COMPLETE REPLACEMENT OF THE MITRAL VALVE

C. N. BARNARD, M.D., M.MED., M.S., PH.D.; V. SCHRIRE, M.SC., PH.D., M.B., F.R.C.P.E., F.R.C.P.; C. C. GOOSEN, A.S.C.T.; and L. V. HOLMGREN, A.I.S.T.

Departments of Surgery and Medicine, University of Cape Town, Council for Scientific and Industrial Research Cardio-Pulmonary Research Group and the Cardiac Clinic, Groote Schuur Hospital, Cape Town

The mitral valve consists of an annulus or ring, two leaflets, chordae and papillary muscles. Chiechi *et al.*,[1] Brock,[2] Bailey and Bolton,[3] Harken and Block,[4] Little,[5] and van der Spuy,[6] have studied and described the function of each of these components.

However, for practical purposes, the normal mitral valve is a flap-type valve.[7-9] The freely mobile anterior leaflet attached to the anterior portion of the annulus and restrained by its chordae and papillary muscles, acts as the flap; the shorter, relatively immobile posterior leaflet, attached to the posterior portion of the ring, with its chordae and papillary muscles, provides the shelf or ridge against which the flap is forced closed during ventricular systole (Fig. 1).

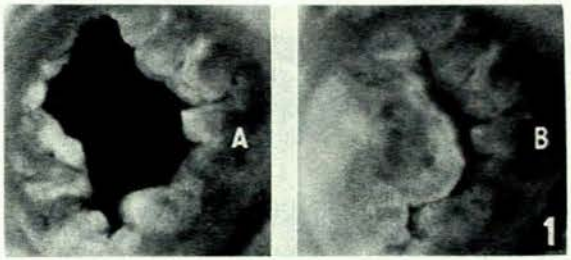
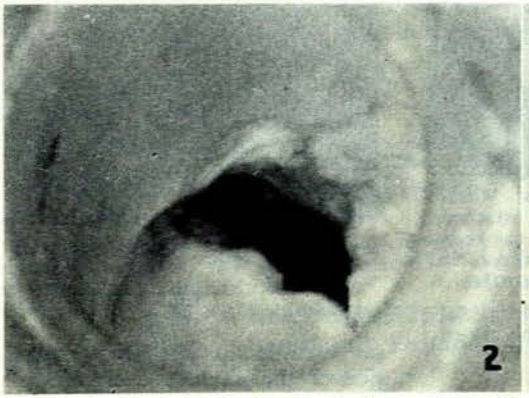


Fig. 1. Normal mitral valve (*A*) in the open position and (*B*) in the closed position. Note the freely mobile anterior cusp which closes like a flap against the much narrower posterior leaflet.

It follows that normal mitral function requires (*a*) an intact flap, (*b*) a freely mobile flap, and (*c*) an adequate posterior shelf, and mitral valve dysfunction will result from a derangement of one or more of these components. Immobility of the flap, owing to commissural fusion, will result in stenosis (Fig. 2). Deficiency of the posterior shelf results in an incompetent valve (Fig. 3).

A combination of these two factors will result in a mixed lesion, i.e. a stenosed and incompetent valve.

At open-heart surgery, reasonable valve function can be restored in most patients by plastic procedures such as mobilizing the flap, building a posterior shelf, or a combination of these two procedures. Such repairs, however, usually are palliative only, since stenosis or incompetence often recur. Furthermore, in a small percentage of cases repeated disease has disturbed the valvular function so extensively that plastic procedures cannot restore it, even temporarily. Thus, there appears to be a definite place for a reliable prosthesis. Such a valve would



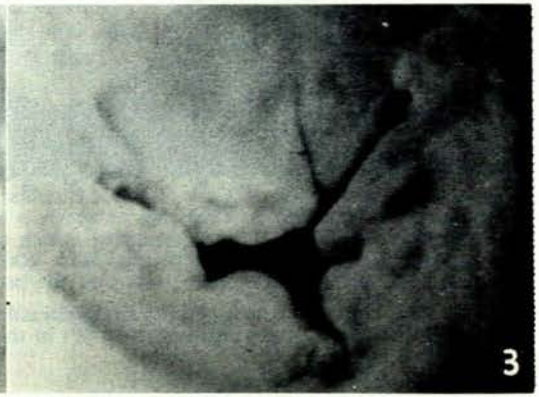


Fig. 2. A stenosed mitral valve in the open position. Note how the fusion of the anterior and posterior commissures prevents the flap (the anterior leaflet) from opening freely.

