OPEN-HEART SURGERY FOR RHEUMATIC DISEASE OF THE MITRAL VALVE:

EXPERIENCE AT THE UNIVERSITY OF CAPE TOWN MEDICAL SCHOOL*

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The early experience of open-heart surgery for rheumatic mitral valve disease at the University of Cape Town has already been reported.[1, 2] With the development of total mitral valve replacement the subject has recently become one of rapid change and development. The material at Cape Town now covers a variety of plastic techniques as well as total valve replacement and a review of this experience is timely.

MATERIAL AND METHODS

The case records of all patients undergoing open-heart repair of rheumatic mitral valve lesions have been reviewed. The majority of the operations were performed by one of us (C.N.B.) and the remainder by another (R.W.M.F.). Commissurotomies that could have been done closed but which were done under direct vision during the course of operations on other valves are not included. For each case the pathology of the valve, the type of repair, the degree and duration of cardiac disability, the presence of pulmonary hypertension and the presence of other valve defects have been recorded. The influence of these factors on operative mortality, morbidity, the immediate quality of the repair, the durability of the repair and the occurrence of complications has been assessed. Most patients have been followed-up as a routine procedure in the Cardiac Clinic by one of us (V.S.). A full reassessment was made annually including radiological and ECG examinations. Postoperative cardiac catheterization was performed only with valve replacements. Two patients could not reattend, and reports from them or their physicians were relied upon.

Selection of Patients

At this institution severity of cardiac disability has never been a contraindication to surgery. In fact many patients have been in the last stage of class IV of the American Heart Association classification.

Until about 2 years ago, however, there was selection according to the pathology of the valve. Since the techniques used up to that time had definite limitations, an attempt was made to select only patients with pure insufficiency. Cases with significant aortic valve disease were also excluded. With the advent of effective prosthetic valves no patient has been refused surgery because of the severity of his condition, the degree of destruction of his valve, or the presence of multivalvular disease.

Surgical Techniques

The techniques used for valve repair have been:

1. *The ivalon baffle*. This was used, as previously described,[1] to compensate for a non-functioning posterior cusp in the presence of a reasonably mobile anterior cusp and a normal or only slightly diseased valve ring. It was placed over the posterior cusp and anchored by several heavy silk mattress sutures which were passed through and tied over more ivalon in the atrium. This technique was combined with annuloplasty stitches when needed.

2. *Annuloplasty*. This was used in the presence of a dilated valve ring and normal leaflets. Heavy silk sutures passed through and tied over ivalon buttresses were used to crimp and shorten the atrioventricular ring at one or both commissural areas. A Wooler-type annuloplasty[3] was used once.

3. *Cusp extension with autogenous pericardium*.[4, 5] This was sometimes used as an alternative to the baffle, but was also used when a baffle was unsuitable, because of shortening of both main cusps and some narrowing of the valve ring. After separating the edge of the valve and its attached chordae from the ring, an appropriately sized and shaped insert was sutured to ring and cusp by multiple interrupted and continuous 3-0 sutures. Annuloplasty stitches were employed as well in some cases.

4. *Replacement of posterior cusp and chordae with an autogenous pericardial cusp with chordal sheets*.[6] With a normal or narrowed ring, a destroyed posterior cusp and chordae, and an anterior cusp with still reasonable function, this technique has been used. The appropriately tailored pericardial cusp was sutured to the valve ring or the remaining destroyed valve tissue with multiple interrupted and continuous 3-0 sutures. The chordal sheets were anchored to the papillary muscles with interrupted sutures and each commissure was closed with a continuous stitch.

5. *Total valve replacement*.[7] When half or more than half of the valve tissue had lost mobility, valve replacement became mandatory. The natural cusp tissue, the chordae and the papillary muscles were excised leaving a narrow rim of remaining valve around the ring. The prosthetic valve ring was mounted either on the ventricular or on the atrial side of this rim and secured in place by 20 or more heavy silk mattress sutures. When the prosthetic ring was on the ventricular side the sutures were passed through and tied over a doughnut of ivalon in the atrium.

6. *Relief of mitral stenosis*. Open operation for mitral stenosis consisted of the separation of anterior cusp and commissural tissue, usually by sharp dissection, the mobilization of fused chordae tendineae and, where necessary, the splitting of papillary muscles.

