Papers and Originals

Prognosis after Operation for Constrictive Pericarditis

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A survey of the published results of the surgical treatment of constrictive pericarditis (Smith and Muller, 1962) showed a fall in the operative mortality from 20% (in 629 patients) before 1950 to 13% (in 776 patients) in the period 1951-61, while the "cures" increased from 44 to 57%. However, the results varied considerably in the 20 series reported since 1950, the operative mortality rate ranging from 3 to 24%. As selection for surgery is bound to be influenced by such figures it was felt that a reappraisal should be made both of the operative risks and of the post-operative course of this condition.

In this country the period of the second world war and the succeeding two decades, comprising an epoch of rapid expansion in thoracic surgery, included what will doubtless prove to have been the peak in the number of operations performed for this disease. The back-log of operable cases was largely abolished and the incidence of new ones was possibly curtailed by the advent of antituberculous chemotherapy. The present report covers the operations of patients subjected to pericardiectomy for constriction at two cardiothoracic units since 1942, and includes a recent assessment of those survivors who could be traced for a follow-up examination.

Subjects and Pre-operative Data

Between April 1942 and August 1964 pericardial resection for constrictive pericarditis was performed at the Middlesex and Harefield Hospitals in 56 patients. These were all primary operations for constriction, though in the same period five of these patients have undergone further operation for recurrent constriction. There were 38 males and 18 females. Their ages

TABLE I.—Age and Rhythm of Patients at Operation

Age in Years:	0-10	11-20	21-30	31-40	41-50	51-60	61-70	Total
Sinus rhythm	4	9	9	7	6	8	2	45
Atrial fibrillation or flutter	0	0	2	2	1	5	1	11
	4	9	11	9	7	13	3	56

at the time of operation are given in Table I. This shows a fairly even distribution from the second to the fifth decade: four patients were under 10 and three over 60. The interval between the first symptoms of constriction and diagnosis was not always easy to determine, sometimes owing to an insidious onset, and sometimes because the effects of an acute pericarditis with tamponade merged imperceptibly with those due to fibrous

constriction. In some 60% of cases, however, the interval was a year or less, and in a further 23% from one to four years.

Aetiology

Pre-operative evidence of tuberculous infection was obtained in 10 out of the 56 patients (18%). This evidence consisted of a positive sputum culture in four patients, and positive culture or guinea-pig inoculation of pericardial fluid in six. Reports of pericardial biopsies taken at operation are available in 51 subjects, and provide firm histological evidence of tuberculosis in six cases, including three who also had pre-operative evidence. One of these is illustrated in Fig. 1. The overall incidence of proved tuberculous infection is therefore 23%. Suggestive histological features, in the form of faint tubercle formation or occasional giant cells, were reported in two more cases, while caseous material, often amidst calcific areas, was common. Though we have not accepted these findings as proof of tuberculosis, they are highly suggestive. In all other cases the histology was non-specific, showing thickened pericardium due to fibrosis or calcification, with a varying quota of inflammatory cells.

FIG. 1.—Pericardial biopsy, showing tuberculous lesion.

Excluding the 10 cases with proved tuberculous infection, six patients gave a history of acute pericarditis with effusion from seven months to eight years before operation. One patient had chronic rheumatoid arthritis. None had a history of trauma with haemopericardium, and there was no instance of

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constriction following suppurative pericarditis. One patient who suffered some years previously from staphylococcal pyaemia with pericarditis and multiple bone abscesses later developed the classical features of constrictive pericarditis, but at operation the pathology was shown to be tuberculous. In the remaining cases (39 out of 56, or 70%) the onset was insidious and the aetiology obscure.

Clinical Presentation

The mode of presentation and the physical signs were typical in the majority of cases. 86% of the patients complained of dyspnoea, 68% of oedema of the legs, and 61% of abdominal swelling. One patient presented with attacks of paroxysmal dyspnoea, a feature often taken to exclude constriction. She was explored as a case of suspected left atrial myxoma, the correct diagnosis being made at operation.

