

Mitral valve replacement in the presence of severe pulmonary hypertension

T. K. KAUL, W. H. BAIN, J. V. JONES,
A. R. LORIMER, R. M. THOMSON,
M. A. TURNER, and A. ESCAROUS

Departments of Surgical and Medical Cardiology and Radiology, Glasgow Royal Infirmary

Kaul, T. K., Bain, W. H., Jones, J. V., Lorimer, A. R., Thomson, R. M., Turner, M. A., and Escarous, A. (1976). *Thorax*, 31, 332–336. **Mitral valve replacement in the presence of severe pulmonary hypertension.** Thirty patients with severe preoperative pulmonary hypertension (pulmonary artery pressure range 90–165 mmHg, mean 118 mmHg) were reviewed following single mitral valve replacement, with prosthetic valves, within the last 10 years (1964–74). The early and late mortality for this group was no different from that of the total series of mitral valve replacements performed over the same period. Marked postoperative clinical improvement was accompanied by corresponding radiological and electrocardiographic changes. In addition there was a statistically highly significant haemodynamic improvement in the 21 survivors (mean survival time 5½ years).

At recatheterization the mean pulmonary artery pressure was 41.5%, the mean wedge pressure 46.3%, and the transpulmonary gradient (PAm–LAm) 36.1% of the preoperative values.

It is concluded that gross pulmonary hypertension is not *per se* a contraindication to mitral valve replacement surgery.

The criteria governing the selection of most patients for mitral valve replacement have become standardized over the past two decades, but opinions have differed when the disease is complicated by severe pulmonary hypertension. Early workers regarded the pulmonary vascular changes associated with long-standing mitral valve disease as irreversible (Emanuel, 1963). In general, it has been held that severe pulmonary hypertension was associated with greater operative risk, higher operative mortality, and a poorer long-term prognosis.

However, a significant reduction in the level of pulmonary hypertension in some patients after mitral valve surgery and a more favourable long-term outlook have been reported (Dalen *et al.*, 1967; Zener *et al.*, 1972). Furthermore, Ward and Hancock (1975) suggested that extreme pulmonary hypertension should be regarded as a positive indication for early operation. In their series there was a high mortality in this group, but the prognosis was nonetheless considerably

better than the natural history of the disease without surgical intervention. In our series in the last five years, only one of 16 patients with gross pulmonary hypertension has died during or after surgery.

PATIENTS

Thirty patients (13 men and 17 women, aged 23–59) were studied following mitral valve replacement (MVR) for isolated mitral valve disease. The preoperative pulmonary arterial systolic pressure exceeded 90 mmHg in all (range 90–165 mmHg). The operations were performed over a period of 10 years (1964–74) and the average period of follow-up was five and a half years. All the patients had symptomatic mitral valve disease for periods of up to 20 years and 11 patients had had a previous mitral valvotomy. The Björk-Shiley tilting disc prosthesis was inserted in nine, the Hammersmith disc valve in one, and the Starr-Edwards ball valve prosthesis in 20

TABLE I
SUMMARY OF HAEMODYNAMIC CHANGES IN 21 SURVIVORS
(mean follow-up period 5½ years)

	Preoperative Mean	±SD	Postoperative Mean	±SD	% Reduction	P Value
Pulmonary arterial systolic pressure (mmHg)	110.5	18.9	47.5	12.4	57.2	< 0.001
Pulmonary arterial mean pressure (mmHg)	74.16	18.5	30.8	5.5	58.4	< 0.001
Wedge pressure mean (mmHg)	30.61	6.3	14.19	3.9	53.3	< 0.001
Transpulmonary arteriovenous pressure gradient (PAm-LAm)	43.52	19.7	15.73	5.69	63.85	< 0.001
Arteriovenous sat. diff. (vol. %)	63.1	20.0	39.14	16.7	37.93	< 0.001

SD=standard deviation

patients. Before operation 19 patients were in grade IV, 10 in grade III, and one in grade II of the New York Heart Association classification. After surgery the patients have all been followed up at regular intervals with, at each visit, a full clinical examination, chest radiograph, and electrocardiogram. In the 21 surviving patients, cardiac catheterization has been performed 1-10 years after replacement of the mitral valve.

RESULTS

There was a progressive fall in hospital mortality over the first five years of mitral valve replacement with maintenance of this improvement in the last five years. In the latter period the hospital mortality for all patients undergoing MVR was 6%.