OPERATIVE APPROACH

The surgical approaches to the mitral valve used in this series have been directly through the left atrium, via a right thoracotomy or a left thoracotomy, and indirectly through the right atrium and interatrial septum via a median sternotomy. With an aneurysm of the left atrium any approach is satisfactory. With a left anterior thoracotomy (with removal of the fourth

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rib) the mitral valve is close to, but often not directly in the view of the operation. With a right anterior thoracotomy (above or through the bed of the fifth rib) the valve is somewhat further away but can be seen more directly and often with less distortion of the heart. The approach through the septum probably involves more retraction of the heart than any other approach. With relative fixity of the heart owing to the adhesions of a previous operation, the sternal and the left thoracic approach become especially difficult. Flexibility in the choice of approach is obviously desirable and the need to repair other valves may determine the issue, but, in our opinion, the right thoracic approach most often provides a satisfactory exposure of the mitral valve.

RESULTS

The results are related first to the techniques used for repair.

The Baffle Operation

Seventeen patients had this operation (Table I). All had disability that placed them in class III or IV of the American Heart Association classification. Pulmonary hypertension (mild)

TABLE I. COMPARISON OF CASES

Technique	Patients	Valve lesion			Previous operations	Calcification	Pulm. hpt.		Other valve lesions
		Sten.	Insuff.	Both			Mod.	Sev.	
Baffle	17	0	13	4	1	1	2	0	1
Pericardium	8	0	5	3	1	2	5	1	3
Replacement	18	2	3	13	17	6	5	10	7

was present in only 2 cases. These cases were selected on the basis of physical signs indicating pure insufficiency, and the surgeon's findings confirmed the accuracy of the selection. Most often the valve at surgery had a shrunken posterior cusp and a reasonably long and pliable anterior cusp and chordae without commissural fusion. In 4 cases there was some commissural fusion which had to be opened before placing the baffle. Calcification was present only once but was not extensive enough to impair the mobility of the anterior cusp. After the initial placement of the baffle, some insufficiency at the commissures was often still present, and annuloplasty stitches were used to correct this in 6 cases. The surgeon felt that a complete or nearly complete cure of the insufficiency had been achieved in 13 cases, with definite reduction in the rest. Where mitral valve disease only was present there was no operative mortality. The operative death was a patient in whom both mitral and aortic valves were repaired. There was an initially satisfactory

TABLE II. DEATHS AND COMPLICATIONS COMPARED FOR DIFFERENT OPERATIONS

	Patients	Operative deaths	Late deaths	Complications	
				SBE	Emboli
Baffle resections ..	17	1	4	4	2
Pericardial autografts	8	0	1	1	0
Valve replacements ..	18	3	2	1	8

haemodynamic response. However, acute renal failure developed and the patient died suddenly 4 days postoperatively. The case was in class IV of the American Heart Association classification, and was on cardiopulmonary bypass for nearly 4 hours (Table II).

There were 4 late deaths among the 16 survivors. All were due to bacterial endocarditis. In 3 patients the organism was a *Staphylococcus aureus* and in one a *Paecilomyces*.[8]

In 2 of these patients episodes of subacute bacterial endocarditis due to *Streptococcus viridans* had occurred before surgery. In 2 cases the infection became evident within 2 months of the operation while in the other 2 it did not manifest until 1 and 3 years postoperatively. All 4 cases had rather gross mitral insufficiency at the time of death and this appeared to have preceded the development of endocarditis in 2 of them.

All the remaining 12 patients had an initially excellent symptomatic response to surgery. In 1 symptoms and signs recurred after 1 year, necessitating later total valve replacement. Another patient, despite the recurrence of gross signs of mitral insufficiency, continues to feel greatly improved symptomatically 24 months after repair. A third has developed tricuspid insufficiency while her mitral valve has become stenotic. The remaining 9 patients are all greatly improved symptomatically and have only minor disability; 5 have signs of moderate mitral insufficiency while 4 have only minimal evidence of mitral valve disease. The status of these patients has not changed from 1 to 2½ years postoperatively (Table III).

