EXPERIENCE IN 100 PATIENTS WITH STARR-EDWARDS PROSTHETIC VALVES IN THE MITRAL AND AORTIC POSITIONS

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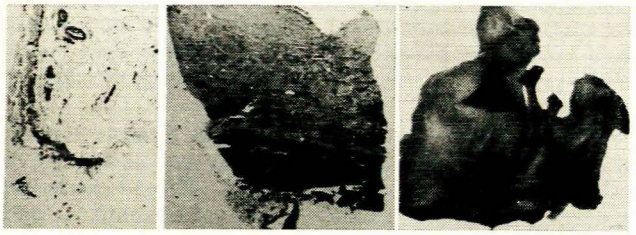
Before May 1962 48 patients underwent open-heart mitral valve surgery in Johannesburg. Different surgical techniques were used, all of which aimed at preserving the natural valve, no matter how diseased it proved to be. The results of these operations have been reported elsewhere.[1] In general the results were poor, and analysis showed that only in certain well-defined pathological states were conservative procedures effective. Mitral annuloplasty was able to control the regurgitation only when the annulus was dilated and the leaflets comparatively unaffected. Even so, the late results are often disappointing since there is a significantly high incidence of recurrent regurgitation after surgery. Lengthening of a shortened posterior leaflet by interposing a pericardial graft at its base can correct regurgitation, provided the anterior leaflet is long and mobile. Again, improvement is often temporary because of thickening and stiffening of the pericardial graft with the development of stenosis (Figs. 1a, b and c). In 5 patients with regurgitation associated with rupture of major chordae tendineae in otherwise healthy valves, chordal replacement restored competence. (Continued observation has shown satisfactory long-term results in the 4 survivors.)



(*a*) (*b*) (*c*)

Fig. 1. Changes occurring in pericardial grafts if used in the mitral position. (*a*) Section through normal pericardium. (*b*) Section through pericardium used as a graft to extend the posterior leaflet of a mitral valve 6 months after implantation. Note the thickening and cellular infiltration. (*c*) Reconstruction of posterior leaflet of mitral valve with pericardial graft. Patient developed progressive mitral stenosis owing to thickening and rigidity of the graft. Valve excised and replaced with a prosthesis 2½ years later.

The high mortality of conservative operations was largely due to failure to improve the haemodynamic state, and it was obvious that many of the valves were often so severely diseased that only replacement could hope to effect a cure. A bicuspid teflon prosthesis[2] of our own design (Fig. 2) was then used in 3 patients, but stiffening and fracture of the prosthetic leaflets occurred (Fig. 3), and its further use was discontinued. In 1960 Starr[3] reported the replacement of the mitral valve with a ball-valve prosthesis (Fig. 4). Since then this valve has been widely used, and many surgeons have demonstrated its effectiveness in relieving the disability of patients with acquired mitral and aortic valve disease.[4-9] Since August 1963 we have performed 100 Starr-Edwards valve replacements and we here relate our experience with this valve used in 60 mitral and 40 aortic operations.

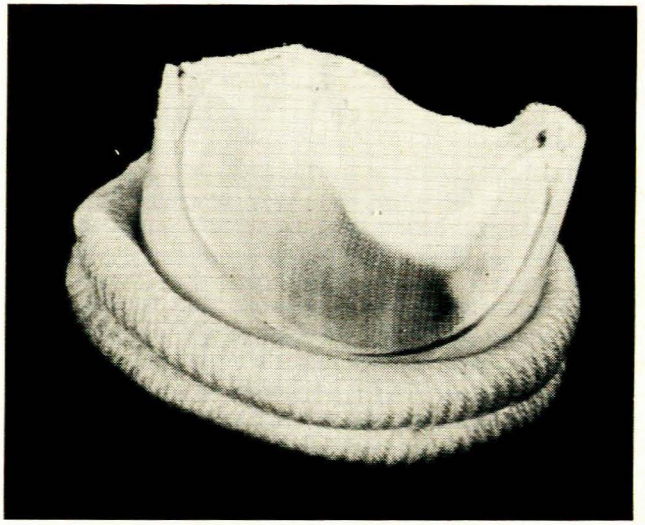


Fig. 2. Bicuspid teflon mitral valve prosthesis.

