VALVE REPLACEMENT OR REPAIR IN MITRAL AND AORTIC HEART DISEASE

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INTRODUCTION

Among the advances in medical science of the last decade is the repair or replacement of the mitral, aortic or tricuspid valve. Progress in this field was made after the introduction of the extracorporeal circulation and perfusion techniques (Key et al 1958). Under direct vision, evaluation of valve dysfunction can be accurately determined and so adequate correction is achieved (Morris et al, 1962; Kay et al, 1960; Lellehei et al, 1958; Scott et al, 1958; Logan et al, 1967). Operations of this kind it is now no exaggeration to state have become a commonplace event and they must be considered amongst the important recent developments in the management of valvular disease of the heart.

This paper deals with the evaluation of the results of surgical treatment in eighteen patients attending the Cardio-Vascular Unit of the Belfast City Hospital. These patients underwent repair or replacement of one or more heart valves in surgical centres outside Northern Ireland in the years between 1963 and 1968.

PRE-OPERATIVE FINDINGS

The age and sex distribution of the patients is shown in Table I. There were ten females and eight males in the series and their ages ranged between 20 and 57 years with a mean of 41.2 years. Table II shows the distribution of the patients into three main groups. Group I comprised ten patients with dominant mitral incompetence with or without tricuspid insufficiency. Five patients in Group II had combined mitral and aortic valve disease of sufficient severity to necessitate replacement of both valves. In Group III there were three patients with aortic valve disease alone.

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TABLE II — The Three Main Groups

| Group | Anatomical diagnosis                      | Female | Male | Total |
|-------|------------------------------------------|--------|------|-------|
| I     | Mitral valve disease (± Tricuspid valve disease) | 5      | 5    | 10    |
| II    | Mitral and aortic valve disease          | 2      | 3    | 5     |
| III   | Aortic valve disease                     | 3      | 0    | 3     |

patients in Group I compared with 6.2 years and 3.3 years respectively for patients in Groups II and III.

Dyspnoea. All patients complained of undue dyspnoea on effort. Of the fifteen patients in Groups I and II, all had at least Grade III/IV incapacity (New York Heart Association 1953), that is they were breathless on minimal exertion and seven were dyspnoeic even at rest.

Angina. Five out of eight patients with aortic valve disease experienced anginal pain. This was usually brought on by effort but in two cases it occurred at rest and produced total incapacity.

Cardiac Decompensation. All eighteen patients were considered to have failure of one or other or both ventricles as judged clinically. In several cases, especially in Group I, this was severe and unresponsive to medical treatment. In no patient was the decompensation attributable to any known reversible cause and therefore had to be presumed to be associated with myocardial impairment often of gross degree.

Atrial Fibrillation. This was present in all patients in Groups I and II but as might be expected in those with pure aortic valve disease sinus rhythm was universally present.

Cardiac Murmurs. A mitral pansystolic murmur of at least Grade III/VI intensity at the apex was audible in each of the ten patients in Group I in which mitral incompetence was the dominant lesion. Two patients in this group had long mitral mid-diastolic murmurs and were considered to have significant mitral stenosis in addition. The five patients in Group II all had mitral incompetence and in each of them aortic diastolic murmurs indicative of aortic insufficiency were also present.

Additional Sounds. A third heart sound of rapid ventricular filling was noted in seven of the ten patients in Group I. In the two patients in this group considered to have significant but not dominant mitral stenosis the additional sound was recorded on the phonocardiogram as an opening snap.

Electrocardiography. The electrocardiograms were consistent with left ventricular enlargement in fourteen patients. In the remaining four, all in Group I, right axis deviation and right ventricular dominance were present.

Radiography. The cardio-thoracic ratio was measured for each patient as an index of generalised cardiac enlargement. Figure I shows cardiac enlargement in a patient in Group I. In every patient the ratio exceeded fifty per cent. In fifteen out of seventeen patients it was greater than sixty per cent, and in seven of these the cardio-thoracic ratio exceeded seventy per cent. All patients in this series had radiographic evidence of left atrial enlargement which varied in size from moderate to aneurysmal.
FIGURE 1
Aneurysmal dilatation of the left atrium.

FIGURE 2
Left ventricular angiogram (trans-septal route) showing gross mitral regurgitation.
Mitral incompetence or aortic insufficiency were assessed by left ventricular angiogram or aortogram. Angiograms and/or aortograms were available in eight patients in all of whom the mitral or aortic insufficiency was considered to be at least Grade III out of IV. Figure 2 illustrates a left ventricular angiogram in a patient with gross mitral insufficiency.

