SURGERY OF THE COMPLICATIONS (OTHER THAN ANGINA) FOLLOWING MYOCARDIAL INFARCTION

By Donald N. Ross

The pathological consequences of a myocardial infarct vary from death of the individual to massive or localised destruction of areas of myocardium. The functional disturbance in those who live will usually depend on the anatomical localisation of the segment of muscle involved.

Since the effects of an infarct are so widespread and varied, one cannot hope to do more than chronicle or deal briefly with the various clinical syndromes resulting and indicate where surgery is applicable.

The best known and most widely treated complications of an infarct present their effects some time after the acute phase—often days or months later. However, we are possibly entering an era when early or immediate treatment may have the effect of ameliorating or avoiding the damaging effects of the muscle ischaemia and we should examine this prospect. Therefore when talking about complications we should distinguish in our minds between the immediate problems and the remote or delayed effects of the infarct. (Table I.)

TABLE I
EFFECTS OF AN INFARCT

IMMEDIATE	—	Death
	—	Pump Failure—Shock
	—	Acute Arrhythmias
DELAYED	—	Healing or Fibrous Replacement
	—	Dyskinetic Area
	—	Aneurysm
	—	VSD
	—	Mitral Regurgitation
	—	Chronic Failure

The immediate pathological events following an ischaemic episode are that the area of muscle is at first deprived of blood and later becomes infarcted, swollen or haemorrhagic with a central necrotic area and congested zone of perinfarction. During this early phase, if the infarct is extensive, the patient may die of acute pump failure or may experience severe dysrhythmias or go into cardiogenic shock.

Theoretically one should be able to operate during this acute phase and remove the vessel obstruction or revascularise while the myocardium is ischaemic but before irreversible infarction has resulted. This can be a rewarding policy in acute ischaemia of the leg where early embolectomy or bypass grafting can certainly prevent loss of tissue.

In support of this prospect we know from our everyday cardiac surgical experience that the myocardium can be and is rendered acutely ischaemic every time we use elective arrest of the heart for periods of perhaps up to one hour. Also these hearts return to full functional efficiency once the blood supply is restored. This implies that short periods of ischaemia are recoverable.

However, the analogy is not necessarily applicable to our discussion since hearts on cardiopulmonary bypass are relieved of their workloads, are asystolic with low oxygen consumption and are uniformly ischaemic without 02 tension gradients across adjacent segments of muscle.

Also, even in these apparently normal hearts, when biopsies of the myocardium are examined at intervals by electron microscopy we have seen evidence of commencing breakdown and dissolution of the nuclear components of the cells within 45 minutes of ischaemia (De Gasperis et al 1971) and on chemical analysis of the muscle there is a marked loss of potassium indicating loss of integrity of the cell membrane (Singh et al 1970).

Furthermore on the experimental side, deliberately induced localised ischaemic myocardial segments in dogs are likely to progress to infarcts after about one hour of ischaemia even after restoration of the blood supply (Mundth et al 1972).

As a consequence, it seems unlikely that one can avoid the relentless pathological progression towards an infarct by early operation and restoration of the circulation unless surgery is instituted within an hour of the acute episode. This is hardly practical although a period of up to six hours has often been loosely suggested in clinical discussion. Even then it will be rare to be able to operate within this time so that in the present state of our knowledge it does not appear possible to reverse the established infarct by direct surgical means alone.

Once an acute infarct is established, can we benefit the patient by an emergency infarctectomy? Here we have the bold pioneer work of Heimbecker (1967) as an example. Although there is again a theoretical case to be made out for such a course of action, it is difficult to know how much this is likely to benefit the patient or whether it will jeopardise his chances of recovery. Additional problems relate to the difficulty in deciding upon the limits of muscle excision and how to deal with the friable tissues (Table II).

A potentially more rewarding line of treatment has been developed, particularly by the Boston group using intra-aortic balloon pumping to maintain a circulation and blood pressure and to assist the crippled heart in cardiogenic shock (Sanders et al. 1972). During the period of pump support an accurate angiographic assessment can be made and revascularisation of the remaining myocardium can be effected. This approach implies that the patient has recovery potential, particularly the brain and kidneys and that some of the ischaemic damage is reversible perhaps through maintaining or opening up collaterals per infarction zone. The method is at present under trial in a number of centres and an objective assessment of its value should be available soon.

