

Rupture of posterior wall of left ventricle after mitral valve replacement

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ABSTRACT Possible aetiological factors, presentation, and management were reviewed in 18 patients with posterior left ventricular rupture complicating mitral valve replacement seen at one centre over six and a half years. The patients were elderly (mean age 57), predominantly women (16 of the 18), and suffering from mitral stenosis. Rupture was much more common after isolated replacement of the mitral valve (16 out of 797 operations) than after double valve replacement (one out of 236) or mitral valve replacement and coronary artery bypass graft (one out of 70). A total of 1221 mitral valve replacements were performed over this period, with an overall incidence of rupture of 1.47%. Damage to the valve annulus occurred five times. On four occasions haemorrhage followed a vigorous response to a bolus dose of an inotrope. With the exception of these features, it was difficult to define specific risk factors. Eleven patients bled while still in theatre; one of them survived long term and another four lived for four to 10 days. Repair after restarting cardiopulmonary bypass made short term survival much more likely. In seven rupture developed after return to the intensive therapy unit; again only one survived long term. In nearly all cases bleeding was at, or just below, the atrioventricular groove.

Rupture probably occurs after endocardial damage to a thin myocardium that has lost the internal buttress of the subvalvar apparatus. With the rise in intraventricular pressure at the end of bypass blood dissects into the myocardium, resulting in a large haematoma and eventual rupture.

Exsanguinating haemorrhage from the posterior aspect of the left ventricle after replacement of the mitral valve was first described by Roberts and Morrow in 1967.¹ The bleeding originated either from a tear at the atrioventricular groove in two cases or from the midpoint of the ventricular wall in a third. Treasure *et al* collected seven cases and described a dissecting haematoma as characteristic of the second type of rupture.² Bjork *et al* formalised this distinction into type I rupture, associated with the atrioventricular groove, and type II, related to the site of insertion of the posterior papillary muscle.³ He described eight cases and reviewed a total of 26 in previously published reports. Cobbs *et al* introduced the term transverse midventricular disruption and reiterated the important point that a dissecting haematoma may cause death from left ventricular

pump failure without going on to rupture.⁴ Miller *et al* argued that dissection in the area between the annular ring and the base of the papillary muscle should be termed type III. Several authors have described a third possible outcome⁶⁻⁸: that of false aneurysm of the left ventricle, when the rupture is contained by adhesions.

Patients at risk are commonly said to be frail, elderly women with the small ventricular cavity and thin myocardium of long standing mitral stenosis who sustain damage to a calcified annulus during excision of the native valve.^{3,4,9,10} The rupture or dissection may occur early, with the chest still open, or late, in the intensive therapy unit. We reviewed 18 patients, whose cases were well documented, seen at one centre over six and a half years. There were two long term survivors.

Methods

During the six and a half years reviewed 1221 (table 1) mitral valve replacements were performed, with

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Table 1 *Incidence of left ventricular (LV) rupture after mitral valve replacement*

Operation	n	No (%) LV rupture
First time MVR	797	16 (2.01)
First time MVR + AVR	236	1 (0.42)
MVR + CABG	70	1 (1.43)
Second time MVR	70	0
MVR + tricuspid procedure	48	0
Triple valve replacement	16	0
Total	1221	18 (1.47)

MVR—mitral valve replacement; AVR—aortic valve replacement; CABG—coronary artery bypass graft.

an incidence of left ventricular rupture of 1.47%. The operations were carried out by seven surgeons, all of whom encountered the complication. Most dissections occurred in 797 patients undergoing replacement of the mitral valve for the first time. Of these 797 patients, 38 (4.75%) died, 14 (37%) owing to rupture of the left ventricle. Although mortality for combined aortic and mitral valve replacement was nearly double that for isolated mitral valve replacement (8.05%), only one of 19 deaths was due to rupture of the left ventricle.