The jugular venous pressure was raised in all cases, usually grossly. Sinus rhythm was present in 80% and atrial fibrillation or flutter (one patient) in 20% (11 patients). Arrhythmias were not confined to the older patients, and five out of the 11 affected were under 45 (Table I). Nor did the presence of atrial fibrillation correlate with the duration of symptoms. Of those in atrial fibrillation 64%, and of those in sinus rhythm 65%, had had symptoms for less than a year. The blood-pressure was in the normal or low normal range in all, and pulse-pressure tended to be small. The liver was enlarged, often markedly, in 95%, there was oedema in 73%, and ascites in 64%. The incidence of Friedreich's sign, pulsus paradoxus, and third heart sound cannot be given accurately owing to the lack of data in the earlier records. Of the 30 patients in whom the signs were looked for Friedreich's sign was present in 44%, pulsus paradoxus in 40%, and a third heart sound in 27%. Associated diseases were uncommon. Two patients had rheumatic mitral incompetence, one chronic rheumatoid arthritis, and one pernicious anaemia.

Investigations

Twelve-lead electrocardiograms (E.C.G.) were available in 44 patients, and all except one showed classical T-wave flattening or inversion in some or all leads. Voltage was low in 80% of cases, often strikingly so. Notched P waves (Evans and Jackson, 1952) were common. Chest radiograms were available in 42 cases. The cardiothoracic ratio (C.T.R.) was less than 0.50 in 64% of cases. In patients with sinus rhythm the mean was 0.49, and in those with atrial fibrillation 0.51. Calcification of the pericardium was seen in 50%. Cardiac catheterization was employed in only five patients, in all of whom the characteristic pressure curves were recorded in the right heart chambers. The diagnosis did not depend on this test in any instance and it was not considered a necessary part of the pre-operative investigation. Angiocardiography was carried out in only two cases where pericardial calcification was absent and the conspicuous clinical feature was pulmonary venous congestion. Plasma proteins were estimated in 32 patients, revealing a low serum albumin or reversed albumin-globulin ratio in five. Liver-function tests (flocculation, alkaline phosphatase, and serum bilirubin) were carried out in 26 cases and showed abnormalities, usually minor, in eight.

Operation

The operations were carried out by six surgeons, the majority (45 out of 56) being performed by one of us. Constrictive pericarditis constitutes an impediment to the diastolic filling of the ventricles. Restriction of movement of the atria is less significant and there has been no evidence that the vena caval flows are seriously affected by the constrictive

process. Ideally surgery should remove all constricting scar tissue from the surface of the heart. This is a counsel of perfection and is rarely necessary. As much scar tissue as possible should be removed from the ventricles, and both layers of the thickened pericardium must be excised. It is easy to find a false plane between the visceral and parietal layers and leave a thin "membrane" of visceral scar tissue to persist with its constricting action.

The exposure of the heart is determined largely by the site of the areas with the most cicatrization and by the presence of additional pathological changes, notably pleural adhesions. A left antero-lateral thoracotomy with or without division of the sternum gives adequate exposure in most cases, but the left ventricle is not so easily freed as it would be through a full postero-lateral incision. On the other hand, an ill patient does not submit so readily to the lateral position and if there has been a previous pleurisy the approach is complicated by laborious freeing of the lung. A longitudinal sternotomy gives admirable access to the front of the heart but makes the left border difficult to reach.

Pericardiectomy is started most easily over the anterior border of the heart. The apex and diaphragmatic surface are next freed, and the dissection carried as far round the left ventricle as possible and upwards over the pulmonary outflow tract. Calcification has an erratic disposition but there is a tendency for it to form in massive bars in the atrio-ventricular grooves. Excision in this area carries the hazard of injury to the coronary arteries, but liberation of the atrio-ventricular ring may be achieved by division of the dense calcium in two or three places, allowing the incised areas to gape. The myocardium is unduly friable and apt to tear, and oozing from the muscle may be appreciable. If burrowing spicules of calcium are eased out of the heart muscle the risk of massive haemorrhage is considerable. In all cases the extent of the operation has to be judged by the state of the patient. In some instances a very limited pericardial resection may give considerable relief with minimal risk; in others the actual liberation of the heart produces such an immediate improvement in the circulatory condition that it permits an extensive pericardial removal to be undertaken.

All except two patients survived to leave hospital. One died from pulmonary oedema on the table, and one in cardiac failure on the day after operation. The operative mortality for the series of 56 primary operations is thus 4%.

