EXPERIENCE WITH PERICARDITIS AT GROOTE SCHUUR HOSPITAL, CAPE TOWN

AN ANALYSIS OF ONE HUNDRED AND SIXTY CASES STUDIED OVER A SIX-YEAR PERIOD

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In Cape Town we have a multiracial society living under the same climatic and geographic conditions, with each race, in the main, adhering to its own traditional customs, particularly as regards diet. The Whites, are mostly derived from Great Britain, Holland and France. They are economically privileged; even the patients who attend the public hospital (Groote Schuur), although representing the poorer section of the White community, earn considerably more than the average non-White. The Cape Coloured community originated mainly from White, Hottentot and 'Malay' stock; only within the last few decades has any Bantu element been added. The Hottentots were the original indigenous population of the Cape. From the socio-economic point of view the Cape Coloured occupy a position midway between that of the White and the Bantu. Like the White, they are a relatively stable population and, for the most part, have lived all their lives in an urbanized community and have been exposed to all the infections and diseases that are common to city dwellers.

The Bantu, descendants of migrant tribes from Central Africa, occupy the lowest place in the socio-economic scale, providing the unskilled and heavy labour. They are an unstable population migrating from the rural parts of South Africa for a temporary stay of one or two years. During this period they attempt to meet their own needs and the needs of their homes in the Reserves.[1] As a consequence there is a strain on their meagre resources which contributes to the development of malnutrition and the consequent lowering of their resistance to infectious disease. Moreover, as they live in overcrowded and often insanitary conditions, it is not surprising that the incidence of infectious disease in this racial group is high.

When it is remembered that the Western Province, of which Cape Town is the capital city, is the chief wine-producing centre of one of the great wine-producing countries of the world, it can readily be appreciated that the effects of alcoholism are often superimposed on those of poverty, overcrowding and malnutrition. One would therefore expect that the diseases associated with the underprivileged, namely deficiency disease and tuberculosis, would occur frequently in the Coloured and Bantu racial groups.

As far as the cardiovascular system is concerned, this results in a great incidence of pericarditis, which is usually tuberculous in origin, of beri-beri heart disease,[2] and probably of 'cardiac failure of unknown origin'.[3]

During the 5 years July 1952 to June 1957, 160 consecutive cases of pericarditis have been reviewed in the Cardiac Clinic, Groote Schuur Hospital, and their course has been followed until August 1958. Pericarditis complicating rheumatic fever, the collagenoses, uraemia and cardiac infarction have been excluded from this study. It is usually not difficult to differentiate pericarditis from 'cardiac failure of unknown origin' and beri-beri, though on occasions special techniques such as cardiac catheterization or even exploratory thoracotomy may be necessary.

This study is concerned with the aetiology, natural history and results of treatment, including surgery, in these 160 cases of pericarditis.

MATERIAL AND METHODS

During the 5-year period under survey the electrocardiographic (ECG) service of the Cardiac Clinic included all in-patients and out-patients attending Groote Schuur Hospital and the 44 in-patient beds of the Somerset Hospital. As previously described,[3-5] all patients suspected of having heart disease have ECGs performed and all tracings have been examined by the author. During the period under review, approximately 22,000 ECGs were studied. Where the diagnosis of pericarditis was suspected on clinical or ECG grounds, the case was selected for special study. In this way 160 consecutive cases of pericarditis were obtained, representing all the cases of pericarditis recognized as such occurring in these two hospitals during the 5-year period under review.

Of the 160 patients, 69 were under the personal care of the author, and 67 were seen in consultation and followed through their illness while in hospital. Of the remainder, 11 were first examined after discharge from hospital, 8 died before they could be examined, and only 5 were never seen and were untraceable.

All patients were advised after discharge to attend the special pericarditis follow-up in the Cardiac Clinic for long-term treatment, usually anti-tuberculous, and observation. Whereas local White and Coloured patients are relatively easily controlled in this way, the Bantu present a far more difficult problem. As their stay in the city is often temporary, they cannot be re-examined over a period of many years. They are frequently untraceable from their local addresses and the importance of prolonged treatment is not usually appreciated. However, with the assistance of a trained

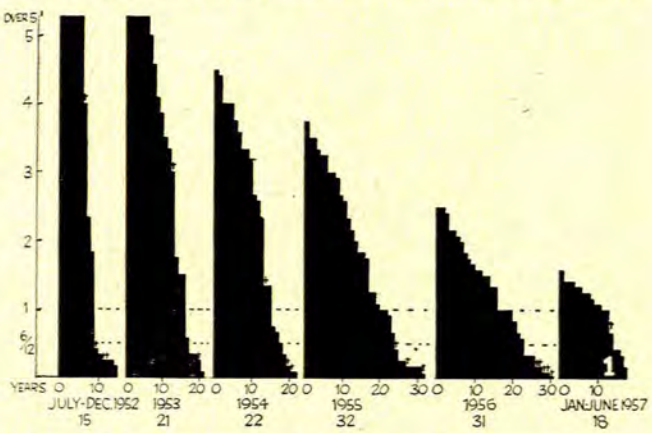

Fig. 1. Follow-up period. 75% of the patients were seen for periods longer than 6 months. Many of the patients seen for less than 6 months (10%) had died of their disease (†). These figures include the 139 cases of tuberculous, probable tuberculous and pericarditis of unknown cause only.

social worker, it was possible to follow up the majority, though reliance on general practitioners' reports were sometimes necessary. Once repatriated to the Territories, follow-up by correspondence was the only possible means, and was usually unsuccessful. During the first year of follow-up, patients were seen frequently but thereafter, except for surgical cases, who were seen more frequently, they were examined at yearly intervals.

