Emergency coronary artery bypass surgery after intracoronary thrombolysis for evolving myocardial infarction

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SUMMARY Sixteen patients underwent emergency coronary artery bypass surgery immediately after intracoronary streptokinase infusion for acute evolving myocardial infarction. Of these, 11 patients had 70% residual stenosis in the recanalised vessel, and in five thrombolysis was unsuccessful. There were no hospital deaths. All the patients sustained myocardial necrosis, the peak activity of creatine phosphokinase correlating with the time to reperfusion. Chest tube drainage (mean 960 ml) was significantly higher than for control patients but did not correlate with the total dosage of streptokinase. No patients had further myocardial infarction or developed recurrent angina.

Selected patients may benefit from coronary bypass surgery after intracoronary streptokinase infusion. If necessary this may be performed immediately with low mortality and morbidity.

The importance of left ventricular function in determining the prognosis of patients with coronary artery disease^{1 2} has directed attempts towards early restoration of coronary blood flow in patients with acute evolving myocardial infarction. The infusion of streptokinase into the appropriate coronary ostium restored patency in 72-95% of patients.^{3 4}

Nevertheless, though successful streptokinase treatment results in thrombolysis it does not remove the underlying coronary artery stenosis, and thus secondary occlusion with reinfarction remains a problem.^{5 6} Serruys *et al* suggested a combination of transluminal angioplasty after successful thrombolysis,³ while Phillips *et al* recommended coronary bypass surgery for stenoses not responding to streptokinase treatment.⁷

To obviate both these problems we adopted a policy of immediate coronary bypass surgery after intracoronary streptokinase infusion in selected patients.

Patients and methods

During 1983, 28 patients admitted to this hospital with acute evolving myocardial infarction⁸ were treated by intracoronary streptokinase infusion. Throm-

bolysis was unsuccessful in five (18%) patients, including two with cardiogenic shock and one with intractable ventricular arrhythmias. Eleven (39%) patients had residual stenosis of >70% in the recanalised vessel and were considered at high risk of reocclusion. These 16 patients underwent immediate coronary bypass surgery, and they constitute the study group.

All 16 patients were male, with a mean age of 53 (range 38-60) years. All presented with unremitting angina associated with ST segment elevation on the electrocardiogram. None had developed Q waves. This was the first coronary occlusion in all patients. The mean interval between the onset of pain and coronary arteriography was 2.2 (range 1-4) hours. All patients had occlusion of one major coronary artery (anterior descending (12), right coronary artery (3), large first obtuse marginal (1)). In each case the left ventriculogram showed segmental wall motion abnormalities associated with the occluded vessel. An initial bolus of 20 000 units of streptokinase was infused into the occluded coronary artery followed by an infusion of 5000 units/min. The mean dose of streptokinase was 200 000 units (range 95 000-340 000 units). In two patients with cardiogenic shock streptokinase infusion was stopped after 15 minutes (equivalent to 95 000 units). A third patient developed intractable ventricular arrhythmias, and the streptokinase infusion was stopped after 100 000

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Fig. 1 Coronary arteriograms (a) before and (b) after successful intracoronary thrombolysis of occluded anterior descending artery. (b) shows residual 90% stenosis (arrow).

units had been given. All recanalised vessels had proximal stenoses of $>70\%$, and in eight patients the stenosis was $>90\%$ (Fig. 1).

The patients were transferred immediately from the catheter laboratory to the operating room. Cardiopulmonary bypass was rapidly instituted between the right atrium and the ascending aorta. Coronary artery bypass surgery was performed using the technique of intermittent ischaemic arrest at moderate hypothermia (30°C). The occluded artery was grafted first so that the time between the onset of pain and complete reperfusion of the occluded artery was less than six hours in each case. All other major vessels with stenoses of $>50\%$ were then serially bypassed. Eight patients received three grafts, five patients two grafts, and three patients one graft. At the end of the operation the patients were given 10 units of cryoprecipitate, 10 units of platelets, and 2 units of fresh frozen plasma.

