Papers and Originals

Long-term Results of Aortic Valve Replacement with the Starr-Edwards Valve

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Summary: Review of the 74 patients undergoing aortic valve replacement with a Starr-Edwards ball-valve prosthesis between October 1963 and December 1967 showed that 16 died during surgery or within the first month after operation, usually owing to myocardial failure; and there were nine late deaths. The remaining patients developed few major complications, and the long-term results of operation are considered satisfactory, no patient being grossly incapacitated and most of them are leading active, symptom-free lives.

Introduction

Severe disease of the aortic valve, in the form of aortic stenosis or aortic regurgitation, inevitably causes disability and death, and surgical relief of these conditions is always desirable. The unsatisfactory operation of closed transventricular aortic valvotomy has been abandoned and most surgeons now reserve the operation of aortic valve reconstruction for the special case where there is no extensive valve calcification and no aortic regurgitation (Scannell and Austen, 1966). Aortic valve replacement with a prosthesis such as the Starr-Edwards caged ball-valve (Starr *et al.*, 1963) is the operation of choice in many centres for calcific aortic valve stenosis or for severe aortic regurgitation, but reports of late complications from this operation have appeared and have encouraged some surgeons to undertake the technically more difficult procedure of aortic valve replacement with a homograft (Ross, 1964; Barratt-Boyes, 1964).

The relief of symptoms after aortic valve replacement with the Starr-Edwards valve is satisfactory, but little is known about the long-term effects; delayed complications, especially peripheral embolism, aortic incompetence, and haemolytic anaemia, are known to occur. The frequency and severity of these sequelae can be determined only by keeping all patients under observation after surgery. In this report the complications of aortic valve replacement with the Starr-Edwards valve occurring over a period of four years are analysed.

Patients and Methods

All patients at St. Bartholomew's Hospital having aortic valve replacement with a Starr-Edwards ball-valve prosthesis in the period October 1963 to December 1967 are included in the study, excluding only those patients who had more than one

valve replaced. There were 60 male and 14 female patients; their ages ranged from 14 to 68 years, the majority being in the older age groups (Table I). Aortic valve replacement was undertaken for aortic stenosis in 51 and for aortic incompetence in 23 patients (Table II). Cardiac failure, even of an advanced degree, was not considered a contraindication to surgery, and many patients were in severe failure in spite of intensive treatment before the aortic valve replacement procedure.

TABLE I.—Age Distribution of 74 Patients at Time of Operation

Age No.	10-19	20-29	30-39	40-49	50-59	60-69
..	1	6	10	19	27	11

TABLE II.—Aetiology and Nature of Aortic Valve Lesion in 74 Patients

	No. of Patients		No. of Patients
Aortic stenosis:		Aortic regurgitation:	
Calcific	45	Rheumatic	8
Rheumatic	6	Dilatation of valve ring ..	8
		Bacterial endocarditis ..	7

The patients were extensively investigated before surgery. The electrocardiogram and chest x-ray films were obtained before surgery in each patient and haemodynamic data from left heart catheterization and angiocardiology were available in most cases. In the earlier patients aortic valve replacement was carried out under profound hypothermia with an arrested circulation, but for the last 32 operations cardiopulmonary bypass with the Kay Cross disc oxygenator or the Rygg or Travenol bag has been used. Coronary arterial perfusion was maintained in these patients by selective cannulation of one or both coronary arteries.

The mortality after surgery has been divided into early deaths, within one month of the operative procedure, and late deaths. Necropsy was performed in every patient dying within one month of surgery, and the findings, together with clinical and operative details, were used to determine the cause of death for each patient. Necropsy was obtained in all but one of the patients who died at a later period after surgery, and in this way the cause of the late deaths could be determined with some accuracy.

All patients leaving hospital after aortic valve replacement are reviewed regularly in the outpatient department. Only one patient, who has emigrated to Australia, has been lost to follow-up. In addition to regular clinical examination the patients have chest x-ray and electrocardiographic examinations.

Results

Sixteen patients died at surgery or within the first month after surgery, an early mortality of 22%. The cause of these early

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deaths is given in Table III. Failure of the heart caused by myocardial ischaemia or from unknown causes accounted for nine of these early deaths. There have been nine late deaths; the cause of these is detailed in Table IV. The number of months survived for eight of these patients is given in Table V.

The non-fatal major complications (Table VI) include both those giving rise to symptoms and also any major abnormality detected on examination, even in the absence of symptoms.