Even without balloon pumping it is feasible to do emergency angiography and vein grafting in cases of intractible cardiogenic shock with established infarcts and we have had some success in this field (Table III).

Before leaving the subject of surgery for the acute episode one should mention the value of pacemakers for acute A-V dissociation and bradycardias. Since a jugular electrode is usually adequate this nowadays hardly rates as a surgical intervention. The excision of ischaemic areas is also theoretically indicated for the control of intractible dysrhythmias unresponsive to medical management (Schlesinger 1971). Although we have tried this we have not so far had a lasting clinical success.

With regard to the more remote or later pathological consequences of an infarct we are on more familiar ground and we think in terms of aneurysm formation, ventricular septal defects and mitral regurgitation.

In terms of pathological anatomy, it is usually an infarct of the anterior descending artery territory, that gives rise to the common aneursym involving the region of the apex and front wall of the ventricle. The infarct may additionally involve the septum with the development of an acute ischaemic ventricular septal

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TABLE II

TREATMENT OF ACUTE INFARCT

Emergency revascularisation or clot removal	—	Within 1-6 hours
Excision of infarct	—	Heimbecker
Circulatory support, coronary angiography and revascularisation	—	Balloon Pumping

TABLE III

ACUTE COMPLICATIONS OF INFARCT
15 EMERGENCY OPERATIONS

	Number	Hospital Mortality	Late
Cardiogenic Shock	3	1	0
Resistant Failure and MR	2	1	1
Resistant Failure and VSD	1	1	0
Intractible rest pain	5	0	0
Resistant Dysrhythmia	4	4	0

TABLE IV

Anterior wall infarct————aneurysm

ventricular septal defect

Posterior wall infarct————mitral regurgitation

TABLE V

TREATMENT OF DELAYED EFFECT OF AN INFARCT

Aneurysm		excise and revascularise surrounding myocardium
Flat Infarct	dyskinesia	revascularise surrounding myocardium
	full thickness destruction (flat aneurysm)	excise
Mitral Regurgitation		valve replacement and revascularisation
Ventricular Septal Defect		support medically if possible and operate at 2 weeks or later

TABLE VI

SURGERY FOR COMPLICATIONS OF INFARCT
(EXCLUDING VEIN GRAFTS)

	Number	Deaths
Infarctectomy (failure)	9	4
Imbrication	3	1
Calcified Infarct	1	0
Aneurysms	9	3
Mitral Replacement	3	0
Mitral Annuloplasty	2	0
VSD	2	0
	29	8

TABLE VII

HEART TRANSPLANTS June 1972
(American College of Surgeons Transplant Registry)

Total number	183
Survivors	27
9% survivors are now over 3 years	
Stanford University Results	
39 cases — 43% survive 1 year	
32% survive 2 years	

defect. Posterior wall infarcts resulting from ischaemic necrosis in the region of the circumflex or posterior descending branch of the right artery are likely to involve the papillary muscles and result in mitral regurgitation and less frequently give rise to a septal defect (Table IV).

The excision of aneurysm is well-established, having been shown to be a feasible and worthwhile procedure by Cooley's group. Their reported mortality was 20% (Cooley and Hallman, 1968) and Favaloro et al (1968) reported 23% mortality. A more recent report from the Hammersmith Hospital (Graber et al 1972) quotes a 21.7% operative mortality, all of which results show a surprising uniformity.

Since the segment of muscle involved in the aneurysm is replaced by fibrous tissue and is excised, little can be gained by additional saphenous vein grafting to the involved artery. However, revascularisation of the remaining muscle where the vessels are stenotic or blocked may improve the prognosis and functional results in these cases which in the past have often had significant residual disability and late deaths.

