

Elective operations for post-infarction left ventricular aneurysms

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Sbokos, C. G., Monro, J. L., and Ross, J. K. (1976). *Thorax*, 31, 55–62. **Elective operations for post-infarction left ventricular aneurysms.** During a two-year period (February 1973 to February 1975) 20 consecutive patients with post-infarction left ventricular aneurysm, seen at the Wessex Cardiac and Thoracic Centre, underwent aneurysmectomy with or without aorta-to-coronary artery saphenous vein bypass grafts, ventricular septal defect closure, or valve replacement.

The diagnoses were established by clinical means, plain chest radiographs, left ventriculography, and selective coronary arteriography. The indications for surgery were uncontrollable congestive heart failure and angina, ventricular arrhythmias, or a rapidly growing aneurysm. Low cardiac indices or high left ventricular end-diastolic pressure were not considered to be contraindications to operation. Resection of the left ventricular aneurysm was performed with the use of normothermic cardiopulmonary bypass with haemodilution. In addition to the aneurysmectomy, four of these patients had concomitant closure of post-infarction ventricular septal defects; four had valve replacements; two had grafts to coronary arteries; and one had both replacement of the mitral valve and a right coronary vein graft.

There were two hospital deaths (10%) and two late deaths (10%), making an overall mortality of 20%. All but one of the deaths were related to coronary artery disease. The survivors are active, and their rehabilitation was satisfactory. The longest survivor is doing well two years after left ventricular aneurysmectomy, ventricular defect closure, and tricuspid valve replacement.

It is evident from our experience and from the reports of others that surgery has an established place in the management of post-infarction left ventricular aneurysm.

Left ventricular aneurysm is a complication of myocardial infarction and seriously reduces the chances of survival. In a collected series of 102 patients with left ventricular aneurysm, Schlichter, Hellerstein, and Katz (1954) found that 73% died within three years, and 88% within five years, due to intractable heart failure, repetitive arrhythmias, recurrent myocardial infarction, or systematic embolization. Left ventricular aneurysm has been reported to occur in 8 to 38% of patients following myocardial infarction (Schlichter *et al.*, 1954; Abrams *et al.*, 1963; Gorlin, Klein, and Sullivan, 1967; Tassel and Edwards, 1972), this wide variation in calculated incidence being caused mainly by difficulty in agreement on an accurate definition of a ventricular aneurysm.

Ventricular aneurysm was present in 3.5% in a series of 2293 myocardial infarctions in 10 800

consecutive necropsies at the Mayo Clinic (Dubnow, Burchell, and Titus, 1965). Abrams *et al.* (1963) found at necropsy that ventricular aneurysms were present in 12.4% of patients with myocardial infarction.

The first successful aneurysmectomy without the use of cardiopulmonary bypass was reported in 1955 by Likoff and Bailey. Since Cooley *et al.* (1958) performed the first excision of a left ventricular aneurysm under cardiopulmonary bypass, resection of ventricular aneurysms has become a well-established surgical procedure (Lam *et al.*, 1966; Cooley and Hallman, 1968; Favaloro *et al.*, 1968; Ibarra-Pérez and Lillehei, 1969; Schattenberg *et al.*, 1970; Kluge *et al.*, 1971; Loop, 1971; Austen, 1973; Merin *et al.*, 1973; Rao *et al.*, 1974; Cooperman *et al.*, 1975). The first British report of successful open excision was by Telling and Wooler (1961).

The recognition of ventricular aneurysms is of practical importance as surgical repair is advocated. Intractable cardiac failure, uncontrolled arrhythmias, and systemic embolism are the main indications for aneurysmectomy. Occasionally other mechanical problems, such as an associated ventricular septal defect or mitral regurgitation, also need correction. Combined revascularization should probably be undertaken in vessels that have considerable narrowing and still supply functioning myocardium (Thind, Blakemore, and Zinsser, 1971; Austen, 1973; Davis *et al.*, 1973; Gerbode *et al.*, 1974; Cooperman *et al.*, 1975).

