CONSTRUCTIVE PERICARDITIS

BY

T. M. D. GIMLETTE

From St. Thomas's Hospital

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A study has been made of 62 patients with constrictive pericarditis. There was considerable variation from the classical description of the disease. It is hoped that the findings may be of use, because both diagnosis and treatment can sometimes cause perplexity.

The aetiology and mode of onset of constrictive pericarditis will be considered first, since these have important effects on the final clinical picture, treatment, and outcome.

Twenty-eight patients developed constriction within a year of an attack of pericarditis ("acute constriction" in Table I and elsewhere). Thirty-four patients developed constriction insidiously; a few of these had a history suggestive of an attack of pericarditis more than a year before constriction developed ("chronic constriction" in Table I and elsewhere).

TABLE I

CAUSES FOUND FOR CONSTRICTION IN THE PRESENT STUDY
(The number of patients with acute constriction given in brackets)

<i>Tuberculosis:</i>	Bacteriological proof only	..	..	..	..	4	(3)
	Histological proof only	..	..	..	..	11	(10)
	Bacteriological and histological proof	..	..	..	..	2	(2)
<i>Tuberculosis (total)</i>	..	..	..	..	..	17	(15)
<i>Pyogenic infection</i>	..	..	..	..	..	1	(1)
<i>Carcinoma</i>	..	..	..	..	..	3	(2)
<i>Radiotherapy (possibly)</i>	..	..	..	..	..	2	(1)
<i>Unknown</i>	..	..	..	..	..	39	(9)
						62	(28)

Tubercle bacilli were found in the pericardial fluid from six patients. Two of these six also provided histological proof of tuberculosis at operation later; two others recovered without operation and now have minimal constriction; in a fifth patient tubercle bacilli were found in pus encountered at operation, although symptoms of constriction of insidious onset had been present for five years; the sixth died of pulmonary tuberculosis a year after operation, no trace of the original infection being found in the pericardium at post-mortem examination.

Histological evidence of tuberculosis was found in 13 patients; pericardial tissue from 41 was examined. Twelve of these 13 had acute constriction, and tissue was examined within a year of its onset. On the other hand, tissue from 7 patients with acute constriction, examined more than a year after the onset, showed no evidence of tuberculosis. The histology of the tissue from apparently non-tuberculous cases and that of parts of the tissue from tuberculous cases was similar. Tuberculosis of the pericardium can evidently result in constriction, but in time all traces of the original infection can disappear and may often do so. Similarly, other causes of constriction may ultimately produce non-specific changes that do not reveal their original nature. The present study has not shown any decline either in the incidence of constriction or in the proportion of proven tuberculous cases over the past twelve years.

None of the patients in the present series had a previous history of myocardial infarction or of significant trauma to the pericardium. Only one patient had coincident rheumatic heart disease. A number of patients had acute pericarditis of unknown cause, possibly attacks of acute benign pericarditis, but no positive evidence has been found that this disease results in constriction.

In eleven patients an ætiology other than tuberculosis is suggested.

One patient in the present series rapidly developed severe constrictive pericarditis following suppurative pericarditis; the pericardium was infected with *B. coli* from a subphrenic abscess. This patient's condition was very bad before operation and he died soon afterwards.

Constriction with secondary carcinoma in the pericardium was observed in three patients. One developed a pericardial effusion containing carcinoma cells: a primary growth in the left lung was irradiated with temporary improvement, but a month later signs of constriction appeared. At necropsy the pericardium was found to be thick and closely adherent; nodules of growth were present. Another had extensive metastases from a carcinoma of ovary; the pericardium was thickened and contained a blood-stained effusion. The third presented with signs of cardiac failure; a little fluid containing bronchial carcinoma cells was removed from the pericardium; radioactive gold treatment was followed by temporary improvement. Two patients had heavy prophylactic irradiation of the mediastinum after removal of seminomata. One had acute pericarditis during therapy; constriction developed within a year and was cured by operation. The other developed constriction insidiously and was not improved by operation.