A more difficult policy is posed by those cases presenting with failure and angiographic evidence of an area of dyskinesia or akinetic muscle. Examination of the myocardium at surgery in these cases often shows an area of disturbed contractility with patchy fibrous replacement interspaced with muscle tissue. Little is to be gained by over-enthusiastic excision of these areas and our policy where the case has been accepted for operation has been to revascularise only, in the hope of restoring co-ordinated activity. Excision is reserved for full-thickness infarcts with uniform fibrous replacement—the-so-called flat aneurysm. Kirklin (1972) has reported on nine cases in cardiac failure where the infarct has been excised and saphenous grafting has been carried out. Three died at operation and four within four months while the two survivors are not improved, so that this is not a particularly rewarding area of surgical endeavour at present.

Mitral regurgitation as a result of an infarct can be sudden and incapacitating, particularly where a papillary muscle is avulsed or damaged. The heart is small and pulmonary congestion is marked. Mitral regurgitation can also result from involvement of the mitral ring with a full-thickness posterior wall infarct, normally with associated papillary muscle dysfunction. In these latter cases the condition may be more chronic with an enlarged heart presenting with breathlessness and failure. Treatment in both instances is by mitral replacement with or without revascularisation.

Ventricular septal defects arising from acute rupture of the infarcted ventricular septum are not common and probably arise in 1% of all infarcts according to Friedberg (1966). Most deaths occur within the first week and only 13% of patients with a ventricular septal defect survive longer than two months. The first surgical attempt at correction was again by Cooley and Hallman in 1957 and surprisingly only 78 cases had been reported up till 1971.

The problem in this condition is that while death is common in the first week, the septum and ventricular wall are excessively fribable and there is the added difficulty of delineating the extent of the necrosis. It is therefore not surprising that most successful cases have been repaired after three weeks and theoretically the best time from a point of view of healing and collagen density is at six weeks. Although rare successes have been reported within eight days of infarction (Dagget et al, 1970) at present the best chance of surgical success arises where one can tide the patient over medically for 2-3 weeks or longer.

Practical points of importance in the early case are to approach the defect through the area of infarcted ventricular wall and to excise this at the same time and also to use generous patches of prosthetic material or Teflon sewn well wide of the defect margins (Tables V and VI).

As a rare complication of an infarct, we have had a patient present with a rigidly calcified posterior left ventricular wall following an infarct, involving the whole thickness of the muscle and the mitral valve ring. The patient was considerably disabled by virtue of the area of non-contractile left ventricle plus the mitral regurgitation. The whole calcified mass and the valve was excised by sharp dissection and the mitral valve was replaced, producing an excellent clinical result.

Finally, we have to face the unpalatable but nevertheless unavoidable fact that we cannot induce low grade fibrous tissue to revert to contractible myocardium. No matter how much we increase the blood supply or improve the ventricular mechanics by fibrous tissue excision or valve replacement, the eventual result will depend on an adequate complement of contractible myocardium remaining. It is these cases in chronic failure and usually without angina that make up a significant proportion of clinical practice. There is generally a history of repeated infarcts, breathlessness is dominant and there is a large poorly contracting heart. At present, we can only offer increasingly stringent anti-failure therapy or consider cardiac transplantation and the time is coming when we shall have to take a second look at transplantation as a form of therapy in rare instances. Although the latest overall figures from the world transplant register are not too encouraging, Shumway's persistence and scientific application make his results look reasonable. (Table VII). Also it seems that the practical immunological aspects of the transplantation problem are again in the doldrums and probably need the boost of additional surgical pressures to exploit new and dormant therapeutic possibilities.

Of course, the dream of a totally implantable mechanical heart has already passed from the realm of science fiction to successful animal application and we may look forward to seeing replacement hearts used in cases of massive infarction—even if used on a temporary basis, within our clinical lifetime.

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SURGERY FOR THE COMPLICATIONS OF MYOCARDIAL INFARCTION

By G. R. Stirling* and Eric Cooper†

Augmentation of coronary blood flow by saphenous vein[1] or internal mammary artery by-pass[2] is now established as an effective method of palliation of angina in patients with myocardial ischaemia due to proximal coronary artery stenosis or occlusion. Because of the acceptable mortality of these procedures their indications have been extended to the treatment of patients with acute coronary insufficiency thought to be in jeopardy from impending myocardial infarction, with encouraging results.