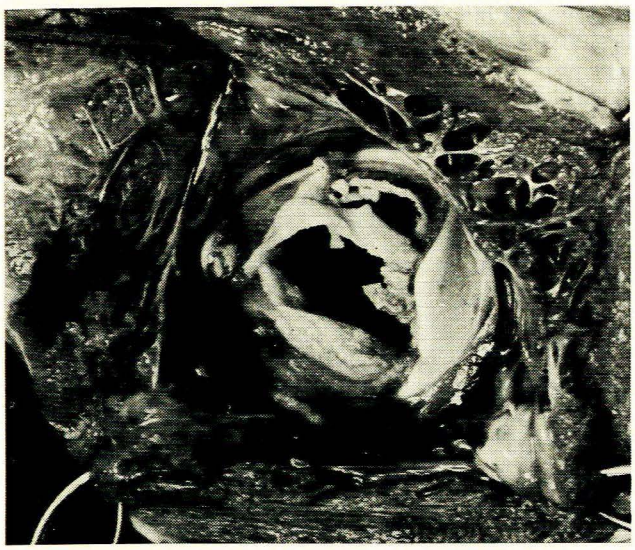


Fig. 3. Teflon prosthesis showing disintegration of a leaflet after 6 months of intracardiac function.

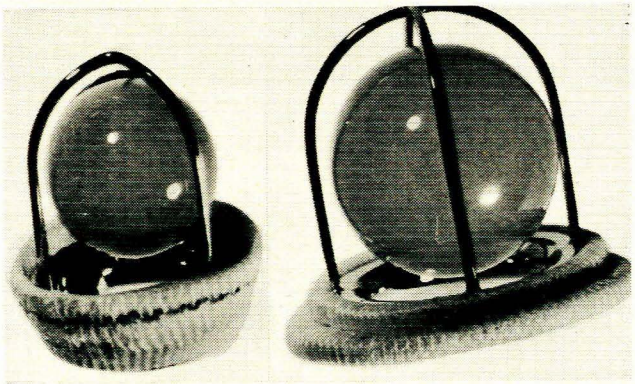


Fig. 4. Starr-Edwards mitral and aortic valve prostheses.

PRE-OPERATIVE AND OPERATIVE DATA

Table I summarizes the pre-operative data of the 60 mitral valve replacements and Table II of the 40 aortic valve replacements. Excluded from this series are 6 patients who underwent simultaneous mitral and aortic valve replacement.

TABLE I. PRE-OPERATIVE DATA IN MITRAL VALVE SERIES

No. of patients	*Age*	*Sex*	*Major lesion*	*Functional disability grading (Amer. Heart Assoc.)*	*Pressures * (mm. Hg)*		*Previous mitral surgery*
					R. vent. syst.	*M.L.A.P.*	
60	6–58 years Aver. 31·6	25 M 35 F	28 MS 32 MI	I– 0 II– 1 III–29 IV–30	53	20	25 (42%) 8 had two or more valvotomies

*Averages in 30 patients with pre-operative catheterization data.

The average age of the patients in the mitral series was 31·6 years. Thirty-five were females and 25 were males. Combined mitral stenosis and regurgitation was usually present, but regurgitation was the dominant lesion in 32 and stenosis in 28 patients. All but one of the patients were severely disabled. Thirty patients were catheterized pre-operatively. The average right ventricular peak systolic pressure was 53 mm.Hg, and the average mean left atrial pressure was 20 mm.Hg. Twenty-five patients (42%) had had previous surgery; in 8 (13%) two or more valvotomies had been performed.