The majority of patients were adequately studied, follow-up study including clinical, ECG and radiological examination. In this way 92% (147) of all patients were personally seen at some time in their illness, and follow-up was possible and adequate in 94%.

The material was divided into two groups. In the first were included tuberculous and probable tuberculous pericarditis and pericarditis of unknown cause, and in the second, pyogenic and a few miscellaneous causes. The follow-up period in the first group is shown in Fig. 1. The majority of patients (75%) were seen for periods longer than 6 months, by which time the condition is usually cured or static. Many of the patients seen for less than 6 months (10%) had not survived this period and had died of their disease. In the second group, the mortality rate was higher but the majority of the survivors were followed up for an adequate period up to 3 years.

The sex, race and age in the 160 cases of pericarditis is analysed in Table I. The maximal incidence was between the ages of 20 and 50 (60%), except in the pyogenic group, where the condition occurred earlier. Males were affected more than 3 times as commonly as females, especially in the tuberculous series. Pericarditis was uncommon in Whites, who made up only 8% of the cases, though the attendance of Whites at the hospital is equal to that of the Coloured.[2] On the other hand, over 50% of the cases of pericarditis occur in the Bantu, though this race represents, only 1/5th of the hospital population.[6] In fact pericarditis is one of the commonest cardiovascular diseases in the Bantu.[3] Pericarditis is far commoner in the Coloured than in the White, but less common than in the Bantu.

In addition to the aetiological diagnosis the cases were analysed according to whether pericardial effusion or constrictive pericarditis was present. (*a*) The diagnosis of pericardial effusion was based on the finding of a pericardial friction rub, aspiration of the pericardial fluid, and clinical and radiological evidence of considerable enlargement of the cardiac outline. On treatment, significant reduction in the heart size occurred. Cardiac compression was often present (tamponade), reflected in jugular venous distension, hepatomegaly or oedema on the right side and pulmonary congestion with dyspnoea on the left. Pleural effusion was common, but it was often inflammatory and not mechanical. On the other hand, tamponade was not invariable; many cases even with big effusions showed no signs of interference with cardiac function. (*b*) In the constrictive pericarditis group, either following absorption of the pericardial fluid or presenting *de novo*, marked evidence of cardiac constriction was present. Pericardial friction rub was absent, pericardial aspiration failed to yield fluid, and the heart size was normal or only moderately increased. Jugular venous distension and hepatomegaly were always present, and oedema, pulsus paradoxus and pulmonary congestion were often found. Treatment of the cardiac failure produced partial relief only. Surgery was performed in 45 cases of constrictive pericarditis and in only one was there still some fluid present, though marked constriction dominated the findings.

DIAGNOSIS

The diagnosis could usually be made by the clinical methods in common use, including ECG and radiology. A history of pain of pericardial origin occurred in about 60% (94) of the cases and was the most valuable single finding extracted from the history. Though occasionally mistaken initially for the pain of ischaemic heart disease, the differentiation was usually relatively easy, because of the stabbing quality of the pain, the relation to respiration and the associated ECG and radiological findings. Pericarditis was often associated with pleurisy (20%) and this frequently gave rise to both pericardial and pleuritic pain occurring simultaneously, unlike that which occurs in ischaemic heart disease. In the Bantu, a history of pain in the centre of the chest almost always indicates pericarditis, as ischaemic heart disease is so exceptional.[7] In the White and Coloured, however, in whom coronary disease occurs so frequently, the differentiation of ischaemic pain from the pain of pericarditis must be made. Difficulty arose chiefly in cases that were considered to have 'benign pericarditis', as in this condition the onset was often so sudden and the duration of pain relatively short. Other symptoms of cardiac involvement, such as effort dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, hepatic pain and systemic congestion, were frequent but were of no assistance in anatomical diagnosis.

At some time in the illness, a pericardial friction rub was heard in 50% of the cases (81) (Fig. 2) and in 40% the friction rub was present on the initial clinical examination.

TABLE I. SEX, RACE, AGE AND AETIOLOGY IN 160 CASES OF PERICARDITIS

Age	*Tuberculous*					*Presumed Tuberculous*					*Unknown*					*Total*	*Pyogenic*					*Miscellaneous*					*Total*
	M	*F*	*W*	*C*	*B*	*M*	*F*	*W*	*C*	*B*	*M*	*F*	*W*	*C*	*B*		*M*	*F*	*W*	*C*	*B*	*M*	*F*	*W*	*C*	*B*	
0—9	5	2	0	5	2	0	0	0	0	0	1	0	1	0	0	9	4	1	1	4	0	0	0	0	0	0	14
10—19	11	5	1	14	1	2	3	0	2	3	0	2	1	1	0	22	1	2	0	3	0	0	1	0	1	0	26
20—29	10	2	0	5	7	5	2	1	0	6	6	1	2	3	2	27	2	3	0	5	0	0	0	0	0	0	32
30—39	11	2	1	2	10	12	2	0	4	10	4	1	0	1	4	31	1	0	0	1	0	0	1	0	0	1	33
40—49	15	0	0	3	12	10	0	1	0	9	4	1	0	1	4	30	1	0	0	1	0	0	0	0	0	0	31
50—59	6	0	0	2	4	4	1	1	1	3	1	0	1	0	0	12	2	0	0	2	0	1	0	1	0	0	15
60	2	2	0	3	1	3	0	0	1	2	1	0	0	0	1	8	0	0	0	0	0	0	1	0	1	0	9
Total	60	13	2	34	37	36	8	3	8	33	17	5	5	6	11	139	11	6	1	16	0	1	3	1	2	1	160

Males (M) 125. Females (F) 35. White (W) 12. Coloured (C) 66. Bantu (B) 82.