It is probable that, in the future, myocardial revascularisation will be importantly directed at the prevention of myocardial infarction or sudden death since these dramatic events frequently begin and end the clinical history of patients with previously undetected coronary artery disease. It is hoped that the extension of surgery into this area will rest securely on conclusions reached from study of the survival of patients currently being submitted to revascularisation surgery. The evidence for this forward step is not yet available.

Unfortunately there is a significant group of patients who, having survived a myocardial infarction, remain seriously disabled by its effects. In many instances heart failure results from an extensive area of infarction and, it seems, there is nothing that surgery can offer. However there is another group of cases in whom the haemodynamic disturbance can be attributed to a local area of infarction, which may give rise to the complications of ventricular aneurysm, ventricular septal defect or mitral valve incompetence, in whom surgery should be seriously considered. The dividing line between these two groups is ill defined but one infers from the evidence currently available that surgery has most to offer in those cases in which the myocardial infarction is localised and well defined, the infarction has resulted from single vessel disease and the remaining coronary arteries are patent, and the haemodynamic disturbance attributable to the lesion is gross.

It follows that very careful and complete evaluation of the anatomy and physiology of the coronary artery tree and the left ventricle are necessary in both diagnosis and assessment. Selective coronary cine-arteriography and two-plane left ventriculography are the basic investigations required but more sophisticated measurements of left ventricular wall movement may complement the latter investigation. Whilst, in many instances a localised ventricular aneurysm or area of akinesia is clearly delineated, when the heart is enlarged current techniques are insensitive in distinguishing between local areas of myocardial infarction and diffuse damage to the left ventricle due to more extensive infarction or to myocardial ischaemia without infarction.

Since the mortality of surgery for the complications of myocardial infarction has been relatively high and the long term outlook after successful repair is still ill defined it is proper to be more highly selective in recommending surgery in this group than in uncomplicated cases with angina with or without myocardial infarction.

VENTRICULAR ANEURYSM

A ventricular aneurysm may be defined as a localised protrusion of the external aspect of the ventricle accompanied by a corresponding protusion of the ventricular cavity. This definition, proposed by Edwards[3] helps to distinguish true aneurysms from healed infarcts with thinning of the ventricular wall or bulging of the cavity into an akinetic scar. Dubnow and his colleagues[4] found ventricular aneurysm to be present in 3.5% of autopsies on cases of myocardial infarction. Douglas and colleagues[5] reported an incidence of 8.7% and Schlichter and colleagues[6] one of 20% in their series of autopsies on patients dying of myocardial infarction. Most ventricular aneurysms lie anteriorly and involve the apex of the left ventricle with variable involvement of the anterior and apical portions of the ventricular septum which may be perforated. They result from obstruction to the left anterior descending coronary artery.

Less commonly the aneurysm involves the inferior portion of the left ventricle and associated involvement of the posterior papillary muscle may interfere with mitral valve function. The combination of extensive inferior infarction with mitral incompetence and aneurysm formation is rarely compatible with long survival but two such cases have been reported as successfully repaired by Szentpetery and colleagues[7].

The natural history of patients with left ventricular aneurysm is very variable. I have observed a patient to live for ten years without disability with a calcified apical aneurysm 8 cm. in diameter. Most patients, however, suffer from mild to severe cardiac failure and, with expansion of the aneurysm, the disability is usually progressive. The haemodynamic effect of the aneurysm is usually described in terms of the paradoxical movement of the aneurysm which, by the outward movement of the infarcted segment in systole, results in a direct subtraction from left ventricular ejection. The aneurysm is usually lined by laminated thrombus, which may result in arterial embolism, but may have the beneficial effect of reducing paradox by splinting the infarcted area. Whether paradox be present or not the effect of the infarction producing the aneurysm is twofold. Firstly there is a reduction in the mass of contracting myocardium in the left ventricle and, secondly the ventricular volume is increased so that the resting tension of the myocardium is abnormally raised and ejection involves a greater energy expenditure.