TABLE III. RECURRENT OR CONTINUED MITRAL INCOMPETENCE

	Patients	Gross		Moderate	Minimal
		No SBE	With SBE		
Pericardial autografts	8	1	1	2	4
Baffle insertions ..	16	2	4	5	5

One patient had a cerebral embolus 5 months postoperatively. She had had subacute bacterial endocarditis several years before the operation but there was no evidence of bacterial endocarditis at the time of the embolism. Atrial fibrillation was present at the time despite attempts at conversion. The hemiplegia has largely resolved and exercise tolerance is excellent. The signs are those of mild mitral insufficiency. This is the only patient on long-term anticoagulants. Another patient, also with atrial fibrillation, had a cerebral embolus 8 months postoperatively, from which she has recovered completely.

Pericardial-cusp Extension and Replacement

Pericardial autografts were used in 8 patients (Table I); 5 had grade IV, 2 grade III and 1 grade II-III disability. 3 had mild and 2 had severe pulmonary hypertension. The valve lesion was stenosis and insufficiency in 3 and pure insufficiency in 4. Unlike the patients in whom baffles were inserted, the anterior cusp was shortened and/or thickened in 6 (partly calcified in 2). All had shortening and loss of mobility of the posterior cusp. Shortening and loss of mobility of commissural tissue contributed to the insufficiency in 5 cases, while excessively long chordae tendineae, with overshooting of the anterior cusp, were present in 4.

In 6 cases extension of the posterior cusp and part or all of the commissural tissue was performed. In 3 annuloplasty stitches were inserted, in 2 the commissural area was obliterated by stitching adjacent anterior cusp and commissural tissue to each other and in 1 a tear in commissural tissue was repaired; 1 patient had in addition a portion of pericardium stitched to the free edge of the anterior cusp and then down to the chordae tendineae.

In 1 patient both anterior and posterior cusps were extended and a new commissural cusp and chordal sheet was provided. Finally, posterior cusp and commissural tissue and chordae were replaced in 1 patient.

In 1 patient some insufficiency remained at one commissural area after repair. In the rest, repair appeared to have been complete. For the first 3 weeks or so after surgery, the clinical findings confirmed this. Murmurs returned thereafter in 4 patients. In one, 1 year after surgery, the clinical features are those of quite marked mitral insufficiency. For 11 months she experienced great subjective benefit but has recently had a recurrence of cardiac failure and is awaiting re-operation. In the patient in whom both cusps were lengthened the return of murmurs coincided with the development of bacterial endocarditis due to *Staphylococcus albus*. He died 2 months after surgery in severe congestive failure. At postmortem a short commissural suture line between the anterior cusp and the new commissural cusp and chordae sheet had given way, presumably as a result of the endocarditis. This patient had mitral stenosis and insufficiency, an immobile valve with calcification, severe tricuspid insufficiency (also repaired), and pulmonary-artery pressure at systemic level (Table II).

Five of the remaining 6 patients have had excellent symptomatic results. 2 have clinical signs of moderate mitral insufficiency: One of these having a recurrence of symptoms with pregnancy. The others have no or trivial incompetence 2 months to 1½ years after surgery. None of these patients is on anticoagulants (Table III).

Total Replacement of the Mitral Valve

18 patients have had total replacement of the mitral valve (Table I). 12 were in class IV and several of these were in the

last stages of cardiac invalidism with cachexia and impaired liver function. 11 previous closed operations on the mitral valve had been performed (3 in one patient). 4 of these operations had been followed by a significant degree of traumatic mitral incompetence. 4 patients had already had attempts at open-heart repair of their diseased valves. In 1 patient a baffle had been used but severe insufficiency had recurred. In the other 3 patients the valves had been too distorted and destroyed for any procedure short of replacement to be of any value. 2 patients had sufficient mobile cusp substance to make a plastic repair feasible and in 1, while a plastic repair could have been done, the result would certainly have been less than optimal. For the rest the valves were too destroyed to allow any technique short of replacement. In 2 cases there was pure stenosis, in 3 pure insufficiency and in the remaining 13 combined stenosis and insufficiency. In 7 patients there was significant disease of other valves (5 cases of tricuspid insufficiency and 2 of aortic insufficiency). 2 tricuspid valve replacements, 2 tricuspid annuloplasties and 1 aortic cusp extension with a Bahnson leaflet were done. In the remainder the other valve lesions were not repaired.

