Early and late results of pericardiectomy in 118 cases of constrictive pericarditis

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ABSTRACT   The medical records of 118 patients (86 male, 32 female, age 10–50 (mean 27) years) who underwent pericardiectomy for constrictive pericarditis at the Christian Medical College Hospital, Vellore, from 1954 to 1985 were reviewed. All had appreciable pericardial constriction. Preoperatively 97 of the 118 were in class III or IV of the New York Heart Association classification and 100 had peripheral oedema or ascites. Tuberculosis was proved as the cause in 72 patients. Pericardiectomy was accomplished through a standard anterolateral thoracotomy (107 cases), median sternotomy (3 cases), or bilateral thoracotomy (8 cases). Postoperatively an apparent low cardiac output state was seen in 34 patients, 12 of whom died. Hospital mortality in the last 12 years was 11%. Mortality was higher in NYHA class III and IV patients. The improved surgical results recently may be related to increased use of inotropic support and prolonged ventilation. At follow up there were 72 patients in whom functional capacity could be assessed; 63 were in class I or II. The poor results of pericardiectomy in some patients are likely to be related to advanced preoperative disability and early pericardiectomy is therefore recommended.

Introduction

Constrictive pericarditis is the end result of a chronic inflammatory process that produces a fibrosed, thickened, constricted pericardium around the heart with limitation of diastolic ventricular filling. As the encompassing scar shrinks the heart is compressed further, especially the right heart and the great veins.

As early as 1898 DeLorme conceived the idea that pericardial resection for this condition might be feasible, and subsequently Churchill in 1929, Becker in 1930, Harrington in 1940, and Blalock in 1941 reported their experience. The past three decades have brought about improvement in the surgical approach and techniques for pericardial resection. In most patients pericardiectomy corrects the haemodynamic abnormalities and produces dramatic clinical improvement. Considerable controversy continues, however, with regard to the early and late results of the various techniques of pericardiectomy.

Our experience with pericardiectomy at the Christian Medical College and Hospital, Vellore, over 30 years provides the basis for this report.

Patients and methods

PATIENTS

The medical records of all patients undergoing pericardiectomy for constrictive pericarditis at this institution from January 1954 to December 1985 were reviewed. Only patients with clinical, operative, and pathological features of pericarditis and constriction were included. Patients with purulent pericarditis or recurrent pericarditis without constriction were excluded. A total of 118 patients met these criteria and the study represents a review of their clinical features, invasive and non-invasive preoperative investigations, early postoperative course, and long term results.

Ages ranged from 10 to 50 (mean 27 (SD 11·5) years) (table 1). The disease was most prevalent in the age range 11–40 years and less prevalent in the very young and old. There was a male preponderance with a male: female ratio of 2·7:1. The duration of the symptoms varied from one to 15 (mean 6) years. Our patients were referred from different hospitals and before referral many were extensively investigated and treated for various suspected hepatic, malignant, and cardiac disorders.

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Table 1   *Age distribution and sex ratio of the patients*

Age (y)	No of cases
1–10	4
11–20	31
21–30	43
31–40	26
41–50	8
51–60	6
Sex ratio (M:F)	3·7:1

Preoperative disability was categorised according to the New York Heart Association classification. At the time of diagnosis two patients (1·7%) were class I, 19 (6%) class II, 57 (48%) class III, and 40 (34%) class IV. All patients had clinical evidence of pericardial constriction; the common physical findings that were striking were raised jugular venous pressure in all 118, peripheral oedema in 99 (84%), ascites in 106 (90%), hepatomegaly in all 118, splenomegaly in 21 (18%), pleural effusion in 64 (54%), muffled heart sounds in 116 (98%), and pulsus paradoxus in 99 (84%).

