

The Management of Intractable Angina Pectoris Using Saphenous Vein Bypass Grafts

EXPERIENCE IN CAPE TOWN

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SUMMARY

Between 1970 and 1972, 54 patients with obstructive coronary artery disease had 84 saphenous vein bypass grafts performed. There was a 7% operative and a 14% late mortality. Significant symptomatic improvement occurred in 93% of the survivors and 63% were totally relieved of angina; 76% of the grafts were patent at the time of recatheterisation.

No correlation between the graft patency rate and size of grafted vessels — the extent of peripheral run-off or flow rate at the time of grafting — could be demonstrated.

Saphenous vein bypass grafting appears to have a place in the management of intractable angina pectoris in the relatively small group whose vessels appear suitable for grafting. There was little evidence of improvement in left ventricular function, and surgery is contra-indicated when left ventricular function is severely impaired.

The superiority of surgical treatment over conservative medical management of patients with less severe disease and in the pre-infarction or crescendo anginal syndrome, remains to be demonstrated.

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Since the introduction of direct myocardial revascularisation by the Cleveland¹ and Milwaukee² teams about 6 years ago there has been widespread enthusiasm for this form of treatment, particularly in the USA. During 1971 about 25 000 vein grafts and Vineberg operations were performed,³ and in 1972 roughly double that number.

This large-scale surgical attack on ischaemic heart disease has occurred despite repeated warnings from more conservative members of the medical profession that the operation could at best be palliative, that the long-term fate of vein grafts was unknown, and that there was no evidence to show that it in any way altered the mortality, or infarction rate, of patients with ischaemic heart disease.^{3,4}

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Recently there have been more and more reports of late failure of grafts owing to intimal thickening,⁵ thrombosis of the arterial segment between the graft and the proximal occlusion,⁶ and of a disturbingly high operative infarction rate.⁷

At the Groote Schuur Hospital Cardiac Clinic we have adopted a conservative approach, and only advised surgery for patients with incapacitating angina pectoris not responding to full medical treatment; in whom at least some of the distal vessels appear angiographically suitable for grafting, and ventricular function is not severely compromised. Aggravating factors such as obesity and hypertension must have been eliminated.

This report describes our results with saphenous vein grafting in this selected group of patients over the past 3 years.

PATIENTS AND METHODS

Fifty-four patients were operated on at Groote Schuur Hospital and the Volkshospitaal, Cape Town, between 1970 and 1972. Only patients with intractable angina pectoris which did not respond to medical management with beta-blockers, usually propranolol, isosorbide (Isordil), and nitroglycerin, were considered suitable for operation. The dose of beta-blockers was increased until the resting pulse rate was reduced to 60 beats/min or less. If there were symptoms of left ventricular failure before or during the use of beta-blockers, digitalis and diuretic therapy were used as well. Bronchospasm occasionally necessitated the use of practolol instead of propranolol.

Grossly obese subjects were accepted only when they had reduced their weight to normal levels. Hypertensive subjects were accepted once their blood pressure was adequately controlled on medical therapy.

All subjects who appeared suitable by the above criteria were submitted to cardiac catheterisation with coronary arteriography, using the Sones technique, and left ventricular angiography. From the information obtained, surgery was advised only if there was a 70% or greater obstruction in the proximal segment in one or more of the major coronary arteries, with a distal segment greater than 1 mm diameter showing a good 'run-off' into a viable segment of the myocardium. Latterly, only patients with adequate left ventricular function were accepted, but early on, 3 patients were selected with severely-impaired left ventricular function, in the mistaken hope that ventricular function would improve.

The 54 patients operated on were selected from over 200 patients investigated with a view to bypass grafting. The majority were rejected for surgery because of a lack of suitable vessels, or because of an inadequate ventricular function.

The indication for surgery in 7 of the 54 patients was crescendo angina. We are at present conducting a controlled trial of surgery for this condition, but for the purpose of evaluating the results of saphenous vein bypass grafts they have been included in the group with chronic stable angina.

The breakdown of the group according to sex, age, and race is shown in Table I. The ratio of White to Cape Coloured patients was 4,4 : 1, and men to women 8 : 1; 29 patients (54%) showed a significant obstruction in all 3 major vessels. The average number of vessels involved was 2,4.