Follow-up

Of the original 56 patients four are known to be dead. The two operative deaths are mentioned above. Of the two late deaths one died 11 years later, four months after a second operation for pericardial constriction; the other died eight years after operation from myocardial infarction. In 18 subjects follow-up assessment has proved impossible, because either they have left the country or their whereabouts is unknown. Six of these had their operations in the war years.

Recent information has been obtained about the remaining 34 patients, all but one of whom have been examined by one or more of us. The duration of the follow-up is summarized in Table II. The longest interval since operation is 19 years and the shortest 8 months, the mean interval being 7.2 years.

TABLE II.—Duration of Follow-up

Duration of follow-up (years)	0-5	6-10	11-15	16-19
No. of patients	12	14	5	3

On reassessment 29 patients (85%) were found to be free from cardiac symptoms and leading normal lives. In this group atrial fibrillation was present in five and atrial flutter in one; with the exception of the patient in flutter these arrhythmias all dated from before operation. Two of these 29 patients had

moderate elevation of jugular venous pressure at rest, but no other abnormal cardiovascular signs were noted.

Of the five subjects with cardiac symptoms four had atrial fibrillation and all had a raised venous pressure. One had rheumatic mitral incompetence, and one was still improving after operation. In only three of the 34 patients, therefore, could symptoms be attributed solely to the original disease. One of these has had a second pericardial resection with much benefit and the second awaits investigation. The third, recently operated on for extrinsic pulmonary stenosis, is described in the next section. It is noteworthy that the first two developed atrial fibrillation during the follow-up period, and were the only patients to do so. If persisting asymptomatic atrial fibrillation is discounted but the two symptom-free patients with elevated venous pressure are added, evidence of circulatory dysfunction, as judged by clinical assessment and attributable to pericardial disease or associated myocardial damage, was present in five out of the 34 patients reviewed (15%). These facts are summarized in Table III.

TABLE III.—*Follow-up Details of 34 Patients*

Symptom-free	..	..	..	..	..	29
Sinus rhythm	..	..	..	..	..	23
Atrial fibrillation	..	..	..	..	..	5
Atrial flutter	..	..	..	..	..	1
Raised J.V.P.	..	..	..	..	..	2
With symptoms	..	..	..	..	..	5
Sinus rhythm	..	..	..	..	..	1
Atrial fibrillation	..	..	..	..	..	4
Raised J.V.P.	..	..	..	..	..	5

Details of the 5 patients with symptoms:

1. Rheumatic mitral incompetence
2. Still improving after operation
3. Improved by second operation; probable myopathy
4. Probable reconstriction or myopathy
5. Extrinsic pulmonary stenosis; improved by third operation

The T-wave and voltage abnormalities in the electrocardiogram had regressed in 40% of those in whom comparison with pre-operative tracings was possible. In 80% the cardiothoracic ratio was unchanged or reduced, and in 20% it had increased; in only two of these, however, was it greater than 0.52. Plasma proteins and liver-function tests were within normal limits in all cases.

Reconstriction

Owing to the incomplete follow-up the total incidence of reconstriction justifying a second operation is not known. Of 39 patients of whom news is available 5 (13%) have had second operations. Two of these were aged 12 and 16 at the time of their first pericardiectomy and were among those with proved tuberculosis; treatment with chemotherapy was not then available. This may have had a bearing on their subsequent course, but the type of operation performed at that time was a limited one. One indeed had active tuberculous lesions at reoperation but is not available for follow-up. The other has been seen and remains well. The third died in failure four months after reoperation and the fourth is limited by rheumatoid arthritis but without cardiac symptoms. The fifth patient merits a more detailed account.

A Cypriot presented at the age of 26 with typical signs of severe constriction. The only unusual feature was a mitral diastolic murmur. There was no history of rheumatic fever. At cardiac catheterization the tracings were consistent with constrictive pericarditis, but the right ventricular pressure was 65/8 mm. Hg. The pulmonary artery was not entered. At operation on 15 December 1954 thickened pericardium was found with haemorrhagic gelatinous material between the layers. An apparently adequate resection was carried out leaving the ventricles free. The operation was followed by improvement, but four years later he was found to have deteriorated with signs suggesting reconstriction. A further exploration of the pericardium was therefore performed on 8 July 1958, but little evidence of constriction was found and the procedure was

limited to decortication of the right atrium. There was again some improvement, but his abnormal signs never wholly regressed and in 1964 he was again found with venous pressure raised to the jaw with hepatomegaly and oedema. A loud systolic ejection murmur was now heard, maximal over the right ventricular outflow tract. The short mitral diastolic murmur was still present. At right heart catheterization the right ventricular pressure was 88/4 and the pulmonary artery pressure 22/12. The site of the gradient corresponded with a calcified bar shown by angiocardigraphy to be 3 cm. in length lying across the right ventricular outflow tract. It was concluded that right ventricular ejection was embarrassed by the extrinsic pressure of this band.