Fig. 3. An incompetent mitral valve in the closed position. Note how the destruction of the posterior leaflet removes the shelf against which the flap closes, resulting in incompetence.

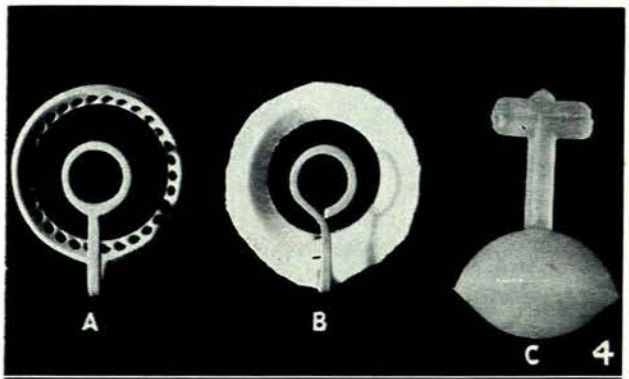
have the advantage that function could be completely restored, no matter how extensive the disease, and restenosis or recurrence of the incompetence would not occur. Cure would be substituted for palliation.

In this communication we report the first 6 consecutive patients without mortality in whom complete replacement of the mitral valve with a new prosthetic valve has produced dramatic improvement with good function for periods extending up to five months. These patients were selected because the disease was advanced and death imminent.

VALVE DESIGN AND INSERTION

Design

The University of Cape Town Lenticular Mitral Prosthesis consists of a stainless-steel ring to which is attached a suspension bar (Fig. 4A). The ring and bar are coated with 'teflon'. The ring is pierced with many holes and, before insertion, is covered with compressed polyvinyl sponge (Fig. 4B). This allows the ring to be stitched into the mitral orifice at operation. The mobile segment of the valve is made from 'silastic',* which is a plastic rubber combination. It consists of a ball in the shape of a lens, a stem and a cross-bar (Fig. 4C).



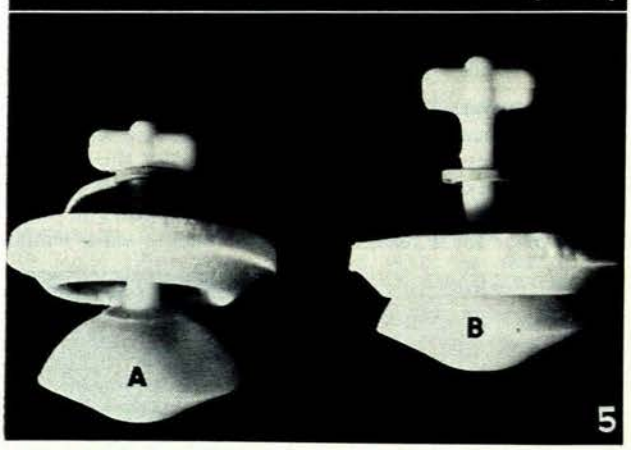


Fig. 4. The University of Cape Town Lenticular Mitral Prosthesis. *A*. The stainless-steel ring to which is attached a suspension bar; *B*. the stainless-steel ring, which has been pierced with many holes and covered with compressed polyvinyl sponge; *C*. the mobile portion of the prosthesis.

Fig. 5. The University of Cape Town Lenticular Mitral Prosthesis, (*A*) in the open position, and (*B*) in the closed position.

