reduction of intrapericardial pressure to normal.
whose pericardial disease was either idiopathic or secondary to radiotherapy in excess of 4000 rads for malignant neoplasm in the mediastinum. None had a clinical history of tuberculosis or a positive second strength tuberculin skin test, and each had negative examinations of pericardial fluid and resected pericardial tissue for tuberculosis and other bacterial and fungal agents. Their symptoms of pericardial disease ranged up to 16 months in duration, and 10 of 13 had had symptoms or signs of active pericarditis during this period, such as fever, pleuritic substernal chest pain, or pericardial friction rub. Most of the idiopathic cases were thought clinically to represent viral infection, but this was largely speculative since none occurred during an epidemic of viral disease or had virological studies adequate for establishment of a specific etiological diagnosis.

The clinical features of the effusive-constrictive cases were generally similar to those of classic chronic constrictive pericarditis, with dyspnea, abdominal distention, and swelling of the ankles the leading symptoms, and jugular venous distention and pulsation, hepatomegaly, ascites, and ankle edema the leading physical signs. There were several features, however, that tended to distinguish effusive from noneffusive constrictive pericarditis and to make the picture of effusive-constrictive pericarditis more like that of chronic tamponade without constrictive (table 2).

Paradoxical pulse was more prominent in the effusive-constrictive cases than in those with noneffusive constrictive, and was similar in degree to that seen in cardiac tamponade (table 2). In general the paradoxical pulse was readily palpable at the radial pulse during quiet breathing in both groups of patients with pericardial effusion, and not palpable in those with classic noneffusive chronic constrictive pericarditis.

A third heart sound ("pericardial knock") was found by clinical auscultation more often in noneffusive chronic constrictive than in either group with effusion (table 2). Phonocardiograms were done in three of the effusive-constrictive cases, two with and one without a clinically audible third sound, and showed a third sound in each instance. The third sounds in these patients occurred from 0.08 to 0.12 sec after the aortic second sound and could not be distinguished clinically or in the graphic recording from the "knock" of chronic constrictive pericarditis.

The electrocardiograms in all three groups showed a similar degree of low voltage and flat or inverted T waves characteristic of pericardial disease. None of the effusive-constrictive cases showed diffuse S-T segment elevation suggestive of acute pericarditis at

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Table 2

<table>
<thead>
<tr>
<th></th>
<th>Effusion-constrictive</th>
<th>Constriction, noneffusive</th>
<th>Effusion, nonconstrictive</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients*</td>
<td>13</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>mean 29</td>
<td>38</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>range (13-47)</td>
<td>(16-58)</td>
<td>(23-64)</td>
</tr>
</tbody>
</table>
| Paradoxical pulse (mm Hg) (inspiratory fall in arterial peak systolic pressure) | mean 21 | 10 | 19
|                         | range (9-37)          | (6-27)                    | (11-26)                   |
| Third heart sound, incidence by auscultation† | 3/13 | 10/15 | 2/7 |
| Kussmaul's sign, incidence in direct right atrial pressure records† | 1/10 | 4/12 | 1/5 |
| Abnormal P waves or atrial fibrillation† | 3/13 | 7/15 | 0/7 |

*Four patients who progressed from effusive-constriction to noneffusive constriction are here included in both groups.
†Proportion of patients tested.
Cardiac ratio in frontal chest X-ray view in effusive-constrictive pericarditis and non-effusive constrictive pericarditis, prior to pericardiocentesis or surgery.

the time of hemodynamic study or operation, although this had been present in previous electrocardiograms in some patients. Abnormally peaked, broad, or notched P waves were less frequent in the effusive-constrictive patients than in those with noneffusive chronic constriction, and the only patient with atrial fibrillation was in the latter group (table 2). No patient in this series had electrical alternans.

Chest X-rays showed enlarged cardiomedastinal silhouette in all of the effusive-constrictive cases, often markedly enlarged (fig. 2). Air was introduced into the pericardial space in most of the patients who had pericardiocentesis, and subsequent X-ray views showed normal or reduced size of the heart, itself, and notable thickening of the parietal pericardium (fig. 3). Only two patients in the chronic constrictive group had distinct cardiac enlargement, both with recurrent constriction after previous pericardiectomy, in one instance 20 years previously. Pericardial calcification was not present in any of the effusive-constrictive cases.