_Haemodynamic Data._ The results of pre-operative haemodynamic investigations were available in twelve patients. Six of these were in Group I and pulmonary hypertension was present in all. In two, this was considered to be mild (P.A. systolic pressure less than 40 mm. Hg). Three patients had moderately severe pulmonary hypertension (P.A. systolic pressure between 40 and 60 mm. Hg). In one patient with tight mitral stenosis as well as incompetence the pulmonary artery systolic pressure exceeded 100 mm. Hg. Four out of five patients with mixed aortic and mitral valve disease had mild pulmonary hypertension, and in one it was moderately severe.

**Operative Results**

Table III summarises the results of operative treatment for the eighteen patients. Eight patients underwent mitral replacement or repair alone. Four patients required tricuspid valve replacement or repair in addition to mitral valve surgery. Four patients had their aortic valves replaced by Starr-Edwards prostheses, and two required combined mitral and aortic valve replacement. Only three patients died during or immediately after operation so the overall mortality was only 16.6 per cent. The cause of death was cerebral embolism in one patient and the syndrome of low cardiac output accompanied by peripheral circulatory failure in the remaining two.

_Complications in the Survivors_  

One patient aged 44 years who had a successful aortic valve replacement with marked improvement in exercise tolerance post-operatively, died suddenly and unexpectedly at home twelve months after operation. A post-mortem was carried out, and demonstrated a fresh myocardial infarction due to coronary occlusion.

Three patients in Group I and one in Group II sustained systemic emboli at one day, six weeks, eight weeks and fifteen months respectively after operation. One patient initially appeared to have severe cerebral damage but in the course of the succeeding few weeks she made a steady recovery and has only minimal residual disability. The other three patients suffered only transient hemiparesis without dysphasia and all made a rapid and complete recovery. All patients were maintained on long term anticoagulants and with the exception of the incident at one
day, the prothrombin times were all at the lower end of the therapeutic scale at the time of the embolic episodes.

Table IV shows long-term results in fifteen patients. Of the fifteen patients who survived operation only one has since died. Amongst the remaining fourteen all except one have benefitted from operative treatment. Ten have had remarkably good results and have returned to work or normal household duties. Three patients have had a fairly good result, but the improvement has not been sufficient to permit them to resume normal activities. Amongst the nine survivors in Group I the initial systolic murmur was abolished in four, reduced in intensity in four, and unchanged in one. In two out of four patients in whom the murmur was reduced in intensity a mitral Starr-Edwards prosthesis had been inserted and following this systolic murmurs of little or no functional significance commonly occur. Seven patients in Group I were noted to have a third heart sound pre-operatively but in only two was this sign present at post-operative follow-up. Amongst the six surviving patients in Groups II and III five had aortic Starr-Edwards prostheses inserted for the treatment of aortic incompetence, and in all but one a short aortic systolic ejection murmur was noted post-operatively. Figure 3 shows pre- and post-operative phonocardiograms in one patient who had mitral incompetence.

All patients prior to operation had cardiac decompensation whereas post-operatively this was present in only three of the fifteen survivors.

The electrocardiogram especially in patients in Groups I and II provided little objective evidence by which to assess improvement post-operatively. However, the electrocardiogram of one patient with calcific aortic valve stenosis demonstrated a remarkable reduction in the degree of left ventricular hypertrophy in the months following surgery. Of the thirteen patients with atrial fibrillation who survived operation only one returned to sinus rhythm post-operatively. This was achieved by D.C. shock thirteen months after operation and now twenty-three months later this patient remains in sinus rhythm with an almost normal electrocardiogram.

Table V compares the pre- and post-operative cardiothoracic ratios. In eight cases there was a reduction in heart size ranging from as little as 1.6 per cent up to 14.5 per cent. Six patients showed a small increase in C.T.R. varying from 0.4 per cent to 4.2 per cent despite considerable functional improvement in most instances. Figures 4 and 5 show the cardiothoracic ratios in a patient before and after mitral annuloplasty in whom there was an overall reduction in cardiac size of ten per cent after operation. Figure 6 illustrates Starr-Edwards prostheses replacing the mitral and aortic valves.
**FIGURES 3A and B**

Pre- and post-operative phonocardiograms of a patient with mitral insufficiency.