This paper presents our clinical experience with the management of 20 consecutive patients by a reasonably aggressive surgical approach.

PATIENTS AND METHODS

During the two-year period from February 1973 to February 1975, 20 patients were referred to the Wessex Cardiac and Thoracic Centre, Southampton for evaluation and treatment of left ventricular aneurysm. Of the 20 patients, 14 (70%) were men and 6 (30%) women. Their ages ranged from 35 to 74 years, with an average age of 58, and 60% were more than 60 years of age.

All the patients had had at least one myocardial infarction, 70% had persistent congestive heart failure, 10% cardiac failure and angina, and 20% had repetitive arrhythmias, varying from rapid atrial fibrillation to intractable ventricular tachycardia or fibrillation (Table I).

TABLE I
PRESENTATION AND INDICATION FOR OPERATION -
20 PATIENTS

Main Symptom	Patients	
	No.	%
Congestive heart failure	14	70
Arrhythmia	1	5
Angina	0	—
Arterial emboli	0	—
Congestive heart failure and arrhythmia	3	15
Congestive heart failure and angina	2	10

There was no preoperative embolization.

An average of 8.5 months (range 19 days to 2 years) had elapsed between the initial infarction and the diagnosis of the aneurysm. The mean time interval between infarction and operation was 13.6 months (range 23 days to 5 years). Only five patients (25%) had systemic hypertension.

There were no pathognomonic physical findings, although clinical examination disclosed a forceful and rather broad systolic expansion of

the chest wall in eight patients. Chest radiographs showed cardiac enlargement and a distinct circumscribed bulging of the extreme left ventricular contour. In our series, the diagnosis could be made on the plain chest radiograph alone in 12 out of 20 patients (Figs 1-3). With one exception, all patients had been studied by left ventriculography, to determine the size and site of the aneurysm, the contractility of the unaffected part of the ventricle, and the presence or absence of mitral incompetence.

Selective coronary arteriography was routinely performed, to identify the distribution of the coronary artery disease and to allow planning of myocardial revascularization. The aneurysm was located in the anterolateral wall or involving the apex of the left ventricle in 17 out of 20 patients (85%). Two were posterior, and one (superior) was situated high at the topmost part of the left ventricle, lateral to the pulmonary artery and just beneath the aortic valve (Fig. 3a).

Four patients were found to have, in addition to their aneurysm, a postinfarction ventricular septal defect, and another four had severe mitral regurgitation due to papillary muscle dysfunction (3 patients) or rheumatic valvular disease (1 patient) (Tables II and III).

In all 17 patients with anterolateral or apical aneurysms, the left anterior descending coronary artery was occluded. In both patients with a posterior ventricular aneurysm, the right coronary artery and/or the circumflex were occluded associated with mitral regurgitation in one patient and ventricular septal defect in the other. In the patient with the superior aneurysm and mitral regurgitation, the left anterior descending coronary artery was normal, but there was significant stenosis of the circumflex and right coronary arteries.

In two of the three patients with associated mitral regurgitation due to papillary muscle dysfunction, the left anterior descending coronary artery was normal, but there was occlusion of either the circumflex or the right coronary artery or both (Table II).

In all patients with postinfarction ventricular septal defect, either one or both of the non-occluded remaining major coronary arteries were also seriously diseased.

The left ventricular end-diastolic pressure was routinely measured and found to be elevated in over two-thirds of the patients.