Pericardial aspiration was successful in 44% (70 cases), the fluid usually being blood-stained and, with one exception, inflammatory in nature. A pericardial friction rub or pericardial aspiration thus proved the diagnosis of pericarditis in 66% of the cases (107). Surgery was performed in 47 cases,

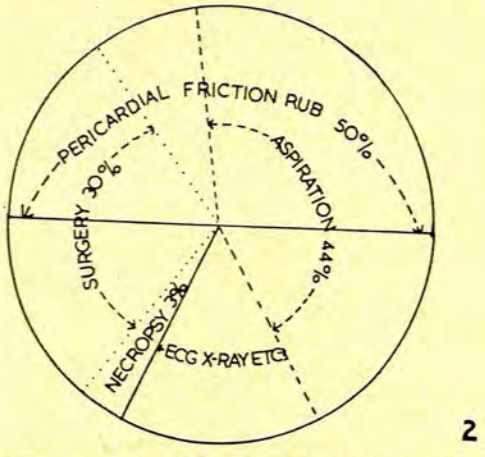

Fig. 2. Diagnosis of pericarditis, 160 cases (see text).

in 23 of whom no friction rub or pericardial fluid had been found. There were 5 cases in whom the clinical diagnosis was confirmed by necropsy only. Thus in 84% of the 160 cases (135) the diagnosis was proved by the presence of a pericardial friction rub, aspiration of pericardial fluid, surgery or necropsy. In the remainder, the diagnosis was based on clinical, ECG and radiological findings (Fig. 2).

The majority of cases could be diagnosed at the bedside. Other investigations included phonocardiography, kymography, venous pressure and circulation times, cardiac catheterization and selective angiocardiography, but these refined methods of investigation were of value in an occasional case only. There remained a few cases in which it was not possible to differentiate pericarditis from 'myocardial failure of unknown origin' with absolute certainty. Two patients early in the series were in fact subjected to thoracotomy to exclude the diagnosis of constrictive pericarditis. The number of cases where the diagnosis was uncertain, however, was extremely small and they were excluded from this series, since they would have no bearing on the statistical value of this study. Common cardiac diseases such as hypertension, coronary vascular disease and valvular disease were readily excluded and did not give rise to difficulty in diagnosis.

AETIOLOGICAL DIAGNOSIS

After excluding rheumatic heart disease, collagenosis, uraemia and cardiac infarction, the common causes of pericarditis in this series were tuberculosis, unknown infections (presumably virus), and pyogenic organisms.

1. *Tuberculous Pericarditis*

Acid- and alcohol-fast bacilli morphologically identical with *M. tuberculosis* were isolated by culture or guinea-pig inoculation from 27% (13) of the 49 cases in whom the pericardial aspirate was examined for these organisms. Many of the fluids in which organisms were not isolated were nevertheless tuberculous. This was proved by the finding of a tuberculous histology at surgery performed at a later stage in 9 cases in whom the bacteriology of the fluid had been negative. There were 2 additional cases in whom pleural-fluid examination was positive for tuberculosis and 1 in whom the ascitic fluid was positive, so that it was fair to regard the associated pericarditis as tuberculous in nature. A positive histology was obtained at surgery in 25 further cases and at necropsy in 3. Absolute proof of tuberculous pericarditis was, therefore, obtained in 44 cases.

In 29 cases there was overwhelming clinical evidence that the pericardial infection was tuberculous, in view of active tuberculosis elsewhere. Active pulmonary tuberculosis was present in 7, in 4 of whom a positive sputum was obtained. Involvement of hilar and peripheral glands by tuberculosis was present in 11 and, in addition to the adenopathy, there was sometimes evidence of involvement of other organs such as the pleural cavity, eyes and spleen. In some cases long-term follow-up revealed the development of glandular calcification radiologically. In 3 cases, miliary tuberculosis was shown by liver biopsy and, in addition, adenopathy and hepatosplenomegaly were present. There were 3 cases with peritonitis, the features of which were those associated with tuberculosis, and glandular enlargement was also present in one of these cases. Meningitis with the features of tuberculosis was present in 2 and osteitis in another 2. One afebrile patient had a loculated pericardial effusion from which sterile pus was withdrawn, as well as deeply haemorrhagic fluid from another loculus. Pleurisy developed later. Constitutional symptoms, pyrexia and response to antituberculous therapy in almost all these cases were additional features suggesting a tuberculous origin of the pericarditis. There were thus 73 cases of pericarditis which were almost certainly tuberculous in origin.

There were only 2 tuberculous cases in Whites (Table I), whereas 34 were in Coloured and 37 in Bantu. Over half the Coloured cases occurred in patients under 20, whereas the peak age in the Bantu was between 30 and 49. This is presumably due to the fact that the Coloured are a stable urbanized population with a high incidence of tuberculosis of all forms and a high infectivity rate, hence the infection of children at a young age. The Bantu are a migrant population with relatively few permantly urbanized families born in Cape Town and the high incidence of the disease between the ages of 20 and 49 is presumably related to the fact that the majority of the 'population at risk' fall between these age groups.[4] In the 37 Bantu patients there were only 2 females; this is in part related to the male predominance in the Bantu population previously mentioned.[6]

2. *Probable Tuberculous Pericarditis*

There were 11 cases who presented for the first time with well developed constrictive pericarditis; in 9 of them operation was followed by the histological finding of fibrous or granulation tissue without tubercle follicles. It is reasonable to assume that the majority of these cases were, in fact, tuberculous in origin, though constrictive pericarditis has been attributed to other causes.[8, 9] Most of the cases were chronic, of long standing, and some even had extensive calcification, so that a negative histology was not unexpected. Moreover, it must be remembered that the intensive and often prolonged antituberculous therapy which preceded histological examination at surgery or necropsy might well destroy all evidence of an underlying tuberculous process. This point was well brought out by one case of proven tuberculosis that presented

with a pericardial effusion from which tuberculous organisms were grown; yet extensive resection of constricting pericardium some time later during surgery revealed no macroscopic or microscopic evidence of tuberculosis. Another example is a case who required two attempts at surgery to relieve him of cardiac constriction. The histology at the first operation revealed tuberculous granulation tissue, whereas the second showed fibrous and chronic inflammatory tissue only. Two of the 11 cases (one a Bantu youth of 19 were admitted for acute inflammatory 'idiopathic' pleural effusion with all the features usually associated with tuberculosis, and calcific constrictive pericarditis was incidentally found, for which surgery was performed.