A third operation was therefore undertaken, the heart being approached by a midline sternotomy. A thick chalky ridge was found overlying and compressing the outflow tract and obscuring the root of the pulmonary artery. This was removed piecemeal with bone forceps. The remainder of the anterior and lateral aspects of the ventricles was pulsating freely. The neighbourhood of the left atrioventricular groove was deeply buried and inaccessible in dense fibrous tissue. Pressures at the conclusion of the operation were 52/10 in the right ventricle and 42/16 in the pulmonary artery. He remains under observation.

Discussion

Tuberculosis is undoubtedly an important aetiological factor in constrictive pericarditis. Infection can occur in several ways. It may be part of a generalized process where miliary tuberculosis or a widespread dissemination includes the pericardium. This type of case does not often develop constriction and in former years the patients did not always live long enough to do so. The next and probably most common form is the one in which all the serous cavities may be involved—poly-orrhymenitis or polyserositis. The pleural cavities may show fleeting collections of fluid ending with a heavily adherent lung, the peritoneum may develop ascites—which has the character of an exudate and not that of a transudate—in association with raised venous pressure, and a pericardial effusion may develop which can produce subacute tamponade before it absorbs and the thickened pericardium undergoes fibrous contraction. The final route of infection is from rupture of caseating tuberculous glands in the mediastinum or lung hilum into the pericardium. This produces a fulminating pericarditis which may well proceed to constriction if the patient survives. We have two examples of this where a large glandular mass suddenly became smaller at the same time as an acute pericarditis developed.

In chronic tuberculous lesions caseation is frequently followed by a non-specific cicatrization, leaving no bacteriological or histological evidence of its tuberculous origin. The chance of recognizing tuberculous scar tissue is more favourable in recent than in long-standing cases. The difficulty in providing conclusive evidence of a tuberculous origin, and the varied criteria used as proof of past or present infection, explain to some extent the diverse emphasis which different authors place on the aetiological role of tuberculosis. Andrews, Pickering, and Sellors (1948) and Evans and Jackston (1952) were prepared to accept all cases of constriction as tuberculous. In the series described by Chambliss, Jaruszewski, Brofman, Martin, and Feil (1951) from Cleveland, U.S.A., bacteriological or histological evidence of tuberculosis was found in 28% of 61 patients. Among Gimlette's (1959) 62 patients in the United Kingdom 38% was attributed to tuberculosis, and among Schrire's (1959) 64 South African subjects 50% at least, and possibly 75%, was so ascribed. In the series in India described by Sen, Parulkar, Chhabria, and Dhruva (1962) 30 out of 40 pericardial biopsies showed characteristic tubercles and giant cells. Afro-Asian races, who are prone to a more florid type of tuberculosis than Nordic races, are particularly liable to fulminating pericarditis. In the present series tuberculosis has been accepted as the causative agent in only 21%, but our criteria have been strict and the true incidence may be higher.

Antituberculous Treatment

A course of antituberculous treatment was given at the time of diagnosis in nearly all patients. The value of this treatment in the absence of proved tuberculosis is debatable. Schrire's impression (1959) was that constriction progressed more rapidly when antituberculous drugs were given, but this may merely reflect a desirable acceleration from the active to the fibrotic form of the disease. We are not convinced that antituberculous drugs have much effect in hastening the process of constriction and if the patient is first seen with a pericardial effusion it will be at least 12 months before the constricting membranes can be peeled or dissected off the heart muscle.

The time has perhaps come to reserve antituberculous therapy for patients in whom the diagnosis is firmly established, and to lay the fear that surgery will activate a dormant tuberculous lesion. There has been no such instance in this series.

