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Myocardial rupture after acute myocardial infarction
Ten year review

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SUMMARY Forty-nine patients with myocardial rupture complicating acute myocardial infarction were managed in our coronary care unit from 1972 to 1981: 33 patients with post-infarction ventricular septal defect, 12 patients with isolated rupture of the free wall of the left ventricle, and four patients with papillary muscle rupture. Nine of 19 patients (47%) who underwent surgical repair of a post-infarction ventricular septal defect survived. The major determinant of survival was the preoperative haemodynamic status. Ten of 13 patients (77%) who developed cardiogenic shock preoperatively died, while none of the six patients who were not in cardiogenic shock died. Survival was not related to the site or size of infarction, extent of coronary artery disease, or magnitude of the left to right shunt. There were no survivors among the 14 patients with post-infarction ventricular septal defect managed without surgical intervention. Seven of the 12 patients with isolated rupture of the free wall of the left ventricle developed mechanical cardiac arrest and died at the onset of rupture, but five patients developed subacute heart rupture and two of these patients survived after urgent surgical repair. Two of the four patients with papillary muscle rupture underwent mitral valve replacement, but both died in the early postoperative period; both patients who were not operated on died. Early detection and early surgical intervention are essential in the management of myocardial rupture complicating acute myocardial infarction.

Rupture of the myocardium after acute myocardial infarction may involve the free wall of the ventricle, the interventricular septum, or the papillary muscles. Rupture of the free wall usually results in an acute haemopericardium with apparent cardiac arrest despite continuing electrical activity, though "subacute" rupture may result in a syndrome resembling cardiogenic shock or, rarely, in the development of a pseudoaneurysm. Septal rupture results in the creation of a left to right shunt, while papillary muscle rupture causes acute mitral regurgitation.

As a result of the improved treatment of ventricular arrhythmias during the early phase of acute infarction, myocardial rupture has become, after myocardial power failure, the most common cause of in-hospital death.[1 2] Despite major advances in surgical technique over the past 15 years, there is still great controversy regarding the optimum time for surgical intervention and the role of preoperative cardiac catheterisation, concomitant coronary revascularisation, and arterial counterpulsation in the management of this problem. We report here our experience of myocardial rupture during the past 10 years and the management approach we have evolved during this time.

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Patients and methods

During the period 1972 to 1981, a total of 8105 patients were admitted to our 30 bed coronary care unit; 2581 (32%) of these had evidence of a definite acute myocardial infarction by WHO criteria.[3] Review of these latter cases disclosed 49 patients with myocardial rupture confirmed at operation or at necropsy. Thirty-three of these patients had a post-infarction ventricular septal defect, two with associated free wall rupture and haemopericardium, and four with associated haemopericardium without apparent free wall rupture. Twelve patients had isolated free wall rupture and four had papillary muscle rupture (Table 1). Twenty of the patients with a post-infarction ventricular septal defect and five of the patients with free wall rupture had been transferred from other hospitals because of acute haemodynamic deterioration.

Cardiogenic shock was defined as systolic arterial

Table 1 *Myocardial rupture after acute myocardial infarction*

		No. of cases
Ventricular septal rupture		33
Isolated septal rupture	27	
Septal rupture with haemopericardium	4	
Septal rupture with free wall rupture	2	
Isolated free wall rupture		12
Papillary muscle rupture		4
Total		49

pressure <80 mmHg, accompanied by oliguria (urine output <20 ml/h for four hours or more).[4] The statistical significance of differences between mean values was assessed by Student's t test. Differences between discrete variables were analysed by Fisher's exact probability test. Results are expressed as mean (range) or mean ± standard error of the mean.