Although late rupture is rare we have observed rupture of a ventricular aneurysm in two cases six days and eleven weeks after infarction with acute pericardial tamponade. Although both patients were treated by aspiration of the pericardium and immediate aneurysm resection both succumbed from inadequate cardiac output in the postoperative period.

A final complication of left ventricular aneurysm is that of recurrent ventricular tachy arrhythmia. Hunt and colleagues[8] have reported successful control of this situation by aneurysm resection. The diagnosis of left ventricular aneurysm may be suspected by the emergence of abnormal systolic pulsation anteriorly over the left ventricle after myocardial infarction. The electrocardiogram shows features of full thickness infarction with persistent S-T elevation in the leads appropriate to the infarction. The chest radiograph may show an abnormal bulge in the contour of the left ventricular border and, in chronic cases, linear calcification may be evident. The presence of aneurysm cannot be excluded by chest radiography and screening so that consideration should always be given to left ventriculography in patients with cardiac enlargement and heart failure after myocardial infarction as this technique is much more sensitive in the detection of ventricular aneurysm.

The final and definitive assessment of the presence, extent and haemodynamic effects of a left ventricular aneurysm are obtained from left ventriculography, haemodynamic assessment of left ventricular function and selective cinecoronary arteriography of the right and left coronary arteries. This data added to the clinical electrocardiographic and radiographic information pro-

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vides adequate information for selection for surgery. Symptomless aneurysms occur and do not merit surgical treatment. However patients with singificant disability from dyspnoea, frank heart failure, arterial embolism or ventricular arrhythmia should be fully assessed and may benefit from surgery. Although many patients with these features may also exhibit angina there is a separate group in which patients, suffering predominantly from angina, also manifest a ventricular aneurysm and in these aneurysmectomy may be advised in addition to myocardial revascularisation.

Since Cooley and colleagues[9] described the first excision and repair of a ventricular aneurysm using cardio-pulmonary by-pass the operation has been widely practised and the principal technical objectives are now clearly defined. Loop[10] in 1971 was able to report on the total experience of the Cleveland Clinic at that time amounting to 301 cases. The surgical apprach is through a median sternotomy. Cardio-pulmonary by-pass is established with the minimal disturbance of the ventricle, which is usually adherent to the pericardium, to avoid embolism due to displacement of mural thrombus during dissection. Ventricular fibrillation should be induced and the aorta cross clamped before the ventricle is mobilised. The left heart should be decompressed with a left atrial or left ventricular cannula. The aneurysm is easily defined and mural thrombus, usually present, is carefully and completely removed. The aortic clamp can now be removed restoring coronary perfusion. The walls of the aneurysm are then resected leaving only a thin layer of scar attached to the adjacent healthy musculature of the ventricle. Closure of the ventricle is achieved by two or three layers of deep sutures usually supported by cushioning strips of teflon felt. Air is evacuated from the left ventricle before final closure. Should there be high grade proximal obstruction of the coronary arteries with a patent distal segment aorto-coronary by-pass with a saphenous vein graft should be performed. The left anterior descending coronary artery is uncommonly available, as its distal obliterated end may be involved in the aneurysm wall, but severe disease of the right or circumflex coronary arteries is, appropriately, corrected in a significant proportion of cases. Monitoring of the left atrial pressure in the postoperative period has proven to be helpful in guiding the policy with regard to transfusion as most patients exhibit evidence of left heart failure in the early postoperative period.

When ventricular aneurysm is complicated by ventricular septal defect the repair can be conveniently carried out through the left ventricle suturing a large overlapping patch to the left side of the septum. In the uncommon situation of an inferior aneurysm associated with mitral incompetence aneurysmectomy should be combined with mitral valve replacement as described by Szentpetery[7].