Right atrial pressure was higher on the average in the effusive-constrictive cases than in those of chronic constriction, and after removal of the pericardial fluid it tended to fall to approximately the level seen in the latter group (table 3). This corresponded to a generally more severe clinical syndrome in the effusive group, and suggested that the degree of constriction was similar in the two conditions, with an added element of cardiac tamponade in those with associated effusion.

Figure 2

Cardiothoracic ratio in frontal chest X-ray view in effusive-constrictive pericarditis and non-effusive constrictive pericarditis, prior to pericardiocentesis or surgery.

Figure 3

Frontal chest X-ray views in a patient with idiopathic subacute effusive-constrictive pericarditis before and after pericardiocentesis with air introduced into the pericardial space. Heart size is normal, and the pericardium is thickened.
Table 3

Comparison of Certain Hemodynamic Features in Three Types of Pericardial Compressive Disease

<table>
<thead>
<tr>
<th></th>
<th>Effusion-Constriction</th>
<th>Constriction, Noneffusive</th>
<th>Effusion, Noneffusive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrial pressure (mm Hg)</td>
<td>Pretap (mean)</td>
<td>Posttap (range)</td>
<td>Pretap (mean)</td>
</tr>
<tr>
<td></td>
<td>18 (11–25)</td>
<td>13 (10–21)</td>
<td>13 (8–19)</td>
</tr>
<tr>
<td>Pericardial pressure (mm Hg)</td>
<td>mean</td>
<td>14</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>range</td>
<td>(6–22)</td>
<td>(–3–+7)</td>
</tr>
<tr>
<td>Right atrial pulse contour*</td>
<td>x only</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>x &gt; y</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>x = y</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>x &lt; y</td>
<td>0</td>
<td>3</td>
</tr>
</tbody>
</table>

*Classified according to the relative depth of the systolic (x) and diastolic (y) dips.

Right atrial pulse contours, classified according to the relative prominence of the systolic x and diastolic y descents, also differed in the three types of compressive pericardial disease. Nonconstrictive tamponade cases showed a predominant systolic descent (pure x or x > y), and noneffusive constrictive cases showed a predominant diastolic descent (x = y or x < y). The effusive-constrictive cases were intermediate, showing predominant x or equal x and y, but never a predominant y descent as was seen in the majority of noneffusive chronic constrictive cases. After removal of the pericardial fluid the patients with nonconstrictive tamponade regained a normal right atrial pulse contour, and the effusive-constrictive patients reverted to a contour with a more prominent y descent. The characteristic x > y pulse of effusion-constriction and the x < y pulse of pure constriction are illustrated in figure 4, in the same patient before and after removal of pericardial fluid.

Kussmaul's sign, defined as a rise in right atrial mean pressure with inspiration, was less frequent in the two effusive groups than in pure constriction (table 2). Kussmaul's sign was often thought to be present on clinical examination of the jugular venous pulse in the effusive-constrictive cases, but in several instances the direct recording showed that this appearance was caused by an inspiratory increase in the amplitude of pulsations without a rise in mean pressure. Pericardial pressure rose with inspiration in the one patient with effusive-constrictive disease who demonstrated Kussmaul's sign. All other patients showed a fall in pericardial pressure with inspiration or, in two instances, no respiratory variation. Pericardial pressure fell with inspiration in the one patient with cardiac tamponade without constriction who had Kussmaul's sign.

Figure 4

Right atrial and right ventricular pressure pulses, before and after removal of pericardial fluid, in a patient with subacute effusive-constrictive pericarditis that followed radiotherapy. The right atrial pulse shows a predominant systolic descent (X > Y) initially and a predominant diastolic descent (X < Y) after removal of fluid. The diastolic dip-plateau pattern in the right ventricular pulse is prominent only after removal of fluid, in association with the X < Y right atrial pulse.
The pericardial fluid in the effusive-constrictive patients was often grossly bloody and always showed high protein concentration (table 4). The characteristics of the fluid were not different in the idiopathic and radiation groups.

Medical therapy with various combinations of digitalis, diuretics, anti-inflammatory drugs, and pericardiocentesis was carried out in all patients before surgery was recommended. Despite temporary improvement the underlying constrictive disease remained very evident. Corticosteroid therapy was used in several instances, and was associated with prompt relief of fever and chest pain, but had no effect on constriction. One patient deteriorated rapidly during corticosteroid therapy and required emergency pericardiectomy.