Five patients had active rheumatoid arthritis. Four had arthritis for some years before the cardiac symptoms. The fifth developed arthritis three months after an acute pericarditis which initiated constriction. All these patients had cardiac enlargement. Three of them were operated upon: only the one with acute constriction was cured, and the others were little if at all improved. In one of them severely damaged cardiac muscle was found at operation. Tuberculosis was not demonstrated in any of them. The segregation of this small group may be artificial, but there is a little evidence that they had more myocardial involvement than usual, and it seems possible that myocardial, pericardial, and joint lesions together were a single disease. The resemblance between the synovial and nodule lesions and the pericardial lesions, often present in rheumatoid arthritis, has been pointed out (Bywaters, 1950). These pericardial lesions are usually symptomless. One patient with rheumatoid arthritis and constrictive pericarditis in whom the pericardium showed lesions "compatible with rheumatoid arthritis" has been described (McMurray *et al.*, 1951). In systemic lupus erythematosus, lesions in the pericardium and myocardium as well as the joints are found, but constrictive pericarditis has not been described. A search for L.E. cells in two of the patients with rheumatoid arthritis in the present series was unsuccessful, and none of the other patients showed features of lupus erythematosus. In one patient chronic constriction and nephrosis arose together.

SYMPTOMS AND SIGNS

Findings of particular use in differential diagnosis, or illustrating important features of the disease, will be emphasized. In most respects there was little difference in the final clinical state between patients with acute and those with chronic constriction, but differences in the incidence of cardiac enlargement, pericardial calcification, and atrial fibrillation will be described.

Symptoms and signs suggesting pulmonary venous hypertension were frequent, equally so in patients with acute and chronic constriction. Their presence implies that there was left as well as right ventricular failure. Fifty-six patients (90%) were orthopnoëic, 12 of them also having paroxysmal nocturnal dyspnoëa. Basal crepitations were present in 36 patients, most of whom were also orthopnoëic, and pulmonary congestion was often noted in X-rays. When the pulmonary capillary pressure was measured at cardiac catheterization, levels comparable with moderately severe mitral stenosis were found. In experimental tamponnade, pressures in the left and right atria have been shown to remain identical (Isaacs *et al.*, 1954). The high systemic venous pressure

observed probably gives a good approximate indication of the pulmonary venous pressure in constriction.

A raised jugular venous pressure was observed in every patient, even in the few without symptoms, and it was usually the last abnormal sign to be lost after successful pericardectomy. In 46 patients (75%) the veins filled to the angle of the jaw in the upright position. Venous pulsation was invariably present, usually with an M- or W-shaped pressure pulse. The early diastolic dip in pressure is the most important feature; this was unaffected by atrial fibrillation. A rising venous pressure on inspiration was observed in some patients, and confirmed by pressure measurements made at cardiac catheterization. This was not due to transient tricuspid regurgitation. In some instances the rising venous pressure was associated with increased loudness of the diastolic sound.

A raised systemic venous pressure is essential to the diagnosis of constriction. Neither the height of the pressure, the type of venous pulsation, nor the respiratory changes are diagnostic; these are all found in right ventricular failure from various causes. However, the presence of venous pulsation of the form described does exclude superior vena caval obstruction and tricuspid disease.

Forty-nine patients had œdema, mainly of dependant parts, but a few had œdema of the face.

The liver was enlarged in 52 patients (85%). Cirrhosis was diagnosed histologically in six; in two there was some fibrosis round the centrilobular veins; and in five others the liver showed venous congestion only. Liver function tests were not done as a routine. In 7 patients out of 18 there was a low serum albumen, and in 6 the thymol turbidity was raised. The tests done were otherwise normal, including those in two of the patients with histological evidence of cirrhosis. Only two patients were ever jaundiced; one died, a month after pericardectomy, following hæmorrhage from œsophageal varices; the other died in coma, which may have been hepatic, six months after pericardectomy. In both these patients the operation failed to relieve cardiac failure. Thus, although the liver was usually enlarged from congestion, cirrhosis or hepatic insufficiency were much less often present and seldom diagnosable clinically. There was no evidence that liver disease advanced after constriction had been relieved. The incidence of evident liver damage and of ascites was the same in acute and chronic constriction. Ascites was clinically demonstrable in 27 patients (44%), all of whom had hepatic enlargement and high systemic venous pressure.

