

State of the vein grafts, native coronary arteries, and myocardium and principal cause of death in patients dying after aortocoronary bypass grafting

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ABSTRACT Fifty five patients with 108 coronary bypass saphenous vein grafts were studied at necropsy. The mean duration of the grafts was 153 days (SD 516). The luminal narrowing of the native coronary arteries proximal to, at, and distal to the vein graft anastomoses and the narrowing of the non-grafted arteries were evaluated planimetrically. Twenty nine per cent of coronary arteries distal to graft anastomoses showed at least 76% narrowing and 50-75% occlusion was seen in 39% of such arteries. Fifty three per cent of non-grafted arteries showed at least 76% luminal narrowing and 26% had 50-75% narrowing. Six patients (11%) had surgically induced dissection of coronary arteries. Seventy seven vein grafts (71%) showed no appreciable luminal narrowing. Problems related to operative technique caused 30% of the deaths.

Coronary atherosclerosis is the commonest disease affecting the heart¹ and ischaemic heart disease has reached "epidemic proportions" in white South Africans.² In the United States 750 000 deaths a year are estimated to be due to coronary occlusion and more than 1 000 000 people are affected with angina pectoris.³ Coronary arterial bypass vein grafting has been the most important advance since 1969 for the management of the patient with moderate to severe symptoms from angina due to obstructive atherosclerotic coronary arterial disease.⁴ Coronary bypass surgery has become the most commonly performed heart operation⁵ in the United States (170 000 operations were performed in 1983). About 150 such operations are currently performed annually in our institution. Several papers give the clinical results of coronary bypass surgery both in our institution⁶ and in other South African hospitals.⁷⁻¹⁰

All patients with unstable angina are potential candidates for reperfusion (thrombolysis, percutaneous transluminal coronary angioplasty, or coronary bypass surgery), or any combination of these; the type of reperfusion used will depend on individual (and hospital) experience and expertise as well as on the

findings in the individual patient.¹¹ Routine angiography after a coronary bypass operation may produce artefactual "occlusion" caused by altered or competitive flow relations.¹² Postmortem studies yield more reliable data. While several reports record the morphological observations in vein grafts and in native coronary arteries of patients who had died after coronary bypass surgery,¹³⁻²² only two^{23,24} give data on the state of the native coronary arteries proximal to, at, and distal to the vein graft anastomoses. The present report emulates the latter two studies and also records the state of the non-grafted coronary arteries and the myocardium. The principal cause of death in each patient is also identified.

Methods

The hearts studied were from 55 patients who died at various times after coronary bypass surgery for obstructive coronary disease. These represented all of the specimens from cases in which this operation was performed and received for examination up till the end of June 1984. Surgically resected failed vein grafts from three additional patients were also examined during the study period. Three surgical services were concerned, but 44 of the patients came from Groote Schuur Hospital. The 55 patients had received a total of 108 saphenous vein grafts.

The patients comprised 43 men and 12 women; the mean age was 50.6 (SD 11.2) years, with a range of

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25–82 years. Atherosclerosis was the indication for operation in 50 patients. The remaining five patients were operated on for the following coronary arterial diseases: catheterisation induced dissection; bilateral postcannulation coronary ostial stenosis; direct trauma to a coronary artery during mitral valve replacement; and syphilitic coronary ostial stenosis (two patients). One patient had coronary bypass surgery after attempted lysis of a coronary arterial thrombus and two others underwent endarterectomy at the time of the bypass operation. Fifteen patients underwent heart valve replacement at the same time as the bypass (13 aortic, two mitral, and one tricuspid valve replacement). Six patients had postischaemic left ventricular aneurysms and three patients underwent aneurysmectomy at the time of the bypass surgery.

The major coronary arteries were sectioned transversely at intervals of about 3 mm and all segments showing 50% or more luminal narrowing were taken for histological examination, with representative samples of less severely diseased or normal arteries. The saphenous vein grafts and their anastomoses were similarly sectioned and sampled. All sections were stained with haematoxylin eosin and elastic van Gieson. To overcome the effects of postmortem collapse of coronary arteries on estimation of the severity of stenosis, true cross sections of the arteries studied were selected from the histological slide and projected on to a sheet of paper. One traced out the perimeter (circumference) of the residual lumen and of the internal elastic lamina, the latter being taken to indicate the original lumen of the vessel. The lengths of these two perimeters were measured with a graphics tablet digitiser board linked to an Apple microcomputer. These perimeters were assumed to be constant after collapsing down from the expanded (perfused) state. Both of these perimeters were converted to circular profiles, which more closely approximate the state of the coronary artery during life, the formulae of Lowe²⁵ being used. The circumferences of the circular profiles were the same as those obtained from the original flattened vessels. Planimetry (using the graphics tablet digitiser board) was carried out on each circular profile, yielding the area contained by the internal elastic lamina and the area contained by the

lumen. The degree of luminal narrowing was assessed as the percentage ratio of the luminal area to the area contained within the internal elastic lamina of the artery. The percentage luminal narrowing was graded as follows: grade 1 narrowing, 0–25%; grade 2, 26–50%; grade 3, 51–75%; and grade 4, 76% or more.