The operative findings agreed with the pre-operative assessment in all patients. The size and site of the aneurysm, the presence of mitral

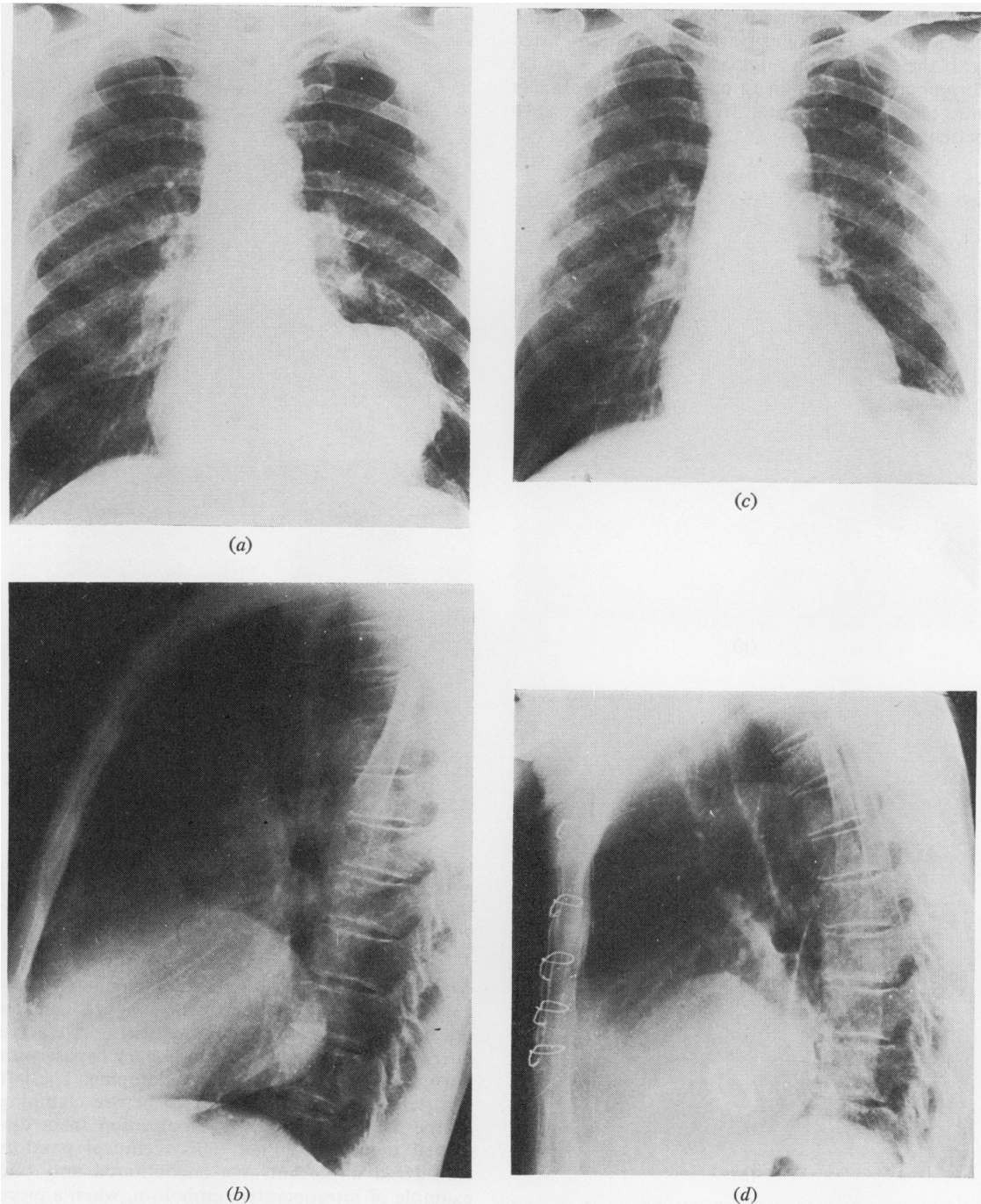


FIG. 1. Chest radiographs in a 69-year-old patient with a left ventricular aneurysm and severe mitral regurgitation, before and after aneurysmectomy and mitral valve replacement. (a) Large aneurysm at the left heart border. (b) Lateral view showing the posterior aneurysm extending up to the atrioventricular groove. (c and d) Postero-anterior and lateral views five months after operation.

regurgitation or ventricular septal defect, and the distribution of coronary artery disease all fitted with the findings on angiocardiography. A mural thrombus was found in 13 patients (65%), old clot was present in 11, and fresh thrombus in two patients.