TABLE II. PRE-OPERATIVE DATA IN AORTIC VALVE SERIES

No. of patients	*Age*	*Sex*	*Major lesion*	*Functional disability grading (Amer. Heart Assoc.)*	*Pressures (mm. Hg)*		*Associated mitral stenosis*
					Average LV aorta gradient *	*L.V.E.D.* †	
40	16–58 Aver. 38	M 25 F 15	AS 19 AI 21	I– 0 II– 0 III–24 IV–16	55	11	14 (35%)

*Average in 11 patients with pre-operative catheter data where AS was dominant lesion.

†Average in 22 patients with pre-operative catheter data.

The average age of the aortic patients was 38 years; males predominated (25:15). Again the lesion was usually a mixed one, but the major lesion was stenosis in 19 and regurgitation in 21. All were seriously disabled. Pre-operative catheterization was performed in 11 patients with dominant stenosis, and the average gradient across the valve was 55 mm.Hg. In 22 patients the average left ventricular end diastolic pressure was 11 mm.Hg. Fourteen of the 60 patients (35%) also had significant mitral stenosis.

In 46 of the 60 mitral valve replacements, the heart was exposed through a left thoracotomy, but in 14 who had a previous left thoracotomy a right approach was used. The average duration of cardio-pulmonary bypass was 66 minutes, and the valve size most commonly used was a 4M Starr-Edwards prosthesis. In 31 (52%) postoperative mechanical ventilation was necessary.

TABLE III. OPERATIVE DATA IN MITRAL AND AORTIC VALVE REPLACEMENT

Approach	*Additional procedure performed*	*Average perfusion time (min.)*	*Coronary perfusion average (ml./min.)*	*Valve size used*	*Postoperative mechanical ventilation*
Mitral L 46 60 R 14	—	66	—	2M– 1 3M–20 4M–39	31 (52%)
Aortic All 40 V.S.P.	15 (37%) Mitral valvotomy	86	700 ml. Average line pres. 200 mm. Hg	9A–18 10A–12 11A– 6 12A– 4	4 (10%)

For aortic valve replacement the heart was always exposed through a vertical-sternal splitting incision. In 15 of the 40 patients a closed transventricular mitral valvotomy was performed before replacing the aortic valve. Average perfusion time was 86 minutes. Coronary artery perfusion averaged 700 ml./min. with line pressures seldom exceeding 200 mm.Hg. The size 9A Starr-Edwards prosthesis was used in 18 patients, the size 10A in 12 patients and the larger valves (sizes 11A and 12A) in 10 patients. In contrast to the mitral valve series, mechanical ventilation was necessary in only 4 patients.

RESULTS

Seventy-three of the 100 patients have survived. The longest follow-up is 2 years. Table IV summarizes the results.

TABLE IV. RESULTS IN MITRAL AND AORTIC VALVE REPLACEMENT

	No. of patients	*Hospital deaths*	*Late deaths*	*Total*	*Longest follow-up*
Mitral ..	60	16 (26%)	4 (7%)	20 (33%)	23 months
Aortic ..	40	3 (7%)	4 (10%)	7 (17%)	24 months
Total	100	19	8	27	

Hospital Deaths

There were 16 hospital deaths among the 60 patients whose mitral valves were replaced. Nine died during or

shortly after surgery because of myocardial failure. Seven of these were in the functional disability grade IV group, while 8 were over the age of 40 years. In 5 previous mitral valve surgery had been performed.

Two patients died of massive pulmonary embolism 10 days and 6 weeks after surgery. Two late deaths were due to haemorrhage, one from the gastro-intestinal tract at 20 days, the other from a secondary intra-thoracic bleed on the 14th day.