Johnson and colleagues[11] indicate that the best results obtain when the aneurysm is large and there is major disease of a single coronary artery. Under these circumstances the risk of aneurysmectomy is less than 10 per cent. However in cases with major obstruction to two or three vessels, in whom the aneurysm is frequently smaller, a much higher risk prevails. Analysis of Loop's data[10] reveals a similar situation. In 45 cases, operated after 1969, in whom aneurysmectomy alone was performed there was but a single death, whereas the mortality in 30 cases submitted to aneurysmectomy and saphenous vein by-pass was 5, a mortality of over 18 per cent. These results do not deny the utility of saphenous vein graft by-pass, in the cases in which this additional technique was used, but point to a more severe stage in the progress of coronary vascular disease in those cases.

The results of successful aneurysm resection are very gratifying in most cases with relief of dyspnoea, angina and arrhythmia in most cases. It is too much to expect that normal haemodynamics should be restored but a high order of palliation justifies this procedure in patients confronted with progressive heart failure.

The modest experience with aneurysmectomy at the Alfred Hospital Melbourne is summarised in Table 1. There were 6 cases three of whom have been greatly benefited by aneurysmectomy at the time of review 9, 12 and 16 months after operation. Two cases, previously referred to, who developed rupture and were operated in a moribund state failed to survive. The sixth patient, a 64 year old male with a large anterior aneurysm associated with a ventricular septal defect, made a satisfactory recovery and left hospital well but died suddenly four months after operation (Table 11).

MITRAL REGURGITATION

When mitral regurgitation complicates myocardial infarction, it is usually a result of obstruction to the posterior descending branch of the right coronary artery or to obstruction of the circumflex branch of the left coronary artery. As a result of posterior infarction the posterior papillary muscle may stretch, lose its contractility or rupture so that the chordal restraints to both mitral valve cusps may be ineffective. Extensive posterior wall infarcts may, by involving the mitral annulus, result in dilatation and subsequent incompetence. Massive mitral incompetence may occur in the first few hours or days after infarction resulting in acute left ventricular failure whereas, in other cases, progressive annular dilatation or chordo-papillary dysfunction may cause a delayed and progressive increase in incompetence with the late onset of symptoms.

Rupture of the chordae tendinae is a common cause of mitral incompetence but definitive evidence of posterior infarction is uncommonly present. In our own experience of 32 cases of ruptured chordae tendinae definite evidence of posterior infarction was present in only two cases. It may be that more discrete infarcts, perhaps involving only the apex of the papillary muscle, are more common and the crude techniques of electro-cardiography and inspection at the time of surgery fail to detect infarction as the mechanism in many cases.

Mitral valve replacement may be life saving to a patient developing mitral incompetence and consequent left heart failure after myocardial infarction. The diagnosis is usually obvious but should be confirmed by left ventriculography. Cine coronary arteriography should also be performed to evaluate the state of the major coronary arteries as reconstructive arterial surgery may also be necessary. Severe mitral incompetence with cardiac failure may occur without the characteristic murmur being audible. Forrester and colleagues[13] described three such cases and reported that, although the papillary muscles were macroscopically normal there were extensive areas of myocardial necrosis at microscopic examination. Our own experience with surgery for mitral incompetence following myocardial infarction is confined to four cases (Table 111). The results in three of these have been very satisfactory. Whereas reconstructive operations have gained little popularity in this situation, one 62 year old male in intractable pulmonary oedema six weeks after myocardial infarction has achieved an excellent result from cusp plication and annuloplasty. He remains symptom free five years after surgery. The other three cases were operated on two years, seven weeks and four months after infarction and in each a Starr Edwards prosthesis was used for mitral valve replacement. Two patients remain well three years and two years after operation but the fourth case, after an early excellent result, died suddenly nine months after surgery, from anterior myocardial infarction. The right coronary artery was by-passed with a saphenous vein in one case.

VENTRICULAR SEPTAL DEFECT FOLLOWING MYOCARDIAL INFARCTION

Perforation of the ventricular septum is an uncommon but serious complication of myocardial infarction. Lee and colleagues[13] estimated the incidence

TABLE I

POST INFARCTION VENTRICULAR ANEURYSM

Case	Size Associations	Time after Infarct	Surgery Type	Result
E.C. M. 50	10 × 15 cms. Clot Femoral Embolus L.V.F.	9 months	Aneurysmectomy	Well at 18 months
K. McC. M. 54	7 × 2 cms. L.V.F. Angina	2 years	Aneurysmectomy Graft R.C.A., L.A.D.	Well 9 months
I.B. F. 58	4 × 5 cms. L.V.F. Angina	7 years	Aneurysmectomy Graft L.A.D.	Slight improvement 1 year

TABLE II

POST INFARCTION VENTRICULAR ANEURYSM AND V.S.D.