TABLE I. AGE, SEX AND RACE OF 54 PATIENTS SUBMITTED FOR CORONARY BYPASS GRAFTING

	Age in years					Total
	21-30	31-40	41-50	51-60	61-70	
Cape Coloured males	0	3	1	5	0	9
Cape Coloured females	1	0	0	0	0	1
White males	3	8	13	12	3	39
White females	0	0	3	2	0	5
	4	11	17	19	3	54

Lipoprotein abnormalities were found in 21 patients and in the 4 patients in the age group 21 - 30 years. The pre-operative electrocardiograms showed evidence of myocardial infarction in 16 cases (30%) and a left ventricular damage pattern in 25 (47%); it was normal in 13 (24%) and of these all except 1 had an abnormal response to submaximal exercise testing.

At cardiac catheterisation, the left ventricular end-diastolic pressure was over 15 mmHg in 30 patients (56%). Left ventricular angiography revealed areas of localised akinesis or hypokinesis in 26 (48%), and aneurysm formation in 4 (7%).

A number of the patients had other diseases or associated conditions which complicated their management. Seven had hypertension; 2 diabetes; 2 significant peripheral vascular disease; and 1 had asthma. One patient had severe calcific aortic stenosis; 1 had severe aortic insufficiency secondary to syphilis, with narrowing of the coronary ostia; a third had mitral incompetence due to papillary muscle dysfunction; and a fourth had had 2 valvotomies for mitral stenosis.

Three patients had had Vineberg implants performed and 1 patient with syphilitic ostial obstruction had had an endarterectomy performed with a temporary relief of symptoms.

During the period under review, 54 patients had 84 saphenous vein grafts inserted. The site of insertion and the number of vein grafts performed are shown in Table II. During these operations 5 patients had Vineberg implants as well, 2 had an aneurysmectomy, and 1 had

both surgical treatments. Two patients had an aortic valve replacement, and 1 a mitral annuloplasty.

TABLE II. ANALYSIS OF THE VEIN GRAFTS CONSTRUCTED: THEIR SITE OF INSERTION AND NUMBER OF GRAFTS

Coronaries grafted	No. of patients
LAD	16 - 29 ² / ₃
RCA	8 - 15 ⁰ / ₃
L. CIRC.	1 - 2 ⁰ / ₃
LAD & RCA	22 - 41 ² / ₃
Other combinations	6 - 11 ² / ₃
LAD, RCA & L. Circ	1 - 2 ⁰ / ₃
Total	54 - 100⁰/₃

LAD = left anterior descending artery; RCA = right coronary artery; L.circ. = left circumflex coronary artery.

Of the aortocoronary grafts (Table II), 25 were single grafts, 28 double and 1 triple. In 6 of the double grafts a Y-graft with a single aortic anastomosis was used; 43 grafts (51%) were inserted into the left anterior descending artery; the right coronary artery was grafted in 34 (41%); and the circumflex artery in 7 (8%).

As many of the patients had extensive 3-vessel disease, and most had only 1 or 2 vessels suitable for grafting, it was recognised that complete revascularisation had not been achieved in many cases.

RESULTS

Operative Deaths

Three patients died within a few hours of the operation and 1 within a month, giving an operative mortality of 7%. Deaths resulted from haemorrhage from the aortic cannulation site in 1; a thrombosed graft in a second; and intractable ventricular fibrillation in a third; all 3 grafts were patent at autopsy. The patient who died a month after surgery had had a recent anterolateral myocardial infarct and a large pericardial effusion due to occlusion of the Y-graft.

Late Deaths

The remaining 50 patients have been followed for a period varying between 2 and 31 months (mean 13,0 months). During this period a further 7 patients have died, giving a late mortality of 14%. The time between surgery and death varied between 9 - 24 months (mean 18,4 months). Of 50 patients who had left hospital, 37 have been recatheterised at periods varying from 1 - 24 months postoperatively (mean 6,2 months).

These patients had 60 grafts, of which 45 (75%) were patent at the time of recatheterisation.

The 7 patients who died later in the postoperative period had all been recatheterised and the graft patency in this group was 83%, not significantly different from the patency rate of the survivors (73%) (Table III),

TABLE III. PATENCY OF THE GRAFT AS DEMONSTRATED BY POSTOPERATIVE ANGIOGRAPHY IN RELATION TO ARTERY GRAFTED AND IN THOSE SUFFERING LATE DEATHS COMPARED WITH THE SURVIVORS

	LAD		RCA		Lt. CIRC.		
	Patent	Blocked	Patent	Blocked	Patent	Blocked	
Patients alive	21	5	10	7	4	1	Patency 35/48 (73%)
Late deaths	5	1	5	1	—	—	Patency 10/12 (83%)
Total	26	6	15	8	4	1	
	Patency 81%		Patency 65%				

although the time interval between surgery and re-examination, and the duration of follow-up between the late death group and the survivors, were similar. Retrospective analysis revealed a higher incidence of electrocardiographic abnormalities and of left ventricular dysfunction as judged by left ventricular cine angiography and left ventricular end-diastolic pressures (Table IV). The incidence of late deaths did not appear to be influenced by the age of the patient or the performance of additional surgical procedures.