When assembled, the cross-bar is passed through the ring of the suspension-bar. In the open position (Fig. 5A) the ball hangs about $\frac{1}{4}$ inch below the ring, suspended by its cross-bar on the arm of the ring. In the closed position (Fig. 5B) the ball is guided by the stem into the ring, to close the opening.

Insertion

Under general anaesthesia, the left side of the heart is approached through a left anterolateral thoracotomy at the level of the bed of the 5th rib. The pericardium is opened exposing the left atrium, left ventricle, right ventricle and right atrial appendage. The mitral valve is now explored *via* the left atrial appendage to determine the extent of the disease and to ascertain whether complete replacement of the valve will be necessary. As soon as this has been decided, the patient is connected to the oxygenator, the venous blood being drained by a single catheter placed in the right atrium through the right atrial appendage, and the arterial blood being returned from the oxygenator through a catheter placed in the left common femoral artery.

In all the patients extracorporeal circulation, maintained by the helix reservoir bubble oxygenator with profound hypothermia, has been used.

As soon as hypothermic failure of the heart occurs, the left atrium is opened and the mitral valve is exposed. The anterior and posterior leaflets of the valve are excised with their chordae and papillary muscles. A prosthesis of suitable size (five sizes are available) is selected, and the ring of the prosthesis is securely sutured to the ventricular surface of the mitral annulus with 12 - 15 interrupted 'O' silk mattress sutures. These sutures are tied on the atrial side of the annulus over a ring of compressed 'ivalon' (Fig. 6).

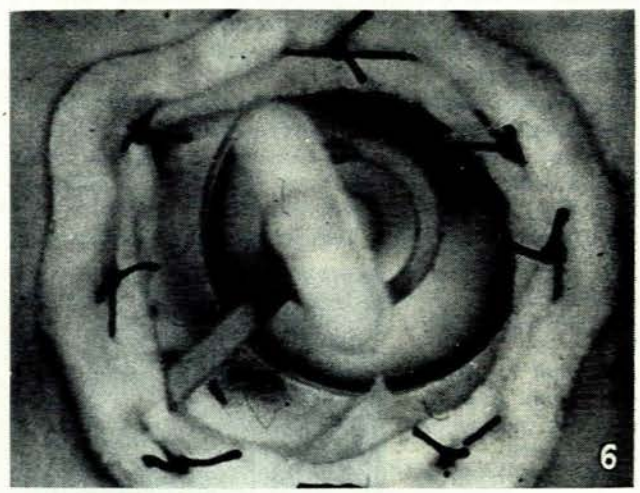


Fig. 6. The mitral prosthesis sutured in position on the ventricular side of the annulus, viewed from the atrium. Note how the sutures are tied over a ring of compressed ivalon.

Rewarming is started as soon as the valve is in position, and the left side of the heart is allowed to fill with blood by keeping the ball of the prosthesis in the open position.

* Manufactured by Dow Corning Corp., Midland, Michigan, USA.

When the heart beat has resumed and all air has been dispelled from the left ventricle, the left atrium is allowed to fill with blood and the atriotomy is closed. Bypass is discontinued and the patient is disconnected from the heart-lung machine. The postoperative care is the same as for patients undergoing open-heart surgery except that anticoagulants are started on the fourth postoperative day and continued indefinitely, and antibiotics are continued for at least 3 weeks after surgery.

CASE HISTORIES

Case 1

J.C., a Coloured male messenger of 29, suffered from rheumatic fever in his youth, followed by asymptomatic heart disease until 1955. Effort dyspnoea then developed, followed by cough, haemoptysis, paroxysmal cardiac dyspnoea, orthopnoea and marked limitation of effort tolerance until he was almost incapacitated. On examination, atrial fibrillation was present, but no frank congestive cardiac failure. The physical signs were those of advanced mitral stenosis and incompetence; this was confirmed by cardiac catheterization and angiocardiography. Mild aortic incompetence was also present.

