Constrictive Pericarditis with Particular Reference to Etiology

By Ross Robertson, M.D., and Craig R. Arnold, M.D.

In 1954, seven cases of constrictive pericarditis were reported from the Vancouver General Hospital and Shaughnessy Hospital.1 During the succeeding years, 14 additional cases of constrictive pericarditis have been treated surgically (table 1). Constrictive pericarditis following trauma or hemopericardium of sufficient severity to cause disability, has not been seen by the authors, although a number of cases of hemopericardium have been treated by aspiration. One patient operated on for mitral stenosis was found to have an adherent constricting pericardium, which was removed at the time of commissurotomy and may possibly have been a result of rheumatic fever. Bacteria such as staphylococci have not been found responsible for constrictive pericarditis in this series.

The belief was firmly held that constrictive pericarditis was usually due to the tubercle bacillus until this year, when five cases of constrictive pericarditis were seen within a few months. Four of these patients had negative tuberculin tests. The fifth was slightly positive to 1/10,000 old tuberculin, but gave a history similar to the other four; had no other evidence of tuberculous infection; and showed no response to antituberculous therapy. Four of these patients were under 40 years of age. One was aged 58. Two of them were hospitalized and diagnosed as nonspecific pericarditis several months before operation. The remaining three had a similar illness for which they did not consult a physician. All of them had their original illness during a period in 1960 when there was an epidemic of 48 cases of nonspecific pericarditis in the Vancouver General Hospital (table 2).

Of the 48 reported cases of nonspecific pericarditis occurring at the Vancouver General Hospital in 1960, five had positive cultures of Coxsackie B-5 from the feces. Unfortunately, the two patients in this series who subsequently developed constriction did not have cultures taken during the acute phase of pericarditis. Two of the five cases of nonspecific constrictive pericarditis operated on in 1961 had specimens of excised pericardium, pericardial fluid, and feces taken at the time of operation and cultured for virus. All were negative as expected, considering the months that had elapsed since their acute attack.

Similar epidemics of nonspecific pericarditis have been reported from other centers.2-5 Some of them have been attributed to the Coxsackie virus.6-8 In 1954, Krook reported the later development of constrictive pericarditis in two patients among a group of 24 cases of acute nonspecific pericarditis.9

Diagnosis

The diagnosis of tuberculous constrictive pericarditis has been described by the author in a previous report.1 It is rarely urgent. The long illness, a strongly positive tuberculin test, and, in the early stages, positive cultures for tubercle bacilli from pleural or pericardial effusions usually make diagnosis easy. Calcification of the pericardium frequently occurs, as shown in figure 1, and facilitates the diagnosis. Even when not calcified, the pericardium is greatly thickened and may be demonstrated by angiocardiograms indicating excessive thickness between the chamber of the left ventricle and the left border of the cardiac silhouette.

The nonspecific type of constrictive peri-
carditis is more difficult to diagnose. Such patients may be treated for long periods for cirrhosis of the liver, myocardial disease, or coronary insufficiency. The onset of constriction may be more acute than in tuberculous pericarditis; so acute that it may become a surgical emergency as in one of our cases (case 1). Fluoroscopy will often reveal a small, quiet heart (fig. 2), but, since the pericardium is not greatly thickened, angiograms are not of much assistance. A thoracotomy may be required to prove the diagnosis.

The differentiation between tuberculous and nonspecific pericarditis is shown in table 3. In our tuberculous cases, the average duration of the illness before operation was 3 years, whereas in the nonspecific cases it was only 10 months. The tuberculous cases were scattered sparsely over a period of many years, whereas the nonspecific cases tended to appear in a group following an epidemic of pericarditis. The pericardium in patients with tuberculous pericarditis was greatly thickened, averaging 9 mm. in thickness, and contained areas of calcification or necrosis with loculated collections of fluid. The nonspecific cases showed a smooth, uniform thickening of the pericardium by proliferating fibroblasts. There was much less tendency for the process to invade the myocardium, as it so frequently does in tuberculous pericarditis, and conse-

**Table 1**

<table>
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<tr>
<th>Year</th>
<th>1961</th>
<th>60</th>
<th>59</th>
<th>58</th>
<th>57</th>
<th>56</th>
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<th>54</th>
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<th>47</th>
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<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Nonspecific</td>
<td>5</td>
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<td>1</td>
<td>3</td>
<td>1</td>
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**Table 2**

<table>
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<th>50</th>
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<th>47</th>
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<td></td>
<td></td>
</tr>
<tr>
<td>Nonspecific</td>
<td>26</td>
<td>48</td>
<td>24</td>
<td>14</td>
<td>17</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>
CONSTRUCTIVE PERICARDITIS