Various sizes of the University of Cape Town lenticular mitral prosthesis were used in 17 patients.[9] In 1 a flexible monocusp prosthesis was used.[10]

There were 3 operative deaths (Table III). The flexible monocusp prosthesis was used to replace a valve with pure mitral stenosis. The unusually small size of the atrioventricular ring and the left ventricle in this case interfered with proper movement of the cusp of any prosthesis of average size and the patient died because of this. The need for an ultra-small prosthesis in stenotic cases was demonstrated by this case and the lenticular prosthesis used in the other case of pure stenosis was particularly small. A 54-year-old woman with very severe pulmonary hypertension and systemic hypertension had a 2-hour period of myocardial ischaemia of sudden onset starting 45 minutes after the end of bypass. This was accompanied by hypotension and followed by signs of cerebrovascular accident, which persisted after effective haemodynamics were established. The patient died 12 hours postoperatively and at postmortem examination there was a fresh cerebral infarction and coronary atherosclerosis, but no fresh coronary occlusion. The remaining death occurred in a 35-year-old woman with pulmonary hypertension. Respiratory function was poor after surgery and she was given intermittent positive-pressure respiration via a cuffed tracheostomy tube. She died suddenly on the third postoperative day, apparently from a complication of the presence of the tracheostomy tube.

The haemodynamic result in all except one of the remaining patients was excellent. Objective evidence of mitral valve and cardiac function showed spectacular returns to normality and this improvement has been maintained in patients followed-up from 4 to 16 months. The symptomatic response has been similarly good, and in patients without subsequent complications, a dramatic return to normal activity from a state of complete cardiac invalidism has been the rule. The patient whose significant tricuspid insufficiency was not repaired made a noticeably slower return to normality than those in whom this lesion was corrected. The one patient with an unsatisfactory symptomatic and haemodynamic result had developed severe and progressive systemic hypertension since surgery. Frequent attacks of paroxysmal tachycardia appeared to affect the function of the valve. She died nearly 2 years postoperatively and at postmortem examination the significant findings were occlusion of a renal artery, probably owing to atheroma and massive hypertrophy of the left ventricle interfering with the movement of the valve.

All patients were initially put on long-term anticoagulants. One had troublesome bleeding into the thigh and was therefore taken off anticoagulants. One week later she had cerebral symptoms lasting a few hours suggestive of a small embolus. She has had no further difficulty in the subsequent 9 months. 6 other patients have had cerebral episodes presumably owing to embolism while on anticoagulants. In one there was a severe hemiplegia with aphasia in whom speech has not recovered. This followed within 2 days of left-atrial cardiac catheterization. In the others the acute episodes were very transient and have left no residue. 5 of these patients are in chronic atrial fibrillation, and 2 in sinus rhythm (Table II).

One patient developed bacterial endocarditis owing to *Staphylococcus aureus*. This became evident following an infected scalp wound which she acquired some 6 weeks postoperatively. Although the fever resolved and the blood cultures became negative on appropriate antibiotic therapy, persistence of signs and symptoms of endocarditis led to the decision to re-operate. Anticoagulants were abruptly discontinued. A massive fresh clot was present in the inactive aneurysmal left atrium and in the vicinity of the valve there was much fibrin deposition, although the valve was not obstructed or hindered in its action by this. The clot was cleared away and the valve replaced by another. The removed clot was sterile on culture. A light growth of *Staph. aureus* was cultured from the ivalon ring of the removed prosthesis. Within 2 weeks of operation blood cultures were again positive. 2 months after surgery both signs and symptoms of bacterial endocarditis were controlled on antibiotics. However, a mitral systolic murmur indicative of a partial failure of the suture line appeared. She developed severe pulmonary congestion and tricuspid insufficiency. At re-operation the atrioventricular ring was the seat of marked bacterial infection. After this had been cleared away as well as possible, her prosthetic valve was replaced by yet another, the prosthesis being anchored on this occasion onto the ventricular side of the ring. A tricuspid annuloplasty was also performed. A light growth of *Staph. aureus* was cultured from the removed prosthetic valve. The infection recurred, and 6 weeks postoperatively she died of cerebral embolism (Table II).