Results

POST-INFARCTION VENTRICULAR SEPTAL DEFECT

The clinical data of the 33 patients with a post-infarction ventricular septal defect are summarised in Table 2. Notable are the older age (mean=65 years)

Table 2 *Post-infarction ventricular septal defect—total group*

No.	33
Age (y)	65 (50–81)
Male:female	19:14
Antecedent hypertension	12
Previous infarction	5
Site of infarction	
Anterior	17
Inferior	13
Anterior and inferior	3
Peak daily serum CK (IU/l)	1100 (412–2145)
Delay to onset of septal rupture (d)	2·6 (0·25–10)
Qp/Qs	3·4:1 (1·6–6·8:1)

Qp/Qs, pulmonary to systemic flow ratio.

and the relatively high proportion of women compared with the general population of patients suffering acute infarction. Septal rupture was associated with a wide variation in serum creatine kinase (CK) levels, in agreement with previous reports,[5,6] and a similar incidence of anterior and inferior infarction. The onset of septal rupture ranged from as early as six hours to as late as 10 days after infarction.

Eight of the 33 patients were rejected from surgical management. Five of these eight patients were more than 70 years old and three had been in cardiogenic shock for more than 24 hours at the time of initial assessment, having been transferred from other hospitals. A further patient was not diagnosed until necropsy.

Twenty-four patients were initially accepted for surgical management. Four of these patients died during the first 24 hours after septal rupture; three died despite arterial counterpulsation, initiated in an attempt to achieve haemodynamic stabilisation preoperatively, and one patient died during preoperative cardiac catheterisation. A further patient died suddenly eight days after septal rupture, while awaiting late elective surgical repair; necropsy disclosed acute free wall rupture in addition to the ventricular septal defect.

Thus, 19 patients finally proceeded to surgical repair. Thirteen of these patients underwent preoperative cardiac catheterisation (Table 3). There

Table 3 *Cardiac catheterisation data*

Case No.	*Extent of CAD*	*LVEF*	*Qp/Qs*
Survivors			
1	LAD	61%	2·8:1
2	LAD	40%	2·5:1
3	LAD, RCA	6%	1·6:1
4	Cx, RCA	67%	5·3:1
5	LAD, RCA	56%	4·1:1
Non-survivors			
6	LAD, RCA	—*	2·5:1
7	TVD	—*	2·4:1
8	LAD	57%	2·5:1
9	RCA	48%	2·2:1
10	TVD	26%	2·0:1
11	LAD	18%	5·0:1
12	LAD, RCA	22%	—*
13	LAD	12%	1·7:1

CAD, coronary artery disease; LVEF, left ventricular ejection fraction; Qp/Qs, pulmonary to systemic flow ratio; LAD, left anterior descending artery; RCA, right coronary artery; Cx, circumflex artery; TVD, triple vessel disease.
*Not calculated.

was no significant difference in the extent of coronary artery disease between the five catheterised survivors (mean 1·6 diseased vessels), and the eight catheterised non-survivors (mean 1·8 diseased vessels). With the exception of case 11, a high pulmonary to systemic flow ratio generally correlated with a higher left ventricular ejection fraction and a low pulmonary to systemic flow ratio correlated with a lower left ventricular ejection fraction. Further, the left ventricular ejection fraction and pulmonary to systemic flow ratio tended to be higher in the survivors, though there was considerable overlap between the groups in these variables. The very low left ventricular ejection fraction of case 3 was attributable to a large left ventricular aneurysm which was successfully resected.

Surgical repair consisted of left ventriculotomy through the region of infarction, Dacron patch grafting of the left side of the ventricular septum over Teflon buttressed sutures, and infarctectomy/aneurysmectomy.[7] One patient required concomitant mitral valve replacement. At operation, one patient was found to have associated free wall rupture with