Non-tuberculous Aetiologies

Excluding the 13 patients with proved tuberculosis, six more in this series gave histories of previous acute pericarditis, but no bacterial agent was specified and in no case, therefore, can a pyogenic organism be incriminated with certainty. The same holds for antecedent virus infections, in which a causal relationship is even more difficult to maintain, though suggestive evidence has been provided by Robertson and Arnold (1962), who postulated a Coxsackie virus infection as the cause of constriction in five patients affected during an epidemic in Vancouver. The relationship between rheumatoid arthritis and constrictive pericarditis has received previous comment and is discussed by Keith (1962). Though one of our patients falls into this group and required reoperation, the relationship may have been coincidental. All authorities are agreed that rheumatic fever is not an aetiological factor in constriction, though the occurrence of constriction in patients with rheumatic heart disease has often been reported and was found in two patients in the present series. The danger in such cases of attributing symptoms to the rheumatic lesion and missing the constriction has been stressed by Kaltman, Schwedel, and Straus (1953). There was no episode of thoracic trauma in any of our patients, and haemopericardium cannot be postulated as a causative factor. The aetiology of constrictive pericarditis is well reviewed by Smith and Muller (1962) and by Schepers (1962).

The diagnostic features of the disease have been amply described by White (1935), Chambliss *et al.* (1951), and Wood (1956, 1961) and require no further elaboration. Our experience confirms that in cases where the acute illness with effusion is witnessed the earliest clue to constriction is the diminution of the cardiac shadow on x-ray films (as the fluid is absorbed) without a concurrent fall in venous pressure. The clinical presentation in this series was in nearly all cases classical and diagnosis seldom gave rise to difficulty. The exception was a woman of 55 in whom surgery was undertaken with a pre-operative diagnosis of left atrial myxoma. This was supported by a history of paroxysmal dyspnoea, left atrial enlargement on the chest film, and an angiocardigram which appeared to show a left atrial filling defect. At thoracotomy non-calcified constrictive pericarditis was found, with a tight constricting band in the atrio-ventricular groove, but no myxoma. This band, visualized in two planes on the angiocardigram, had been responsible for the so-called left atrial filling defect. Successful pericardial resection was performed. The reverse error, that of mistaking an atrial myxoma (in this case right atrial) for constrictive pericarditis, has been reported (Emanuel and Lloyd, 1962). Whether to explore for presumed residual constriction or reconstriction may be a greater problem than at the initial operation.

Medical treatment with bed rest, sodium restriction, and diuretics was employed in the interval between diagnosis and operation, and fluid retention can be counted on to show some

response to such measures. The value of digitalis in this condition has been questioned but we have used it frequently without noting any harmful effects. The wisdom of surgical relief, once the diagnosis is established, is indicated by the 80% mortality in untreated cases within five years of established disability (Schepers, 1962). The unnecessary postponement of operation, moreover, will result in further myocardial atrophy

FIG. 2.—Chest radiograph showing pericardial effusion in a 5-year-old girl at the time of an acute pericarditis.

with prolongation of the recovery phase. Steroid therapy has been employed by some to discourage fibrosis, but we have no experience of this treatment, the value of which would be hard to establish.

The operative mortality of 4% falls in the lowest range of those reported and is encouraging inasmuch as many of the patients had their operations in the early days of pericardial resection at these two centres. There has been no operative death in the 30 operations performed over the last 10 years.

FIG. 3.— Same patient as in Fig. 2, but three years later, when pericardial constriction had developed. There is gross ascites.

Myocardial Atrophy

The present series affords evidence of an excellent result in 85% of the patients followed up. Abnormal physical signs were abolished in nearly all cases, and if atrial fibrillation or elevated venous pressure persisted they were often unaccompanied by symptoms. These residual abnormalities could in several cases be explained, at any rate in part, by an unrelated cardiac lesion. If such a lesion is excluded and residual or recurrent constriction is also ruled out the dominant factor governing these patients' prognosis must be the amount of myocardial damage associated with or caused by the original pericardial disease. In an earlier report (Sellors, 1946) it was stated that "one of the fundamental points of constrictive pericarditis is that the heart muscle is normal in nearly every case." This calls for qualification, but in so far as it implies the reversibility of changes it remains substantially true. Myocardial atrophy was demonstrated histologically

in 11 fatal cases of constrictive pericarditis (Dines, Edwards, and Burchell, 1958), confirming the earlier observations of Roberts and Beck (1941). These workers were able to reproduce the appearance in dogs by constricting the heart with linen tape. Their studies showed a significant reduction in the mean diameter of myocardial fibres in both the human and the dog heart. In the former the mean fibre diameter in five specimens was 9.9 microns, compared with 13.9 microns in normal hearts. They considered this disuse atrophy to be reversible, a view consistent with the slow recovery often seen after successful pericardiectomy. The probable contribution of a myocardial factor in the constrictive syndrome is further emphasized by Deterling and Humphreys (1955) but the mechanism of the myocardial dysfunction is not clarified.