Annuloplasty

Only 2 patients had annuloplasties performed. Both had pure mitral insufficiency with dilated valve rings with excellent preservation of cusps. Despite evidence of excellent valve function in the early postoperative period, mitral insufficiency recurred in both. One patient died a year after surgery from ventricular fibrillation which occurred during attempted correction of atrial fibrillation.[11] The other continues to enjoy an excellent symptomatic result 9 months postoperatively, despite recurrence of the clinical features of gross mitral insufficiency.

Ten other patients had annuloplasty stitches inserted in addition to the placement of baffles or pericardial autografts. In 6 of these patients there was some (not necessarily severe) recurrence of mitral insufficiency after initial apparently complete correction.

Commissurotomy

Three patients with recurrent mitral stenosis were operated on under direct vision. 2 had had closed-finger dilatations 6 years before and one 3 years before the second operation. At operation the fused commissural areas were separated and chordae and papillary muscles were mobilized. In one patient calcium was crushed and removed. These patients have been followed-up for $2\frac{1}{2}$ - $4\frac{1}{2}$ years and have maintained excellent symptomatic results with clinical evidence of only minimal mitral stenosis.

Repair of Traumatic Mitral Insufficiency

In 1 patient closed valvotomy with the Tubb's dilator or a stenotic valve resulted in gross mitral incompetence. She was not benefited by surgery and ultimately developed intractable cardiac failure. At open operation there was contraction of the free edge of the valve rather than commissural fusion, thus making wide opening of the valve impossible. In attempting to achieve a wide opening the posteromedial commissural tissue had been split out to the ring and the posterior cusp and commissural tissue had been sheared off the atrioventricular ring. These were sutured back, but the split in the commissural tissue was incompletely repaired so as to increase the orifice size. The patient has had an excellent symptomatic result with signs of mild mitral stenosis and insufficiency on clinical examination.

There were 7 other patients in whom traumatic mitral incompetence had been produced by closed valvotomy. One patient with the same pathology as the 1 above was treated by pericardial-cusp extension and incomplete suturing of the split commissural area. The remaining cases had severe thickening and immobility of all tissues. Rather than being merely fused the commissural areas were obliterated by dense fibrosis, or calcification. In other words there was no plane of cleavage

capable of being split, and when extra force was applied with the Tubb's dilator in order to achieve a satisfactory opening, the tissues tore at whatever happened to be their weakest point. This was often in the substance of the anterior cusps to one or other side. The recognition of this type of valve is not always possible pre-operatively but it should be possible on the operating table. In one case a pericardial cusp with chordal sheets was inserted. The remaining cases had total valve replacement.

DISCUSSION

Ideally open-heart repair of the mitral valve should result in the attainment of a haemodynamically normal valve without operative mortality and morbidity and the long-term maintenance of normal haemodynamic function and freedom from complications. With all these should go a return to normal life and an absence of symptoms.

Mortality

An analysis of the factors resulting in mortality is given in Table IV.

In this series there has been no operative mortality in 21 cases of isolated pure mitral insufficiency (22 operations). In 16 cases of isolated combined stenosis and insufficiency there were 3 operative deaths (21 operations) while in 5 cases of isolated pure stenosis there was 1 death (6 operations). The overall mortality of 3 in 42 cases of isolated mitral valve disease (49 operations) is considerably better than the usual 25% reported from other centres.[12-14] In 11 patients with significant disease of other valves (7 of whom had operative procedures performed on those valves) there were 2 operative deaths. The total operative mortality for the series is 4 from 49 cases and 56 operations.

TABLE IV. FACTORS IN MORTALITY

Factor	*Patients*	*Operative deaths*
Solitary pure MI	21	0
Solitary MI and MS	16	3
Multiple valve operations	7	2
Calcification	12	3
Pulmonary hypertension	12	4
Previous heart operations	14	4

MI=Mitral incompetence. MS=Mitral stenosis.

In analysing the causes of this mortality several factors come to light. All deaths were in cases in class IV of the American Heart Association classification. All patients had had previous cardiac operations. Mortality was undoubtedly influenced by the presence of multiple valve lesions. This situation imposes considerable technical problems involving the operative approach and the performance and duration of effective cardiopulmonary bypass. Even higher mortalities for correction of multiple valve lesions have been recorded from elsewhere.[12] However, there have been several striking successes in these cases especially when the tricuspid has been the other valve involved. Experience with the correction of mitral and aortic lesions has shown this to be the more difficult problem.