TABLE III.—*Causes of the 16 Deaths at Surgery or During the First Month Thereafter*

	No. of Deaths
Myocardial infarction or coronary embolism ..	6
Myocardial failure cause unknown ..	3
Technical trouble with valve insertion ..	3
Tamponade ..	2
Ruptured ascending aorta ..	1
Clot involving valve ..	1

TABLE IV.—*Causes of the Nine Deaths One Month or More After Aortic Valve Replacement*

	No. of Deaths
Massive clot in valve ..	2
Ruptured ascending aorta ..	2
Escape of ball from cage ..	1
Dislocation of valve from seating ..	1
Septicaemia ..	1
Myocardial infarction ..	1
Unknown ..	1

TABLE V.—*Late Deaths. Time of Death for Each Complication*

	Time of Death (Months)		Time of Death (Months)
Myocardial infarction ..	6	Ruptured ascending aorta ..	3
Septicaemia ..	9		3
Dislocation of valve ..	13	Massive clot in valve ..	19
Ball out of cage ..	37		4

TABLE VI.—*Major Complications in 49 Survivors*

	No. of Patients		No. of Patients
Arterial embolism ..	13	Heart block ..	5
Leak around prosthesis ..	9	Anaemia ..	3
Left ventricular failure ..	8	Infected sternum ..	2
Haemorrhage ..	7		

Discussion

The major cause of death soon after operation was failure of the myocardium to maintain an adequate cardiac output. In six patients the myocardial failure can be definitely related to abnormality of the coronary arterial system, and myocardial infarction or coronary emboli were found at necropsy. In three patients the clinical features indicated a failing myocardium, but no evidence of severe ischaemia was obtained at necropsy. The ventricle of each of the patients was grossly hypertrophied and a considerable degree of fibrosis was noted on histological examination. However, it was not always the patients with the most severe myocardial hypertrophy preoperatively who failed to maintain an adequate circulation in the postoperative period and some other factors must have played a part. A detailed histological examination of the coronary arteries with post-mortem coronary angiography was not performed and we cannot exclude trauma to the intima by coronary cannulation, described as a fairly common occurrence by Fishman *et al.* (1968), as a contributory factor. These nine deaths from myocardial failure, representing an early mortality of 12%, are difficult to avoid with present techniques and may represent an irreducible minimal mortality of this operation for our patients. As experience has been gained there have been fewer deaths from unrecognized cardiac tamponade or from difficulties in positioning the valve due to a narrow aortic ring or a constricted ascending aorta.

Originally long-term anticoagulant therapy was not used routinely after aortic valve replacement, but since the death of the first patient in the series, 19 months after surgery, from massive clotting in the prosthetic valve, all patients are given warfarin on the third postoperative day and a prothrombin time twice the control is maintained in hospital and after discharge. Prothrombin estimations are performed regularly at this hospital

or at hospitals near to the patient's home. Only one further death from massive clot involving the aortic valve has occurred since the policy of permanent anticoagulation was begun, and this happened at a time when anticoagulant dosage was reduced in a patient admitted to hospital with a massive haematemesis. Thrombus formation on the prosthetic valve is now recognized as an important late complication of cardiac valve replacement and was present in 17 of the 20 patients studied at necropsy by Roberts and Morrow (1967).

Anticoagulant therapy has been less successful in the prevention of systemic embolism. In 10 patients cerebral embolism occurred at surgery or within the first week after surgery. The usual course for these patients was the presence of a mild hemiplegia in the postoperative period with subsequent full recovery. Dysphasia or weakness of one limb can still be detected at routine follow-up in only two.

Three patients have sustained a clinically detectable embolism after discharge from hospital. One patient, who was not receiving anticoagulants, developed a hemianopia 17 months after surgery. She is now on long-term anticoagulant therapy and has had no further incidents over the next 30 months. The other two late embolic incidents occurred, at five and six months respectively, in patients on long-term anticoagulants at a time when control of the therapy was adequate. Both have some permanent sequelae; in one there is weakness of the right arm and leg, and the other patient has a mild dysarthria. We now have only one patient not receiving long-term anticoagulant therapy. This patient suffered from a chronic anaemia before operation and control of the anaemia was achieved only when anticoagulants were discontinued. He remains well, four years after surgery, and has had no embolic incidents.

Long-term anticoagulant therapy has carried with it a significant number of bleeding episodes. In the immediate postoperative period cardiac tamponade was recognized, being relieved by a second thoracotomy in two patients, but another two patients (in whom the diagnosis was delayed) died of tamponade. Seven further patients have suffered one or more bleeding episodes outside the immediate postoperative period. Three patients bled from a known duodenal ulcer or hiatus hernia. In four bleeding occurred into the gastrointestinal tract without there being a demonstrable lesion, and in only one of these was the anticoagulant therapy excessive. Anticoagulation was continued in all but two of the patients who have had a major bleed. One patient had a chronic anaemia from chronic loss of blood into the gastrointestinal tract and the anaemia came under control only when anticoagulant drugs were discontinued. One patient had a massive haematemesis without demonstrable lesion on barium meal examination, and further attempts to treat him with anticoagulants were deemed inadvisable. He died five months later, but permission for necropsy was not obtained and we do not know if this death was a result of stopping the anticoagulant treatment.