Case	Size Associations	Time from Infarct	Surgery Type	Result
E.E. M. 64.	10 × 8 cm. V.S.D., Clot L.V.F.	6 weeks	Aneurysmectomy Patch V.S.D.	Well 4 months Sudden Death
I.M. F. 61	10 × 8 cm. R.V. and L.V. L.V.F. Rupture Tamponade Arrest	6 days	Aneurysmectomy Patch V.S.D.	Operative Death
F.F. M. 46	10 × 12 cm. R.V. and L.V. Clot Arrest	11 weeks	Aneurysmectomy Direct Repair	Low Output Death 4 Days

TABLE III

POST INFARCTION MITRAL INCOMPETENCE

Case	Clinical State	Time from Infarct	Surgery Operation	Result
S. McK. M. 62	L.V.F.	6 weeks	Leaflet Repair Annuloplasty	Symptomless 5 years
W.S. M. 54	Angina L.V.F.	2 years	M.V.R. R.C.A. Graft	Symptomless 3 years
M.S. F. 54	L.V.F.	7 weeks	M.V.R.	Mild Failure 2 years
W.C. M.55	L.V.F.	4 months	M.V.R.	Well for 9 months Sudden Death

TABLE IV

POST-INFARCTION V.S.D.

Case	Associations	Time (days)	Surgery Type	Result
*E.E. M. 60	L.V.F. Arrhythmia Aneurysm	40	Patch Repair Aneurysmectomy	Well until sudden Death 4 months
*I.M. F.57	L.V.F. Aneurysm Rupture Tamponade	6	Direct Repair Aneurysmectomy	Operative Death
*F.F. M.46	L.V.F. Aneurysm Arrest	77	Patch Repair Aneurysmectomy	Low Output Death 4 Days
F.G. M.48	L.V.F.	24	R.V. Repair with Patch	Continued L.V.F. Death 5 Months

*These patients also mentioned in Table II.

as 1 to 2 per cent in patients dying of myocardial infarction. In 35 per cent of cases there is an associated ventricular aneurysm[14]. The defect is usually inferior and apical but it may occur more superiorly or posteriorly in the septum[15]. Importantly there may be multiple perforations in 40 per cent of cases and the surgeon may have great difficulty in locating these from the trabeculated right ventricular aspect of the septum.

Perforation usually occurs during the first two weeks following myocardial infarction[13, 14] and, in most instances, in the first week.

Sanders, Kern and Blount[16] documented the very grave prognosis in a review of 91 cases. In their series only 50 per cent of patients survived the first week and 13 per cent survived two months. No doubt the large left to right shunt which occurs adds an intolerable burden to the already struggling left ventricle with its function already depressed by recent infarction.

In an excellent review by Kitamura, Mendez and Kay[14] early surgical repair of the defect is advocated. The high early death rate in these cases precludes long delay until some healing has occurred around the margins of the perforation. They advise operation through the adjacent area of infarcted left ventricle and stress the importance of exposing and repairing the defect from the left side. Visualization is easier, one is less likely to overlook additional defects and a large patch can be placed on the left side or the septum overlapping the margins of the defect. It is hoped that the high incidence of persistence or recurrent defects may be reduced by this manoeuvre. They further stress the importance of excision of an associated ventricular aneurysm as their studies clearly indicate a much poorer survival rate in those cases in which aneurysmectomy was not performed. Kitamura estimates the survival rates after surgery as 58 per cent at two months, 50 per cent at six months and 32 per cent at one year. Although these results leave no room for complacency it is clear that aggressive planned surgery may significantly improve the prospects of survival in this condition. Better results may well follow earlier surgery, the use of the left ventricular approach and aneurysmectomy when it is appropriate. Full investigation with angiocardiography and selective coronary cine arteriography is necessary to establish the diagnosis. Particular attention is paid to the recognition of associated ventricular aneurysm, the exclusion of mitral incompetence and the identification of remediable lesions in the major coronary vessels. Concomitant revascularisation may prove beneficial in many cases.