The operative findings in the effusive-constrictive cases were generally uniform in that each patient showed extensive thickening of both the visceral and parietal pericardial layers, with a free pericardial space containing 100–500 ml of fluid under some tension. The degree of thickening of the visceral pericardium and the degree of its adherence to the underlying myocardium was markedly variable from patient to patient, and occurred in different areas of the heart in individual patients. The visceral constriction was often more severe over the right side of the heart than elsewhere and usually extended over the venae cavae; none of the patients in this series had any localized constriction of the venae cavae, however. In the four patients who had surgery in a later noneffusive constrictive phase there was a distinct plane of dissection.

Table 4

<table>
<thead>
<tr>
<th>Case</th>
<th>Volume (ml)</th>
<th>Specific gravity</th>
<th>Protein (g/100 ml)</th>
<th>Red blood cells (ml/100 ml)</th>
<th>White blood cells (no./ml)</th>
<th>Polymorphs (%)</th>
<th>Mononuclear (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50</td>
<td>1.027</td>
<td>4.5</td>
<td>1</td>
<td>350</td>
<td>42</td>
<td>58</td>
</tr>
<tr>
<td>2</td>
<td>200</td>
<td>1.032</td>
<td>6.0</td>
<td>(bloody)</td>
<td>1000</td>
<td>100</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>235</td>
<td>3.7</td>
<td>13</td>
<td>2</td>
<td>1400</td>
<td>24</td>
<td>76</td>
</tr>
<tr>
<td>4</td>
<td>325</td>
<td>3.6</td>
<td>(nonbloody)</td>
<td>2</td>
<td>36700</td>
<td>95</td>
<td>5</td>
</tr>
<tr>
<td>5</td>
<td>300</td>
<td>3.7</td>
<td>16</td>
<td>3</td>
<td>1400</td>
<td>27</td>
<td>73</td>
</tr>
<tr>
<td>6</td>
<td>330</td>
<td>4.0</td>
<td>&lt;1</td>
<td>2</td>
<td>360</td>
<td>28</td>
<td>72</td>
</tr>
<tr>
<td>7</td>
<td>325</td>
<td>4.0</td>
<td>(bloody)</td>
<td>2</td>
<td>3600</td>
<td>95</td>
<td>5</td>
</tr>
<tr>
<td>8</td>
<td>205</td>
<td>3.7</td>
<td>16</td>
<td>3</td>
<td>1900</td>
<td>24</td>
<td>76</td>
</tr>
<tr>
<td>9</td>
<td>375</td>
<td>3.6</td>
<td>(nonbloody)</td>
<td>2</td>
<td>16900</td>
<td>0</td>
<td>100</td>
</tr>
</tbody>
</table>

Cases 1–4 followed radiotherapy; cases 5–13 were idiopathic. In cases 4 and 11 the fluid was obtained at operation only; in all others by pericardiocentesis prior to surgery.

Table 5

<table>
<thead>
<tr>
<th>Classification of operative result</th>
<th>Effusion-constriction*</th>
<th>Constriction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Operative death</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Late death</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Excellent or good</td>
<td>10†</td>
<td>8</td>
</tr>
<tr>
<td>Fair or poor</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

Surviving patients, duration of follow-up completed as of Jan. 1970 (yr)

| <1 | 1  | 1 |
| 1  | 1  |   |
| 2  | 3  | 1 |
| 3  | 4  | 2 |
| 4  | 1  |   |
| 5  | 1  |   |
| 6  | 1  |   |
| 7  | 1  |   |

*Four patients operated upon in the noneffusive constrictive stage after previous effusion-constriction are included here in the effusion-constriction group only.

†Includes one late death unrelated to heart disease.
between the parietal and visceral layers, and both layers appeared to contribute to the constriction.

The operative procedure consisted of removal of all of the parietal pericardium except the portions containing the phrenic nerves, and the removal of various amounts of the visceral pericardium, depending on the extent and severity of visceral constriction. The visceral pericardiectomy was often difficult and time-consuming, requiring sharp dissection of many small fragments. Usually the anterior and lateral surfaces of the heart could be decorticated well, but not the posterior and diaphragmatic areas.