Table 3

Tuberculous Pericarditis Versus Nonspecific (Virus?) Pericarditis

<table>
<thead>
<tr>
<th></th>
<th>Tuberculous</th>
<th>Virus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration (average)</td>
<td>3 years</td>
<td>10 months</td>
</tr>
<tr>
<td>Epidemic</td>
<td>0</td>
<td>Onset during epidemic of pericarditis</td>
</tr>
<tr>
<td>Tuberculin</td>
<td>Positive</td>
<td>Usually negative</td>
</tr>
<tr>
<td>TB culture</td>
<td>Positive early; negative late</td>
<td>Negative</td>
</tr>
<tr>
<td>Pathology of excised pericardium</td>
<td>Calcium and necrosis</td>
<td>Fibrosis</td>
</tr>
<tr>
<td></td>
<td>9 mm. thick</td>
<td>2-3 mm. thick</td>
</tr>
</tbody>
</table>

frequently removal was much easier and the myocardial action much more vigorous following operation.

The typical history of tuberculous pericarditis is well known and has been shown by cases in a previous article. Nonspecific (virus?) pericarditis is typified by the following cases. The contrast between these and those who have tuberculous constrictive pericarditis is remarkable.

Case Reports

Case 1

This patient, E.M., was a 34-year-old man who gave a history of having developed a dry cough in April 1960. His wife had the same complaint. This symptom lasted 4 to 5 weeks and gradually cleared. He was well during the summer and until Christmas 1960, when he had a recurrence of paroxysmal dry cough lasting 3 weeks; it never completely cleared. This was associated with a constricting discomfort along both costal margins. He noticed gradually increasing fatigue, some shortness of breath, and in March 1961, the gradual appearance of edema, epigastric discomfort, and some diarrhea.

He was admitted to hospital on March 25. His face was flushed. The veins in the neck and arms were greatly engorged. There was moderate swelling of the ankles and abdomen. The left chest contained a moderate-sized pleural effusion. The liver was slightly enlarged. The apex beat was not palpable and the heart sounds were tachycardia in quality. There were no cardiac murmurs. X-rays showed a moderately enlarged heart with a cardiothoracic ratio of 17 to 32 cm. On fluoroscopy, no pulsation could be seen on the left heart border. Aspiration of the pericardial sac was attempted without success. The tuberculin skin test was negative.

On the morning following admission, while reaching over to a chair for the urinal, he lost consciousness, stopped breathing, and became deeply cyanosed. Artificial respiration was administered mouth-to-mouth by a nurse, reviving him after a few minutes.

At operation the next day, the pericardial sac contained about 300 ml of blood-stained fluid. The parietal pericardium was about 6 mm. thick and was adherent to the visceral pericardium in only a few places, so that it was removed and the visceral pericardium was left. The pericardectomy was not technically difficult. Pathologic sections of the tissue removed showed fibrous connective tissue of varying densities in which were actively proliferating fibroblasts and numerous chronic inflammatory cells. No particular or specific histopathologic features were noted, and the pathologic diagnosis was chronic nonspecific pericarditis. Following operation the signs of cardiac failure rapidly disappeared. At the present time he is working full time with no disability.

Case 2

S.G., a 31-year-old man, was in good health until October 1960, when he developed “influenza” with cough, malaise, fatigue, and epigastric discomfort and distention. He remained in bed for 3 days, then returned to work but developed increasing dyspnea and edema of the ankles. On November 16, 3 weeks after the onset of his illness, he was admitted to the Vancouver General Hospital where he was found to have a friction rub over the precordium. Chest x-ray showed an enlarged heart with a cardiothoracic ratio of 18 to 34 cm. The cardiac silhouette suggested fluid in the pericardial sac. The tuberculin test was negative. Blood pressure was 110/90. On January 20, 1961, he was discharged from the hospital and rested at home. He felt well until April 2, when the edema of the ankles recurred, along with epigastric fullness and dyspnea on exertion. He had gained seven pounds in weight.
Table 4  

Results of Pericardiectomy (21 Cases)  

<table>
<thead>
<tr>
<th>Operative deaths – 4</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cause: Silicosis with right heart failure</td>
<td>1</td>
</tr>
<tr>
<td>Fibrosing pleurisy and pneumonitis right lung</td>
<td>1</td>
</tr>
<tr>
<td>TB pericarditis, active</td>
<td>1</td>
</tr>
<tr>
<td>Myocardial or liver failure</td>
<td>1</td>
</tr>
<tr>
<td>Survivors – 17</td>
<td></td>
</tr>
<tr>
<td>Working full time</td>
<td>13</td>
</tr>
<tr>
<td>Working part time</td>
<td>1</td>
</tr>
<tr>
<td>Not traced since discharge</td>
<td>2</td>
</tr>
<tr>
<td>Died 6 months after surgery of myocardial infarction</td>
<td>1</td>
</tr>
</tbody>
</table>