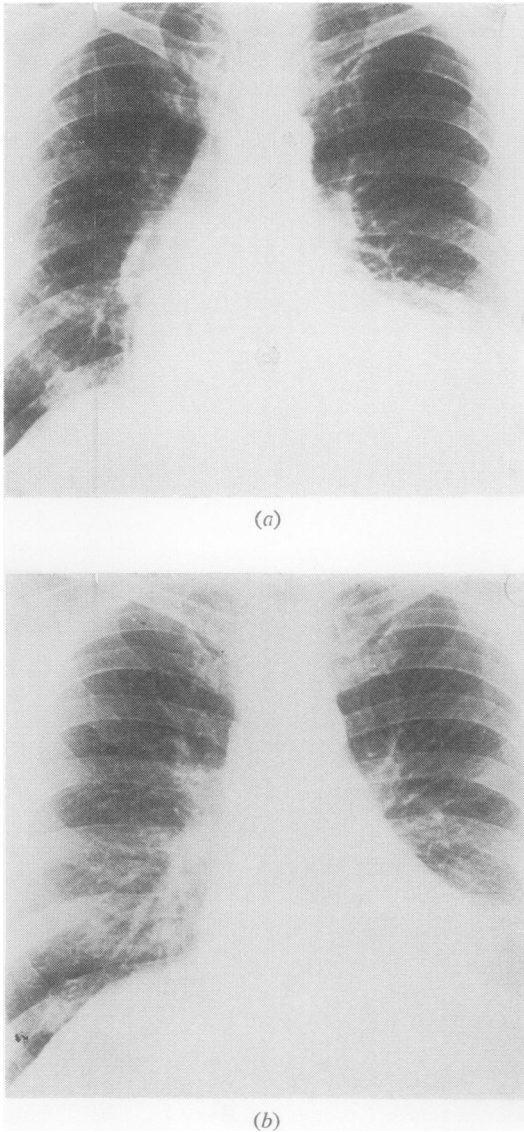


FIG. 2. (a) Chest radiograph showing a large left ventricular aneurysm in a 58-year-old man. (b) The postoperative film, two months after resection of the aneurysm, showing a considerable decrease in heart size.

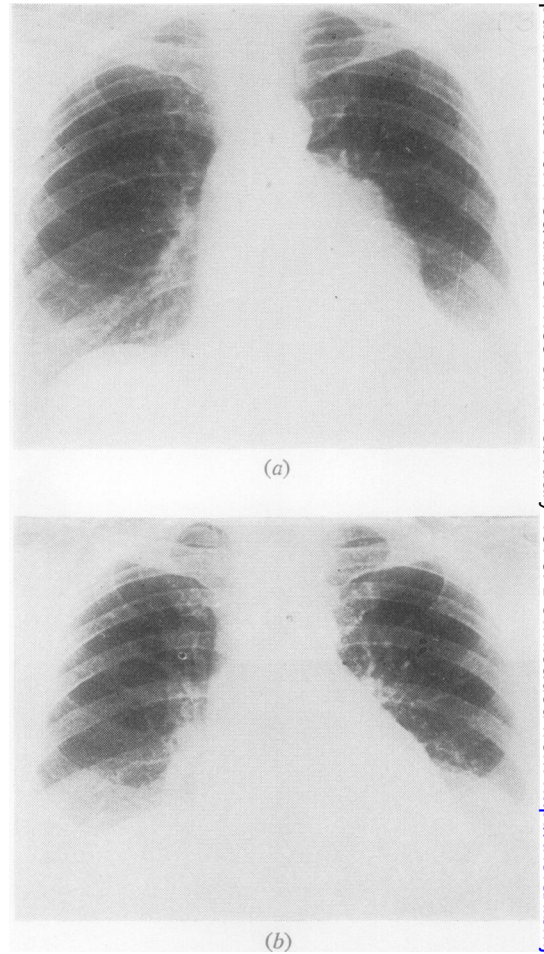


FIG. 3. Chest radiographs (a) before and (b) two months after resection of an aneurysm arising from the upper portion of the free wall of the left ventricle and mitral valve replacement.