The importance of relieving constriction around the atrio-ventricular groove, referred to by Paul, Castleman, and White (1948), was appreciated early in this series and none of the patients followed up show evidence of significant residual atrio-ventricular obstruction. In one patient, referred to earlier, the symptoms of left atrial obstruction, including paroxysmal dyspnoea, were shown at operation to be caused by a tight constricting band of thickened pericardium. The importance of such localized constrictions has been emphasized by Mounsey (1959), who first described extrinsic pulmonary stenosis created in this way. Our own patient, whose history is recounted above, is an instructive example of this uncommon lesion. The facts suggest that obstruction to right ventricular outflow dated from before his first operation in 1954, when the right ventricular pressure was 65/8. The presence of a constricting band at this site was not suspected, however, and no deliberate exploration of the area was made either at the first operation or at the re-exploration four years later. The diagnosis only became apparent with the cumulative evidence of a systolic murmur clearly of ejection type, calcification over the outflow tract, the gradient at catheterization, and the appearance on angiocardiography, and was much supported by Mounsey's report of a similar case. In his patient signs of mitral, aortic, and pulmonary obstruction were all present. J. L. Barros (personal communication, 1965) encountered a third example of this condition, and has successfully relieved pulmonary artery constriction by a pericardial band which was responsible for a gradient of 106 mm. Hg across the stenosis. We interpret the short mitral diastolic murmur in our patient as evidence of distortion of the atrio-ventricular ring by fibrous or calcific bands; significant obstruction of left atrial emptying is excluded by a normal pulmonary wedge pressure (mean 8 mm. Hg).

Coronary occlusion by the fibrotic process seldom occurs, and though calcific infiltration may sometimes burrow deep between the muscle fibres the functional disturbance so caused appears small, for symptoms are relieved without their removal. The degree of myocardial fibrosis, which, as Burwell (1957) points out, is also incapable of resection, is impossible to assess clinically. Though the E.C.G. might be expected to reflect it, the severity of the pre-operative changes does not correlate with the post-operative course.

Electrocardiographic Changes

It is known from experimental work (Kisch, Nahum, and Hoff, 1940) that the electrocardiographic changes produced by minor surface injury predominate over those due to deep injury, and it follows that persistence of abnormal T vectors after pericardiectomy cannot be taken as a quantitative index of myocardial damage.

In the present series reversion of the E.C.G. to a normal or near normal pattern was seen in 40% of patients. Clinical improvement was correspondingly satisfactory, as it was in the majority who failed to show E.C.G. improvement. The discrepancies between clinical course, E.C.G., and radiological findings are illustrated in Figs. 2 to 8.

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Cardiac enlargement during the follow-up period, a more reliable sign of myocardial damage than the E.C.G., was seen in only six of our patients, and in only two is the C.T.R. now greater than 0.52. This lends support to the opinion that

FIG. 4.—Pre-operative radiograph and E.C.G. of same patient as in Figs. 2 and 3. The E.C.G. shows typical T-wave changes.

FIG. 5.—Radiograph and E.C.G. of same patient as in Figs. 2-4, 10 years after operation. She is now 18 and symptom-free. Note the persisting T-wave changes, but no increase in heart size (C.T.R. 0.49).

irreversible or progressive myocardial injury is the exception rather than the rule in this condition. Further evidence for this view is provided by Fitzpatrick, Wyso, Boshier, and Richardston (1962), who found normal right atrial and right

ventricular pressure tracings in seven out of eight patients after complete pericardial resection.

The disturbance of liver function by low cardiac output or chronic venous congestion was far less severe on routine chemical tests than might have been expected from the considerable hepatic enlargement often present. There is no evidence of any permanent derangement of liver function after the return of a normal circulation.

FIG. 6.—Pre-operative radiograph and E.C.G. of man of 39. Note the right pleural effusion and widespread T wave changes.