In the immediate postoperative period the development of myocardial necrosis was monitored by serial creatine phosphokinase measurements and electrocardiographic changes. Serum creatine phosphokinase activity was measured at eight, 16, and 24 hours after admission to hospital. Electrocardiograms were recorded on the first, second, third, and seventh postoperative day.

Results

There were no hospital deaths. Four patients required positive inotropic support (dobutamine $5\text{--}12\text{ }\mu\text{g/kg/min}$) and one balloon counterpulsation. Six patients received a lignocaine infusion (2 mg/min) for ventricular arrhythmias. The mean blood loss through the chest tube drain was 960 (SD 385) ml. This was higher than for control patients but did not correlate with the total dose of streptokinase given

Fig. 2 Relation between blood loss and streptokinase dose.

Table Indices of myocardial necrosis

Case No	Occluded vessel	No of grafts	Interval to reperfusion (hours)	New ECG changes	Peak CPK activity (IU/l)
1	LAD	1	2	Normal	540
2	LAD	1	2	Normal	890
3	LAD	2	2	R	990
4	LAD	3	2	R	2500
5	RCA	1	3	Q	1800
6	LAD	2	3	R	1900
7	LAD	3	3	R	1950
8	LAD	2	3	R	2100
9	Lat Cx	2	3	Q	2500
10	LAD	3	3	Q	3500
11	RCA	2	4	Q	2800
12	LAD	3	4	R	3000
13	LAD	3	4	Q	3700
14	LAD	3	5	R	2000
15	LAD	3	6	R	2500
16	RCA	3	6	Q	5500

LAD, left anterior descending; Lat Cx, lateral circumflex; RCA, right coronary artery; ECG, electrocardiogram; R, loss of R wave progression across anterior chest leads; Q, new Q waves; CPK, creatine phosphokinase.

($r=0.36$) (Fig. 2). One patient required re-exploration for excessive haemorrhage.

All patients had myocardial necrosis confirmed by serial electrocardiogram changes and enzyme studies. The Table summarises the data of individual patients. All four patients with occlusions of the right coronary artery or obtuse marginal vessels and two with occlusions of the anterior descending artery developed new Q waves. Eight patients with occlusions of the anterior descending artery showed a loss of R wave progression on serial electrocardiograms. In two patients (Table, cases 1 and 2) the electrocardiogram returned to normal. The postoperative value for creatine phosphokinase activity was abnormally raised (mean 2323 (SD 1561) units) with a positive MB isoenzyme in all patients. Three patients with occlusion of the anterior descending artery underwent successful thrombolysis within two hours of the onset of

pain. The highest value for creatine phosphokinase activity in these patients was <1000 units (Table, cases 1–3). Figure 3 shows the correlation between the peak value for creatine phosphokinase activity and the time interval to reperfusion ($r=0.67$, $p<0.01$).

Follow up in this series was short (mean 8 months). Nevertheless, to date no patients have experienced further angina or suffered myocardial infarction.

Discussion

An analysis of the results of the 63 coronary care units in Boston, Massachusetts, showed no perceptible fall in the hospital mortality from acute myocardial infarction between 1973–74 and 1978–79.⁹ The most rational approach to the management of acute evolving myocardial infarction is the restoration of anterograde blood flow. After coronary artery occlusion in dogs, necrosis begins in the subendocardial region and spreads outwards to the subepicardial layer. This wavefront of necrosis can be arrested by reperfusion,¹⁰ the final size of the infarction being a function of the duration of ischaemia and the ischaemic tolerance of a given patient. This in turn depends on the number and size of collateral vessels compensating for the occluded artery.