Subacute bacterial endocarditis caused 2 hospital deaths in the mitral series. In the first, friable thrombus was dislodged from the left atrium when palpating the valve before cardio-pulmonary bypass. This resulted in severe cerebral involvement, and for 4 months mechanical ventilation was required. Urinary and pulmonary infection supervened. At postmortem examination exuberant infected thrombus was present on the prosthesis sufficient to obstruct the flow through the valve (Figs. 5 and 6). The

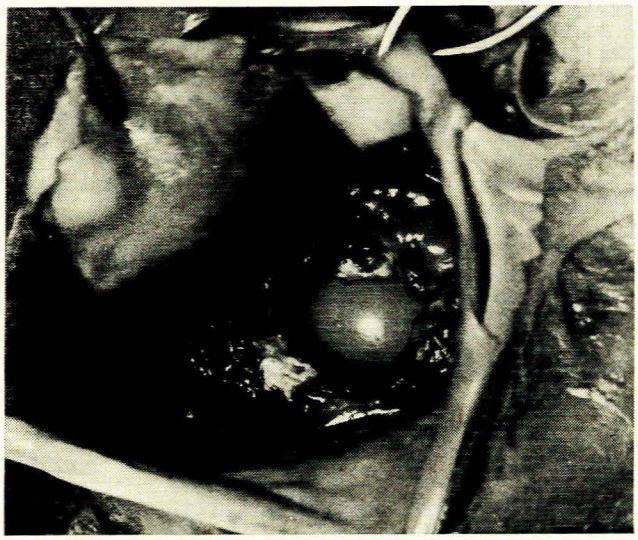


Fig. 5. Thrombus formed on prosthetic valve owing to subacute bacterial endocarditis. The atrial aspect shows thrombus partially obstructing orifice of valve.

second patient developed an early empyema followed by the signs of bacterial endocarditis. Despite energetic antibiotic therapy he died one month later. Autopsy confirmed the diagnosis.

A sudden death which occurred 2 months after operation was probably due to an acute arrhythmia. The only abnormality found at necropsy was a small area of haemorrhage on the left side of the interventricular septum closely related to one of the struts of the prosthetic valve cage (Fig. 7).

Three of the aortic valve patients died in hospital. One death was due to the surgical error of inserting too large a prosthesis (9A) into a narrow aortic root. He died on the third day during an attack of severe angina pectoris. Autopsy showed that the prosthetic annulus was obstructing the orifice of the left coronary artery. The other 2 patients died of haemorrhage. In the first, ventricular fibrillation occurred while dissecting the dense adhesions of a previous operation. In attempting to free the heart rapidly so as to institute cardiac massage, the ventricle was torn and the blood loss was uncontrollable. The other patient died suddenly and unexpectedly a week after surgery. At necropsy a large left ventricular sub-endocardial haemorrhage was found to be due to retrograde dissection of blood from the site of the prosthesis.

Fig. 6. Ventricular aspect of prosthetic valve. Infected clot has formed on the metal struts of the valve.

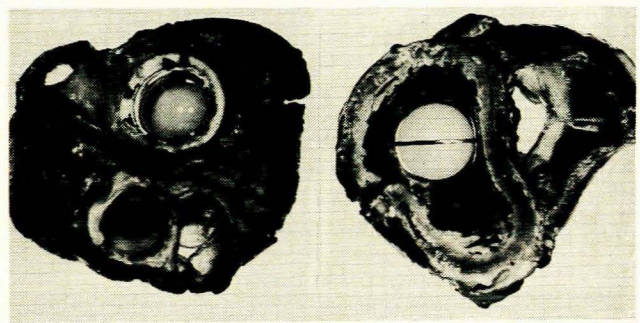


Fig. 7. Appearance of Starr-Edwards valve 2 months after insertion. There is no thrombus formation, and tissue ingrowth has started into the cloth of the prosthetic ring.

Late Deaths

There were 3 late deaths among the mitral group. The first occurred exactly a year after operation. On the day of death this young man played a strenuous game of rugby and collapsed at home a few hours later. An exhaustive necropsy examination failed to show evidence of mechanical valve failure or other cause of death. It is possible that an arrhythmia was responsible. Three patients died of massive systemic arterial embolization 10, 11 and 12 months after surgery. All 3 patients were on anticoagulation therapy and were symptomatically well. Necropsy was refused in all these patients.