Other factors that may have influenced the mortality have been calcification of the mitral valve (2 deaths in 12 cases), immobility of the mitral valve (not entirely synonymous with calcification—2 deaths in 15 cases) and severe pulmonary hypertension (more than 80 mm.Hg—3 deaths in 12 cases). In several cases these factors co-existed. However, it was never possible to show a direct link between them and the patient's death, and more patients with these features did well than did badly. Thus they are in no sense a contraindication to surgery, but they do demand the greatest possible vigilance and attention to detail to enable these difficulties to be overcome.

Since, in the great majority of cases, a definite improvement in valve function was achieved, the adequacy of the repair has not been a factor in mortality. Before the advent of prosthetic valves 2 patients survived procedures at which nothing was done for their valves. One subsequently died when too large a flexible monocusp valve was inserted, leading to failure of valve function, while the other had an excellent result following the insertion of a University of Cape Town lenticular prosthesis. Nevertheless, the advantage of having good valve function in the immediate postoperative period must be admitted and this is especially important in the group of cases with destroyed valves, in whom this can only be produced by a valve replacement.

Morbidity

The factors that seemed to contribute to operative mortality also influenced postoperative morbidity. Many class-IV patients with difficult valves previously operated on, had very long perfusions especially when other valves were also diseased. Evidence of reversible renal damage and hepatic damage occurred in several of these. In a non-specific way many of these patients took several weeks to 'get over' their operation. The continued presence of a tricuspid lesion led to prolonged convalescence in 1 patient in this series and has been observed in patients subjected to closed valvotomy. By contrast the most striking responses were seen in 2 very severely disabled patients in whom mitral and tricuspid lesions were both corrected.

Completeness and Durability of Repair and Symptom Relief (Table III)

The objective assessment of the completeness of repair must be separately considered from symptom relief. Clinical examination may indicate an inadequate repair, yet the symptoms may be markedly relieved.

The ideal in all cases is clearly to achieve a valve that is neither stenotic nor insufficient. Only 1 technique has resulted in this consistently and that is *total valve replacement*. If a properly designed valve of the appropriate size is correctly stitched in place, normal resting haemodynamics must inevitably result. The late development of insufficiency can only result from breakage or pulling loose of the valve. Fracture of a part of the prosthesis with long-continued use is a possibility but from clinical experience it can already be stated that the durability is more than 1 year. Continued *in vivo* and laboratory experience will ultimately define the limits (if indeed there are any) of durability of artificial valves. Pulling loose of a valve may occur from failure of host, invasion of the invasible part of the ring, or failure of the sutures to hold, or both. One valve in this series pulled partly loose. Bacterial endocarditis had invaded and weakened the tissues of the atrioventricular ring.

The use of the baffle technique only seldom resulted in an ideal repair. However, about one-third of the patients have minimal murmurs and a quiet cardiac action with reduction in cardiac size and another third have mild

mitral insufficiency. The durability of initially successful baffle repairs seems to have been good. Most patients have maintained their early postoperative status well.

The pericardial-autograft cases are rather too small a group for adequate analysis, but it appears as if ideal results may be somewhat easier to attain. However, there has also been some early recurrence of murmurs. Although animal work has shown a loss of function of some pericardial autografts,[5] thus far the human cases with mitral valve inserts seem to show no progression of murmurs over periods of a year or more.

Analysis of the causes of failure to achieve perfect repairs shows the common factor of insufficiency at the commissural areas. Among 6 surviving patients, in whom significant mitral insufficiency remained or returned after baffle insertions, 5 had insufficiency at the commissures. Annuloplasty stitches were used in an attempt to correct this in 3. Seven of 8 patients with pericardial autografts had some commissural insufficiency. All had some sort of procedure to correct this. 2 of these patients had gross recurrences (one associated with bacterial endocarditis) and 4 had some return of mitral insufficiency murmurs. The commissural areas then, either because of loss of substance or because of stiffness and 'ridging', frequently presented a problem in plastic repairs. The recognized techniques for annuloplasties did not solve this problem. New techniques are currently being tried in an attempt to do so.