Results of various preoperative investigations are summarised in table 2. Radiological evidence of pericardial calcification was present in 25 cases (fig 1) and pleural effusion in 63. Cardiac catheterisation and angiocardiography were performed in 84 patients and were diagnostic of a constrictive or restrictive process in all. A characteristic dip and plateau pattern was seen in the right ventricular wave forms with equalisation of end diastolic pressures in all four cardiac chambers and pulmonary artery (fig 2). A ratio of systolic to end diastolic pressure of less than 3:1 was seen in all patients. The mean (SD) values of the preoperative intracardiac pressures were as follows: right atrium (mean) 18 (5) mm Hg, right ventricle (end diastolic) 20 (6) mm Hg, pulmonary artery (diastolic) 21 (7) mm Hg, left atrium (mean) 21 (5) mm Hg, left ventricle (end diastolic) 21 (5) mm Hg. The mean (SD) cardiac index was 1·8 (0·6) l/min/m.^{2}

On the basis of the clinical data and operative and pathological findings we were able to identify a specific aetiological factor, tuberculosis, in 72 (61%) of the 118
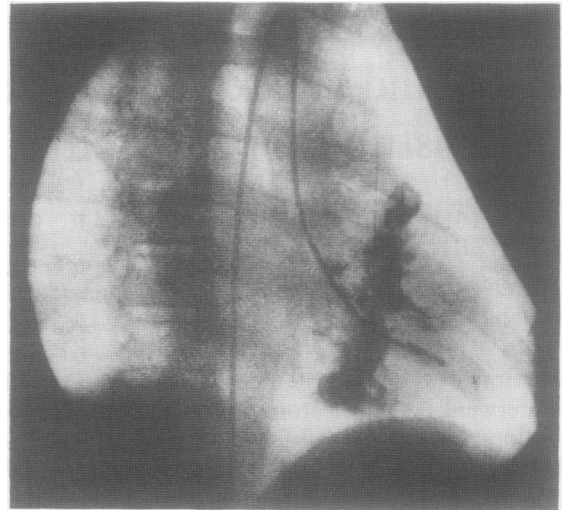

Fig 1   *Left anterior oblique view showing dense calcification in the pericardium at the atrioventricular groove and a catheter in position for angiography.*

patients. In a few cases tubercle bacilli were found in the fluid and debris removed at surgery. In about 12 cases in the tuberculous group we found an effusive constrictive pericarditis.[1] Two patients in the non-specific group also had mitral stenosis and a past history of rheumatic fever.

OPERATIVE PROCEDURE
Pericardiectomy was performed through a standard left anterolateral thoracotomy through the 4th or 5th intercostal space with provision for transecting the sternum when necessary. Early in our experience a bilateral submammary thoracotomy was used in eight patients.[2] Median sternotomy was carried out only in three cases, where there was extensive calcification of the pericardium. Cardiopulmonary bypass was not used for any patient. The process of decortication was generally terminated on the left side as soon as the left atrioventricular groove was reached after mobilisation and retraction of the left phrenic nerve. On the right

Table 2   *Results of preoperative investigations*

| Investigation	No of patients	Abnormality detected	Positive	
			No	%
Chest radiograph	118	Pericardial calcification	25	21
		Pleural effusion	63	53
		Cardiomegaly	70	59
Electrocardiograph	118	Low voltage QRS complex	89	75
		Atrial arrhythmias	9	10
Fluoroscopy	60	Evidence of restriction	42	70
Echocardiography	78	Pericardial thickening	33	42
Cardiac catheterisation	84	Increase in end diastolic pressures	84	100
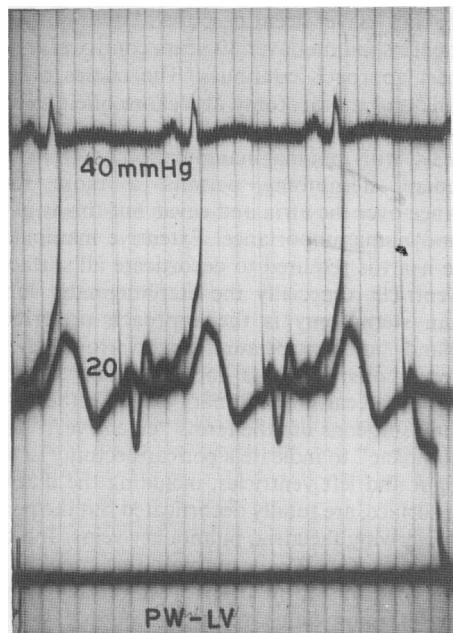
Fig 2 *Pulmonary wedge (PW) and left ventricular (LV) pressure curves depicting the classical equalisation of end diastolic pressures.*