In 1960 Boucek and Murphy infused fibrinolytics into the aortic sinuses of patients with acute myocardial infarction, reporting circumstantial evidence that the manoeuvre resulted in the re-establishment of blood flow.¹¹ This technique was refined by Rentrop *et al* who reported the first successful cases of intracoronary thrombolysis in 1979.¹² The sooner the treatment is started, the greater its chance of success.^{13 14} Successful streptokinase treatment has been associated with improved thallium-201 uptake^{15–17}

Fig. 3 Relation between peak value of creatine phosphokinase (CPK) activity and time to reperfusion.

and improved wall motion¹⁸⁻²⁰ in the ischaemic areas. The clinical results of thrombolysis have also been encouraging.^{6,18} In the German multicentre study mortality was 7% among patients in whom reperfusion occurred and 24% among those in whom the occlusion persisted.⁶ After successful thrombolysis reocclusion has been reported in 17%³ to 25%⁵ of patients. Serruys *et al* suggested concomitant balloon dilatation of the residual stenosis after successful thrombolysis, reporting no late reocclusions in 18 patients undergoing this procedure.³ Krebber *et al* have used thallium-201 scintigraphy as a predictor of the salvageability of myocardium immediately after intracoronary thrombolysis to assess patients suitable for early coronary revascularisation.²¹ No details of the patients undergoing this procedure are given, however, nor is the time interval between medical revascularisation and surgical reperfusion stated.

Berg *et al* have been strong advocates of immediate coronary bypass surgery for acute evolving myocardial infarction.⁸ DeWood *et al* reported appreciable improvement in global function and regional wall motion in selected patients if surgical reperfusion was performed within six hours of the onset of symptoms of anterior infarction.²² Phillips *et al* used immediate coronary bypass surgery for 21 patients in whom streptokinase treatment failed.⁷ The mean interval to reperfusion was 10 hours in this group, four of which required re-exploration for haemorrhage. Becher *et al*, however, found no increase in bleeding complications in patients operated on within 24 hours of thrombolysis compared with those undergoing surgery after this period.²³

The results of our study support the aggressive approach in treating patients with acute evolving myocardial infarction.^{7,8,22} Thrombolytic treatment had failed in five patients, two of whom were in cardiogenic shock, while in eight the residual stenosis after successful thrombolysis was >90%. The anatomy of the latter group of patients was more severe than that reported by Serruys *et al*, who found that only 19% of 64 patients undergoing thrombolysis had a residual stenosis of >70%.³ In no case did the combination of rapid intracoronary thrombolysis followed by immediate coronary bypass surgery prevent myocardial damage. We consider, however, that it may have limited its extent. This was particularly apparent in two patients in whom blood flow was re-established within two hours of the onset of pain (Table, cases 1 and 2). In these patients peak creatine phosphokinase activity was <1000 IU/l and the electrocardiogram returned to normal. The follow up period was short, but to date there have been no deaths, mechanical complications of infarction, or further ischaemic episodes.

Theoretically, all patients having acute evolving

myocardial infarction should be revascularised at the earliest possible opportunity. As a prelude all patients will undergo coronary arteriography to delineate the anatomy. Successful thrombolysis with intracoronary streptokinase then provides additional time before a decision regarding surgery must be taken. The length of time depends partly on the residual coronary lesions. Some may be so minor that the infarction can be allowed to run its course and elective surgery planned at a later date when further symptoms occur.²⁴ This more elective approach would allow the use of the internal mammary artery which must now be considered as the conduit of choice for the anterior descending artery.^{25,26} In other situations a more aggressive approach may be required. This may take the form of immediate balloon angioplasty³ or early surgery.

Since this was not a prospective randomised trial with follow up haemodynamic data, we are unable to conclude that this is the preferred method of treatment. The study simply shows that immediate coronary bypass surgery can be performed effectively after intracoronary thrombolysis. Though postoperative chest tube drainage was significantly higher in this group of patients than after elective coronary bypass surgery, haemorrhage was not an insuperable problem. Indeed in the dosage given (95 000–340 000 units) there was no correlation between the dosage and total blood loss. Though Jutzy *et al* have shown no difference in the systemic fibrinolytic state assessed by serial determinations of fibrinogen and euglobulin lysis time,²⁷ it is likely that the larger doses (>500 000 units) given intravenously^{28,29} may preclude immediate surgery.

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