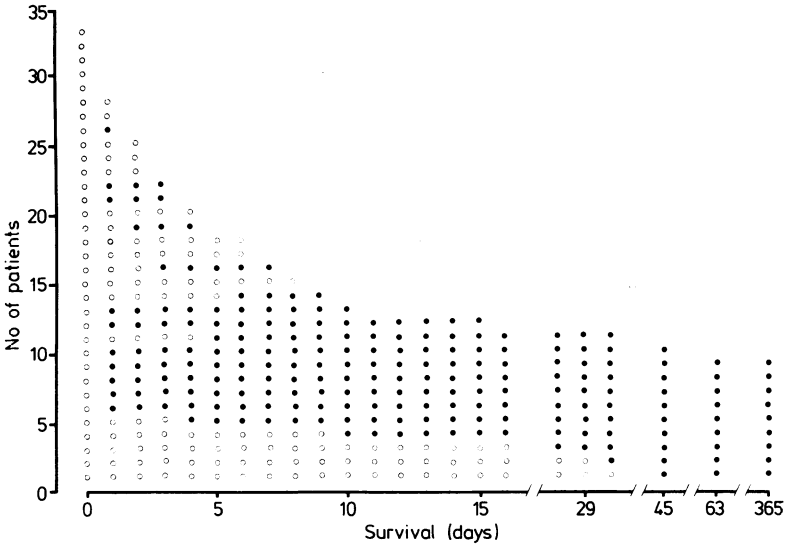

Fig. Post-infarction ventricular septal defect survival—total group. Each open circle (○) in the first column represents one of the patients at the onset of septal rupture. If surgical intervention occurred, the patient is then represented by a solid circle (●), starting on the day of surgery. Disappearance from the chart indicates death of the patient.

haemopericardium and four further patients had haemopericardium with imminent free wall rupture; two of these developed free wall rupture intraoperatively.

The early mortality of the total group of 33 patients (Fig.) was extremely high, 55% of patients dying within one week of septal rupture. Most early deaths (13 of 17) occurred in those patients managed without surgical intervention. Only four of the 33 patients survived longer than eight days without surgical intervention. The overall survival was only 27%, all nine survivors having undergone surgical repair. The early mortality of the 19 surgically treated patients was less than that of the total group, 26% (5 of 19) dying within one week of septal rupture. Nine of the 19 surgically treated patients (47%) survived to hospital discharge. All four patients who underwent late repair, between day 10 and day 45, survived. The remaining 15 patients underwent earlier operative intervention within one week of septal rupture and five survived: four of 10 patients who underwent repair within 24 hours and one of five patients who

Table 4 *Post-infarction ventricular septal defect (VSD): surgically treated patients*

	Survivors	*Non-survivors*	*Probability*
No.	9	10	
Age (y)	61±2·5	61±2·2	NS
Male:female	6:3	6:4	NS
Antecedent hypertension	6	2	NS
Previous infarction	1	1	NS
Site of infarction			
Anterior	5	7	
Inferior	4	2	NS
Anterior and inferior	0	1	
Peak daily serum CK (IU/l)*	1059±173	1220±160	NS
Delay to onset of septal rupture (d)	2·8±0·8	1·6±0·6	NS
Systolic BP when murmur first noted (mmHg)	109±11·5	95±7·6	NS
Qp/Qs	3·5:1 (1·6–6·8:1)	2·8:1 (1·7–5·6:1)	NS
Extent of CAD† (13 patients)	1·6	1·8	NS
Systolic BP preoperatively (mmHg)	86±6·7	60±7·6	<0·05
Preoperative oliguria	3	10	<0·05
Preoperative counterpulsation	2	7	
No. of patients revascularised	5	2	
Recurrent postoperative VSD	3	3	

*Normal value ≤130 IU/l.
†No. of vessels with >50% stenosis.
NS, not significant.

underwent repair between day 1 and day 6.

The clinical data of the surgically treated patients are presented in Table 4. Survivors could not be distinguished from non-survivors on the basis of age, sex, site of infarction, peak serum CK level, systolic arterial pressure at onset of ventricular septal defect, or the size of the left to right shunt. By the time of surgical repair, however, all of the 10 non-survivors were in established cardiogenic shock (systolic arterial pressure 60±7·6 mmHg with extreme oliguria), and seven of these patients were on counterpulsation support. In contrast, the preoperative systolic arterial pressure of the nine survivors was significantly higher (86±6·7 mmHg, $p<0·05$), and, though three survivors developed acute oliguria, surgery was performed within six hours of the haemodynamic deterioration. Only two of the non-survivors received preoperative counterpulsation support. Hence, the immediate preoperative haemodynamic status of these patients appears to have been the major determinant of survival, but this difference in haemodynamic status was not apparent at the onset of septal rupture.