PREOPERATIVE FINDINGS

Posterior left ventricular rupture occurred in 18 patients, of whom 16 (88%) were women, aged 43–67 (mean 57), and two were men, aged 57 and 63 (table 2). Preoperative weight was known for 16 patients and ranged from 35 to 94 (mean 57) kg. Only one patient weighed more than 66 kg. Three patients, all women, were receiving thyroid

replacement treatment for previous hypothyroidism. They had been biochemically euthyroid (thyroid stimulating hormone concentration in the normal range) at the time of catheterisation three to six months before surgery.

Preoperative coronary arteriography was performed in 11 of the 18 patients and gave normal results in nine. Another two had their coronary vessels described as normal at necropsy, but no information existed on five. Only one of the patients with an abnormal coronary arteriogram required coronary artery bypass grafting, for tight stenoses of the left anterior descending and right coronary arteries.

Three patients had undergone four closed mitral valvotomies, nine, 10, 16, and 27 years previously. Two others had been offered this operation 15 and 20 years before mitral valve replacement. These long intervals indicate the chronicity of mitral valve disease in this group. Eight patients had predominant or pure mitral stenosis, and another eight were described as having mixed disease. Only two had predominant mitral incompetence and only two, one of whom required aortic valve replacement, had any degree of aortic valve disease.

INTRAOPERATIVE FINDINGS

Only four of the 18 patients had pericardial adhesions, which were completely divided by sharp dissection in three cases. The left ventricle was described as small in seven cases and the myocardium as soft, flabby, or thinned in eight. In four of eight patients with a calcified valve calcification extended into the annulus. The subvalvar apparatus was described as shortened or fused in 13.

Table 2 *Characteristics of patients and possible risk factors*

Case No	Age (years)	Sex	Other disease	Cardioplegia	Annular damage	Valve type	Bolus inotropes ((+) hypertension)
EARLY RUPTURE							
1	50	F			+	BS	
2	55	F				BS	
3	63	F	CAD		+	CE	+
4	57	F			+	BS	
5	62	M	MVY		+	CE	
6	62	F		+		BS	
7	43	F		+		BS	
8	54	F	RTRT AI	+		CE	+
9	65	F		+		BS	
10	45	F	MVY	+		CE	
11	57	F		+		CE	+
LATE RUPTURE							
12	63	M		+		CE	
13	57	F		+		CE	
14	58	F		+		BS	+
15	56	F		+		CE	
16	63	F		+		CE	
17	61	F	RTRT/CAD/MVY	+	+	BS	(+)
18	57	F	RTRT	+		BS	(+)

CAD—coronary artery disease; MVY—mitral valvotomy; AI—aortic incompetence; RTRT—receiving thyroxine replacement treatment; CE—Carpentier-Edwards valve; BJ—Bjork-Shiley valve.

Table 3 Management and outcome of rupture occurring early (while patient was still in theatre)

Case No	Time to onset (min)	CPB restarted	Other manoeuvres	Reached ITU	Outcome
1	0				DOT, never off CPB
2	10			+	Rebled in ITU, died at 2 hours
3	30	+	Cx Graft, IABP	+	Renal failure, died at 10 days
4	5	+	Cx Graft, IABP	+	LCO, died at 4 days
5	5	+		+	Renal failure, died at 8 days
6	5				DOT massive disruption
7	10	+	IABP	+	Survived
8	20				DOT (bled after decannulation)
9	10			+	Rebled in ITU, died at 8 hours
10	10	+	Transatrial repair		DOT, massive disruption
11	20	+	Transatrial repair	+	LCO, died at 4 days

CPB—cardiopulmonary bypass; Cx graft—bypass graft to a branch of the circumflex coronary artery; IABP—intra-aortic balloon pump; ITU—intensive therapy unit; DOT—death on table; LCO—low cardiac output.

