Follow-up of Starr-Edwards Mitral Valve Replacements in Children


SUMMARY

This article discusses 34 children between the ages of 6 and 16 years with a Starr-Edwards mitral valve replacement, who have been followed-up over a period of 8 months to 8 years. The principal causes of late deaths were subacute bacterial endocarditis, and embolic episodes. The main complication was recurrent rheumatic fever affecting the aortic or tricuspid valves. Valve dysfunction was not a major problem, and an adult-size valve could usually be inserted.

Of the 29 survivors, 14 are markedly improved. For the reasons mentioned, 10 remain cardiac grade II, while 3 are still significantly incapacitated. From this experience we conclude that when a Starr-Edwards valve replacement is indicated, 70% of children will have good long-term palliation.


It is now more than a decade that the Starr-Edwards ball valve prosthesis has been in use for mitral valve replacement. Recently several reports regarding the long-term follow-up of these observations mainly concern adults. The prevalence in Bantu children of relentless rheumatic mitral regurgitation, refractory to medical treatment, has been previously reported by us.

We are thus in the unique position of having a significant series of children in whom Starr-Edwards valves have been inserted.

Although it is still the policy of the Johannesburg Department of Thoracic Surgery to conserve these valves whenever possible, there is an appreciable number of these children in whom the valve mechanism is completely destroyed, and there is no alternative but a complete replacement. We have consistently used the Starr-Edwards prosthesis except for a short period when mounted tissue valves were inserted. Because of early failure their further use was discontinued and we reverted back to the Starr-Edwards valve. This is a report of our experience with the long-term follow-up in these children.

MATERIAL

The series of 48 children who underwent mitral valve replacement with a Starr-Edwards prosthesis is shown in Fig. 1. In 47, the lesion was due to rheumatic fever, but in 1, severe regurgitation resulted from the repair of a complete form of atrioventricular canal. There were 12 hospital deaths; 2 were lost to study. The remaining 34 children have been followed-up carefully and form the basis of this report.

The age distribution, race and sex in these remaining 34 patients are shown in Figs 2a and 2b. The average age was 11.8 years. Twenty-four were Bantu, 6 Coloured and 4 White. Thirteen were males and 21 females. A preponderance of Bantu, and females, is again observed.
Pre-operative functional grading was done on the basis of the New York Heart Association classification. Thirty-three were cardiac grade IV and only 1 a cardiac grade III (Fig. 3). They were all, therefore, severely incapacitated. Cardiac failure was controlled only by intensive medical treatment, and usually this had to be maintained up to the time of surgery. The average cardio-thoracic ratio was 68%, and in 13 who underwent pre-operative catheterization, the average right ventricular pressure was 54 mmHg and the average mean left atrial pressure 21 mmHg.

Surgery was performed with the standard cardiopulmonary bypass and the approach was through either a left or right thoracotomy, or via a sternum-splitting incision. This was determined by any previous heart surgery and on the preference of the surgeon. The size and model of the Starr-Edwards valve used is shown in Fig. 4.

In 14 children a valve with a Silastic ball and uncovered struts (model 6000 and 6120) was used, while in the rest the new model (6310 and 6320) with a metal ball and covered struts, was inserted. It is interesting to note that the majority of hearts accommodated a No. 3M, and in 5 a No. 4M valve was inserted.
RESULTS

Late Deaths

There were 5 late deaths in our 34 patients. The causes and time of death are shown in Fig. 6. Two died of subacute bacterial endocarditis, 9 months after leaving hospital. One developed this on an old type of valve and the other on a cloth-covered prosthesis. One patient had a fatal massive cerebral embolism 2 years after surgery. A fourth patient had recurrent rheumatic fever with subsequent tricuspid regurgitation, and died of a pulmonary embolus in the 4th postoperative year. The fifth patient had numerous recurrent episodes of rheumatic fever, and in the 8th postoperative year was admitted to hospital in frank left-ventricular failure, secondary to overwhelming aortic regurgitation. Unfortunately he died before surgery could be effected.

Complications

The complications encountered in this group are shown in Fig. 7. Nineteen of the 34 showed one or other problem.

Recurrent rheumatic fever occurred in 8 (21%) of the children and caused death in 1. The remaining patients have all developed aortic valve dysfunction, mainly aortic regurgitation. At present, however, the lesions are not haemodynamically significant. Most children received prophylactic antibiotics after surgery, but many of the
Bantu children failed to return for regular observation, and so antibiotic cover was inadequate.

Subacute bacterial endocarditis developed in 4 of our patients, and was fatal in 2. In 1, with vigorous antibiotic therapy, this was cured, but a perivalvular leak remained. She has been operated on again, and at present, despite mild cardiac failure, there is nothing to suggest a continuation of the endocarditis. Another patient is still in hospital and at the time of writing is receiving intensive antibiotic therapy.

Thrombo-embolism was observed in 5 patients, an incidence of 15%. This is not dissimilar to what has been reported by other authors. In 4, these emboli were cerebral events developing 2 - 3 years after surgery. One patient died, 1 is left with a hemiplegia, the third has mild residual hemiparesis, but the fourth has fully recovered. In 1, a fatal pulmonary embolism occurred 4 years after surgery.

Valve dysfunction occurred in 3 patients. In the first, 7 years after surgery, signs of ball variance appeared. With catheterization, a significant diastolic gradient was found across the valve. At surgery, obstruction was found to be due to uniform swelling of the Silastic ball, which notably reduced the effective valve orifice (Figs 8a, 8b and 8c).

Two other patients remain in mild failure and show little reduction in heart size. Some obstruction to flow through the valve is present and 1 patient, recatheterized,
showed an elevated pulmonary wedge pressure. In both these cloth-covered model 6320 valves were inserted. Haemolysis has been seen in only 1 patient. It remains mild and the child is easily controlled by oral iron therapy. A perivalvular leak due to subacute bacterial endocarditis has been seen in the 1 patient already mentioned. She was re-operated upon and the lesion corrected.

**Functional Status**

The functional status in the remaining 29 patients is summarized in Fig. 9. One patient deteriorated to a cardiac grade IV, 7 years after surgery, due to valvular dysfunction. As mentioned, she was re-operated upon and at the last visit she was thought to be a cardiac grade II. Four patients remain cardiac grade III. In 1.

**CONCLUSIONS**

Our experience with these 34 children is shown in Fig. 10. If we assume that the 2 patients lost to follow-up have died, the figure for late deaths is 21%. Twelve per cent of children are not improved, but the rest, i.e. 67%, are well, some are back at school, some are employed as domestic servants and others have gone to work in the industries. With the knowledge that the vast majority of these were cardiac grade IV, doomed to
We wish to thank Mr P. Marchand for advice in the preparation of this article and for allowing us access to data of those patients he operated on.

REFERENCES


Boeke Ontvangst: Books Received


