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Repair of moderate aortic valve lesions associated with other pathology: an 11-year follow-up[☆]

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Abstract

Objectives: The presence of moderate aortic valve (AV) lesions associated with other pathologies that require surgery presents a problem since ignoring or replacing the valve seems unsatisfactory. AV repair can be an attractive alternative if shown to perform satisfactorily. **Methods:** To evaluate this possibility, all consecutive AV patients who underwent operation between July 1988 and July 1999 were reviewed. Out of 1764 AV patients, 239 (14%) underwent repair and 86 (study group) had moderate lesions associated with mitral (73), tricuspid (33), coronary disease (5) and others (8). Mean age was 28 years (range 2–66); 78% were rheumatic, 71% were in sinus rhythm and 71% in NYHA class III–IV. **Results:** There were seven hospital deaths (8%) and three patients were lost to follow-up (95% complete). Late mortality was 8% and 10-year actuarial survival was $86 \pm 4.5\%$ (excluding hospital mortality). There were four (5%) embolic events (actuarial freedom $94 \pm 3.5\%$). Twenty-one patients required reoperation with two mortalities. The AV was not touched in five patients. In the remaining 16, the AV was replaced. Only one patient had isolated AV replacement while in all others, additionally, the mitral, tricuspid, or both required surgery. All reoperated patients had rheumatic etiology. Actuarial freedom from AV dysfunction at 8 years was $68 \pm 7.5\%$. **Conclusions:** Repair of associated moderate AV lesion is worth considering even in a predominantly young rheumatic population. © 2001 Elsevier Science B.V. All rights reserved.

Keywords: Rheumatic aortic valve; Aortic valve repair; Multiple valve disease

1. Introduction

Aortic valve (AV) repair has regained a lot of interest recently. This is probably due to the superiority of corrective surgery over replacement particularly in the atrioventricular area [1,2]. Because of the young age of our patients infected with rheumatic valvular disease we developed an interest in valve repair techniques. Reconstructive surgery for AV disease was utilised enthusiastically and widely initially [2,3]. However, as longer follow-up became available it became obvious that these techniques have their limitations particularly in patients with severe AV pathology [4]. The repair of AVs with moderate lesions appeared more satisfactory. Since the presence of moderate AV lesions associated with other pathologies that require surgery represents a problem (ignoring or replacing the valve seems unsatisfactory), AV repair can be an attractive alternative if shown to perform well. We therefore reviewed

retrospectively our AV repair population and evaluated those patients that fit these criteria.

2. Patients and methods

2.1. Patients

All consecutive AV patients who underwent operation between July 1988 and July 1999 were reviewed. Out of 1764 AV patients, 239 underwent repair (14%). Ninety-four patients had isolated AV repair whilst 145 patients had AV repair plus other kinds of surgery and from these, 86 patients had moderate AV lesions. These 86 patients constituted the study group.

All patients had preoperative evaluation by transthoracic echocardiography. Coronary angiography was performed to rule out coronary artery disease in older patients (>40 years for males and >45 years for females). The mean age of the study group was 28 ± 15 years with a range from 2 to 66 years. There were 29 males (34%) and 57 females (66%). The etiology of the AV disease was rheumatic in 68 (79%), congenital in nine and other etiologies in nine patients. The

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NYHA functional class was III–IV in 71% of the patients. Left ventricular function was normal or only mildly impaired in 87%.

Six patients (7%) had pure aortic stenosis, 52 (60%) regurgitation and 28 (33%) had mixed stenosis and regurgitation. Sinus rhythm was the predominant rhythm in 71% of the patients while 29% were in atrial fibrillation/flutter.

The AV disease was associated with mitral valve disease in 40 patients, with mitral and tricuspid valve disease in 33 patients, with coronary artery disease in five and with various other pathologies like a ventricular septal defect, a subaortic membrane and ascending aortic aneurysm in eight.

2.2. Methods

Surgery was performed with routine cardiopulmonary bypass with single or bicaval venous cannulation and a body temperature around 32°C. Antegrade and retrograde blood cardioplegia were used routinely. Transesophageal echocardiography (TEE) was used in all patients to assess the valve lesions preoperatively and the result of the repair postoperatively.

The AV leaflets were thickened in 56 patients. Commissural fusion was noted in 38 patients with only six valves exhibiting calcification. Only three valves were bicuspid. Gross annular dilation was seen in seven patients but an additional 33 patients had some degree of annular dilation.