There were 7 patients who presented with pericardial effusions which resolved, and in whom, with the absorption of the fluid, the signs of well-marked constrictive pericarditis developed. A long history of ill health, prolonged pyrexia, and response to antituberculous treatment, was present in all. One of these cases refused surgery, another died after operation, and both came to necropsy, where evidence of tuberculosis was not found, but an intense chronic inflammatory fibrous constriction of the heart was present.

A further 26 cases were assumed to be tuberculous because of the long history of chronic ill health, often dating from 3–12 months before admission. Over half of these had prolonged pyrexia, constitutional symptoms, and gradual resolution of all symptoms and signs while on antituberculous therapy. The pleural cavity was involved as well as the pericardium in the majority of cases and in 8 of them ascites (polyserositis) was also present, in the absence of cardiac constriction.

There were thus 44 cases who were regarded as probably tuberculous, though the aetiological diagnosis remains presumptive only.

The rarity of the condition amongst the Whites is again shown by its occurrence in only 3 cases (Table I). The majority of the cases (33) occurred in the Bantu; only 8 Coloured were affected. Most of the Bantu cases were encountered between the ages of 20 and 49 and there were 5 Bantu female cases.

3. *Pericarditis of Unknown Cause*

In 22 cases of pericarditis the aetiological diagnosis could not be determined and it was not possible with any degree of confidence to differentiate tuberculosis from 'virus' pericarditis. Examination of Table I shows that of these 21 cases 11 were Bantu, 6 Coloured and 5 White. Thus the overwhelming incidence of the disease in the Bantu is again manifest, particularly between the ages of 30 and 49. If anything, this suggests that tuberculosis is the commonest cause of pericarditis in this group, because virus disease, unlike tuberculous infection, is no respecter of wealth and privilege and indeed, is more often associated with the more favoured section of the population than the reverse. On clinical grounds, 9 cases were thought to fit better with the diagnosis of 'acute benign pericarditis'[8, 10, 13] in that the illness was acute in onset, sometimes associated with fever and upper respiratory symptoms, and of short duration. Recovery was rapid and complete, although 2 cases had many relapses over a period of several months before ultimately recovering. The pericarditis appeared to be 'dry' in 6 of these cases, since there was no increase in the heart size in association with the pericardial friction rub.

In 11 cases the clinical bias was towards tuberculosis but, in the absence of bacteriological proof or evidence of organ tuberculosis or severe systemic illness, no certain diagnosis could be made. The Mantoux skin reaction was of no help, because a positive reaction was obtained in most cases of pericarditis on whom the test was performed and, on the other hand, a negative Mantoux was occasionally encountered in a proven case of tuberculosis. The nature of the effusion was also not of any real help in diagnosis. Haemorrhagic effusions were encountered in 48 of 66 cases in which the nature of the effusion was noted, the quantity of blood varying from a slight staining to an amount that made the fluid appear to be venous blood, except that it did not clot and the packed cell volume was less than that of venous blood. A heavily blood-stained effusion was often associated with tuberculosis, but merely because tuberculosis was the usual cause of pericarditis. Haemorrhagic effusions, often fairly heavy, were encountered in pyogenic pericarditis, malignant pericarditis, and pericarditis of unknown origin; even in one case of myxoedema the pericardial fluid was haemorrhagic. There is often a traumatic element to the blood-staining of the pericardial fluid and, probably because of the constant action of the heart, the pericardial fluid soon becomes uniformly blood-stained.

The degree of diagnostic overlap between pericarditis of unknown or virus origin (including benign pericarditis) and tuberculous pericarditis is considerable, and certain diagnosis rests on bacteriological proof. Two cases are given below to illustrate the difficulty.

Case 1

J.M., a Bantu female of 42, was admitted on 2 June 1955 with a story that she had been quite well until 2½ weeks before admission, when she felt cold and shivery and sweated profusely, especially in the mornings on rising. Effort dyspnoea followed and progressed rapidly until she was orthopnoeic and suffered continuous pain in the chest and a feeling as if her chest had 'closed up'. Epigastric pain and pain in both flanks developed, more marked on the right, and she also complained of a backache felt in the lower thoracic region. A cough was present from the onset of the illness; at first it was associated with blood-stained sputum, but later it became extremely severe and she began to cough up large amounts of frothy white sputum with the features of pulmonary oedema. Anorexia was present from the onset of the illness and was associated with dysphagia, nausea and vomiting. Swelling of the legs and coldness of the feet completed the history.

On examination, she was seen to be obese and in great distress with orthopnoea. Marked oedema of the sacrum and legs was present with jugular venous distension but, because of the obesity, the venous pressure could not be measured. The liver reached 10 cm. below the costal cartilage and was tender. The peripheral pulses were small, with cold extremities, tachycardia, pulsus paradoxus and a blood pressure of 100/40 mm. Hg. The apex was impalpable, but dullness to percussion extended to the right and the left beyond the normal limits, with soft heart sounds and a distant triple rhythm. Bronchial breathing was present at both bases. There was no pyrexia and protein was present in the urine.