TABLE IV. FACTORS ASSOCIATED WITH THE LATE DEATHS

	Patients alive	Late deaths
Pre-op. ECG normal	16	0
Ischaemia/infarction	14	7
Pre-op. LV function		
Normal	15	2
Abnormal	15	5
Pre-op. LVEDP		
Normal	15	1
Elevated	15	6
Other diseases		
Absent	22	3
Present	8	4

Vein Graft Patency

Thirty-four grafts were studied less than 6 months after surgery, and 23 were patent (68%). Of 26 grafts studied 6 months or later after surgery, 22 were patent (85%). The over-all patency rate was 75%. The patency rate in 1971 and 1972 was a little better than that during 1970 (Table V).

TABLE V. VEIN GRAFT PATENCY ANALYSED BY YEAR OF OPERATION

	1970	1971	1972
Vein grafts patent	15 - 68%	21 - 81%	9 - 75%
Vein grafts blocked	7	5	3
Grafts not yet studied	0	2	15
Total No. of grafts	22	28	27

There was no correlation between the flow obtained through the graft at the time of surgery and the subsequent fate of the graft. There did not appear to be any difference in patency between those judged to have a relatively poor distal 'run-off' angiographically (72%), and those with a good distal 'run-off' (77%), nor was there any difference in patency in those grafts inserted into vessels with a diameter of 1,5 mm or over, as compared with grafts into smaller vessels less than 1,5 mm diameter.

Older patients (over 45) in the series appeared to fare less well, with a patency rate of 69% compared with a patency rate of 84% in the younger group under 45.

Although 5 different surgeons were involved in these operations, there was no significant correlation with patency rate.

Subjective Results

Complete relief of angina was reported by 27 of the 43 survivors (63%) at the time of the last follow-up. A further 13 (30%) claimed a significant improvement in the frequency and severity of the angina. Symptomatic improvement thus occurred in 93% of patients. At least 1 patent graft was demonstrated at catheterisation in 87% of the survivors, and there was a good correlation between the patency rate and symptomatic improvement as seen in Table VI. There was, however, 1 notable exception.

TABLE VI. RELATIONSHIPS BETWEEN SUBJECTIVE IMPROVEMENT AND VEIN GRAFT PATENCY

	All patent	Half patent	All blocked	Not restudied	Total
No angina	14	4	1	8	27 - 63%
Less angina	4	3	1	5	13 - 30%
No improvement	0	1	2	0	3 - 7%
	18	8	4	13	43 - 100%

A patient claiming complete relief of symptoms for 2 years after surgery, had a blocked graft at the time of

re-examination, without evidence of intra-operative or postoperative myocardial infarction. He had, however, had an aneurysmectomy and a Vineberg implant performed, in addition to his vein grafts.

Operative and Postoperative Myocardial Infarction

Five patients had ECG evidence of myocardial infarction in the operative period, and 2 patients developed myocardial infarction 6 months and a year after surgery, respectively. Of the operative infarctions 1 occurred during induction of anaesthesia and this patient was subsequently shown to have 2 patent grafts. One occurred in a patient with 1 blocked and 1 patent graft, and 3 occurred in patients with all grafts occluded.

One of the late infarcts occurred in a patient with extension of the atheromatous process to involve a previously patent right coronary vessel, and in the other an inferior infarct developed despite patent grafts in right and anterior descending vessels. In this case the circumflex artery was obstructed but could not be grafted at the time of surgery.

Left Ventricular Function

Postoperative studies revealed that left ventricular and end-diastolic pressure was unchanged in 14 cases, rose by 5 mmHg in 11, and fell by 5 mm or more, in 11.

Left ventricular cine angiography showed no change from the pre-operative angiogram in 25 patients. New areas of hypokinesis or akinesis were seen in 5 patients, and only 2 of these were clinically recognised as having had postoperative myocardial infarction. In only 2 patients did previous areas of hypokinesis return to normal function.

Four patients had evidence of severe left ventricular dysfunction, pre-operatively. Three have died from this cause, 14, 16 and 19 months postoperatively, respectively. The fourth patient made an excellent recovery after combined grafting of his occluded anterior descending artery and mitral annuloplasty for severe mitral insufficiency secondary to papillary muscle dysfunction.