Concomitant coronary revascularisation was performed in seven patients: five of the nine survivors and two of the 10 non-survivors. There was no difference, however, in the extent of coronary artery disease between survivors and non-survivors in the 13 patients in whom this was assessed and perioperative infarction was not shown in any of our patients.

Six patients developed recurrent ventricular septal defect between day 1 and day 6 after surgical repair. The site of infarction had been anterior in four patients and inferior in two. Reoperation was performed in two cases, one patient surviving to hospital discharge. Two patients survived to hospital discharge without reoperation.

All nine hospital survivors were still alive after the minimum follow-up period of one year and six are still alive after a mean follow-up period of five years (range one to nine years). One patient died suddenly two years after discharge and two patients died after a further acute myocardial infarction four years after discharge. Two of these latter patients had undergone concomitant coronary revascularisation. All six remaining survivors are in New York Heart Association functional class I–II.

FREE WALL RUPTURE

The clinical data of the 12 patients with isolated free wall rupture are presented in Table 5. Like patients with septal rupture, they tended to be older, with a higher proportion of women than the general population of patients suffering acute infarction. Again, a wide variation in peak serum CK levels was noted and the onset of rupture varied from two hours to 11 days after infarction.

Table 5 *Free wall rupture*

No.	12
Age (y)	61 (45–81)
Male:female	7:5
Antecedent hypertension	4
Previous infarction	2
Site of infarction	
Anterior	5
Inferior	2
Anterior and inferior	2
Postero-lateral	2
Infero-postero-lateral	1
Peak daily serum CK (IU/l)	1168 (332–2140)
Delay to onset (d)	3 (2 hr–11 d)
Mode of onset	
Sudden death	7
Subacute heart rupture	5
No. of survivors	2

In seven patients, the onset of free wall rupture was manifested by apparent cardiac arrest, characterised by electromechanical dissociation, and the diagnosis was made at necropsy. Recurrent chest pain usually heralded the onset of rupture. The remaining five patients were transferred from other hospitals with a diagnosis of cardiogenic shock and were treated by arterial counterpulsation. The correct diagnosis was not made until necropsy in two of these patients, one of whom died on the day of admission and the other, four days after admission. The diagnosis of subacute heart rupture was made in one case after insertion of an intra-arterial cannula, when arterial paradox was noted. He recovered transiently after pericardiocentesis, but rapidly deteriorated and died because of rapid reaccumulation of haemopericardium. Another patient was diagnosed when arterial paradox and Kussmaul's sign were noted several hours after the start of counterpulsation, and angiocardiography was performed immediately, echocardiographic facilities not at the time (1972) being available. During left ventriculography, contrast material was seen to enter the pericardial sac, associated with further acute haemodynamic deterioration. He underwent immediate open pericardial drainage and infarctectomy and is still alive and well today, 10 years later, now aged 73 years. The last patient, in 1981, was correctly diagnosed at the time of initial clinical assessment and the diagnosis was confirmed echocardiographically, followed by immediate surgical repair. He is still alive and well after 21 months follow-up, aged 48 years. Coronary revascularisation was not attempted in either patient.

Hence, a premortem diagnosis of free wall rupture was made in only three cases and was strongly suspected at the time of initial clinical assessment in only one case. Nevertheless, review of the case records of all five patients with subacute heart rupture disclosed that systemic venous hypertension was prominent with little or no clinical or radiological evidence of left

ventricular failure, a finding that should have suggested the correct diagnosis.[8] Importantly, both patients in whom immediate surgical repair was instituted are alive and well today.

PAPILLARY MUSCLE RUPTURE

Only four patients, three men and one woman, aged 53 to 75 years, were identified with papillary muscle rupture during the period of review. All patients had suffered an inferior myocardial infarction and all developed a new systolic murmur and acute left ventricular failure, preceded by recurrent chest pain, from several hours to 13 days after infarction. Two patients underwent mitral valve replacement after cardiac catheterisation, but both died in the early postoperative period. One patient was managed conservatively because of his advanced age (75 years) and the diagnosis was confirmed at necropsy. The final patient was diagnosed only at necropsy, though review of the case record showed the typical clinical features listed above. All four patients with acute papillary muscle rupture died.