In the same period 16 patients with severe pulmonary hypertension underwent MVR. None died in hospital and there has been one late death.

The overall survival rate for the group was 50% as compared with 58% for the others in the total series (Fig. 1).

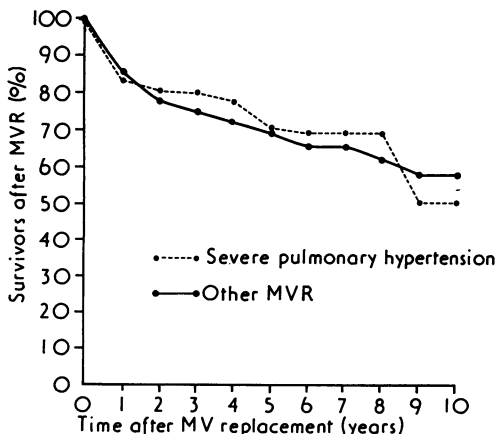


FIG. 1. Overall survival after mitral valve replacement.

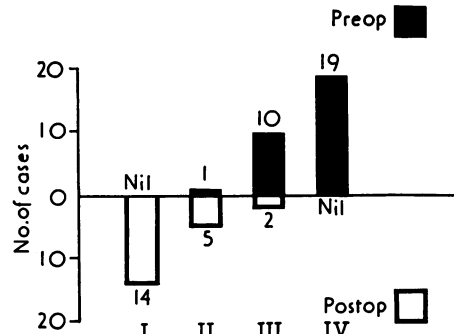


FIG. 2. Clinical gradings.

The findings at recatheterization of the 21 survivors are summarized in Table I. These show that there was a highly significant fall in both left atrial and pulmonary pressures after valve replacement in all. The mean pulmonary arterial wedge pressure was 12 ± 3 mmHg on recatheterization in 17 out of 21 patients, values at the upper limit of normal. The mean pulmonary arterial pressure did not return to normal but fell considerably postoperatively, being 30 mmHg or less in 11 (52%).

The values of the pulmonary arteriovenous pressure gradient (PAm-LAm), which bears a linear relationship to pulmonary vascular resistance¹ (Wood, 1954) fell significantly in all patients. On the basis of Wood's findings, the estimated values of pulmonary vascular resistance were 10-30 units in 15 patients and 6-10 units in six patients before surgery and fell to less than 6 units in all but two patients.

The changes in clinical gradient are shown in Figure 2. Of the 21 survivors, 14 had improved to

$$^1 \text{PVR} = \frac{\text{PAm} - \text{LAm}}{\text{CO}}$$

PVR = pulmonary vascular resistance; PAm = mean pulmonary arterial pressure in mmHg; LAm = mean left atrial pressure in mmHg; CO = cardiac output in l/min

grade I when seen 6–12 months after operation. The two patients shown who remained in grade III had paraprosthetic leaks, subsequently requiring resuture of the valve. Both have improved. In all others, early improvement has been satisfactorily maintained.

There was radiological evidence of reduction in heart size, as noted in serial comparable chest radiographs (Table II). The cardiothoracic ratio was reduced in 11 patients (52%). This change in transverse diameter was due principally to a reduction in size of the left atrium and right ventricle. The latter was in keeping with electrocardiographic regression of right ventricular hypertrophy in 12 (57%) patients according to standard criteria (Goodwin and Abdin, 1959). There were no rhythm changes (Table III). Three of the four cases with increased transverse cardiac diameter and increased left and right ventricular sizes had some paraprosthetic leak. Dilatation of the main pulmonary artery remained unchanged in 16 (76.3%), regressed in four (19%), and apparently increased in one. A reduction in pulmonary venous congestion was noted in 15 (17.4%) patients, and this improvement has been maintained.