Because of the gross distortion and destruction of the valve cusps, prosthetic valve replacement was required. No attempt was made to repair the aortic valve. The postoperative course was highly satisfactory, with striking recovery of cardiac function and complete disappearance of symptoms. Five months after the operation the patient was re-investigated and left-heart catheterization was performed. After this procedure he developed an abnormal temperature. The anticoagulant therapy was uncontrolled. Two weeks later he developed a sudden right hemiplegia from which he improved rapidly.

Case 2

W.B., a White female of 40, suffered from recurrent rheumatic fever as a child; at the age of 30 gross symptoms of effort dyspnoea, paroxysmal nocturnal dyspnoea and severe disability had developed, so that mitral valvotomy was required, with improvement for six years. Thereafter, her symptoms all recurred until she was completely disabled despite continuous treatment. Examination revealed sinus rhythm and the signs of severe mixed mitral stenosis and incompetence, confirmed by cardiac catheterization and angiocardiography.

Replacement of the mitral valve resulted in complete disappearance of her symptoms. The patient has returned to full activity and is well four months after surgery.

Case 3

S.R., a White male of 35, had rheumatic fever at the age of 12 and lived a somewhat restricted life until the age of 26. Progressive effort dyspnoea then developed culminating in paroxysmal nocturnal dyspnoea and congestive cardiac failure, not controlled by cardiac treatment. Cough and haemoptysis added to his difficulties. On examination he had atrial fibrillation and the signs of severe mixed mitral stenosis and incompetence and aortic stenosis and incompetence, confirmed by cardiac catheterization and angiocardiography.

At operation the grossly distorted mitral valves were excised and replaced by the prosthetic valve. Calcium was removed from the aortic valve leaflet and a Bahnson cusp inserted into the non-coronary cusp. The postoperative course was extremely stormy with renal shut-down, and cerebral and hepatic involvement. After a long protracted convalescence in hospital, he gradually recovered. The improvement in his haemodynamic state was striking and four months after operation he had a normal effort tolerance and was symptom-free.

Case 4

J.J., a Coloured female of 45, had her first mitral valvotomy by finger fracture in 1955 for severe symptoms caused by critical mitral stenosis. Freedom from symptoms followed until 1957, despite an inadequate opening, when all her complaints gradually recurred, culminating in paroxysmal nocturnal dyspnoea and severe disability. Re-valvotomy was performed on 5 December 1960, the mitral valve having restenosed to the minimal size of less than 1 sq. cm. found at the first operation. A Tubb's dilator was inserted through the left ventricle and the lateral commissure was split, the split extending into the anterior cusp, producing gross regurgitation. Intractable congestive cardiac failure followed, requiring constant hospitalization.

On 23 January 1961 mitral valvoplasty was attempted, using cardiac bypass, the tear in the anteromedial leaflet of the mitral valve being sutured and the commissures split, with some initial improvement in the patient's condition. During the following 18 months she deteriorated and spent most of her time in hospital with chronic congestive cardiac failure from mitral and tricuspid incompetence. Advanced right heart failure resulted in gross oedema, recurrent ascites requiring repeated paracenteses, and severe fluctuating jaundice. There was no response to intensive treatment with digitalis, diuretics, spirolactone and salt restriction.

On 14 June 1962, after three months' intensive therapy, during which the jaundice subsided, the mitral valve was excised and replaced, and annuloplasty of the tricuspid valve, which had become stretched by the congestive cardiac failure, was performed. The postoperative course was surprisingly smooth, considering the grave condition of the patient before operation. The ascites cleared rapidly and her cardiac status improved remarkably. She was finally discharged with signs of moderate tricuspid valve disease only. Four months after operation she was back to normal activity and symptom-free, though oral cardiac therapy was still required.

Case 5

J.F., a White female aged 45, suffered from several attacks of rheumatic fever between the ages of 20 and 30. Moderate exertional dyspnoea led to an exploratory mitral valve operation elsewhere, at the age of 35, when she was found to have too much incompetence for closed surgery. Thereafter her condition deteriorated progressively, cardiac failure with tricuspid incompetence and cardiomegaly developing, and radioactive iodine being used to control her condition.