Discussion

Nine of 19 patients (47%) who underwent repair of a post-infarction ventricular septal defect survived. This compares well with the experience of other centres.[7 9 10] The major determinant of survival was the immediate preoperative haemodyamic status of the patients. In this finding, we are in complete accord with Radford *et al.*[11] who found that the major determinant of perioperative mortality in their patients was the presence of cardiogenic shock, only 27% of their shocked patients surviving, compared with 82% survival in those who were not shocked preoperatively. Forfar *et al.*[12] reported similar findings. Importantly, we were not able to distinguish surgical survivors from non-survivors at the time of initial diagnosis of septal rupture.

The poor predictive value of the pulmonary to systemic flow ratio in post-infarction ventricular septal defect has been noted previously.[9 11 12] We believe this finding probably reflects the dependence of the pulmonary to systemic flow ratio on the interplay between the left ventricular ejection fraction and the anatomical size of the ventricular septal defect. Thus, an identical pulmonary to systemic flow ratio might be produced by a small anatomical defect in the presence of a high left ventricular ejection fraction (presumably, a high chance of survival) and by a large anatomical defect in the presence of a low left ventricular ejection fraction (presumably, a low chance of survival). This hypothesis would also explain the inability of Radford *et al.*[11] to demonstrate any predictive value for the left ventricular ejection fraction or the combination of the left ventricular ejection fraction and pulmonary to systemic flow ratio, as the anatomical size of the ventricular septal defect was not considered.

Previous reports[5 7 9] have noted the site of myocardial infarction and hence the site of septal rupture to be a major determinant of survival, reporting lower survival in those patients with inferior infarction and posterior ventricular septal defects. The reasons given for this have been the greater difficulty of adequate repair of posterior ventricular septal defects and the more frequent involvement of the papillary muscles, sometimes necessitating mitral valve replacement. Surgical survival, however, was not related to the site of infarction in our series of patients and only one patient required mitral valve replacement. Similarly, the incidence of recurrent ventricular septal defect postoperatively was not higher in those with inferior infarction. This possibly reflects the uniformity of our surgical approach; that is all septal defects, whether anterior or posterior, were approached via a left ventriculotomy with Dacron patching of the left side of the septum. Many of the posterior defects reported by others have been approached via a right ventriculotomy with patching of the right side of the septum. The disadvantages of this latter approach have been discussed previously by Loisance *et al.*[7] Crosby *et al.*,[13] using the same surgical approach as our unit, have also reported a more favourable outlook for surgical repair of posterior ventricular septal defects.

The optimum time for surgical repair of septal rupture remains controversial. Because of the higher mortality of their patients undergoing early repair, Giuliani *et al.*[14] and the Boston group[10] previously suggested that surgery should be deferred until at least three weeks after myocardial infarction, if possible. We contend, however, in agreement with others[5 7 9] and most recently the Boston group,[11] that such an approach may not produce maximal survival. Only four of our 33 patients survived longer than eight days without surgical intervention. None of these four patients required any preoperative haemodynamic support and, not surprisingly, all survived late, semielective surgical repair. In contrast, the remaining 15 surgically treated patients required earlier surgical intervention because of the development of refractory cardiac failure or shock, nine patients receiving preoperative counterpulsation support. The major determinant of survival in these patients was the immediate preoperative haemodynamic status, rather than the timing of surgery. Those five early operated patients who were not in prolonged cardiogenic shock survived. Further, five of the early operated patients had associated haemopericardium, and free wall rupture was present in one case and developed intraoperatively in two further cases. None of these patients would have

survived without early surgical intervention.