There were 4 late deaths after aortic valve replacement. Three died of subacute bacterial endocarditis, 3, 4 and 5 months after operation, despite intensive antibiotic therapy.

Staphylococcus albus was the organism cultured from the blood in all of them. A fourth death occurred suddenly 3 months after operation. No information was available regarding the mode of death, and a postmortem examination was refused. He was known to have coronary artery disease, and it is likely that death resulted from coronary thrombosis.

Follow-up

Our patients are seen regularly, but only a few postoperative catheterizations have been done. Extensive postoperative haemodynamic studies have been undertaken in other centres where the Starr-Edwards valve is used.[8,11,12] These have shown that both the aortic and mitral prosthetic valves are haemodynamically satisfactory. Table V summarizes the follow-up data of our patients.

TABLE V. MITRAL AND AORTIC REPLACEMENT: FOLLOW-UP OF PATIENTS

	No. of patients	*Improved*	*Unchanged*	*Worse*	*Residual lesion*	*Emboli*		*Anticoagul.*
						Transient	*Permanent*	
Mitral	40	39	0	1	0	5	2	32
Aortic	33	33	0	0	5	4	0	25
Total	73	72	0	1	5	9	2	57

In the 73 survivors late cerebral embolization has occurred in 11. Nine of the 11 incidents have been transient with full recovery within minutes to a few days. Two mitral patients had cerebral emboli 5 and 8 months after surgery which have resulted in permanent neurological disturbance. Eight of the 11 were being maintained on anticoagulation therapy. Six of the 7 mitral patients were in atrial fibrillation when the emboli occurred. Two of the patients with aortic prostheses have mild regurgitation owing to ring leaks. A third aortic patient, also fibrillating at the time of embolization, had a heavily calcified stenotic mitral valve opened at the time of valve replacement.

Five aortic patients have signs of aortic regurgitation. In 3 this is insignificant; only a soft early diastolic murmur being present at the left sternal border. One patient has a widened pulse pressure, but despite this he has improved remarkably and the heart has reduced in size. The fifth patient had more severe regurgitation with anaemia which was recurrent despite blood transfusion and iron therapy. Re-operation was therefore necessary and the prosthesis was replaced. He is now asymptomatic. In 3 of these 5 patients one of the silk sutures broke on tying. In the other 2 massive calcification of the aortic annulus was present, and despite all attempts to remove as much calcium as possible, the valves did not seat satisfactorily.

Seventy-two of the 73 patients are markedly improved and reduction in the heart size has resulted. Most have returned to full activity. The one poor result is the patient with hemiplegia following a massive cerebral embolism.

DISCUSSION

Most of the deaths occurring in the mitral group were due to myocardial failure which at present is unpreventable and often unpredictable. This happened in 8 patients, but never after aortic valve replacement. The functional disability grades of both groups were similar, and rheumatic valvulitis was the pathology in the vast majority in both groups. Possible explanations for the differing incidence of myocardial failure are as follows:

Mitral patients with valve lesions of sufficient severity to warrant valve replacement have usually suffered from a more protracted illness than those with aortic valve disease and consequently have suffered from myocardial strain for a longer time. Also, pulmonary hypertension with right heart strain is characteristic of mitral rather than of aortic valve disease, so that generalized myocardial damage is most severe in mitral cases. When the mitral valve is replaced, two-thirds of the base of the left ventricle is splintered by the rigid prosthesis, while prosthetic aortic replacement only interferes with the myocardium over a small area of the interventricular septum.[13] Also, in mitral valve replacement the papillary muscles are excised, and this may interfere with left ventricular function because of the loss of an anchoring mechanism between the left ventricular myocardium and the base of the heart.[14]

Respiratory support with mechanical ventilation during the postoperative phase was more frequently necessary in the mitral series (52% : 10%). Pulmonary vascular disease and a large heart which encroaches on damaged lungs is common in severe mitral disease. In such conditions the addition of a thoracotomy can easily precipitate respiratory failure. Another reason for the higher incidence of mechanical ventilation is the greater frequency of cerebral embolism during mitral valve surgery. This occurred in 11 of the 60 patients in the mitral series. (In 10 this was due to air and 9 patients recovered completely.)