Cardiac pain was rare after the acute inflammatory stage; only four patients in the present series had probable anginal pain on effort. This may have been due to restricted activity or possibly to destruction of the sensory nerve supply of the heart (Daley, 1957).

A diastolic sound was heard in 42 patients (67%), including some only mildly affected by the disease; probably it was present in others. It was not particularly associated with pericardial calcification. In a few patients the diastolic sound was accompanied by a thrust felt over the præcordium. It has been suggested that the diastolic sound in constrictive pericarditis occurs earlier than the third sound sometimes present in other types of heart failure, and becomes later after pericardectomy (McKusick and Harvey, 1955). A phonocardiogram was obtained from one patient in whom symptoms persisted after pericardectomy. A sound characteristically early in diastole was recorded, but a second thoracotomy revealed no constriction. It is concluded that the diastolic sound in constriction is not essentially different from protodiastolic gallop.

The systolic blood pressure was of little value in diagnosis; it was usually within normal limits and seldom abnormally low. One patient was hypertensive (B.P. 220/120) before developing constriction of insidious onset; this reduced the blood pressure to 150/100; after operation, which greatly improved him, the pressure returned to the previous level. Possibly in this case left ventricular hypertrophy brought to light constriction hitherto latent. In two patients a pressure of 160/100 with albuminuria led to an initial diagnosis of acute nephritis.

Pulsus paradoxus exceeding 10 mm. Hg in normal breathing was observed in 43 patients (69%), including all those subsequently cured by pericardectomy. Atrial fibrillation made paradox difficult to detect but did not abolish it. Although paradox does occur in other conditions, including

myocarditis, where the properties of the myocardium are changed, marked paradox is a useful contributory sign in the diagnosis of constrictive pericarditis.

The Valsalva manœuvre, observed in some patients, caused the type of circulatory response usual in cardiac failure, and similar changes in the pulmonary circulation were detected at cardiac catheterization.

RADIOLOGY

Cardiac enlargement, assessed radiologically, was present in 22 out of 58 patients. The thickness of the pericardium itself, seldom more than 0.5 to 1.0 cm., accounted for little of the enlargement; in very few patients some fluid was still present. Enlargement often took place after pericardectomy, but many patients had true cardiac enlargement already. This indicates the importance of myocardial disease in constrictive pericarditis. Table II suggests some correlation between cardiac enlargement and poor prognosis

TABLE II
CARDIAC ENLARGEMENT AND OPERATIVE RESULTS

	Died	I.S.Q. or improved	Cured
Cardiac enlargement	4	9	3
No cardiac enlargement ..	6	8	12

Pericardial calcification was detected in 27 patients (42%); it was best seen by fluoroscopy and was usually densest on the diaphragmatic surface of the heart or in the atrioventricular groove. Five out of the seventeen with proven tuberculosis had pericardial calcification. In one patient calcification developed two years after pericardectomy without recurrence of symptoms; in another it was noted three months after the onset of tuberculous pericarditis; in the others the time of onset was uncertain. Calcification was commoner in patients with chronic constriction. Calcification and atrial fibrillation were frequently associated. Patients with calcification generally had a less successful result from pericardectomy (Table III).

TABLE III
PERICARDIAL CALCIFICATION AND OPERATIVE RESULTS

	Died	I.S.Q. or improved	Cured
Calcified pericardium	4	13	3
No calcification seen	6	4	12

ELECTROCARDIOGRAPHY

Of all 62 patients, atrial fibrillation was permanent throughout in 19, transient after operation in two, and permanent after operation in one. Table IV indicates that fibrillation is unfavourable. Also it was often associated with cardiac enlargement and pericardial calcification. A resting heart rate over 90 was unusual. Atrioventricular conduction was normal in all patients with sinus rhythm.