Another factor that seemed significant in relation to postoperative mitral insufficiency was bacterial endocarditis. There was severe mitral insufficiency in all 4 'baffle patients' and in the 1 'pericardial-autograft patient' dying of bacterial endocarditis, and mitral insufficiency developed in the 1 'prosthetic-valve patient' who had endocarditis. In the 'pericardial-autograft patient' it was thought that the endocarditis led to the breakdown of a suture line. This certainly was the case in the patient with a prosthetic valve. In the other 4 cases the mitral insufficiency preceded the development of bacterial endocarditis in at least 2 and it is hard to be sure which was cause and which was effect.

In the evaluation of post-repair mitral insufficiency it is apparent that when the surgeon recognizes that there is residual insufficiency at the time of operation, there is always clinical evidence of this postoperatively, possibly to a significant degree. Sometimes, despite an appearance of adequate correction at operation, insufficiency becomes evident later. In the cases that developed significant mitral insufficiency after repair there was generally good clinical evidence of this by 1 month after surgery. The left-atrial pressures taken during the operation invariably showed improvement even in the cases subsequently revealed as inadequately repaired.

Symptom relief has occurred in all patients not suffering complications, including 2, 1 with a baffle and 1 with a pericardial-autograft insertion, who have clinical features of significant mitral insufficiency. There is presumably a critical and quite high level of regurgitation at which symptoms develop.

Complications

The most important complications of mitral valve repairs are bacterial endocarditis and emboli.

Bacterial endocarditis has killed 5 patients with plastic repairs and 1 patient with a valve replacement. It has been associated with mitral insufficiency in all 6 cases and may have contributed to it in 4 of them. It seems rather more prone to develop on baffle repairs but no procedure is immune from it. Although infection may occur at surgery it may also develop a year or more later, presumably from bacteraemia. The outcome has been invariably fatal. It may be an argument in favour of prosthetic valves that the infection cannot attack the valve mechanism and destroy it as it can when natural or autograft valve tissue is still in place. Nevertheless the eradication of infection in the presence of foreign material remains extraordinarily difficult, although this has been reported.[12]

Emboli are particularly the problem of patients with prosthetic valves with atrial fibrillation. Laboratory experience demonstrated the great tendency to clotting induced by the presence of a prosthetic valve[10] and the need for anticoagulants to control it.[15] Clinical experience has confirmed this and has shown that in the properly anticoagulated patient in sinus rhythm the danger is slight, but that emboli do tend to occur in the patient who is fibrillating. Fortunately most of these are small and rarely either incapacitating or fatal. That the baffle technique may occasionally be complicated by emboli is shown by the 2 cases in this series. The pericardial technique both experimentally and clinically seems free of this complication.

Comparison of Techniques

In comparing the merits of the various techniques for repair of mitral insufficiency, as shown by this study, the annuloplasty operation fares very badly because of lack of durability of repair. The baffle technique is somewhat limited in application, being difficult to use effectively in cases of combined stenosis and insufficiency. Its complication rate is high, but some excellent results have been achieved with it. The pericardial-autograft techniques (particularly the inserts) are more versatile in their application, and complete haemodynamic correction is feasible by their use in virtually all cases suitable for plastic repair. However, the durability of these repairs is still uncertain. The complication rate may be less than that for other techniques. For certain valves prosthetic replacement is mandatory, but sometimes, when the precarious condition of the patient makes it essential to ensure a perfect haemodynamic result, it may be safer and quicker to replace a relatively mobile valve rather than attempt to repair it. The biggest disadvantage of prosthetic valves is the need for long-term anticoagulants. This has forced the use of plastic techniques in some patients from areas where proper anticoagulant control could not be guaranteed. The long-term durability of the prosthetic valve used in this series is still not completely established.

TABLE V. OVER-ALL STATISTICS

Patients	*Operations*	*Deaths*		*Complications*	
		Early	*Late*	*SBE*	*Emboli*
49	56	4	8	6	8

SUMMARY

Experience at the University of Cape Town Medical School with 56 open-heart operations in 49 patients with mitral valve disease is presented. There were 4 operative deaths and 8 late deaths. Bacterial endocarditis was the most serious complication and occurred 6 times. Postoperative emboli occurred in 9 patients (Table V).

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