THE OPERATION

Access to the heart was by median sternotomy, and standard cardiopulmonary bypass with ascending aortic and bicaval cannulation was used. Pulsatile perfusion has been part of our routine since 1978. Five patients (cases 1–5) had ischaemic arrest with or without topical hypothermia, but cold potassium cardioplegia was used with the other 13. Ten patients received Björk-Shiley tilting disc prosthetic valves and eight received Carpentier-Edwards porcine heterograft valves (table 2). These were the standard prostheses during the period under review. An interrupted suture technique was used in 10 patients, and varying degrees of continuous suture in eight, all of whom had received Björk-Shiley valves. Apical venting of the left ventricle was performed in 11 patients, of whom eight had Björk-Shiley valves. A soft cannula was used for this purpose, and the metal vents criticised by Björk *et al* were never inserted into the ventricle.³

Damage to the annulus during excision of the native valve was recognised on five occasions. In four it was related to calcification extending beyond the valve leaflets. One had what was realised to be over zealous resection of a small annulus.

MANAGEMENT OF DISSECTION

The predominant manifestation was haemorrhage,

Table 4 Management and outcome of rupture occurring late (after patient had returned to intensive therapy unit)

Case No	Time to onset (min)	CPB restarted	Repair attempted	Outcome
12	5			Died
13	1		+ Direct suture	Survived
14	36			Died
15	20			Died
16	2	+	+ Transatrial repair	Died
17	20			Died
18	8			Died

except in one patient (case 1), in whom a huge dissecting haematoma caused left ventricular pump failure without rupture. She could not be weaned from bypass and died on the table. Including this patient, rupture occurred in 11 while they were still in theatre with the chest open; these patients thus sustained “early” dissection. The remaining seven had unheralded bleeding after their return to the intensive therapy unit; they thus sustained “late” dissection.

Early rupture occurred in 11 patients (table 3), in all instances within 30 minutes after stopping bypass. Four died in theatre, including one with only a large haematoma. Bypass was restarted in one of these, but the ventricle was irretrievably damaged. Seven had a repair that permitted return to the intensive therapy unit, and one of these subsequently survived. Four lived for from four to 10 days before succumbing to the effects of low cardiac output. If possible, bypass was restarted and the heart decompressed. If the heparin was reversed and the patient decannulated attempts were made to suture the tear in the beating heart. In most cases repair was attempted from the outside, using pledged sutures for small holes or buttresses of strips of Teflon for larger tears. Definite damage to the circumflex coronary artery was recognised at the time in two patients and a saphenous vein graft applied to a distal branch. Both these patients lived for more than a week. The circumflex coronary artery or its branches and also the coronary veins probably suffered damage during unsuccessful attempts at repair. Indeed, both the long term survivors had evidence of perioperative myocardial infarction. Early return to bypass would seem to be an important step in attempting repair, as table 3 shows.

Late rupture Seven patients suffered unheralded, catastrophic haemorrhage in the intensive therapy unit (table 4). The latent period after operation

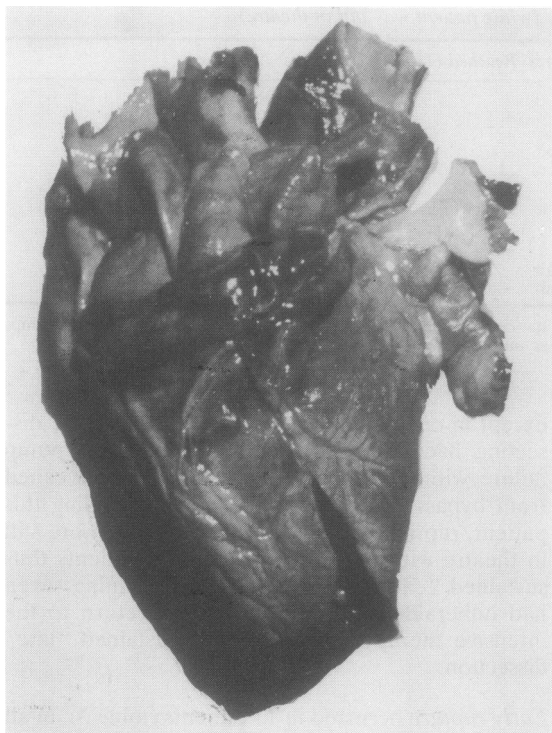


Fig 1 Haematoma in fat of atrioventricular groove. An extensive tear can be seen on the epicardial surface.