Twenty-one patients in sinus rhythm had bifid P waves in some or all leads, the second peak of the P wave often being much the larger. Bifid P waves persisted after operation and cure in some

TABLE IV
ATRIAL FIBRILLATION

	Operated			Unoperated
	Died	I.S.Q. or improved	Cured	
Atrial fibrillation	3	10	—	6
Normal rhythm initially ..	7	7	15	14

patients. They were not correlated with any other cardiographic abnormality or clinical feature. A similar incidence has been reported by others (Evans and Jackson, 1952).

Low QRS voltage was present in 41 patients, uncorrelated with the severity of the disease or the thickness of the pericardium. S-T segment displacement rarely persisted after acute pericarditis. Low or flat T waves were almost always seen; abnormal T wave inversion was present in more than half.

A normal initial record was not obtained in any patient. The usual changes—low QRS voltage and flat or inverted T waves—are nonspecific; bifid P waves are of more diagnostic value. After operation the cardiogram became normal in a few cured patients; abnormalities persisted in many cured, and in all who were still incapacitated.

Cardiac catheterization was carried out in 10 patients. The pressures at comparable sites in each patient were remarkably similar, and resembled those described by other observers both in patients and in experimental animals with constrictive pericarditis (Hansen *et al.*, 1952; Isaacs *et al.*, 1952). The pressures recorded from within the venæ cavæ and heart gave no indication that there was any obstruction to the flow of blood through these veins or through the atria or tricuspid valve. Nor was any convincing evidence of such a state found subsequently at operation. The distance of the catheter in the right atrium from the right heart border occasionally demonstrated pericardial thickening or effusion.

The form of the pressure pulse found in the right ventricle was characteristic, and has been frequently described. Removal of the constricting pericardium does not cause immediate change in the form of this pressure pulse; this was observed in one patient in the present study and one described by Hansen *et al.* (1952). This suggests that the diastolic dip is caused by changes in the myocardium, and it has in fact been reported in various myocardial conditions, particularly in amyloid disease (Gunnar *et al.*, 1955). A ratio of over 33 per cent between the diastolic and systolic pressures in the right ventricle has been regarded as indicative of constrictive pericarditis (Yu *et al.*, 1953), but in the course of the present study two patients with myocarditis were encountered in whom the ratio was over 50 per cent.

TREATMENT AND RESULTS

Twelve patients believed to have tuberculous pericarditis were given a preliminary course of streptomycin (Fig. 1). In two a symptomless state was reached with minimal constrictive signs. One died of pulmonary embolism before operation. Nine patients operated upon after streptomycin had no better results than other comparable patients. There seems little risk of post-operative disseminated tuberculosis: only one patient died of pulmonary tuberculosis a year after operation, despite streptomycin treatment, and none died with disseminated tuberculosis.

The results of operation in patients with acute constriction are better than in patients with chronic constriction (Fig. 1). Several patients with acute constriction would probably have fared better if pericardectomy had been done sooner. Three operated upon 1–15 years after constriction first developed were not cured. The rest, operated upon within a year of the onset of constriction,

FIG. 1.—62 patients are shown, grouped according to the onset of constriction and outcome. Patients with proven tuberculosis in black, patients treated with streptomycin indicated by "s".

nearly all did well. Patients who had no symptoms, and no signs other than a jugular venous pressure raised up to 5 cm., were regarded as cured. Of the 42 patients operated upon, 15 (36%) were cured, 17 (40%) were unchanged or improved, and 10 (24%) died.

The results of pericardectomy for chronic constriction were much less good: both the post-operative mortality and morbidity were much higher. Apart from chronicity, certain features that suggested myocardial damage in contrast to mechanical constriction were unfavourable. Age was one of the most important (Table V). Cardiac enlargement was unfavourable (Table II), suggesting dilatation from myocardial disease. Rheumatoid disease suggested a general disorder which may include myocardial disease. Sometimes the heart muscle was infiltrated with dense pericardial fibrous tissue and the two layers were inseparable, particularly in areas of heavy calcification. Where the two layers of pericardium were separable, the parietal layer was usually removed easily; the thinner visceral layer still caused constriction in some instances, and was difficult to remove without damaging the coronary vessels. A layer of fat beneath the visceral pericardium was often

TABLE V
AGE AND OPERATIVE RESULT

	Died	I.S.Q. or improved	Cured
Age over 50	2	5	—
Age under 50	8	12	15

found and it made the situation of both vessels and heart muscle difficult to locate. Atrophy of the heart muscle made it liable to perforation.

