Early and long term results of re-operation for coronary artery disease

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Abstract

Objective—To define the incidence, possible causes, operative procedure, and early and medium term results of patients undergoing reoperation for coronary artery disease.

Design—A retrospective analysis of patients undergoing reoperation in one hospital during a 10 year period.

Setting—A regional cardiothoracic centre.

Patients—115 patients had reoperation for recurrent angina, 1–17 years (mean (SD) 7.4 (3.9)) after primary revascularisation.

Main outcome measures—They received 279 grafts (2.4 grafts per patient); 58% of the grafts were anastomosed to previously grafted vessels. The internal mammary artery was used in 87% of patients who required grafts to the left anterior descending coronary artery.

Results—Reoperation accounted for 8.3% of the total number of patients who underwent coronary bypass grafting. Graft failure alone or in combination with other factors was judged to be the cause of recurrence of symptoms in 87%. 42% of patients had two or more coronary risk factors. The early mortality was 5.2% and the actuarial survival at five and 10 years was 90.4% and 88.4% respectively. 85% of the survivors had initial complete relief of angina and 14% had partial improvement. Freedom from recurrent symptoms at five and 10 years was 66.6% and 34.6% respectively.

Conclusions—Vein graft failure either alone or in combination with progression of native coronary disease is the main cause for symptomatic deterioration after bypass grafting. Reoperation can be performed with slightly increased risk and can give good early and medium term results.

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Despite the encouraging impact of coronary artery bypass grafting on the natural history of ischaemic heart disease, the procedure remains a palliative one. Recurrence of angina occurs in 3.5%–4.5% of patients each year after coronary artery bypass grafting

(CABG).¹ The incidence of reoperation is increasing because of the progressive nature of the disease.² It is reported that reoperation could carry a higher morbidity and mortality^{3,4} with a variable chance of symptomatic improvement.^{5–8} The aims were to analyse our experience with reoperation in an attempt to define the possible factors causing recurrence of angina after the original operation, and to characterise the early and long term results of reoperation.

Patients and methods

Between January 1981 and December 1990 a total of 115 patients had reoperations for recurrence of their symptoms after primary CABG. These patients represent 8.3% of the total number who underwent coronary operations during this period. Fourteen patients in this group had had their first operation in other hospitals, and were referred to Harefield Hospital for their reoperation. There were 109 men and six women with an age range of 36–71 (mean 53.7 (7.6)) years. Figure 1 shows that the time interval between the two operations ranged from one to 17 (mean 7.4 (3.9)) years. Seventy one percent of patients had their second operation five years or more after the first. According to the Canadian Heart Association Classification of Angina, nine patients were in class II, 66 in class III, and 40 in class IV (fig 2). Table 1 shows the incidence of different risk factors amongst the patients. Forty two percent of patients had two or more of the listed risk factors.

ANGIOGRAPHIC FINDINGS

A total of 294 grafts were inserted in the first

Figure 1 Time interval to repeat CABG.

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Figure 2 The severity of angina in patients presenting for repeat CABG.

Table 1 Preoperative risk factors

	Patients (n)	% of total group
Family history	48	41.7
Previous MI	35	30.4
Smoking	30	26.1
Hyperlipidaemia	21	18.3
Hypertension	17	14.8
Obesity	7	6.1
Diabetes mellitus	4	3.5

MI, myocardial infarction.

operation with a mean of 2.6 grafts in each patient. Of these, the long saphenous vein was used in 288 instances, and the internal mammary artery (IMA) in six patients.

The angiographic study before reoperation showed that 229 grafts were occluded or severely diseased (77.9%) and 65 were patent (22.1%). Five out of the six IMAs used at the first operation were still patent at reoperation. Figure 3 shows the incidence of occlusion of different grafts. The possible causes of deterioration of symptoms were assessed angiographically and table 2 lists these. Graft failure was the sole reason in 46.1%, and in association with progression of native disease or incomplete revascularisation or both, graft failure was implicated in a further 42.5% (total of 88.6%) of cases. Left ventricular function was assessed as good (ejection fraction (EF) >60%) in 75%, mildly impaired (EF 40%–60%) in 16%, and poor (EF <40%) in 9% of patients.