The ECG showed low voltage in the limb leads, with slight ST-segment depression and non-specific T-wave flattening over the left ventricle. The X-ray of the chest showed gross cardiac enlargement and bilateral pleural effusions.

Before aspiration could be performed she died suddenly in the early hours of the morning on the day of admission. At necropsy, extensive fibrinous pericarditis was found with at least 1,200 c.c. of deeply blood-stained haemorrhagic effusion in the pericardial cavity. Bilateral pleural effusions were present and the liver was congested. The visceral and subepicardial layers of the pericardium were thick and tough, and the myocardium and endocardium were macroscopically normal. The histological investigations showed organization of a fibrino-haemorrhagic pericardial effusion, with normal myocardium and no evidence of tuberculosis. Extensive

hepatic necrosis was present, and marked pulmonary oedema. The cause of the condition was not established, but tuberculosis appears to have been excluded, as no evidence of this disease could be found in the chest or elsewhere.

Case 2

K.M., a Bantu male between 60 and 70 years of age, was admitted on 2 July 1954. He had been quite well until 3 months before, when he began to notice effort dyspnoea, which progressed until, shortly before admission, he was considerably disabled. From the onset he had been having attacks of paroxysmal nocturnal dyspnoea. He had suffered with headache, vomiting and general ill-health for 2 months before admission. For the same period, he had been aware of abdominal discomfort and a stabbing pain in the left chest, pleuritic in nature, associated with a chronic non-productive cough.

On examination, he was found to be apyrexial. Pitting oedema of the sacrum and ankles was present, with moderate jugular venous distension and a 5-finger firm, tender hepatomegaly. The peripheral pulses were normal with a blood pressure of 180/70 and pulsus paradoxus. The apex could not be determined, nor were any cardiac pulsations present. Dullness on percussion was increased to the left and right and the heart sounds were normal. Fine basal crepitations were heard on both sides. Bilateral optic atrophy was incidentally found.

The ECG showed T-wave inversion in the anterior chest leads. On X-ray, bilateral basal effusions were seen, with enlargement of the cardiac shadow.

Five days after admission, 900 c.c. of blood-stained fluid was removed from the left pleural cavity, and 300 c.c. of fluid from the pericardial cavity, so heavily blood-stained that it was at first thought to be cavity blood. However, the fluid failed to clot and the haemocrit was lower than that of venous blood. After introduction of air into the pericardial cavity the effusion was clearly shown to be pericardial. Treatment was commenced with streptomycin, PAS and INH. After a month in hospital the patient's general condition had improved sufficiently for him to be transferred to a convalescent home, where he died suddenly the day after admission.

Necropsy showed a small amount of straw-coloured fluid in the peritoneum. The pericardium was markedly thickened, with complete obliteration of the anterior aspect of the sac by dense adhesions. The greater part of the sac contained laminated and organizing blood, with marked thickening of both layers of the pericardium. The outer surface of the heart was ragged, showing haemorrhagic areas, fibrous adhesions and organized blood clot. There was slight dilatation of the ventricles but, apart from this, the heart itself was normal. A marked splenomegaly and moderate hepatomegaly were present. The histology showed no evidence of tuberculosis in the pericardium or elsewhere.

Had these two patients not come to necropsy, the clinical diagnosis of tuberculous pericarditis would have been very reasonable. In both cases deeply haemorrhagic pericardial effusions were present and, in one case, there was a long history of preceding illness associated with left pleuritic pain. The cause of the pericarditis was not established but a bacteriological aetiology appears to have been excluded. A virus aetiology, e.g. Coxsackie,[14, 15] is possible but the condition could hardly be termed 'benign' pericarditis. The difficulty of making a definite diagnosis of tuberculosis in the absence of strong collateral evidence is emphasized. Price *et al.*[16] describe a case of death from cardiac tamponade with massive haemorrhagic pericardial effusion which they attributed to 'benign idiopathic pericarditis'. In a review of the literature they were able to find only 4 previously reported fatal cases.

4. *Pyogenic Pericarditis*

There were 17 cases of pyogenic pericarditis, purulent fluid being withdrawn in 4 on pericardial aspiration. The condition usually developed as part of a generalized septicaemic process. In 5 cases, it spread by contiguity from a lung abscess, pneumonia or empyema. Osteitis was the primary focus in 2, skin sepsis or abscesses with pyaemic spread in 5, and post-abortal sepsis in 3. In 1 case, infection followed a stab wound in the chest and in 1 no source of infection could be found, even after necropsy. The usual causal organism was *Micrococcus pyogenes aureus* (4), but others were streptococcus, pneumococcus and *Klebsiella pneumoniae*. In several cases the causal organism could not be identified.

All the patients were Coloured except for one White. No Bantu case was encountered in this series, though one case has subsequently been seen. Suppurative pericarditis appears thus to be rare in the Bantu, as is bacterial endocarditis. Most of the patients were under 30 and, though there were more males than females affected, there was less disproportion than in the other groups (Table I).

5. *Miscellaneous Causes*

The presenting symptom in 3 cases of malignant disease was pericardial involvement manifesting as acute chest pain. One patient required repeated aspiration to relieve him of tamponade, the primary lesion being in the lung; one developed pericarditis following on perforation of an oesophageal carcinoma; one was a young girl with intrathoracic lymphoma and extension to the pericardium.

A fourth case had well developed myxoedema. Routine screening of the chest revealed massive pericardial effusion which was producing no symptoms or signs of cardiac compression. The pericardial fluid was not inflammatory, although the protein content was 7 g.%. This patient recovered completely on thyroid therapy.

Comment

In the 160 cases of pericarditis, tuberculosis was far and away the commonest cause, with proof in approximately 50%. In another 25%, tuberculosis was the probable but not completely proven cause. In 13%, the cause of the condition was not known, though tuberculosis was favoured in many. Of the remainder, 10% were pyogenic in origin and 2% malignant.