Many patients took up to two years to reach maximum improvement after operation. Delayed improvement was not clearly associated with long duration of symptoms, cardiac enlargement, or active tuberculosis: the gradual restoration of atrophied heart muscle and the slow healing of active tuberculous lesions were probably its most important causes. Most of the patients with active tuberculosis at the time of operation made a good recovery eventually.

Recurrence of symptoms after adequate pericardectomy was unfavourable and attributable to hypertension, myocardial disease of some kind, or to some other non-pericardial condition, cardiac enlargement always being present. There was no instance of recurrence of pure constriction after an adequate and apparently curative pericardectomy. On the other hand, persisting symptoms after an inadequate pericardectomy were sometimes cured after a second or third operation.

CONCLUSIONS

Constrictive pericarditis can arise from a number of different conditions, all capable of producing an indistinguishable end result. Possibly different causes tend to produce distinct varieties of constrictive pericarditis. This is difficult to demonstrate, but a few patients in the present series, particularly those with rheumatoid disease and those having had radiotherapy, may have had significantly more myocardial damage than the others. Patients can be divided for comparison into those with acute constriction developing rapidly after an attack of pericarditis, and those with chronic constriction, developing insidiously often with no evidence of initial acute pericarditis.

The findings in acute and chronic constriction, once established, are alike in most respects. These have been briefly described and the usual presence of pulmonary as well as systemic venous hypertension is stressed again. There are, however, some differences between acute and chronic constriction. Patients with chronic constriction more often have cardiac enlargement, atrial fibrillation, pericardial calcification, close adherence and fibrous infiltration of the heart muscle, and are generally older. In fact myocardial disease is often preponderant in chronic cases. Prognosis and treatment largely depends upon whether mechanical constriction or myocardial disease predominates. Any indications in the case history or physical signs that illuminate this question are therefore important.

The familiar part of constrictive pericarditis is the mechanical effect of an unyielding shell round the ventricles. This impairs their capacity to accept inflowing blood, and possibly also their contractility. The signs in constrictive pericarditis that are most characteristic and useful in diagnosis are evidence of changes in the pericardium itself, although not all are evidence of constriction. Such signs are the combination of a small "quiet" heart with marked pulsus paradoxus, pericardial calcification, high venous pressure and its effects, and certain electrocardiographic changes.

Impairment of the myocardium is in some cases a more important cause of disability than the mechanical constrictive effect of the pericardium. The myocardial lesion is partly due to atrophy, which may be reversible. It is also partly due to destruction or disease of heart muscle, generally irreversible, resulting either from the same process that caused the pericardial lesion, such as tuberculosis or possibly collagen disease, or from some other separate but complicating condition. The myocardial lesion produces signs of heart failure, including cardiac enlargement and a diastolic sound, both of which are unspecific.

Acute constrictive pericarditis demands surgical treatment; mechanical constriction predominates, and the diagnosis is not usually difficult. The sooner pericardectomy is done, the better the results, which emphasizes the importance of early diagnosis. Operation should not be delayed more than one or two weeks unless the signs of constriction are rapidly diminishing; the severe case may be regarded as an emergency. Streptomycin fails to prevent constriction in a large proportion of cases of tuberculous pericarditis. In chronic constriction medical treatment should be preferred to begin with, particularly if many of the features unfavourable for surgery and mentioned earlier are present. Nevertheless these patients tend to lose ground, and then many are helped and a few

cured by pericardectomy. Doubt about the diagnosis, particularly in certain chronic cases with much myocardial damage, may be resolved only by thoracotomy. In such cases, however, the results of pericardectomy are usually not good.

SUMMARY

Causes of constrictive pericarditis are described. The relation of different causes and of acute and chronic onset to variations in the disease is discussed.

Symptoms and signs are described, with emphasis on those of value in diagnosis and in the provision of information about the mechanism of the disease.

The results of pericardectomy are described. The merits of the operation in acute and chronic constriction and its indications are discussed.

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