The AV was repaired using various techniques [3]. These techniques are directed towards the functional improvement of the diseased AV. Usually, multiple techniques are needed for repair of a particular valve. These include commissurotomy (38), leaf plasty (24), commissural resuspension (24) and subcommissural annuloplasty (42). Subaortic membrane/fibrosis was resected in four patients. In one patient an aortic ring was used [5]. Neither cusp extension nor entire annular reduction techniques were used in this group of patients. The mitral valve was replaced in 38 patients and repaired in the others. The tricuspid valve was repaired all the time. The mean cardiopulmonary bypass time was 137 ± 60 min with a range of 39–303 min and mean aortic cross-clamp time was 88 ± 37 min with a range of 23–114 min.

2.3. Statistical analysis

Values were expressed as means \pm standard deviation or as median. The actuarial survival and event-free curves were calculated using the Cox Life table method.

3. Results

3.1. Immediate result

In the operating room, immediate TEE demonstrated a satisfactory result of the AV repair in all patients. Thirty-

nine patients had no or trivial AR and 47 patients had mild AR. About 50% of the patients demonstrated a small gradient across the AV, mean 20–25 mmHg. This was more often seen in patients who had subcommissural annuloplasty. In three patients, the mitral valve repair was not satisfactory and the valve had to be replaced with a second pump run.

3.2. Mortality

There were seven hospital deaths (8%), five with biventricular failure, one with severe right ventricular failure and severe pulmonary hypertension and one brain death related to coagulopathy and bleeding.

3.3. Morbidity

There were no major morbidities. One patient needed a permanent pacemaker. At discharge, 72% of the patients were in sinus rhythm, 3% in junctional rhythm and 24% in atrial fibrillation.

3.4. Anticoagulation

Anticoagulation in the survivors is demonstrated in Table 1.

3.5. Follow-up

The patients are followed up in a dedicated valve clinic with clinical evaluation and transthoracic echocardiography at 3 months, 6 months, then yearly and more frequently as indicated. Patients were followed up for up to 11 years. Mean follow-up was 5 years. The follow-up was 95% complete with three patients lost to follow-up.

Seven patients died late: four of cardiac cause, one following a stroke and two of unknown causes. Actuarial survival at 10 years was $86 \pm 4.5\%$ (Fig. 1). There were four embolic events, three fully recovered and one was fatal. Actuarial freedom from thromboembolic events was $94 \pm 3.5\%$ (Fig. 2). There was one incident of endocarditis in this group and one patient needed hospitalisation and blood transfusion with anticoagulation related haemorrhage. Excluding patients who needed reoperation, 96% of the survivors are asymptomatic in NYHA class I–II. Two patients are in severe congestive heart failure related to tricuspid valve disease and are managed medically. Thirty patients (66%) are in sinus rhythm whilst the others are in atrial fibrillation/flutter and one patient with a permanent pacemaker. Thirty-one patients (68%) have almost a normal

Table 1
Anticoagulation in the survivors as demonstrated anticoagulation

Drugs	#	%
Aspirin	19	24
Low dose warfarin	9	11
Full anticoagulation with warfarin	20	25
None	31	39

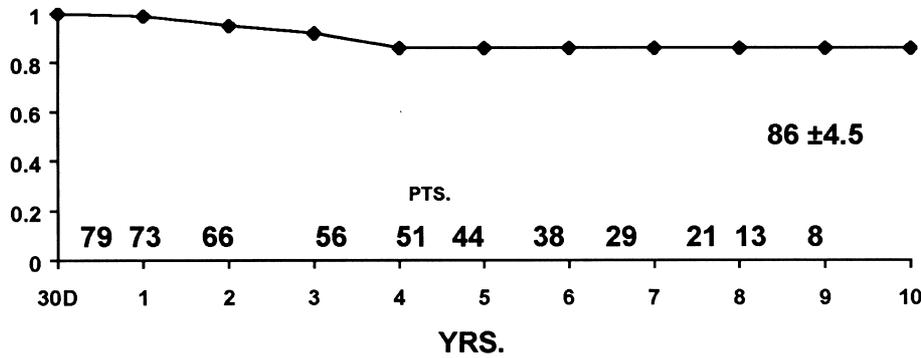


Fig. 1. Actuarial survival curve.

function of the repaired AV by echocardiography. The rest of the patients demonstrated some deterioration in the AV function but are clinically doing well and are still being followed up.

3.6. Reoperation

Twenty-one patients needed 23 reoperations with two patients needing two reoperations. Among these patients, there was one hospital mortality and one late mortality of a patient with sickle cell disease who developed a sickling crisis. The mean interval between surgeries was 4 ± 2.9 years with a range from 11 days to 10 years (median 3.8 years). All reoperated patients had rheumatic etiology and the mitral valve was involved in all but two patients in whom the tricuspid valve disease was the predominant lesion. One patient had endocarditis.