Table 2 Causes of recurrent angina

	Patients (n)	% of total group
Graft failure	53	46.1
Progression of underlying disease	13	11.4
Incomplete revascularisation	2	1.7
Progression and graft failure	37	32.2
Graft failure and incomplete revascularisation	5	4.3
Graft failure, progression and incomplete revascularisation	5	4.3

Figure 3 Angiographic findings before reoperation. LAD, left anterior descending coronary artery; Diag, diagonal branch; Trif, tri-furcation; CX1, circumflex branch 1; CX2, circumflex branch 2; RCA, right coronary artery; PDA, posterior descending coronary artery.

OPERATION DATA

One IMA previously anastomosed to the left anterior descending coronary artery (LAD) was damaged during dissection, being adherent to the undersurface of the sternum. Intermit-tent aortic cross clamping and core cooling to 30°C was used in 88 patients (76.5%), and cardioplegia with St Thomas's solution in 27 patients (23.5%). During reoperation 279 grafts were inserted with a mean of 2.4 grafts per patient. Eighteen patients had single grafts, 42 had double grafts, 43 had triple grafts, and 12 had quadruple grafts. At reoperation 51 patients had grafts only to previously grafted vessels (44.3%), 51 had regrafts as well as grafts to vessels not grafted before, and only 13 patients (11.4%) had grafts to vessels not grafted at the first operation. Figure 4 shows the site and pattern of grafting. Fifty eight percent of the grafts were inserted to vessels previously grafted because of total occlusion or extensive graft atherosclerosis. Forty two percent of the grafted vessels were not grafted before and showed evidence of progression of native vessel disease. The LAD was grafted in 83 patients. Ninety two percent of them had been previously grafted, whereas 8% had not. Out of the 83 patients, 70 had single IMA and two had double IMA grafts.

Fourteen patients had twenty endarterec-tomies at reoperation and two had the aortic valve replaced with aortic homografts. All patients were prescribed aspirin and dipyridamole in the postoperative period.

Figure 4 Sites and pattern of grafting at reoperation. Abbreviations as in fig 3.

Table 3 Risk factors and mortality

Patient	Cause of death	Risk factors
1	CVA	Smoking, obesity, family history
2	CVA	Hypercholesterolaemia, hypertension, obesity, impaired left ventricular function, family history
3	LCOP	Previous myocardial infarction, hypertension, impaired left ventricular function, family history
4	LCOP	Previous myocardial infarction, impaired left ventricular function, smoking
5	CVA	Previous myocardial infarction, impaired left ventricular function, smoking
6	LCOP	Previous myocardial infarction, smoking

CVA, Cerebrovascular accident; LCOP, Low cardiac output.

Mortality and morbidity

Six early deaths occurred within 30 days of reoperation (5.2%). The cause of death was low cardiac output in three and cerebrovascular accident in three. In all of the early deaths, at least two risk factors were present (table 3). Four of the early deaths had poor left ventricular function before operation. Table 4 shows the postoperative complications. The actuarial survival rate at five and 10 years was 90.4% and 88.4% respectively (fig 5).

RELIEF OF SYMPTOMS AND LATE OUTCOME

Complete relief of symptoms was achieved in 93 patients (85.3%) for at least one year. Fifteen patients (13.8%) had incomplete relief of their angina and one patient (0.9%) had no improvement. Those who had initial disappearance of angina remained free of symptoms for a mean period of 42 (22) months. Freedom from recurrence of angina at five and 10 years was 66.6% and 34.6% respectively (fig 6). After a follow up period between one and 10 years (mean 64 (32) months), 43 patients had no symptoms, forty three had class I or II angina, and 14 patients had class III or IV angina. Five patients underwent a third operation without deaths. Early postoperative angiography was performed for 47 patients within the first year after reoperation. This was done as a routine postoperative assessment in 43 patients and to assess residual symptoms in four patients. Out of 121 grafts examined 85.7% were patent (fig 7).