ranged from one to 36 hours. Six of them died, three before any useful attempt at repair could be made. One patient bled almost immediately on return to the intensive therapy unit; there was a well demarcated tear 3 cm from the atrioventricular groove on the posterior wall of the ventricle. It proved possible to control this with buttressed sutures; the chest was packed, and after a stormy course the patient made a good recovery. In the patient who bled at two hours (case 16) cardiopulmonary bypass was restarted in the intensive therapy unit and the valve removed. Despite repair under direct vision, haemorrhage and extension of the haematoma occurred when discontinuation of bypass was attempted.

SITE OF RUPTURE

In 15 of our patients the dissection reached the epicardial surface at, or just below, the atrioventricular groove. In many the spreading haematoma beneath the fat in this area made precise localisation difficult (fig 1). Of the other three patients, one had only a large dissecting haematoma and one a vertical tear, originating in the atrioventricular groove but running towards the apex. The third, who survived a repair without resort to bypass, had a hole in the

ventricle 3 cm from the atrioventricular groove. All three had haematoma surrounding the tear and all had damage to the annulus recognised early in the operation.

Discussion

The incidence of posterior left ventricular rupture is usually said to be 0.5–2% of all mitral valve replacement operations. Our figure of 1.47% fell within this range. The complication was commonest in first time mitral valve replacements and in this group was the most important cause of perioperative death. The coexistence of aortic valve disease seemed to have a protective effect, possibly because the thickened or hypertrophied ventricular myocardium was resistant to tearing. Associated ischaemic heart disease was not a risk factor as most patients had normal coronary arteriograms.

In general terms, the patients in our review conformed with the impression given by other series.^{2–4,9,10} They tended to be frail, elderly women, with predominant mitral stenosis. That three patients were receiving thyroid replacement treatment supports our clinical impression that these patients do badly after surgery. The left ventricle was described as small and the myocardium “poor” in many cases, although these judgements were usually made retrospectively. These features did not, however, separate the patients who sustained rupture in any definite way from the bulk of our patients undergoing mitral valve replacement, who also tended to be women in late middle age. Preoperative risk factors would seem to be general rather than specific and may merely predispose some groups of patients to the possibility of subsequent mishap.

A large number of intraoperative manoeuvres have been blamed for precipitating rupture. Early reports suggested simple mechanical causes, such as excessive resection of the annulus, particularly during second time surgery,^{3,11} or “button holing” of the ventricle during division of papillary muscles.^{2,3,9–11} The calcified annulus is especially susceptible to damage.^{2–4,10} It would seem, however, that in none of our patients was there a type II rupture with the implication of damage at the root of the papillary muscle. Only one of our patients had undivided adhesions posteriorly, although many have considered this to be important.^{2,3,11} The struts of heterograft valves have been suggested as initiating the dissection, particularly if the apex of the ventricle is raised.^{4,9,12} Others have denied that this is of any importance.¹¹ In our series roughly equal numbers of tilting disc and bioprosthetic valves were inserted. Bioprosthetic valves were not associated