Table 3 *Postoperative complications*

Complication	No	%
Atrial fibrillation	39	33
Premature ventricular contractions	6	5
Low cardiac output	34	28
Respiratory insufficiency	25	21

side the decortication was limited to the right atrioventricular groove. Partial pericardiectomy has been shown both haemodynamically and clinically to be adequate. The extent of further resection of the pericardium overlying the atria or the intrapericardial portion of the cavae and pulmonary veins depends on the adequacy of the exposure and the need for it. In only two instances, where a gradient of 6–8 mm Hg had been documented by preoperative catheterisation, was decortication accomplished at the cavoatrial junction. Our impression is that constriction of the cavae and pulmonary veins is rarely evident. Particular attention was directed to the excision of constricting epicardial layers. We had no hesitation in leaving islands of calcified pericardium infiltrating the myocardium.

Results

EARLY POSTOPERATIVE RESULTS
Nineteen patients died within 30 days of surgery, giving a hospital mortality of 16%. In the last 12 years, however, only six of 52 patients (11%) died in hospital. The causes of death were an apparent low cardiac output state (12), bleeding from the great vessels after laceration (3), respiratory failure (1), and cardiac arrhythmias (3). In the patients with an apparent low cardiac output the poor cardiac performance was not the result of unrelieved constriction.

Not infrequently the surgeon noticed minimal dilatation of the heart during pericardiectomy. Low cardiac output was diagnosed when hypotension, cold extremities, a weak pulse, and oliguria were evident, and varying degrees were observed in the early postoperative period in 34 patients (table 3), even though a satisfactory pericardiectomy had been carried out. An appreciable number of these patients required inotropic support with dopamine to improve cardiac function, and some required ventilatory assistance.

Among the 21 patients who were in functional class I and II (NYHA) before operation, there was only one operative death, whereas among the 97 in classes III and IV there were 18 deaths (18·6%).

The operative mortality was analysed from 1954 to 1973 and from 1974 to 1986. There was a favourable decline in the operative mortality from 16% during 1954–73 to 11% during 1974–86, possibly related to improvements in perioperative care. During the latter period patients were in more advanced stages of the disease.

Preoperative chest radiographs and electrocardiograms were available for all patients. There was no significant difference in operative mortality relating to pericardial calcification on the chest radiograph or to low voltage QRS complex or atrial arrhythmias in the preoperative electrocardiogram (p > 0·05).

FOLLOW UP
Of the 99 survivors, 78 were followed from periods ranging from two to 30 (mean 9 (SD 6·5) years. Patients with this disease often belong to a low socioeconomic group, and once symptom free they failed to report for follow up. Of the 78 patients who have been followed, most are in good or excellent condition. None required reoperation for recurrent constriction. Of the 72 subjects in whom functional capacity could be assessed, 59 were in functional class I, four in class II, and nine in class III (NYHA). There were six late deaths. The cause was unrelated to pericarditis in four patients, but two died of progressive cardiac failure.

Discussion

Although the constrictive effect of chronic pericarditis affects all four cardiac chambers and the intrapericar-

dial portions of the cavae and pulmonary veins, the only important haemodynamic abnormality that is consistent is impairment of ventricular diastolic filling.[3-9] There is an apparent encasement of the ventricles with impaired systole as a result of myocardial lesions. These two factors offer a plausible explanation for the low stroke volume, pleural effusion, ascites, oedema, and engorged neck veins. Several patients showed altered liver function, some with a protein losing enteropathy as described.[10] The diagnosis is frequently overlooked for many years, early clinical findings being subtle and the specificity of the common non-invasive tests low.[3 11 12 13]