Discussion

The need for reoperation after primary myocardial revascularisation is increasing. This is probably due to the increasing number of CABG operations, the progressive nature of the disease, and the development of accelerated disease in the vein grafts in the absence of effective methods of prevention. Previous

Table 4 Perioperative morbidity

	Patients (n)	% of total group
Bleeding	3	2.9
Perioperative myocardial infarction	4	3.8
Cerebrovascular accident	4	3.8
Arrhythmia	14	12.2
Complete heart block	1	
Left bundle branch block	1	
Right bundle branch block	2	
Atrial fibrillation	9	
Supraventricular tachycardia	1

Figure 5 Actuarial survival of patients after reoperation (n = 115).

studies have shown that the occlusion rate of saphenous vein grafts is around 12%–20% during the first year, and 2%–4% annually for four to five years with subsequent doubling of the rate.⁹ It is estimated that 1.8%¹⁰ to 5.5%¹¹ of CABG operations are reoperations. In our study, 8.3% of the total number of CABGs were reoperations during this period. This incidence is higher than average, possibly due to the fact that Harefield Hospital tends to act as a tertiary referral centre.

The time interval between the two operations ranged from 1–17 (mean 7.4 (3.9) years). Seventy three percent of patients required their reoperation five years or more after the first. A recent report has found that the time to reoperation is becoming longer, as a result of decreased early graft occlusion.¹² This may be a reflection of improving technique with the recognition of the importance of minimising the damage to the vein during harvesting.¹³ The possible causes of recurrent angina seem to be changing. In our study, graft failure was the sole reason in 46.1% of cases; there was an association with other causes in 42.5% (total 88.6%). Progression of native vessel disease alone or combined with other causes was found in 49.9% of our patients. Early reports,¹⁴ showed incomplete revascularisation to be implicated in a high percentage of cases. Our findings are comparable with more recent reports.^{3 15} The number of grafts per patient increases with surgical experience.¹⁶ In this series, 279 grafts were inserted with a mean of 2.4 grafts for each patient. Janardhan *et al*⁵ has

Figure 6 Freedom from recurrence of symptoms after reoperation (n = 115).

Figure 7 Graft patency of 121 grafts at one year after reoperation. Abbreviations as in fig 3.

recently reported 2.5 grafts for each patient. Most of our grafts to the LAD (91.6%), were inserted into previously grafted vessels. This is attributed to the low use of the IMA in the first operations in this cohort of patients. It was found also by Pidgeon *et al.*,¹⁵ that 77% of LAD grafts at reoperation were to previously grafted vessels. The lack of use of the IMA to LAD was found to be one of the predictors of reoperation by Cosgrove *et al.*¹⁶ The superiority of the IMA over saphenous vein grafts is well documented and is due to several factors including endothelial production of large concentrations of endothelium dependent relaxing factor (EDRF) by arterial grafts.^{17,18} The IMA was used in 87% of patients who required graft to the LAD in our series of reoperations.

Hospital mortality in our series was 5.2%, which is comparable with previous reports.^{3,4,19} Four out of the six early deaths occurred in patients with impaired left ventricular function. The increased risk of reoperation is explained by advancing age, progression of vascular disease, and deterioration of ventricular function.³ The incidence of perioperative myocardial infarction in our patients was 3.8%. It was reported by others to be around 7.1%.^{3,14} The higher incidence of perioperative myocardial infarction may be due to embolisation from previous grafts at the time of manipulation of the heart or the graft. Therefore we advocate in agreement with other authors that the grafts to be replaced should be tied off before any cardiac manipulation.²⁰

Angina was completely relieved for at least one year in 85% of our patients. This group remained symptom free for a mean period of 42 (22) months. Partial improvement was noted in 14% of patients, but 1% had no change in symptoms. Although some previous reports,⁶⁻⁸ have documented less favourable results, more recently, Lytle *et al.*, reported that 50% of patients were symptom free five years after the second operation.³