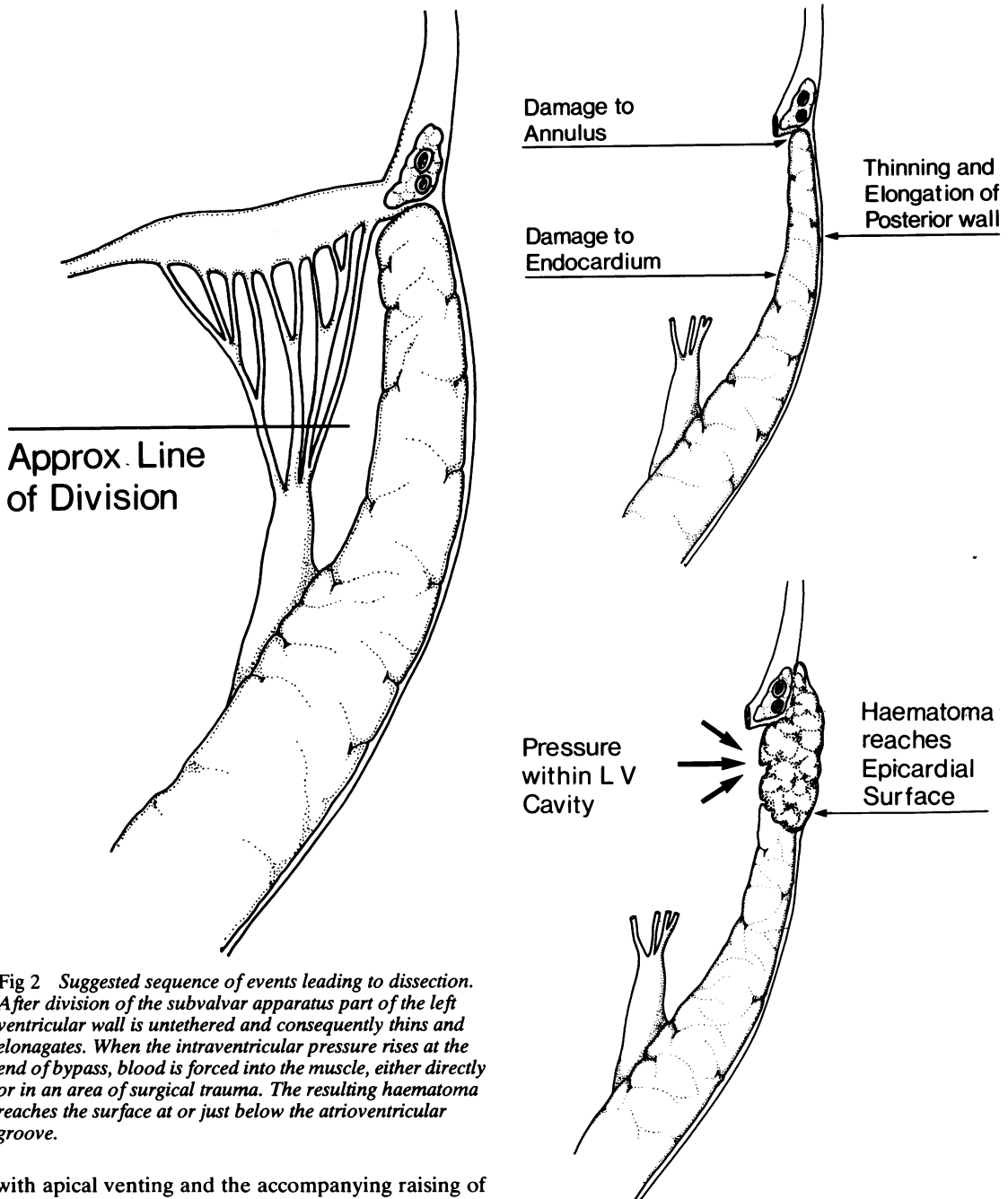


Fig 2 Suggested sequence of events leading to dissection. After division of the subvalvar apparatus part of the left ventricular wall is untethered and consequently thins and elongates. When the intraventricular pressure rises at the end of bypass, blood is forced into the muscle, either directly or in an area of surgical trauma. The resulting haematoma reaches the surface at or just below the atrioventricular groove.

with apical venting and the accompanying raising of the heart. In only one case did the surgeon feel that the site of the endocardial tear was related to a heterograft valve strut.

The potentially harmful effects of intraoperative hypertension in this context were first pointed out by Katske *et al.*⁹ In our series the incidence of the use of an inotrope immediately before the appearance of

bleeding was striking. Five patients (table 2) were given a bolus dose of inotrope (adrenaline in four), in all of whom cardiac action subsequently became very vigorous: in four instances bleeding supervened and the other patient bled after 36 hours. A further two patients who sustained late rupture had systolic



Fig 3 Encocardial aspect of heart shown in figure 1. The initial horizontal breach in the myocardium can be seen several millimetres below the valve ring. Note also that the chordae tendinae are divided well beyond the tips of the papillary muscles.

hypertension that required vasodilator treatment (unusual after mitral valve replacement) in the post-operative period. Finally, three of the patients who bled in the intensive therapy unit did so after being washed, turned, or extubated, manoeuvres which were noted to cause a rise in systolic blood pressure.