OUTCOME

1. *Tuberculous Pericarditis*

Of the 73 cases of proven tuberculous pericarditis, 74% (54) were admitted with effusions, 18% (13) with established constriction, and 8% (6) with ECG evidence of pericardial involvement as part of the generalized tuberculous process. 45% (33) developed constrictive pericarditis and 50% (37) came to surgery (Fig. 3A). 16% (12) died.

Of the 54 pericardial effusions, 60% (33) developed constrictive pericarditis as the effusion resolved, of which the majority (28) came to surgery. Of the 5 cases who did not, 2 died of the effects of cardiac constriction and 3 passed through a phase of constriction which was as intense as most of the surgical cases, but after a long period of morbidity eventually recovered

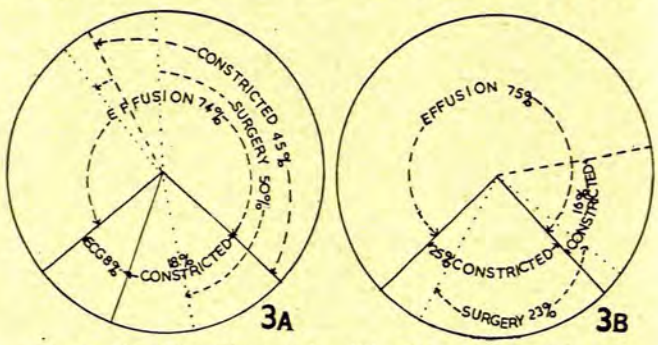

Fig. 3. (A) Tuberculous pericarditis, 73 cases (see text). (B) Probable tuberculous pericarditis, 44 cases (see text).

completely. The very high percentage of patients developing constriction in this group of cases may in part be explained by the fact that absolute proof of tuberculosis was frequently obtained at surgery and this led to inclusion of the case in this group. A very high incidence of constriction, however, was found in cases with a pericardial fluid positive for tuberculosis. Thus, of the 13 such cases, 9 came to surgery, 1 died and 3 recovered, though 2 of the latter went through a phase of constriction. 30% (15) of the patients with effusions completely recovered and returned to full health. Of the remaining 6 patients, 2 died during the early stages of the disease, 2 died of the effects of chronic cor pulmonale due to associated chronic pulmonary tuberculosis, and 2 came to surgery. One patient recovered completely from pericarditis with effusion, and surgery $2\frac{1}{2}$ years later for lung abscess revealed no pericardial pathology.

Of the 13 cases presenting with constriction, 7 came to surgery, 3 recovered without surgical intervention, 2 died, and 1 remained unchanged during the period of observation.

The 6 patients with ECG evidence of pericardial involvement as part of the generalized tuberculous process recovered completely on prolonged antituberculous treatment, with recovery of the ECG.

2. *Probable Tuberculous Pericarditis*

Of the 44 cases of probable tuberculous pericarditis, 75% (33) were admitted with effusions and 25% (11) with established constriction. 16% (7) developed constriction, 23% (10) came to surgery (Fig. 3B). 7% (3) died.

Of the 33 effusions, 21% (7) developed constrictive pericarditis as the effusion resolved, but only one of these came to surgery; 4 passed through a phase of constriction but after a long period of morbidity eventually recovered completely; 2 died of the effects of cardiac constriction; 70% (23) recovered completely and returned to full health. The remaining 3 cases were lost sight of before they had completely recovered, and could not be traced.

Of the 11 cases presenting with constriction, 9 came to surgery and 2 remained in chronic constriction for as long as they were observed. There were 4 cases with fairly marked pericardial calcification.

3. *Pericarditis of Unknown Cause*

Of the 22 cases in this group, 2 died and came to necropsy (details outlined above); the remainder recovered completely with no sequelae, apart from a persistently abnormal ECG in the occasional case. Effusions were present in 13, and 1 had an asymptomatic partially calcified pericardium, which remained unchanged.

4. *Pyogenic Pericarditis*

Of the 17 cases, 5 died of sepsis or cardiac tamponade. The remainder recovered completely, with 1 exception, who was left with mitral incompetence resulting from an associated acute bacterial endocarditis. An interesting finding in 2 cases was a persistently abnormal ECG as the only evidence of past cardiac involvement. No case of cardiac constriction was encountered in this group.

PROGNOSIS

Pericardial Effusion (Tuberculous, Probable Tuberculous and Cause Unknown)

The usual case of pericarditis, on admission to the wards, has a pericardial effusion, of which the aetiology is at first not clear. The majority are in fact tuberculous (see above) and in this series were treated as such. There were 100 cases presenting with pericardial effusion. Cardiac tamponade during the florid effusive phase was a common finding, but usually the effusion resolved or was aspirated and all evidence of interference with cardiovascular function disappeared. However, 40% developed constrictive pericarditis with the absorption of the effusion (Fig. 4A). This figure is unduly high because of special selection of the material. Groote Schuur Hospital is the centre for cardiac surgery of the whole Cape Province and cases who develop constrictive pericarditis during the course of their illness elsewhere are referred for surgery to this hospital. Thus, 8 of the 40 cases who developed constrictive pericarditis were referred for