Vein graft atherosclerosis was shown in this study and in other reports,^{3,14} to be the main reason for recurrent angina after CABG. Many pathophysiological studies implicate platelets^{21,22} and lipids²³⁻²⁵ as possible mediating factors in the development of atherosclerotic lesions. There has been evidence that anti-platelet treatment has an effect in improving graft patency after CABG.²⁶⁻²⁸ Cholesterol lowering treatment has been shown recently to

induce regression of disease in native vessels as well as bypass grafts.^{29,30}

To enhance the benefit of primary myocardial revascularisation, it has been our policy to use at least one IMA routinely, follow a meticulous technique during harvesting of the vessel, prescribe antiplatelet treatment post-operatively, and achieve strict control of hyperlipoproteinaemias.

In conclusion, reoperation for recurrent angina can be performed with slightly increased risk, and can achieve good symptomatic relief for a period of up to 10 years. The main causes of deterioration in symptoms after the first operation are failure of the vein graft followed by progression of native vessel coronary disease. This study highlights the need for developing methods for effectively preventing such problems in these patients.

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ABSTRACTS IN CARDIOLOGY

Spasm and sudden death

Sudden deaths in which necropsy shows a structurally normal heart and no cause of death are a rare phenomenon but cause great distress to relatives who are trying to understand the event. Some insight into the cause of this phenomenon can be gained by investigating patients who have been resuscitated from out-of-hospital ventricular fibrillation (VF). The paper by Myerburg *et al* reports that coronary artery spasm in subjects without obstructive atherosclerotic disease can precipitate VF.

This condition could not be recognised by any pathological feature at necropsy but could be suspected if anginal type chest pain had been reported during life. Having said this, coronary spasm cannot be said to be common, occurring in no more than 1.4% of subjects who present with out-of-hospital VF. The further question of whether spasm can precipitate VF in subjects who have co-existent obstructive coronary disease remains to be answered.

MJ DAVIES

Life-threatening ventricular arrhythmias in patients with silent myocardial ischemia due to coronary artery spasm

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Abstract

Background. Silent myocardial ischemia in patients with coronary atherosclerosis is associated with an increased risk of adverse cardiac events, including sudden death. The relation between silent ischemia and the initiation of potentially life-threatening arrhythmias has not been defined, however.

Methods. A part of a long-term study of sudden cardiac death, silent myocardial ischemia, coronary artery flow, and response to intravenous nitroglycerin, 100 mg, was studied. Twenty-four patients with previously documented coronary artery flow-limiting lesions who had an out-of-hospital cardiac arrest who had an out-of-hospital cardiac arrest, were investigated. Interventions to alter structural causes of cardiac arrest and to clarify potential associations between silent myocardial ischemia, due to coronary artery disease, and the occurrence of characteristics of life-threatening ventricular arrhythmias were studied by both invasive and noninvasive techniques.

Results. Silent myocardial ischemia was associated with the initiation of life-threatening

ventricular arrhythmias in five patients with induced or spontaneous focal coronary artery spasm (or both). These patients were identified among a group of 254 patients with documented cardiac arrest who were examined between 1980 and 1991. In two of the five patients, chest pain, rather than ischemia, was associated with the onset of the ventricular arrhythmia. Only one of the five had an inducible arrhythmia during electrophysiologic testing. Titration of the dose of a calcium entry blocking agent (verapamil, 10 mg/min) in anticipation against the ability of verapamil to provoke spasm was successful in preventing both the provocation of spasm and the initiation of all five patients who were tested.

Conclusions. Silent myocardial ischemia due to coronary artery spasm can initiate potentially life-threatening arrhythmias in patients without flow-limiting structural coronary artery lesions. The role of silent ischemia, regardless of both in the initiation of life-threatening arrhythmias in larger groups of patients with structural coronary artery lesions remains to be defined. *ON Line J Med 1992;326: 1801-7.*