The patients who required adrenaline in theatre probably already had a dissection that was interfering with left ventricular function. We would suggest that the atrioventricular groove is palpated before resorting to this step in a patient who is slow to come off bypass. Furthermore, the patients who bled late after a hypertensive episode may also have been harbouring a contained haematoma. Björk *et al* described a patient with haematoma in the atrioventricular groove that neither interfered with function nor went on to rupture. The same phenomenon has been seen several times by one of us (WHB).

Katske *et al* were the first to suggest that the elongation of the left ventricle, which occurs when the valve is detached from the papillary muscles, might expose an area of weakness.⁹ This was

appropriately termed "untethering" of the ventricle by Cobbs *et al*.⁴ The concept was further developed by Miller *et al*, who went as far as leaving part of the posterior leaflet in place to avoid weakening the wall.⁵

Thus a set of circumstances, none of them unique or specific, can easily be imagined that together might lead to rupture. Detachment of the papillary muscles leaves an area of left ventricular wall relatively unsupported. If the patient is frail and elderly and her mitral valve stenotic the myocardium will be relatively thin already. Operative trauma either at the annulus, or just below, when the fused subvalvar apparatus is divided breaches the endocardium. Alternatively, the tear occurs *de novo* when intracavitary pressure rises at the end of the bypass. Then, as the heart takes over the circulation, blood is forced into the muscle and the dissection starts. It is more likely to proceed if the heart beats vigorously and develops a high systolic pressure, either due to inotropes or raising of afterload (fig 2). Cardioplegia and the very flaccid heart may make the initial breach of endocardium more likely, as may the traction required if posterior adhesions are not fully divided.

If this argument is accepted the finding of disruption in the left ventricular wall, even though the haematoma externally is at the atrioventricular groove, is easily explained. The findings illustrated in figure 3, with a horizontal tear and dissected myocardium below rather than extending from the valve ring, are common. Although most if not all of our cases appear to be of Björk's type I, it may well be that many should really be described as type III (Miller *et al*⁵) or transverse midventricular disruption (Cobbs *et al*⁴).

Once rupture has occurred attempts to suture the tear by raising the beating heart, particularly if it contains a heterograft valve, will probably just extend the dissection. Table 3 shows the importance of restarting bypass promptly. Sutures should be buttressed with large pledgets or strips of Teflon. In theory, delineation of the dissection inside the heart by removing the valve should permit more accurate repair, but this was not borne out by our results. Celemin *et al* recently described an intraventricular pericardial patch to cover the ventricular aspect of the tear, apparently with good results.¹³

Successful repair, particularly of a tear in the atrioventricular groove, is likely to compromise the circumflex coronary artery. Both the long term survivors from this series had evidence of perioperative infarction. Two patients had bypass grafts to distal branches of the circumflex and both lived for more than a week. This would seem to be an important step if vascular damage is suspected. The intra-

aortic balloon pump was used in three patients, including one of the survivors. It facilitates reduction of afterload, and this would theoretically help to maintain the integrity of a repair.

CONCLUSION

Rupture of the wall of the left ventricle would appear to be a rare but unavoidable accompaniment of mitral valve replacement. Factors resulting in a fragile myocardium, together with operative trauma, seem to be important in the aetiology. In particular, the use of bolus doses of inotropes may precipitate an incipient rupture. If a tear occurs bypass should be reinstated if at all possible and repair performed on a decompressed heart. Bypass grafting to distal branches of the circumflex coronary artery and reduction of afterload with the intra-aortic balloon pump may be helpful.

We thank the cardiac surgeons of Glasgow for permission to report details of their patients.

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