OPERATIVE TECHNIQUE Excision of the left ventricular aneurysm was performed in 18, and a plication technique in two patients. All were operated upon using cardiopulmonary bypass with normothermia and aortic cross-clamping. The left ventricle was never manipulated before clamping of the aorta, so avoiding embolization from dislodged mural thrombus. This technical point is considered important. We encountered only one example of intraoperative embolism, when a piece of thrombus was discovered in and was removed from the left coronary ostium. This illustrates the need for exploration of the coronary ostia if the ventricle fails acutely after bypass.

TABLE II

CORRELATION OF CORONARY ANATOMY, LOCATION OF ANEURYSM AND ASSOCIATED LESION - 20 PATIENTS

Anatomical Distribution of Coronary Diseases	Location of Aneurysm	Associated Lesion	No. of Cases
Occluded LAD with good RCA and CCA	Anterolateral	—	10
Occluded LAD and CCA with good RCA	Anterolateral	—	1
Occluded LAD and RCA with good CCA	Anterolateral	VSD	3
Multiple CAD	Anterolateral	MR	1
Occluded CCA with good LAD and RCA	Anterolateral	—	2
Occluded CCA and RCA with good LAD	Posterior	VSD	1
	Posterior	MR	1
	Superior	MR	1

Occluded = more than 80% stenosis
 LAD = left anterior descending coronary artery
 RCA = right coronary artery
 CCA = circumflex coronary artery
 CAD = coronary artery disease
 VSD = ventricular septal defect
 MR = mitral regurgitation

TABLE III

LEFT VENTRICULAR ANEURYSM: CLASSIFICATION IN 20 PATIENTS

Pathology	Number of Patients	Hospital Death	Late Death
Simple LAD aneurysm ¹ with good RCA and CCA	10	1	2
Simple LAD aneurysm needing circumflex vein graft	1	—	—
Ventricular aneurysm + VSD	4	1	—
Aneurysm needing valve replacement	4	—	—
Aneurysm needing MVR + RCA vein graft	1	—	—

¹Left ventricular aneurysm in the region supplied by the left anterior descending coronary artery which is occluded
 MVR = mitral valve replacement

Once the aneurysm had been opened and clot removed, the aortic clamp was released in some patients. In the remainder the whole procedure was carried out with the aorta cross clamped. The average ischaemic time was 22 minutes

Once the aneurysm had been incised over its summit and any thrombus removed, the left ventricular cavity was carefully inspected and the relationship of the infarct to the septum and papillary muscles was defined. Excision of the sac was carried out, leaving a narrow rim of fibrous tissue to provide support for sutures.

Often quite extensive areas of fibrous scar had to be left on the septal side, but this did not impair the mechanical results, possibly because (in patients who have not progressed to septal rupture) the full thickness of the septum was not involved in the infarct.

Ventricular septal defects were closed with interrupted mattress sutures tied over Dacron felt buttresses with a Dacron patch, and always from

the left side. The ventriculotomy was closed using interrupted or continuous heavy silk or mersilene mattress sutures over a double strip of Teflon felt, followed by a second continuous over and over suture.

An additional surgical procedure was performed in combination with aneurysmectomy in half the patients (Table IV). Four patients underwent simultaneous closure of postinfarction ventricular septal defect. One of these had severe tricuspid regurgitation (not due to ruptured chordae or papillary muscle), and an attempt was made to control this by annuloplasty. This failed, and tricuspid valve replacement (with a Starr Edwards valve) the next day was followed by immediate improvement and an excellent late result. Three patients had concomitant mitral valve replacement; one an aortic valve replacement with an antibiotic sterilized aortic homograft; one had a saphenous vein bypass graft to the circumflex coronary artery; and another underwent mitral valve replacement and vein bypass graft to the right coronary artery in addition to aneurysmectomy.