One death occurred, in a boy of 16 at 37 months after surgery, from the escape of the ball from the cage. There had been no sign of the incipient failure of the valve, and the boy was leading an active life until his death within 20 minutes of the escape of the ball. Starr *et al.* (1966) reported 17 instances of degeneration of the aortic ball valve, three of which led to expulsion of the ball, and they found that in all but one case there was some abnormality of implantation that might account for greater than normal trauma to the ball. A major leak around the prosthesis or impingement of the aortic root on the ball pathway was thought to accelerate degeneration of the ball in their patients. The silicone ball in our patient had undergone gross colour change and suffered considerable surface damage, but no mechanical factors were noted that might have predisposed to this change.

An early diastolic murmur indicating leak around the ring of the prosthetic valve was present in nine patients. This sign has persisted unchanged in eight, one patient dying 13 months

after surgery when the valve became grossly displaced from its seating. In the remaining eight patients the aortic regurgitation would be judged to be mild on clinical examination, but it is interesting to note that five of these require long-term oral diuretic therapy to prevent the development of cardiac failure. In only eight patients in the series have oral diuretics been continued for more than three months after surgery, and a leak alongside the aortic valve appears to be the most important factor in determining the occurrence of cardiac failure in the late postoperative period. None of our patients has come to reoperation, but it is likely that the aortic incompetence is caused by the giving way of one or more of the anchoring sutures. Scannell and Austen (1966) report this as a cause of significant aortic regurgitation in seven of their patients.

Heart block is a well-recognized complication of aortic valve replacement and may be caused by injury to the bundle of His either by sutures or by trauma when the calcific material of the diseased aortic valve is removed (Gannon *et al.*, 1966). Five of our patients developed complete heart block at operation which remained permanent thereafter. One patient has died, four months after surgery, from unrelated cause (thrombus formation within the prosthetic valve), three patients are leading active lives, free from failure and from syncopal attacks, at 35, 31, and 25 months respectively after operation, and only one has received an implanted pacemaker. The QRS complex is usually narrow and the heart rate relatively fast in our patients with surgically induced heart block, suggesting that the lesion is in the neighbourhood of the atrioventricular node and the idioventricular focus is high in the conducting system. We do not regard heart block as a particularly ominous complication of aortic valve surgery.

Three patients have developed significant anaemia after discharge from hospital. In two the anaemia was caused by chronic loss of blood into the intestinal tract and the red cell survival time was normal. One patient had a shortened red cell survival time and presented a picture of a mixed haemolytic and iron-deficiency anaemia. This patient had a partial gastrectomy for a duodenal ulcer some years before and the haemoglobin is now maintained at nearly normal levels by oral iron therapy alone. The absence of any severe haemolytic anaemia in our patients is probably accounted for by the absence of any severe regurgitant leak. Brodeur *et al.* (1965) encountered haemolytic anaemia due to trauma to the red cells after aortic valve replacement, but anaemia was present only when there was increased turbulence produced by a leaking valve. In their patients the more severe anaemias were associated with the greater degrees of aortic regurgitation.

FIG. 1.—Reduction in heart size following aortic valve replacement. A, before surgery. B, one year after surgery.

The immediate results and follow-up of 146 patients who were treated by homograft replacement of the aortic valve have been reported by McDonald *et al.* (1968). The early mortality of 19% after this operation is comparable with our figure of 22% when a Starr-Edwards valve prosthesis is used. After homograft aortic valve replacement there was no need for long-term anticoagulant therapy and late systemic embolism was not seen. The use of an aortic homograft was, however, associated

with a considerably higher incidence of postoperative aortic regurgitation.

In general, the long-term results of aortic valve replacement have been most satisfactory. A considerable reduction in heart size (Fig. 1) is evident in patients operated on for aortic incompetence, and an improvement in the electrocardiogram (Fig. 2)

FIG. 2.—Electrocardiograms.

is the rule. Only two of the long-term survivors lead restricted lives and no patient is grossly incapacitated. Forty-seven patients, at an average time of 28 months after surgery, are leading active, symptom-free lives and most are in gainful employment. Some have taken up active sports, and this includes patients who were in left ventricular failure before operation. When the severity of the left ventricular muscle damage before operation and the poor outlook for these patients without operation are considered, we feel that the benefits of the procedure are remarkable. While we hope that improvements in technique will reduce the mortality and morbidity of valve replacement, we find the present operation undoubtedly worth while.

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