Systemic arterial embolization or thrombosis of the prosthetic valve occurred in 14 patients. Ten were in the mitral series and 4 in the aortic group. Serious emboli have only occurred in the mitral patients. The embolic phenomena which occurred in the aortic group were transient, and in 3 of the patients a prosthetic ring leak was present which may have been responsible for thrombus formation. Embolization is therefore not a serious problem in aortic valve replacement but is a distinct threat to patients with prosthetic mitral valves. Most patients were under anticoagulation therapy at the time the emboli occurred, though 2 were Bantu patients in whom, because of practical difficulties, postoperative anticoagulation is not practised. It seems that anticoagulation therapy has little influence in preventing clot formation on prosthetic valves. A worrying feature has been that embolic episodes occur at any time after valve replacement (5 months to 13 months), and more than one episode has occurred in the same patient. Valves of various designs are now being used (Fig. 8), and extensive investigations are being done to improve them. It is thus likely that in time better prostheses may become available.

In all 3 aortic patients with bacterial endocarditis a *Staphylococcus albus* was the causative organism and was probably introduced at the time of surgery. With more rigid theatre discipline and adherence to strict aseptic precautions, combined with more effective pre- and postoperative antibiotic coverage, this complication has not recently occurred and it is hoped that in future it may be virtually eliminated.

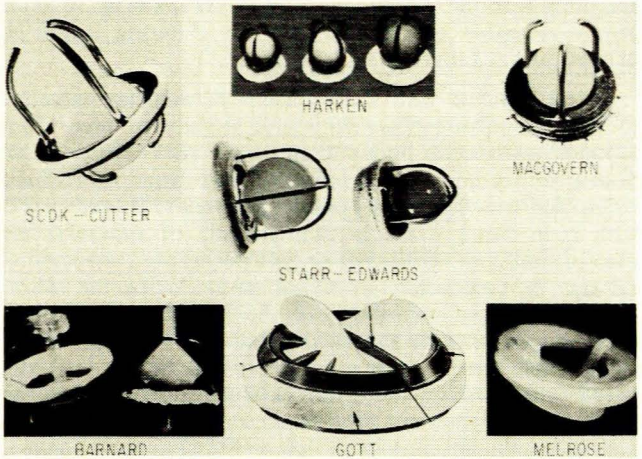


Fig. 8. Prosthetic valves of different designs currently being used for mitral or aortic valve replacement.

SUMMARY AND CONCLUSIONS

The results of 100 Starr-Edwards valve replacements are presented. Sixty were mitral operations and 40 were aortic replacements. There were 16 hospital deaths and 4 late deaths in the mitral series and 3 hospital deaths and 4 late deaths in the aortic group. The longest follow-up is 2 years.

The Starr-Edwards prostheses in both aortic and mitral positions have given gratifying haemodynamic and clinical improvement in almost all the surviving patients. The advantage of immediate absolute correction of the lesion must, however, be balanced against possible future hazards to a patient totally dependent on such a valve. Although failure or dysfunction of these valves has not occurred in this series, certain serious late complications have arisen.

Late emboli remain the most serious hazard, and it may be that particular subjects have a tendency to clot formation. Unfortunately there is no way yet of identifying such patients. To add to this anxiety the danger of embolization does not decrease with the passage of time. They may occur weeks or even years after surgery, and multiple episodes can occur in the same patient. Infection on the valve is a fatal complication, but with improved therapy this may become less frequent.

Prosthetic valve replacement should be reserved for patients with serious disability until such time as the problem of embolization is solved either by the development of better valves or as a result of a fuller understanding of the clotting mechanism in man.

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