Tuberculosis is the leading cause of constrictive pericarditis in the Third World and in our study 72 patients (61%) had proved tuberculosis according to histopathological findings. Two patients also had mitral stenosis and a past history of rheumatic fever. McCoughan and colleagues[14] were able to identify a specific aetiological factor in only 27% of their patients and Blake and colleagues[15] in only 34%. In the very early report from Massachusetts General Hospital tuberculosis was the proved cause in only 17%.[16] Tuberculosis remains a major cause of constrictive pericarditis in Third World countries where pulmonary tuberculosis remains endemic.[12 17-19] Non-infective processes leading to a pericardial inflammatory response include irradiation,[20-22] haemopericardium,[11 13 23] collagen diseases,[11 24-26] and metabolic infiltration.[27] Cardiac surgery has also been implicated; the incidence of constrictive pericarditis after cardiac surgery is reported to be around 0·15%.[28 29 30]

The striking male to female ratio of 2·7:1 in this study is at variance with the ratio of 1:3 reported by Wood[31] and 1:1 reported by Gupta.[32] The disease occurred most frequently in the second, third, and fourth decades of life in our series, as noted by Levine.[33] The presence of ascites without oedema may lead to an erroneous diagnosis of abdominal tuberculosis,[31] though in most of our patients ascites was associated with peripheral oedema. We assume that ascites and peripheral oedema are in great measure the result of the protein losing enteropathy. The high incidence of cardiac arrhythmias reported by Wood was not seen in our series, perhaps because our patients were younger.

Contrary to the belief that the cardiac shadow on the chest radiograph in constrictive pericarditis is either normal or slightly enlarged,[34] we found that it was definitely enlarged in half of our patients. Low voltage QRS complexes with typical T wave changes were found on the electrocardiograph of most of our patients.

In 1928 Churchill[35] performed the first successful pericardiectomy for constrictive pericarditis. Subsequently several different operative techniques and approaches have been described.[34 11-13 29 36] Despite experience spanning over 30 years controversy about the best approach continues. The choice of a left anterolateral thoracotomy at Vellore offers excellent exposure of the anterolateral and inferior surface of the heart, with minimal manipulation.

Median sternotomy permits a more radical clearance over the atria and cavae but this is of little haemodynamic importance. Extensive manipulation of the heart is required to decorticate all surfaces of left ventricle, especially the diaphragmatic surface. Median sternotomy is the approach preferred by Copeland and associates,[36] who routinely use cardiopulmonary bypass for pericardiectomy. We have not used cardiopulmonary bypass hitherto.

Many surgeons use the terms "extensive", "total," or "complete" to indicate a pericardiectomy in which the right and left ventricles, including the diaphragmatic surface, are totally decorticated but the pericardium overlying the atria, pulmonary veins, and cavae is left in place.[11-13] We concur with this approach and believe that this form of pericardiectomy achieves optimal haemodynamic relief. Resection of adherent pericardium over the atria is considered hazardous; in the earlier part of our study, when decortication was extended to these areas, laceration of the inferior cavae (two cases) and right atrium (one case) led to a fatal outcome. The importance of unrecognised constricting epicardial peel has been described by Harrington[4] and recently by Walsh and associates.[37] Wherever this constricting peel was very adherent and calcified we had no hesitation in leaving islands of pericardium, after making multiple incisions, to avoid injury to major coronary vessels.

The hospital mortality after pericardiectomy for chronic constrictive pericarditis remains at 5–15%. In our series hospital mortality bore a strong relation to preoperative functional state. Our mortality in the last 12 years (11%) is similar to that in other reports.[14]

Consistently good late results have been reported in many large series of patients undergoing pericardiectomy for constrictive pericarditis and our experience is similar. From our own experience and a review of published reports it is evident that a few patients develop a low cardiac output state after pericardiectomy regardless of the operative approach and the adequacy of pericardial resection. Operative, clinical, haemodynamic, and necropsy findings indicate that impaired cardiac performance is usually secondary to myocardial dysfunction rather than unrelieved constriction.[6 8 20 27 38] The most common cause of myocardial dysfunction is myocardial atrophy.[38] Cardiac dilatation after pericardiectomy also gives rise to a low cardiac output syndrome postoperatively. The results of radical pericardiectomy have been good[3] but whether this will prevent cardiac

dilatation from poor myocardial function post-operatively is doubtful. Surgical results will continue to improve if the diagnosis is established and pericardiectomy accomplished before pronounced disability ensues.

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