in a few days. He was readmitted to the hospital on April 4. The liver was palpable three fingerbreadths below the costal margin, the lung bases were clear, and the neck veins were distended. The blood pressure was 120/100. There was no visible pulsation of the heart on fluoroscopy. A diagnosis of constrictive pericarditis was made. At operation on April 17, 1961, the left chest was found to contain 1,200 ml. of straw-colored fluid, which had not been present 5 days previously. The pericardial sac was firm and rigid and showed no visible pulsation. The parietal pericardium was about 3 mm. thick. It was adherent to the visceral pericardium in a few small areas over the left ventricle posteriorly. The parietal pericardium was excised over the entire surface of both ventricles. A few small areas of thickened visceral pericardium were also removed from the left ventricle. Elsewhere the visceral pericardium was thin and did not restrict cardiac activity. Following excision of the pericardium, cardiac pulsations were good. Following operation signs of cardiac failure rapidly disappeared. Eight months after operation, the patient was working full time with no disability. He could climb three flights of stairs without dyspnea. The blood pressure was 170/90. On fluoroscopy his heart was normal in size and was pulsating well.

Surgical Technic

Further experience has confirmed our belief that complete freeing of both ventricles is essential for a good result. In order to do this effectively, we have found that a left anterolateral thoracotomy through the fifth interspace is necessary for adequate exposure. Occasionally the sternum is transsected in the line of the incision if better exposure is required over the right ventricle. Recalling the experiences of Blakemore and Lawrence and their associates, we have been constantly on the alert for a constriction of the vena cava or pulmonary veins, and have frequently removed thickened pericardium from these areas, but we have not yet seen a case where there was evidence of venous stenosis, either by angiocardiogram or at operation, as experienced by Stojanovic and Cooley and co-workers.

We believe that early operation is indicated in nonspecific constrictive pericarditis and may be life-saving. In our experience, tuberculous constrictive pericarditis does not respond well to pericardiectomy during the active phase.

Results

Surgical results in 21 cases are shown in Table 4. There were four operative deaths, as follows:

One death was attributed to silicosis and right heart failure. The patient was a 56-year-old man, who had been a hard-rock miner for many years. There was diffuse fibrosis of both lungs and the greatly thickened pericardium contained numerous tubercles.

The second death resulted from respiratory failure. The patient had tuberculous pericarditis and fibrosing pleurisy with atelectasis and fibrosis of the right lung.

The third patient died of myocardial failure. He had active tuberculous pericarditis at the time of operation. The process infiltrated the myocardium to a considerable depth. Pericardiectomy was associated with profuse bleeding.

The fourth death was attributed to liver failure from marked venous congestion and atrophy. Operation had been too long delayed.

Of the survivors, 13 are well, working full time, and complain of no disability. One is able to work only part time because of excessive fatigue. Two patients were discharged from the hospital in good condition, but it has not been possible to trace them. One patient responded well to operation, but died 6 months later of myocardial infarction.

Recurrence of the constriction has not been seen in our cases; this is in agreement with the experience of Paul Wood.

Discussion

We recognize that the viral etiology of nonspecific constrictive pericarditis has not been
proved, in that positive cultures have not been obtained. However, the evidence in favor of virus infection is strong in our five cases operated on in 1961. Tuberculosis and other bacterial infection was excluded. The five cases followed an epidemic of virus pericarditis. The clinical course was typical and differed considerably from tuberculous constrictive pericarditis. The excised pericardium was smooth and moderately thick, unlike the greatly thickened and necrotic or calcified pericardium of tuberculosis. It is expected that when routine virus cultures are taken during an epidemic of nonspecific pericarditis and the patients are followed, some of them will be found to develop constriction and the etiology will be thus proved. In the meantime we would suggest that young adults showing early signs of cardiac failure without a murmur be questioned carefully regarding an attack of "influenza" some months previously, particularly if it was associated with precordial distress and the possibility of constrictive pericarditis carefully considered as has also been suggested by Roberts. 9

Conclusion

Tuberculous constrictive pericarditis is becoming less common in British Columbia, along with other forms of tuberculosis.

Virus infection appears to be responsible for a number of cases of nonspecific constrictive pericarditis seen recently. This is a more acute form of pericardial constriction, occurring a few months after the original attack of pericarditis, and likely to present as a surgical emergency. Early operation is essential.

Tuberculous pericarditis does not respond well to pericardiectomy during the acute phase. If diagnosed in the acute phase, chemotherapy should be given for at least several months prior to operation.

Both ventricles must be freed completely of the constricting pericardium for a good functional result. A left thoracotomy gives the best exposure for this purpose.

References

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ROSS ROBERTSON and CRAIG R. ARNOLD

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