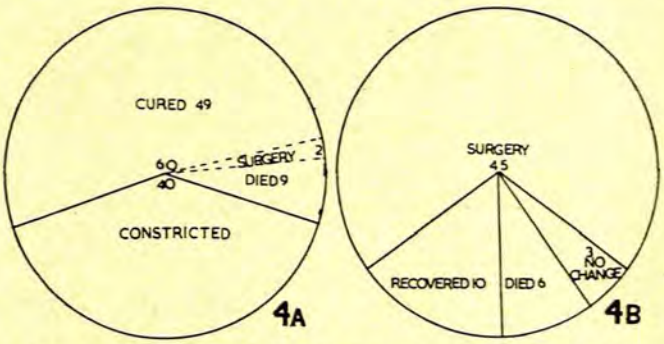

Fig. 4. (A) Prognosis in 100 cases of pericardial effusion (see text).
(B) Prognosis in 64 cases of constrictive pericarditis (see text).

special treatment. These cases in themselves probably represent only a small percentage of the total number of cases of effusion occurring in other centres. Of the 92 local cases admitted to this hospital with effusion, 32 developed constriction. All cases who recovered completely from the acute illness without the development of cardiac constriction remained well throughout the period of observation. It was possible to see 57 of the 60 patients for periods ranging from 1 to 6 years at repeated intervals and in these no form of cardiac involvement was observed to develop, once the patient had recovered from the acute stages of the disease. Of the 57 patients, 48 were enjoying full health and living an active life at their last examination, 7 having died during the acute phase of the disease and two many months later of chronic cor pulmonale, resultant on chronic fibroid pulmonary tuberculosis.

Constrictive Pericarditis

There were 64 cases of constrictive pericarditis, of whom 40 had been observed to go through the stage of effusion to constriction and 24 had presented with constriction. Of these, 45 came to surgery, 6 died of constriction, 10 spontaneously improved and completely recovered, and 3 were unchanged during the period of observation (Fig. 4B).

TREATMENT

Medical Treatment (Tuberculous, Probable Tuberculous and Cause Unknown)

(*a*) *Antituberculous therapy.* Antituberculous treatment was given in 80% of the cases and in 52% it was continued for a period of 6 months to 2 years or more. The treatment consisted in all cases of 1 g. of streptomycin daily, usually for

several months, 8–20 g. of PAS daily and INH in the usual dosage of 300 mg. daily. All three drugs were used together or in rotation in 77% of the treated cases. The development of constrictive pericarditis while on treatment did not seem to depend on what combination of drugs was used; it occurred with streptomycin and PAS, with streptomycin and INH, with streptomycin, INH and PAS, or even when no treatment at all had been given. Though it could not be proved, the impression was gained that constriction occurred more rapidly while patients were on antituberculous therapy and that before the introduction of the antituberculous therapy the development of constriction, if the patient survived the infection, appeared to have taken longer. Even when constriction occurred within 6 weeks, surgery revealed dense fibrous and granulation tissue. Though the simultaneous occurrence of pulmonary tuberculosis and tuberculous pericarditis was seen in 7 cases, tuberculous sequelae after the patient had recovered from pericarditis were uncommon and occurred only twice. Thus, one case developed Addison's disease and diffuse glandular and pulmonary tuberculosis 18 months after he had recovered from pericardial effusion which had not been treated. The other developed pulmonary tuberculosis while in a mental asylum 2½ years after recovering from constrictive pericarditis. Apart from these two exceptions patients remained free from tuberculous manifestations throughout the period of study.

(*b*) *Corticoid therapy*. An attempt was made to assess whether the administration of corticoids would prevent the development of cardiac constriction. While under antituberculous cover, cortisone was prescribed with a loading dose of 300 mg. and a maintenance dose of 100 mg. daily for several weeks. At a later date, 60 mg. of prednisone a day with a maintenance dose of 20 mg. was substituted. There were 14 cases of pericardial effusion selected for treatment with 14 alternate control cases receiving antituberculous treatment alone. No difference in outcome was noticed between the two groups. Of the 14 cases receiving corticoids and antituberculous treatment 4 developed severe constrictive pericarditis requiring surgical interference. The post-operative healing appeared to be somewhat delayed, but there was no spread of the tuberculous process in these cases. One patient who was mentally abnormal before treatment developed a psychosis during the post-operative period, but this gradually settled.

(*c*) *Cardiac therapy*. Treatment with digitalis was routine wherever evidence of tamponade or of cardiac constriction was present. In a few cases only it was administered alone with good resultant diuresis, but generally speaking mercurials were used at the same time. Though diuresis was usually obtained, the evidence of cardiac constriction remained unchanged in that the jugular venous pressure remained elevated and the liver remained distended. These drugs, however, helped to get rid of oedema and fluid in the serous cavities and temporarily relieved the urgent symptoms.

Surgical therapy

Of the 139 cases under discussion, 47 came to surgery and will be described in detail elsewhere.[17] Two of these patients were operated on during the phase of pericardial effusion. In one, the first of the series, the patient was collecting fluid rapidly after repeated effusions, the signs of cardiac tamponade remained unrelieved, and constitutional symptoms and pyrexia persisted, despite intensive therapy. At surgery, the parietal pericardium was removed and the fluid drained. The patient, however, failed to respond to treatment and ultimately succumbed with secondary infection and multiple bone sinuses. The second patient received antituberculous treatment for 6 months, during which period fluid accumulated rapidly, requiring repeated paracenteses, and he was bedridden. The parietal pericardium was markedly thickened and the signs of tamponade persisted. Surgery was eventually advised. The parietal pericardium was excised and the thickened fibrinous visceral pericardium partially removed. The post-operative course was gratifying, with immediate recovery, and long-term follow-up showed complete disappearance of all symptoms and signs. The remaining 45 cases can be subdivided into a group of 32 where constriction had been present for under 1 year, and a group of 12 where it had been present for more than 1 year.