Freedom from reoperation at 8 years was 63.4 ± 7.4 (Fig. 3). The AV repair was stable and did not need intervention in five of these patients. In the remaining 16 the AV was replaced. Echocardiography demonstrated mild to moderate valve dysfunction in four patients but severe valve dysfunction in all the others with regurgitation in eight, pure severe stenosis only in one and mixed regurgitation and stenosis in seven. Excluding the five patients who maintained good AV repair, the actuarial freedom from AV dysfunction after repair is therefore $68 \pm 7.5\%$ (Fig. 4). The AV was replaced with a Carbomedics bileaflet mechanical prosthesis in 12 patients, with a Perimount pericardial bioprosthesis in two, a Hancock II in one and an aortic homograft in one.

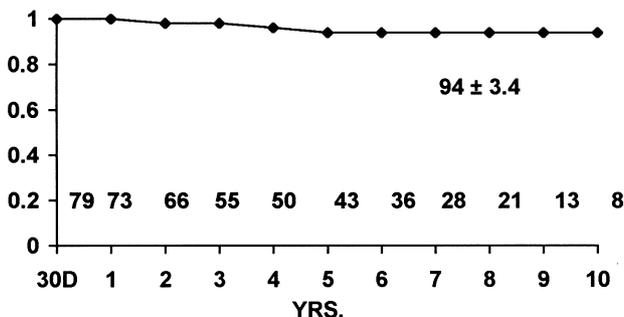


Fig. 2. Actuarial freedom from thromboembolism.

4. Discussion

There has been a renewal of interest recently in reconstructive surgery for the AV. Most surgical techniques used nowadays were described many years ago [2,3]. It remains however, that the single most important factor of AV repair is the quality of the AV leaflets. There must be enough leaflet tissue of good quality to satisfactorily restore normal valve function. Generally, multiple techniques are required in repairing the AV and probably no single technique can be expected to achieve satisfactory results [6]. Cusp extension of all three cusps with a single strip of glutaraldehyde-treated autologous pericardium is reserved for severe valve pathology and is actually a sort of valve replacement [7]. Single cusp extension is more demanding and unreliable because of the difficulty in achieving a good geometry with unequal leaflets [8]. Neither of these techniques was utilized for this study group in whom the AV was repaired, preserving the native leaflets. The natural history of mild AV disease after mitral valve surgery was studied by Vaturi et al. [9]. Their findings indicated that among patients with rheumatic heart disease a considerable number have mild AV disease at the time of mitral valve surgery. Yet, most do not progress to severe disease and AV replacement is rarely needed after a long follow-up period. In their opinion, prophylactic valve replacement is not indicated in these cases. However, the mean age in their study group was 61 ± 13 years. At that age, the rheumatic process is inactive. In our young patient population the likelihood of repeated attacks of rheumatic fever exists and in our experience progression of a mild or moderate disease does occur.

Hence, the presence of moderate AV lesions associated with other pathologies that require surgery presents a clinical problem. Ignoring the valve or replacing it seems unsatisfactory. AV repair under these circumstances can be an attractive option.

In our clinical set-up where our patient population is mostly young and predominantly with rheumatic valvular disease, we are frequently faced with this problem. We adopted the policy of attempting to repair the AV in all such patients. Over the years we accumulated significant experience.

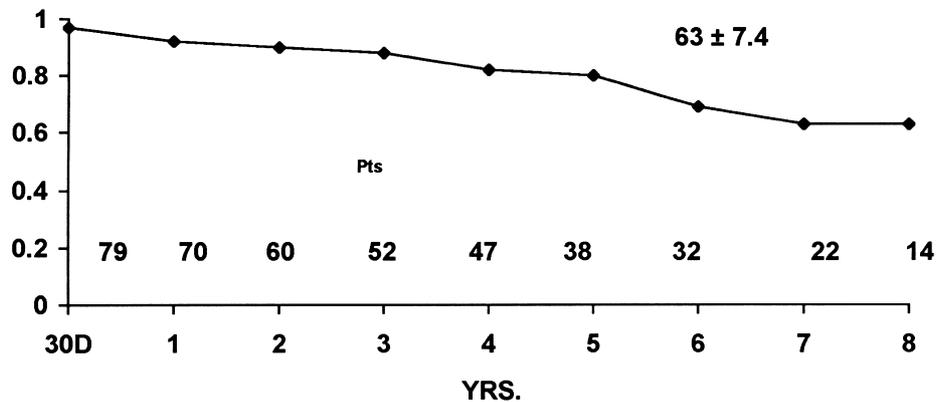


Fig. 3. Actuarial freedom from reoperation at 8 years.

It was always feasible to repair the AV using the operative techniques of commissurotomy, edge unrolling and thinning (leaf plasty), annuloplasty and free edge resuspension. Immediate TEE demonstrated a satisfactory AV repair.