| Patient | Operative Treatment          | Pre-op | Post-op | C.T.R. | Percentage change C.T.R. Post-op. |
|---------|------------------------------|--------|---------|--------|----------------------------------|
| 1       | Mitral valve replacement     | 84.4   | 88.6    | + 4.2  |
| 2       | Mitral and tricuspid replacement | 90.2  | 88.6    | - 1.6  |
| 4       | Mitral annuloplasty          | 74.0   | 59.5    | -14.5  |
| 5       | Mitral annuloplasty          | 77.7   | 71.5    | - 6.2  |
| 7       | Mitral valve replacement     | 76.3   | 78.8    | + 2.5  |
| 8       | Mitral annuloplasty          | 55.1   | 64.7    | - 9.7  |
| 9       | Mitral and tricuspid replacement | 85.0  | 82.5    | - 2.5  |
| 10      | Mitral annuloplasty          | 64.5   | 55.4    | - 9.1  |
| 11      | Mitral valve replacement     | 60.6   | 61.4    | + 0.8  |
| 13      | Aortic valve replacement     | 63.3   | 54.7    | - 8.6  |
| 14      | Mitral valve replacement     | 78.1   | 78.5    | + 0.4  |
| 15      | Mitral and aortic valve replacement | 59.8  | 61.5    | + 1.7  |
| 17      | Aortic valve replacement     | 62.2   | 62.5    | + 0.3  |
| 18      | Aortic valve replacement     | 60.6   | 57.5    | - 3.1  |
| Mean    |                              | 70.7   | 68.9    |        |
FIGURES 4 and 5
Pre- and post-operative chest X-rays of a patient with mitral valve disease.

FIGURE 6
Starr-Edwards prostheses replacing the mitral and aortic valves.
DISCUSSION

It is obvious from the clinical, electrocardiographic, radiological and haemodynamic data presented that the eighteen patients in this series had by any standard advanced valvular heart disease. Every individual had at least Grade III out of IV dyspnoea and cardiac decompensation pre-operatively. Further the mean cardiothoracic ratio before operation in these patients was seventy-one per cent. Half of the patients in Group I with dominant mitral insufficiency had in addition tricuspid insufficiency due to stretching and dilatation of the right ventricle and tricuspid ring. Taking all these factors into account, a relative quantitative assessment of the clinical state of each patient was attempted by arbitrarily awarding points for the presence of each one and so the total pre-operative score was calculated. Thus points were awarded according to the degree of dyspnoea and cardiomegaly, for the presence of tricuspid insufficiency and cardiac decompensation. A similar calculation was made for each patient after operation and the results of this comparative study are presented in the histogram (Figure 7).

In all cases, except one patient who had persistent mitral insufficiency there was a considerable reduction in the post-operative score. This was brought about almost entirely by the disappearance of cardiac decompensation and tricuspid
insufficiency coupled with a considerable reduction in the grade of dyspnoea after operation. The high cardiothoracic ratio for this series with little reduction in heart size post-operatively (mean ratio sixty-nine per cent compared with seventy-one per cent pre-operatively) suggests that in many patients in addition to valvular disease, there must have been considerable rheumatic myocardial damage. This could result from long standing atrial and ventricular dilatation with consequent loss of elasticity. Nevertheless the majority of patients showed considerable improvement, sufficient in thirteen cases to allow a return to near normal living. It is therefore a reasonable assumption that earlier operative intervention might well have produced still better results by preventing irreversible myocardial damage and progressive increase in the size of the heart. This might have produced a much better long-term prognosis for the patients in this series, the outlook for whom in the last analysis must rest on the degree of irreversible myocardial damage prior to operation. This conclusion is in agreement with the findings of Morris (1962) and Logan et al (1967).

Clearly in the majority of our patients the optimum time for surgery had passed by the time treatment was carried out, yet in spite of this the operative mortality was remarkably low (16.6 per cent) and only one of the survivors failed to benefit. The encouraging results in this series concur with the experience of Emanuel (1968) who reported a successful outcome in three patients with advanced rheumatic heart disease.

SUMMARY AND CONCLUSION

The results of valve replacement or repair in eighteen patients with advanced mitral, aortic, and mitral and aortic heart disease have been described. Three patients died either during operation or in the immediate post-operative period. Another death occurred from unrelated disease twelve months after operation. The survivors have been followed for a mean period of two years and two months. Ten patients have had a moderately good result, and three a fair result from surgical treatment.

Despite the considerable functional improvement in all but one patient, there has been little reduction in heart size post-operatively. It is assumed that this indicates irreversible myocardial damage and on this factor the ultimate result of operation will depend. Nevertheless it is considered reasonable to recommend operative correction in suitable patients, even when the disease is advanced, in the expectation of achieving a return to more normal living at least in the short-term follow-up period.

Recent advances in open valve surgery and cardio-pulmonary bypass techniques mean that many patients with severe valve damage who formerly were rejected as unsuitable for operation must now be considered candidates for cardiac surgery.

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