Our own experiences, with four cases, re-inforces Kitamura's conclusions (Table IV). In one early case, in whom repair was carried out from the right ventricular approach, a persistent defect contributed to death occurring five months after operation. The three other cases all had extensive left ventricular aneurysms and repair was conducted from an incision through the aneurysm which was later excised. Rupture of the aneurysm with cardiac tamponade prompted aspiration and emergency operation in two cases but neither survived. The fourth case, a 64 year old male, made a reasonable recovery after patch closure of the ventricular septal defect and aneurysmectomy but he continued in moderate but controlled heart failure for four months at which time he died suddenly. Autopsy revealed a satisfactory repair of the lesions but there was extensive scarring of the septum and a wide area of the remaining left ventricular wall.

SURGERY FOR CARDIOGENIC SHOCK

Most patients dying as a result of the development of complications of myocardial infarction do so in the first two weeks following the onset of infarction. It is notable that most of the successes following surgery for ventricular aneurysm, ventricular septal defect or mitral incompetence have been achieved in a selected group who have already survived four weeks or more. The mode of death in the vast majority of cases dying in the first two weeks is that of cardiogenic shock, a complication which carries a mortality of the order of 80 to 90 per cent. Heimbecker[18] focussed attention on the potential for salvage in this group by aggressive surgery when he described successful infarctectomy in a patient threatened with death from massive anterior myocardial infarction in 1968. His clinical success followed extensive animal experimentation in which the scope and limitations of infarctectomy were evaluated.

It is clear that the techniques of myocardial revascularisation, infarctectomy, aneurysm repair, septal repair and mitral valve replacement are applicable in the acute phase and may be life saving. The potential case group is enormous and the problems in selection remain large, but pilot studies encourage some confidence in the view that surgery will have a continuing and productive role in the salvage of patients otherwise destined to die of cardiogenic shock. Mundth and colleagues[19], in reporting their experience with this problem, indicate the need for early mechanical circulatory assistance for cardiogenic shock. In their experience, with intra-aortic balloon pulsation, there were 5 long term survivors in the first 26 patients. This experience encouraged them to adopt a more aggressive policy in which patients who failed to respond in the first twelve hours of circulatory assistance were selected for complete evaluation by coronary arteriography and left ventriculography. Where it seemed feasible emergency surgery was then carried out continuing the circulatory support up to and often after surgery. In a second series of sixteen patients submitted to circulatory support, cardiac investigation and then surgery, 7 long term survivors were achieved. In 3 saphenous vein bypass grafts were the sole surgical manoeuvres, in two infarctectomy was combined with coronary artery grafting, in one infarctectomy alone was used and in another saphenous vein graft and mitral valve replacement was carried out.

Despite these remarkable successes it is probable that most patients in refractory cardiogenic shock have infarcted an intolerably large proportion of their left ventricular myocardium and nothing short of cardiac replacement will conceivably offer them a future.

The successes that have been achieved have resulted from a massively funded enterprise which has allowed the development of a highly trained team with expensive equipment and facilities readily available at all times. Such resources are available in a very few major institutions in the more affluent countries. The question of priorities cannot be ignored. In 1972 it would seem that the requirement is for this work to be confined to a few major centres so that the techniques can be refined and simplified and the guidelines for patient selection can be more clearly defined before more general application is encouraged.

CONCLUSION

Techniques have been developed for the treatment of ventricular aneurysm, ventricular septal defect and mitral incompetence following myocardial infarction. Encouraging results have been achieved in the application of these techniques to selected patients surviving the acute phase following infarction.

The more common and serious problem of early death from cardiogenic shock is being aggressively attacked by the use of mechanical circulatory assistance, cardiac investigation and emergency surgery. The results of these endeavours have met with some success but further evaluation in appropriately equipped pilot centres is necessary before more general application is justified.

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