The additional time taken to repair the AV after dealing with the original pathology did not appear to adversely affect the overall result. None of the in-hospital mortalities were related to that. In addition, with regard to reoperations, only one patient required a reoperation because of worsening AV pathology alone. Putting all this in perspective, the adopted policy seems reasonable.

Our follow-up, which was 95% complete and with a range of up to 11 years demonstrated that the AV repair holds well, particularly if associated with non-rheumatic etiology. This is of particular importance in developed countries where a surgeon is often faced with patients coming for coronary artery bypass surgery who have associated moderate AV disease. The tendency, in general, is to ignore the AV initially. When the AV disease becomes severe, which can happen in a short period of time, the patient is faced unfortunately with a reoperation at a significantly higher risk [10]. This risk, however, seems to be lessened if the AV replacement is remote after prior coronary artery bypass grafting [11]. Repairing the valve at the initial surgery can help avoid such a problem [12].

The 21 patients who needed reoperation all had rheumatic valvular disease. This seems to be the only obvious factor associated with failure. No specific technique applied seemed to cause failures more than others. However, more need for reoperation was seen in valves that had combined stenosis and regurgitation to start with.

Though 21 patients needed reoperation during the follow-up period, the AV repair was stable in five patients and was not touched at reoperation. Hence, actuarial freedom from AV dysfunction at 8 years was calculated as $68 \pm 7.5\%$. In addition, four other patients had only mild to moderate AV dysfunction and had it not been for the 'other valves' deteriorating, these patients probably would not have been reoperated on.

We therefore feel more optimistic about the validity of this approach than Bernal et al. [13] who reported on a similar group of patients with a mean follow-up of 18.8 years. They followed up a group of 53 patients who had AV repair for mild or moderate rheumatic AV disease at the time of mitral or mitral/tricuspid valve surgery for almost two decades. The freedom from AV deterioration at 22 years was 25.3%. However, the series started in 1974; TEE was not available and intraoperative testing was only made by direct observation of the repaired AV. We believe that in our series the availability of TEE clearly

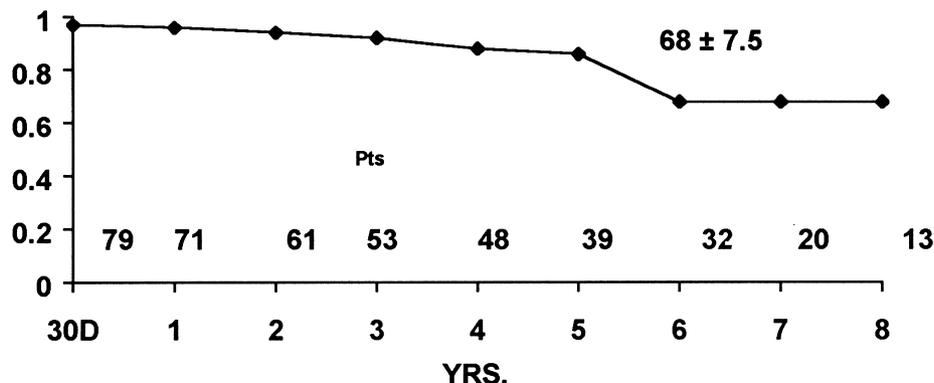


Fig. 4. Actuarial freedom from AV dysfunction at 8 years.

made an impact on ensuring that the patient leaves the operating room with a good repair, which probably will ultimately affect long-term results.

Of course, the only way that one will be able to answer the question as to whether it is worthwhile taking this approach is a prospective randomised study. Such a study would be extremely difficult to conduct and will take an unrealistically long time to finish. In summary, the data presented demonstrate that repair of associated AV lesions is worth considering even in a predominantly young rheumatic population.

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Appendix A. Conference discussion

Dr O. Alfieri (Milan, Italy): Moderate aortic insufficiency can be very well tolerated by patients for a long period of time. What is the evidence to support an aggressive treatment of moderate aortic insufficiency associated with other valve pathology?

Dr Halees: In patients with rheumatic valve diseases, we feel that the presence of aortic valve lesions, when the patient goes for other valve surgery might adversely affect the outcome if it was left alone. In our population of patients we can prove that the disease is progressive, so you don't want to end up with a situation where a patient comes to you with severe mitral and moderate aortic valve lesion. Then 2 years down the road you have a good mitral valve repair or prosthesis but needing to have a reoperation for progression of aortic valve disease.

In addition, maybe in the West, a similar situation is met with the association of aortic valve diseases and coronary artery disease. I know in our series, this has not been common, but I believe that the repair in these patients could be very stable unlike patients with rheumatic heart disease.

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