TABLE IV
ANEURYSMECTOMY, ASSOCIATED PROCEDURES, AND OPERATIVE RESULTS IN 20 PATIENTS

Procedure	Number of Patients	Hospital Death	Late Death
Ventricular aneurysmectomy	10	1	2
Ventricular septal defect closure and aneurysmectomy ¹	4	1	—
Mitral valve replacement and aneurysmectomy ²	3	—	—
Circumflex coronary artery vein graft and aneurysmectomy	1	—	—
Mitral valve replacement, right coronary artery vein graft, and aneurysmectomy	1	—	—
Aortic valve replacement (homograft) and aneurysmectomy	1	—	—

¹In one patient plus tricuspid annuloplasty, but without satisfactory result: reoperation on first postoperative day and tricuspid valve replacement with Starr Edwards prosthesis. The final result was excellent.

²In one patient there was a history of mitral valve disease many years before myocardial infarction and ventricular aneurysm development.

Cardiac failure and arrhythmia were the most common postoperative complications and responded to medical treatment. There was one re-exploration of the wound for tamponade.

RESULTS

The hospital mortality was 10% (two hospital deaths) and there were two late deaths (10%), giving an overall majority of 20% (Table V).

Of the two hospital deaths, one was a 57-year-old man who had a myocardial infarction 23 days before operation for ventricular septal defect

TABLE V
MORTALITY - 20 PATIENTS

Death	No.	%
Hospital	2	10
Late	2	10
Total	4	20

closure and plication of left ventricular aneurysm. He had had two episodes of cardiac arrest but was successfully resuscitated. He underwent another cardiac arrest on the day of operation, and an emergency procedure was carried out. He made reasonable progress and was extubated on the second postoperative day, but the day after he suffered another cardiac arrest, and attempts to resuscitate failed. Necropsy revealed long-standing multiple coronary vessel occlusion.

The second hospital death was in a 62-year-old man, who underwent aneurysmectomy for a large ventricular aneurysm five months after the causative myocardial infarction, and who died at operation from low cardiac output due to diffuse coronary artery disease. Necropsy showed a very small residual left ventricular cavity, with evidence of severe widespread coronary artery disease.

Of the late deaths, one was a man of 44 with preoperative arrhythmias, including on one occasion ventricular fibrillation and circulatory arrest. He initially did well postoperatively but died suddenly three months after operation. Necropsy disclosed a recent myocardial infarction.

The second late death was directly due to a recurrence of the aneurysm. Eleven months after aneurysmectomy, the patient, again symptomatic, required reoperation for a false aneurysm which had developed at the site of the original ventriculotomy. After resection of the false aneurysm and repair of the ventricle, the small and non-compliant residual ventricle caused the patient's death on the operating table.

The postoperative course of the survivors was smooth, with an average hospital stay of 18 days after operation. All but one are leading active lives; the latter still requires medical treatment for diffuse coronary artery disease.

DISCUSSION

The major mechanical complications of myocardial infarction are ventricular aneurysm, rupture or dysfunction of papillary muscles, ventricular septal defect, and acute cardiac rupture (Abrams *et al.*, 1963; Dubnow *et al.*, 1965; Gorlin *et al.*, 1967; Tassel and Edwards, 1972).

The wide variation (8% to 38%) in the incidence of aneurysm following myocardial infarction is probably explained by the different diagnostic criteria used (Schlichter *et al.*, 1954; Schattenberg *et al.*, 1970; Kluge *et al.*, 1971). As a left ventricular aneurysm may develop as early as two days after infarction, or remain unrecognized for several years, an aneurysm should be suspected in any patient following myocardial infarction whose recovery is not prompt or complete, especially when there is persistent congestive heart failure (Lam *et al.*, 1966; Cooley and Hallman, 1968; Favaloro *et al.*, 1968; Schattenberg *et al.*, 1970). In our series, the earliest diagnosis was made 23 days after causative myocardial infarction. Predisposing factors, such as hypertension which increases the transmural tension exerted against the functionless portion of the ventricular wall, reported by others, were not conclusive in our series (Schlichter *et al.*, 1954; Harley, 1969; Davis and Ebert, 1972). Aneurysm formation is more commonly seen in males, and although younger people are not exempt, most occur in the 60-70 year age group.