Pregnancy in 1955 (age 38) was interrupted and a hysterectomy performed, following which she developed pulmonary embolism. Inferior vena caval ligation was performed with temporary benefit. The following year she developed subacute bacterial endocarditis for which she was successfully treated. Investigation in the Cardiac Clinic at Groote Schuur Hospital in 1958 showed gross mitral incompetence, atrial fibrillation and an aneurysm of the left atrium, retrograde aortic angiocardiography excluding aortic incompetence.

Several months later she developed a sudden right-sided hemiplegia with dysphasia. Before operation total cardiac and neurological incapacity was present, and she suffered from severe pain in the right chest, owing to the giant left atrium. Repair of the mitral valve under cardiac bypass was attempted on 4 November 1959, severe mitral incompetence being present, corrected by means of an ivalon baffle as described elsewhere.[5] After a stormy postoperative course, during which she required tracheostomy and assisted respiration, she was eventually discharged with more residual neurological than cardiac disability. The pain in the chest disappeared and she improved so much that she was able to go on an overseas trip, remaining well for a year.

The symptoms then gradually recurred, until she was incapacitated with uncontrollable heart failure, severe right-sided chest pain, haematuria (attributed to renal embolism) and persistent haemoptyses (attributed to recurrent pulmonary embolism), despite long-term anticoagulant therapy. The signs were those of gross mitral and tricuspid incompetence, atrial fibrillation and a giant left atrium. Neurologically she had improved considerably.

Mitral valve replacement was performed on 13 September 1962, the patient being discharged after four weeks much improved and free of pain.

Case 6

J.D., a White male of 39, had his first mitral valvotomy performed in 1957 for severe symptoms caused by tight mitral

stenosis. A heavily calcified valve was noted at this time. Marked relief followed for four years. An intercurrent pulmonary infection then precipitated atrial fibrillation followed by recurrence of his symptoms.

Within a year his activity was markedly limited and he continued having attacks of paroxysmal cardiac dyspnoea while in hospital under full treatment. On examination, atrial fibrillation, jugular venous distension and hepatomegaly were present with the signs of pure mitral stenosis, severe pulmonary hypertension and tricuspid incompetence; heavily calcified mitral valves were seen on screening.

At operation on 1 October 1962 nothing could be done to the valves by closed valvotomy or under direct vision, prosthetic valve replacement being necessary. There has been marked improvement in haemodynamic function since the operation.

SUMMARY AND CONCLUSIONS

Disordered function of the mitral valves, usually rheumatic, is the commonest cause of valvular disease of the heart. Better understanding of normal and abnormal mitral valve function has resulted in the development of new surgical techniques designed to repair the haemodynamic disturbances produced by disease.

Probably the commonest sequel of rheumatic valvulitis is mitral stenosis, which can be readily relieved by closed mitral valvotomy. When mitral incompetence is present, however, either in pure form or combined with stenosis, cardiac bypass is required. The valves can usually be mobilized, freed and, if necessary, lengthened or buttressed by means of autogenous tissue, e.g. pericardium or some sort of plastic material. A small but appreciable number of patients, however, have such disturbed function that the valves cannot be adequately opened or mobilized even under direct vision. Function can only be restored by complete excision of the valves and replacement by a prosthesis.

The tendency for re-stenosis or recurrence of incompetence is ever-present. A prosthetic valve would have the great advantage of producing cure rather than palliation. However, two exacting requirements must be fulfilled before such a prosthesis can be substituted for the patient's own valve. The prosthesis must open and shut competently many millions of times over a period of many years and clots must not form on the valve.

In this communication we present preliminary results on a new prosthetic device which has been tried in 6 desperately ill patients with no operative mortality. Return to normal cardiac function has been truly remarkable up to a five-month period of observation. One patient, however, has developed a hemiplegia five months after operation presumably owing to an embolus. This danger is always present in all patients with rheumatic mitral valve disease and atrial fibrillation, even when having anticoagulants. Further study will be required before the long-term value of this prosthesis can be assessed.

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