FIG. 7.—Same patient as Fig. 6, three years after operation, showing almost complete return to normal of E.C.G. despite moderate persisting cardiomegaly (C.T.R. 0.52). He is symptom-free.

FIG. 8.—E.C.G.s of man of 25 at the time of operation. A, pre-operative tracing; and B, tracing at follow-up 11 years later. T-wave changes persist despite absence of symptoms and a normal cardiac shadow.

The life expectation of patients operated on for constrictive pericarditis is still unknown, but the present evidence suggests that reconstruction after adequate resection is uncommon, that reoperation, when necessary, is usually beneficial at little greater risk than the first operation, and that myocardial damage resulting in permanently abnormal cardiac function occurs in at most 15% of patients, while residual symptoms are even rarer. In comparison with an operation such as mitral valvotomy, therefore, it is reasonable to regard pericardiectomy as a curative rather than modifying procedure, and to accord any new sufferer an optimistic prognosis.

Summary

The results of pericardial resection for constrictive pericarditis in two centres since 1942 are presented. The operative mortality in 56 first operations was 4%. There has been no death in the last 30 cases.

A recent follow-up examination has been conducted in 34 patients, the intervals since operation ranging from 19 years to 8 months (mean 7.2 years). The majority (85%) were found to be free of cardiac symptoms and were leading normal lives. The incidence of symptoms or signs attributable to continuing constriction or associated myocardial damage was in the region of 15%. There were two late deaths, one eight years later (from myocardial infarction) and the other 11 years later (after operation for reconstruction). Five out of 39 patients of whom news is available have had second operations for constriction.

Bacteriological or histological proof of tuberculosis was obtained in 23% of the 56 patients.

The prognosis after adequate pericardial resection is good, and disability from progressive myocardial dysfunction is rarely seen.

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Geographical and Tribal Distribution of the African Lymphoma in Uganda

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The geographical distribution of the African lymphoma (Burkitt, 1963; Wright, 1963) on the Continent of Africa has been shown to correspond closely with certain environmental conditions, notably temperature and humidity (Burkitt, 1962). This geographical localization, together with the age incidence of the tumour, has formed the basis for the hypothesis that this tumour may be caused by an arthropod vectored virus. Most of the previous reports have dealt with the distribution of the tumour in Africa as a whole and have of necessity been rather superficial in certain aspects. This report gives a more detailed analysis of the age, tribal, and geographical distribution of 450 histologically proved cases of this tumour seen in Uganda over the past eight years.

In Giemsa-stained imprint preparations of the African lymphoma the lymphoid cells have a characteristic morphology (Wright, 1963). These cells are not found in other types of lymphoma or leukaemia (Pulvertaft, 1964) and we regard them as diagnostic of the African lymphoma. Many of the features of these cells can be recognized in sections of formalin-fixed tissue, enabling the diagnosis of African lymphoma to be made on histological criteria (Wright, in preparation). Although some of the clinical features of the African lymphoma, such as multiple jaw tumours and bilateral ovarian tumours, are almost pathognomonic of the condition, the diagnosis in all the cases recorded here was confirmed by cytology or histology.

Topography, Vegetation, and Climate

Topography and Vegetation

Most of the northern and eastern regions of Uganda lie at altitudes varying from 2,000 to 4,000 feet (600 to 1,200 m.) above sea level (Fig. 1). Within these regions the only district

above 4,000 feet is Karamoja, an arid semi-desert region sparsely populated by nomadic tribes. With the exception of the thinly populated area containing the Queen Elizabeth Game Park (marked G.P. on Fig. 1), all the western region is over 4,000 feet above sea level. Much of this area, including Kigezi District and the south-western part of Ankole (Fig. 2) is over 5,000 feet (1,500 m.) above sea level. Most of Uganda consists of low rolling hills and plains covered by grass or bush savannah. There are large areas of papyrus swamp, particularly around Lake Kyoga and along the course of the Nile. To the west the broad grasslands of Ankole District rise to the steeply wooded hills of Kigezi District and the snow-capped peaks of the Mountains of the Moon. These highland areas are a continuation northwards of the huge mountainous plateau that forms most of Rwanda and Burundi and extends into north-western Tanzania. Most of the

Fig. 1.—Map of Uganda showing altitudes above sea level.

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