The anterolateral free wall and the apex of the left ventricle supplied by the anterior descending branch of the left coronary artery are the most usually affected areas. This is confirmed in the present series (Table II).

Posterior aneurysms are associated with occlusion of the right or circumflex coronary artery or both. Although posterior myocardial infarctions are common, development of a posterior ventricular aneurysm is not. This is possibly explained by the fact that such infarcts can involve the posterior papillary muscle and the resulting severe mitral regurgitation superimposed on the acute myocardial infarction precipitates fatal left ventricular failure (Abrams *et al.*, 1963; Favaloro *et al.*, 1968; Harley, 1969; Ibarra-Pérez and Lillehei, 1969; Schattenberg *et al.*, 1970; Loop *et al.*, 1973). In our series, both posterior aneurysms (and the superior aneurysm) were associated with mitral regurgitation (two) or ventricular septal defect (one).

Patients with aneurysm are usually symptomatic and often severely incapacitated by heart failure, arrhythmias, and angina, which frequently prove refractory to medical treatment. Peripheral embolism and rupture of a true aneurysm are the least common complications. As the development of an aneurysm markedly reduces life expectancy, aneurysmectomy should be carried out with markedly improved overall survival (Schlichter *et al.*, 1954; Dubnow *et al.*, 1965; Cooley and Hall

man, 1968; Key, Aldridge, and MacGregor, 1968; Thind *et al.*, 1971; Davis and Ebert, 1972; Rosenthal, Daroca, and Cohen, 1972; Rao *et al.*, 1974).

Left ventricular angiography outlines the aneurysm and confirms the integrity of the septum and the mitral valve. Selective coronary angiography indicates the feasibility of direct myocardial revascularization, which probably improves the long-term results (Davis *et al.*, 1973; Merin *et al.*, 1973; Gerbode *et al.*, 1974; Cooperman *et al.*, 1975). The usual haemodynamic parameters, such as the left ventricular end-diastolic pressure and the cardiac index, appear to have relatively little prognostic value (Key *et al.*, 1968; Manvi, Allen and Ellestad, 1974; Cooperman *et al.*, 1975). The majority of patients in our series had an elevated left ventricular end-diastolic pressure, but here there was no correlation with the operative results.

As time is needed for the necrotic wall of the left ventricle to be converted into fibrous tissue, a time interval of three to six months between myocardial infarction and aneurysm resection has been quoted as critical to the results of surgery (Cooley and Hallman, 1968; Favalaro *et al.*, 1968; Kluge *et al.*, 1971; Gerbode *et al.*, 1974). In our series, three patients were operated upon within three months of infarction; it is felt that no arbitrary time lapse should be allowed to affect clinical judgement as to the timing of surgery. All patients who underwent surgery for arrhythmias were relieved of them, and in all survivors in congestive heart failure before surgery the cardiac status has markedly improved. The immediate and late results of resection of left ventricular aneurysms in our small series compare very favourably with others (Cooperman *et al.*, 1975).

The relatively small number receiving coronary bypass grafts does not indicate a reluctance to combine aneurysmectomy with a revascularization procedure. It reflects that fact that, in 18 of 20 patients, either the remainder of the coronary arterial tree was not sufficiently badly diseased to require grafting, or the diseased coronary arteries were considered not suitable for grafting.

We are grateful to Dr. Alan Johnson and to Dr. Neville Conway for cardiological help with these cases, and to Dr. Ivan Hyde for radiological assistance.

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