Histopathologic examination of the resected portions of pericardium showed fibrotic thickening, fibrin deposition with organization, and variable nonspecific inflammation. Proliferation of pericardial mesothelial cells was frequent, and one patient showed marked foreign body reaction to cholesterol crystals. The findings were not distinctively different in the idiopathic and radiation cases. There were two deaths in the early postoperative period, both due to acute cardiac dilatation and congestive heart failure in young men with idiopathic subacute effusive-constrictive pericarditis. Postmortem examination showed no specific myocardial disease in either, but their hearts were small, with myocardial atrophy and focal hyaline and fatty degeneration. A third patient, who had had 8000 rads to the mediastinum for Hodgkin's disease, had a poor clinical result with persistent congestive heart failure and died 6 months after pericardiectomy. Postmortem examination showed extensive myocardial and endocardial fibrosis similar to that seen in other patients with severe radiation heart disease. The three patients who died of heart disease were all in the group of nine who had surgery while in the effusive-constrictive phase.

Late follow-up in the remaining 10 patients has shown excellent relief of pericardial constriction, manifested by complete relief of symptoms and by the finding of normal venous pressure and jugular venous pulse on examination. None has demonstrated recurrent constriction or clinically significant residual myocardial disease during the follow-up (table 5). One patient died 5 years after pericardiectomy from carcinoma of the colon; postmortem examination showed no evidence of myocardial disease or cardiac constriction.

The operative results in the 11 patients with chronic constriction not known to have been preceded by an effusive-constrictive phase are also given in table 5. The only death occurred in a patient with severe radiation heart disease who failed to improve and who died one month after pericardiectomy; postmortem examination showed radiation myocardial fibrosis and, elsewhere in the body, widespread recurrent reticulum cell sarcoma. Another patient with less severe radiation heart disease had moderate improvement from pericardiectomy but continues to show signs of restrictive heart disease, presumed caused by myocardial fibrosis. Another patient, with idiopathic pericarditis and two previous pericardiectomies had moderate improvement followed by deterioration, and 1½ years after surgery had a fourth pericardiectomy. At this time a histopathologic diagnosis of pericardial mesothelioma was established, and it was considered likely that this accounted for her previous pericardial disease as well. The remaining eight patients have maintained excellent clinical results during the follow-up period to date (table 5).

Discussion

The type of effusive-constrictive pericarditis discussed here has been seen in the past chiefly in association with active tuberculous pericarditis.1,2 Many of the clinical features were described by Wood1 as characteristic of the subacute stage of tuberculous pericarditis. The earliest reports of pericardiectomy for tuberculous pericarditis warn of the necessity of removal of a constricting visceral layer in the presence of effusion.3,4 In recent years tuberculosis has accounted for a much smaller proportion of both active and chronic constrictive pericarditis than it did in the past, particularly in the American white population groups.5,6 Not one patient with active tuber-
culous pericarditis was seen by the cardiology
or cardiac surgical services in the Stanford
University Hospital during the 1960's despite
an active program encompassing over 3000
cardiac operations and a like number of
cardiac catheterization procedures during this
period. On the other hand, nontuberculous
constrictive pericarditis with preceding acute
pericarditis and with pericardial effusion has
long been recognized, and there is an
increasing number of reports of such cases,
many of which pass through the effusive-
constrictive phase. The number of cases in
the present series that followed radiother-
apy reflects a large program at Stanford aimed
at potential cure by radical radiotherapy of
Hodgkin's disease and related neoplasms.
Some diseases not represented in the present
series, such as rheumatoid arthritis, post-traumatic pericarditis, etc., are also increasingly reported in association
with subacute constrictive pericarditis.
Neoplastic invasion of the pericardium may
also lead to the effusive-constrictive syn-
drome.

The development of carbon dioxide injec-
tion, radiopaque contrast angiocardio-
graphy, radioactive isotope scanning and ultrasound echocardiography for the detection
of pericardial effusion has greatly facil-
itated the evaluation of patients with pericar-
dial disease. Pericardiocentesis, not long ago
discouraged for use except in emergencies, is
now used often and effectively because the
presence of fluid can be detected with assurance ahead of time, and the position of
the needle tip can be monitored by electrocar-
diography. These developments of the 1960's
have allowed the effusive-constrictive pericar-
ditis syndrome to be studied physiologically
and identified with precision prior to surgery.