In the first group, surgery was advised in patients who had passed through the fluid phase of pericardial effusion and had developed constrictive pericarditis. The period required to pass from the effusive to the constrictive phase varied from 6 weeks to several months. Almost all the patients were labourers whose livelihood required them to return to full activity as soon as possible. It is probable that after a prolonged illness resolution would have occurred in some of the surgical cases, as took place in several patients who refused surgery and ultimately recovered completely. With surgery, however, the period of morbidity is markedly reduced and the recovery rate high. Two patients required 2 surgical attempts each before they improved. Patients were regarded as cured if they were completely symptom-free, the jugular venous pressure was normal, and there was no hepatomegaly, no abnormal findings on clinical examination, and normal cardiac movement on radioscopy. Full return of the ECG or X-ray to normal was not included amongst the criteria of cure. 86% (26) were completely cured, with return to full heavy labour and no recurrence over a prolonged period of observation. If the patient was symptom-free but signs of cardiac constriction, viz. slight jugular venous distension and hepatomegaly, persisted, the result was regarded only as improved; this was the position in 2 cases. One patient developed constriction again, but refused re-operation. Three died, 1 of massive post-operative pulmonary embolism 10 days after surgery, 1 of exhaustion following an acute toxic psychosis, and 1 of uraemia, surgery having been advised as a desperate measure because of rapidly increasing renal failure.

In the second group, the patients had been constricted for over a year but only one case had probably been constricted for many years and had cirrhosis of the liver. Nine were regarded as cured and 3 as improved; 2 required re-operation before they were cured and calcification was present in 4.

The jugular venous pressure and hepatomegaly did not improve as a rule immediately after surgery though symptomatic improvement often preceded objective change. It often took from weeks to months before the venous pressure gradually returned to normal and the liver returned to its normal size. Antituberculous therapy was continued in all the tuberculous cases and in most of the unproven cases for many months.

Of the 47 cases submitted to surgery, 35 were completely cured, 7 improved, 4 died and 1 re-constricted; 4 patients required re-operation.

DEATHS

In the 139 cases of pericarditis (tuberculous, probable tuberculous and of unknown cause) there were 16 deaths (11%). Four died as the result of the underlying tuberculous disease, 4 of the effects of constrictive pericarditis, and 4 after surgery. There were 2 deaths from pericarditis of unknown cause. Two patients died after pericardial aspiration—1 after the removal of a large quantity of fluid, presumably from acute cardiac dilatation, and 1 a week after instillation of air into a cardiac chamber followed by cerebral air embolism. (Pericardial aspiration is not without risk and during the last 2 decades 3 deaths have occurred in this institution after the needling of cardiac failure of unknown cause to exclude pericardial effusion.) One patient died several years after a surgical cure from an unassociated condition.

SUMMARY AND CONCLUSIONS

1. A series of 160 consecutive cases of pericarditis occurring during a period of 5 years were studied and their course followed. The possible period of observation to date of writing varied from 1 to 6 years. The follow-up was considered adequate in 94% of the cases. Pericarditis due to rheumatic fever, collagenosis, uraemia and cardiac infarction were excluded.

2. The incidence of pericarditis was unquestionably highest in the Bantu. In the Coloured the disease is common, whereas in the White it is rare. When it is remembered that the White and Coloured in Cape Town are each 5 times as numerous as the Bantu the markedly disproportionate involvement of the latter becomes more evident. In all races and almost in every decade males far outnumber females, and this is not due solely to the disproportionate number of males in the Bantu population. The malady is commonest from adolescence to middle age.

3. The diagnosis was made in the majority of cases on the history of chest pain, the presence of a pericardial friction rub, aspiration of the effusion, surgery, or necropsy. Almost all cases presented either as pericardial effusion or as constrictive pericarditis, though the development of constriction in a case of effusion was common.

4. Tuberculosis was found to be the commonest proved cause of pericarditis, accounting for 50% of the cases. In another 25%, tuberculosis was the probable, but not completely proved, cause. In 13% the cause of the condition was not known, though tuberculosis was favoured in many. Of the remainder, 10% were pyogenic in origin and 2% malignant.

5. Of the cases of proved tuberculosis, 75% presented with effusion and 18% with constriction. There was a high incidence of constriction (60%) in the cases presenting with effusion, so that ultimately over 60% of this group of cases developed constriction. Cases with a pericardial fluid positive for tuberculosis are particularly liable to constrict and this occurred in 11 of the 12 patients that survived long enough for this to develop.

6. Of patients with probable tuberculous pericarditis, 75% were admitted with effusions and 25% with established constriction. The development of constriction in this group of cases with pericardial effusion was less common, occurring in 16% so that ultimately 40% of this group were constricted.

7. There were 22 cases of pericarditis of unknown cause with 20 complete recoveries. Two died; their clinical and necropsy findings are described. The aetiology was regarded as virus but the outcome belied the use of the term 'benign pericarditis'.

8. There were 100 cases of pericardial effusion of tuberculous, probable tuberculous and unknown cause. Constriction occurred in 40%. All patients who recovered from the acute illness without the development of constriction remained free of all cardiac signs or symptoms (apart from occasionally persistent ECG abnormalities) throughout the period of study.

9. There were 64 cases of constrictive pericarditis, 40 of whom had been observed to pass from the stage of effusion to constriction. Surgery was performed on the majority, but 10 cases improved and recovered completely on antituberculous treatment alone.

10. Prolonged antituberculous treatment was given in the majority of cases and appeared to promote the development of constriction. Corticoid therapy was ineffective in preventing this. Though active pulmonary and organ tuberculosis associated with pericardial effusion was not rare, the subsequent development of tuberculosis once the condition had settled was exceptional and occurred in only 2 cases.

11. Surgical treatment was highly successful in relieving cardiac constriction and reducing morbidity. The operative mortality was low and re-constriction rare.

12. Patients with pyogenic pericarditis either succumbed during the acute illness or recovered completely, though the ECG occasionally failed to return to normal.

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