Results in Crescendo Angina

Seven patients came to surgery for crescendo angina without preceding myocardial infarction. One died at surgery; 1 had a recurrence of angina 6 months later, and 5 are asymptomatic. None developed myocardial infarction in the operative period.

DISCUSSION

The results obtained in our series of 54 patients with saphenous vein bypass grafting with a 7% mortality, a 75% graft patency rate, and significant relief of symptoms in the majority of patients, is similar to results reported from other sources,⁸⁻¹⁰ but falls far short of the excellent results achieved in Cleveland¹¹ with an operative mortality

of under 2% and a graft patency rate of 83%. The reasons for our less favourable results are probably multiple.

Our series is quite small and with more experience many technical problems may be eliminated. It is also probable that with greater experience, cases will be more carefully selected to exclude those in whom the vessel diameter and 'run-off' appear to be adequate angiographically, but in whom the surgeon reports the vessel wall to be extensively diseased, making the anastomosis technically very difficult, or impossible.

It is also to be expected that by our selection of patients on the basis of severe intractable, medically unresponsive angina pectoris, we are dealing with a group of patients with extensive 3-vessel disease which is more often diffuse rather than localised to the proximal segments. Earlier cases with less severely affected vessels may give much better mortality and graft patency rates, but these may equally turn out to have a better long-term prognosis and response to medical management without surgical intervention. This information is as yet not available.

Our series does not confirm the previously-claimed relationship between graft patency and distal vessel 'run-off' flow rate at the time of graft insertion, or size of the grafted vessels.¹⁴

Since our patients have only been re-examined once, we do not know how many of our graft failures are early, presumably due to thrombosis and technical problems, or late, due to fibrous intimal proliferation.⁵ Significant narrowing of the vein graft suggestive of fibrous intimal proliferation was seen in 2 patients studied 6 and 9 months after surgery, and in both cases it occurred in vessels with poor 'run-off' into the distal arterial segment. The graft patency rate of those catheterised after 6 months, (85%), compared with a patency rate of 68% in those catheterised before 6 months, is probably the result of early re-examination being undertaken in patients becoming asymptomatic after surgery.

Some of our apparent graft failures may have arisen from technical difficulties in catheterising the graft at the time of re-examination. In at least 1 patient, in whom we were not able to show a graft postoperatively, a patent vessel was found at later surgery for cardiac transplantation.

In conformity with some other reports, we have very little evidence of improvement in ventricular function after successful bypass grafting. In only 2 cases did previous hypokinetic segments of the left ventricle return to normal function. In other reports^{13,14} as many as 51% of wall-motion defects were improved by successful revascularisation. The variable and unpredictable effect of the operation on left ventricular dynamics and function presumably reflects the varying pathological processes underlying areas of hypokinesis or akinesis seen on ventriculography, some of which are reversible (ischaemic), and others irreversible (infarcts, fibrous scars, and aneurysms).

Myocardial infarction in the operative period was in most cases due to failure of the vein graft or to unrelieved ischaemia in an area not amenable to grafting. The 2 cases of late postoperative myocardial infarction were related to incomplete revascularisation in 1, and extension of the atherosclerotic process in a previously unobstructed vessel, in another.

Our relatively high late-mortality rate of 14% has occurred chiefly in patients with poor ventricular function and severe heart failure before surgery (3 cases). Of the remainder, all had severe 3-vessel disease and complete revascularisation had not been possible for them. In 1 case the vein graft was found severely obstructed at the aortic anastomosis at the time of re-examination. In only 1 patient was the fatal outcome somewhat unexpected. This was a sudden death occurring in an asymptomatic young man with good ventricular function and 2 well-functioning grafts to right and anterior descending arteries. Even here, however, the circumflex artery was known to be diseased, but able to be grafted. A postmortem examination was not performed.

In conclusion, we feel that a large-scale surgical approach to all forms of coronary artery disease is not justified, and the current enthusiasm for this form of treatment is likely to wane.

Surgery in the mildly symptomatic or asymptomatic patient has yet to be shown to be superior to medical management, from both the point of view of control of symptoms and long-term survival.

Surgery in patients with severe ventricular dysfunction and left ventricular failure is generally contra-indicated, but a small place exists for this form of treatment in the severely symptomatic patient not responding to medical

treatment, and in whom distal vessels suitable for bypass grafting are demonstrable; but with a more careful selection the yield of operable cases is likely to be smaller rather than greater.

The value of bypass grafting in acute coronary insufficiency, crescendo angina, and impending myocardial infarction, is still being evaluated.

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