TABLE II
RADIOGRAPHIC CHANGES IN 21 PATIENTS WITH SEVERE PULMONARY HYPERTENSION AFTER MITRAL VALVE REPLACEMENT

Chest Film	Decrease	Unchanged	Increase
C-T ratio	11	6	4
MPA	4	16	1
RV	13	4	4
LV	4	13	4
LA	15	5	1
Pulmonary venous congestion	15	4	2

TABLE III
ELECTROCARDIOGRAPHIC FINDINGS IN 21 PATIENTS WITH SEVERE PULMONARY HYPERTENSION BEFORE AND AFTER MITRAL VALVE REPLACEMENT

ECG Finding	Before Operation	After Operation
Sinus rhythm	3	3
Atrial fibrillation	18	18
Right ventricular hypertrophy	21	9

DISCUSSION

Patients with severe pulmonary hypertension have been reported to be at two to three times greater

risk in the early postoperative period than those with lesser degrees of pulmonary hypertension undergoing identical surgical procedures (Starr, Herr, and Wood, 1967; Najafi *et al.*, 1969). Furthermore, the long-term prognosis of these patients has been described as poor (Barclay *et al.*, 1972). However, our results indicate that neither early nor long-term survival after mitral valve replacement is influenced by the severity of the pulmonary hypertension. Nine patients in our series died, but seven of these deaths resulted from pre- or postoperative complications, unrelated to the level of their pulmonary artery pressure, such as air embolism, haemorrhage, wound infection, and subacute bacterial endocarditis. Two patients died from respiratory failure in which the degree of pulmonary hypertension could have played a part. In the last five years only one patient with gross pulmonary hypertension has died during or following surgery.

In the 21 patients in whom postoperative haemodynamic measurements were made, the mean pulmonary arterial wedge pressure was normal at rest in 17. The rise in left atrial pressure in mitral valve disease has been described as the most significant factor in the development of pulmonary hypertension with consequent vasoconstriction of the pulmonary arterioles (Wood, 1958). Some cases are further complicated by organic changes in the pulmonary vasculature, but these changes are moderate (Harris and Heath, 1962) and are probably reversible after mitral valve surgery (Ramírez, Grimes, and Abelmann, 1968). Even severe degrees of pulmonary hypertension can regress markedly following adequate decompression of the left atrium after closed mitral valvotomy (Werkö *et al.*, 1953; MacKinnon, Wade, and Vickers, 1956; Emanuel, 1963), open mitral valvotomy (Zener *et al.*, 1972), and mitral replacement (Dalen *et al.*, 1967; Zener *et al.*, 1972). Indeed prosthetic valve replacement usually allows a better decompression of the left atrium than most closed mitral valvotomies (Braunwald *et al.*, 1965) and this is in accord with the near normal values for left atrial pressure that we obtained in this series at the time of recatheterization.

We have also observed a marked fall in pulmonary arterial pressure and in transpulmonary arteriovenous pressure gradient in these patients. The transpulmonary pressure gradient has been shown to be related linearly to pulmonary vascular resistance for a given pulmonary blood flow (Wood, 1954) and to be related to the severity of

the pulmonary hypertension. Although pulmonary vascular resistance is regarded as a useful index of severity of pulmonary hypertension, its validity has been questioned (Rudolph and Nadas, 1962) and its interpretation may be difficult (Fritts and Cournand, 1959; Braunwald, Braunwald, and Morrow, 1962). Hamer *et al.* (1968) suggested that higher values of PVR do not necessarily indicate more severe disease of the pulmonary vasculature. The concept of PVR may be valid for steady flow situations where the resistance varies with the pressure difference across the circuit, but higher values of PVR may be related to low cardiac output as well as to an increase in PAm-LAM (Hollinrake, Baidya, and Yacoub, 1973). Reeve *et al.* (1966) and Zener *et al.* (1972) have reported a significant fall in the PVR in the late postoperative period following mitral valve replacement in the presence of extreme pulmonary hypertension. Thus it appears that an early regression of pulmonary hypertension after mitral valve surgery is due to relief of the vasoconstrictive element due to a fall in the left atrial pressure. Eventual regression of pulmonary pressures and pulmonary vascular resistance in the late postoperative period to normal or near normal may be due to reversal of the organic changes in pulmonary arterioles and the medium-sized vessels.

CONCLUSION

The clinical improvement found in this series has been supported by haemodynamic, electrocardiographic, and radiological changes.

It seems from this study that patients with severe pulmonary hypertension are not at greater risk in the early or late postoperative period than those patients who have a lesser degree of pulmonary hypertension, while in both groups in recent years the mortality has been low. Furthermore, favourable long-term survival after mitral valve replacement has been accompanied by significant clinical improvement in these patients.

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- Requests for reprints to: T. K. Kaul, FRCS, Cardiothoracic Surgical Unit, Royal Infirmary, Glasgow G4 0SF.