There is a clear advantage in performance of
pericardiocentesis in a cardiovascular labora-
tory where electromanometric measurements
of the pressures in the right atrium and
pericardial space may be made. In the absence of these facilities much the same
information could be obtained by properly
performed saline manometric measurements,
much as is usually done during lumbar spinal
puncture. If the right atrial or central venous
pressure remains elevated after reduction of
the intrapericardial pressure to normal there
must be a disorder in addition to cardiac
tamponade, and often this will be visceral
constrictive pericarditis. There are many
features that would be helpful in identification
of other causes, such as right ventricular
myocardial failure, tricuspid valve disease, vena caval obstruction, etc.

The reason for the different atrial pulse
contours in tamponade and constriction has
not been fully elucidated. The right ven-
tricular pulse in cardiac tamponade may not show the dip-plateau pattern which is characteristic
of constrictive pericarditis, and this corre-
sponds to the diminished or absent y descent
in the right atrial pulse in tamponade.
Flowmeter studies indicate that in constrictive
pericarditis, with a right ventricular dip-
plateau and an x = y right atrial pulse, there
are peaks of forward blood flow in the venae
cavae with both the x and y dips, whereas in
tamponade there is a peak of forward flow
only in systole with the prominent x descent.
This has been discussed in terms of elastic
and inelastic forms of cardiac compression.

Tamponade is an elastic form of compression,
exerting a cushion-like action throughout the
cycle, so that the least compression is in
systole when the heart size is smallest. Chronic
constriction is inelastic and the heart is
compressed abruptly when it exceeds a critical
size during the diastolic filling; ventricular
filling evidently occurs almost entirely in the
protodiastolic phase, and filling is at least
relatively more rapid than ejection. Rapid
systolic ejection associated with well-function-
ing myocardium has also been suggested as
the basis for the prominent x descent in the
right atrial pulse of some patients with
constrictive pericarditis.

The more prominent paradoxical pulse in
cardiac tamponade than in chronic constric-
tion has also been well described but not fully
elucidated. Paradoxical pulse is now recog-
nized as occurring in association with the
normal phasic inspiratory augmentation of
venous return to the heart. Flowmeter studies indicate that in tamponade this phasic variation in venous return with respiration is present just as it is normally, but in chronic constriction it is not present. Perhaps the difference lies in the principle that in tamponade the compressing force itself is decreased by the inspiratory fall in intrapleural pressure surrounding the heart, whereas in chronic constriction the inelastic constraining force is independent of surrounding pressure variations.

Mounce pointed out that the third heart sound in constrictive pericarditis is likely to be absent when the ascent of pressure following the y dip in the right ventricular pulse is not so rapid as usual. This might explain the absent third sound in the patient illustrated in figure 4, but a systematic review of the right ventricular pressure pulses did not show a less distinctive dip-plateau contour in the effusive-constrictive cases than in those of noneffusive constriction. Muffling of heart sounds by intervening fluid was suggested by Wood as an explanation for absent third sounds with cardiac tamponade; this would be supported by the one patient in the present series who had a third sound in the phonocardiogram that was clinically inaudible.

The natural history of effusive-constrictive pericarditis appears to be the progression into noneffusive chronic constrictive pericarditis, usually in less than a year. This was not altered by corticosteroid therapy. All of the cases in the present series, which were identified by studies during combined cardiac catheterization and pericardiocentesis, required surgery either sooner or later.

Whether surgery is best advised during the active effusive-constrictive phase or during the later chronic constrictive phase is not easily determined. Surgery has often been considered to be more readily accomplished in the effusive stage, but this perhaps applies more to the parietal pericardiectomy than to the more difficult visceral stripping procedure. In this series the policy was adopted of advising surgery whenever the signs and symptoms indicated sufficiently severe constriction to warrant it. The surgical results were generally satisfactory in both groups. The two patients who died after surgery in the effusive-constrictive phase were unusually severe cases and would not have been expected to survive until a later noneffusive stage. If adequate pressure records are obtained during pericardiocentesis the decision for or against early pericardiectomy is usually made without difficulty.

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