As the diagnosis of septal rupture can now be confirmed rapidly by Swan-Ganz catheterisation at the bedside, angiographic diagnosis is no longer essential. The hospital mortality of these patients cannot be related to the extent of coronary artery disease in our experience, nor in the experience of others[7 9 15]; nor has concomitant coronary revascularisation been shown to improve survival. Daggett *et al.*[10] strongly advocate revascularisation, though there was no improvement in the hospital survival of their revascularised patients and only one late death occurred in their group of non-revascularised patients.[7] Similarly, Williamson *et al.*[15] were not able to show any benefit from revascularisation. The whole question of whether revascularisation of patients with established acute infarction is of any value remains highly debatable.[16]

We now believe that for those patients who are deteriorating rapidly after acute septal rupture, prompt surgical repair, after confirmation of the diagnosis by Swan-Ganz catheterisation, offers the best chance of survival and should not be delayed by the performance of left ventriculography or coronary angiography. Further, while arterial counterpulsation was a valuable supportive measure in our experience, early surgical repair would obviate the need for this measure preoperatively in most cases. Arterial counterpulsation remains an invaluable postoperative supportive measure in many cases. For those patients who are initially haemodynamically stable after septal rupture, preoperative coronary angiography and, if indicated, coronary revascularisation at the time of septal repair seem reasonable procedures, in the absence of conclusive data on the question of revascularisation.

Nevertheless, in our experience, the great majority of patients who develop a postinfarction ventricular septal defect die without early surgical intervention, and it is not possible to identify at the time of septal rupture the small number of patients who will survive long enough to undergo late surgical repair. Hence, we now advocate surgical repair within 24 hours of septal rupture, even in those patients who initially appear haemodynamically stable. The results of very early surgical repair reported by others tend to support this viewpoint. Loisance *et al.*[7] reported four deaths in 17 patients undergoing early repair (mean 46 hours) compared with three deaths in seven late operated patients (mean 12 days). Montoya *et al.*[5] reported a 72% survival in seven patients undergoing repair less than two days after septal rupture compared with four survivors of 11 patients operated on two to 28 days after rupture.

Importantly, the long-term prognosis of patients who survive surgical repair of a post-infarction ventricular septal defect is comparable with that of post-infarction patients without mechanical complications. Though three of our patients died two and four years after repair of their ventricular septal defects, their deaths were attributable to their underlying coronary artery disease (sudden death or further acute myocardial infarction). All three had been in New York Heart Association functional class I-II before their subsequent coronary event.

Though little can be done for those patients who develop mechanical cardiac arrest at the onset of free wall rupture, an appreciable number of patients develop subacute heart rupture, surviving long enough after the onset of rupture to be potentially salvageable. We have previously[8] stressed that the classical features of cardiac tamponade are unreliable in this setting and that one should suspect the diagnosis in any patient who, in the post-infarction period, develops the sudden onset of hypotension, often heralded by recurrent chest pain, associated with prominent systemic venous hypertension in the absence of significant left ventricular failure. This was the case in all five of our patients with subacute heart rupture. The diagnosis can now be confirmed rapidly by the echocardiographic demonstration of fluid in the pericardial cavity, followed by immediate surgical repair. Coronary revascularisation is not necessary for good long-term survival in these patients. Hence, preoperative coronary angiography is not necessary and wastes valuable time. Contrast ventriculography is also unnecessary, since the advent of echocardiography, and may well be hazardous in this situation. The excellent prognosis of those patients who can be diagnosed and transferred to surgery rapidly justifies an aggressive approach to the detection and management of this condition.

Papillary muscle rupture has been a distinctly rare occurrence in our experience when compared with septal rupture, though these two conditions are usually said to have the same incidence.[6] It is not really possible to distinguish these two conditions on clinical grounds alone, but Swan-Ganz catheterisation will usually resolve the issue. Our small experience of only four cases precludes any general conclusions. The high association of this condition with inferior infarction was borne out in our small sample.

We wish, in conclusion, to emphasise: (1) the relatively common occurrence of myocardial rupture after acute myocardial infarction, (2) its potentially remediable nature, and (3) the good long-term prognosis of those patients in whom successful surgical repair can be achieved. In these conditions, early diagnosis and early surgical intervention are essential in achieving maximal patient survival. The advent of Swan-Ganz catheterisation and echocardiography, together with arterial counterpulsation and advances in surgical

technique, have made these objectives attainable.

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