Results

The 108 vein grafts in the 55 patients were anastomosed between the aorta and the following coronary arteries: left anterior descending branch of the left coronary artery, 46 grafts; a diagonal branch of the left anterior descending artery, six grafts; circumflex branch of the left coronary artery, 27; and the right coronary artery, 29 grafts. The mean duration of the vein grafts was 153 (SD 516) days with a range of 0–2555 days. The degree of narrowing of the native coronary arteries is indicated in table 1.

Fifty three per cent of non-grafted arteries showed more than 75% luminal narrowing by atherosclerosis and 26% showed 25–50% narrowing. Twenty nine per cent of coronary arteries distal to the site of vein graft anastomoses showed more than 75% luminal narrowing (fig 1). These latter arteries were present in 22 patients, six of whom showed acute myocardial necrosis; three of the latter group also showed thrombosis of the relevant vein graft (fig 2). Seventeen of these 22 patients had died less than 14 days after operation (mean (SD) survival 1.9 (3.9) days). Thirty nine per cent of arteries distal to the vein graft anastomosis showed grade 3 luminal narrowing. Only one quarter of acutely thrombosed vein grafts had grade 4 (76% or more) arterial luminal obstruction distal to the anastomosis.

Only one patient underwent bypass grafting for coronary dissection, which had been induced by cardiac catheterisation. Six other patients (11%) had recent coronary arterial dissection at the level of the vein graft. None of these patients had associated aortic dissection, all had been operated on for coronary atherosclerosis, and in each case the dissection had not been noted at the time of the bypass surgery. The left anterior descending coronary artery was affected in five instances and two patients also had a dissection of the left circumflex coronary artery. All six of the

Table 1 Grades of arterial narrowing (as assessed by planimetry) in grafted and non-grafted coronary arteries of patients undergoing postmortem examination after coronary bypass surgery

Coronary arteries	No (%) with grade of luminal narrowing			
	1 (<25%)	2 (25–50%)	3 (51–75%)	4 (>76%)
Non-grafted (n = 53)	3 (6)	8 (15)	14 (26)	28 (53)
Proximal to anastomosis (n = 108)	1 (1)	8 (7)	40 (37)	59 (55)
Distal to anastomosis (n = 108)	19 (17)	16 (15)	42 (39)	31 (29)



Fig 1 Obstruction in arterial segment distal to vein graft anastomosis. The three sections are different levels of the same arterial segment. (Elastic van Gieson, $\times 2$.)

surgically induced dissections produced severe luminal obstruction. Table 2 lists the pathological changes (fig 3–6) observed in the 108 saphenous vein grafts that had been implanted in 55 patients. Seventy seven vein grafts (71%) were free of appreciable luminal narrowing. Intimal fibroplasia appeared to be compensated for by graft ectasia associated with medial atrophy.

The two patients with dehiscence of the aortic vein graft anastomosis and the two with vein grafts of

Table 2 Pathological alterations observed in 108 coronary bypass saphenous vein grafts in 55 patients

	No of cases
Denuded intima with scanty fibrin deposits	8
Thrombosed vein graft	16
Recanalised vein graft	2
Vein graft converted into fibrous cord	1
Vein graft too short	2
Dehiscence of aortic anastomosis	2
Ectatic vein graft	1
Intimal fibroplasia	8
Atherosclerosis like changes	2

inadequate length had not been operated on at our institution. Only three out of the 16 patients with thrombosed vein grafts had associated thrombosis of the native coronary to which the graft had been anastomosed. Four of the 16 patients with thrombosed vein grafts had grade 4 luminal narrowing of the coronary artery distal to the graft anastomosis. Thus poor runoff of blood from the vein graft played only a minor part in the aetiology of the thrombosis. The mean (SD) heart weight of the 55 patients studied was 518 (131) g and table 3 indicates the state of the myocardium in these patients.

Nine patients (23% of those who died up to six days after operation) showed healed transmural necrosis of the left ventricular myocardium; one of seven patients dying seven to 30 days after operation showed this change, which was not observed in any of the nine patients who died more than 30 days after operation. The principal causes of death in each of the 55 patients with vein grafts having a necropsy are summarised in table 4. Problems related to operative technique accounted for 30% of the deaths in this series.



Fig 2 Thrombosed vein grafts. A—Thrombosed vein graft ostium in aorta. B—Anastomosis between thrombosed coronary artery and vein graft. (Elastic van Gieson, $\times 8$.)

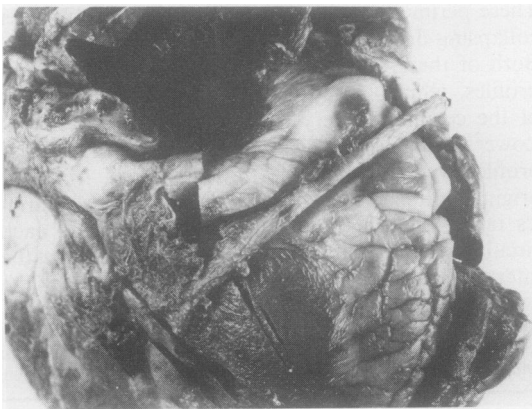


Fig 3 A too short vein graft to posterior descending branch of right coronary artery grooving the underlying myocardium. Distention of the heart during life amplifies this abnormality and further compresses the vein graft lumen.



Fig 4 Graft anastomosis site shows vein (above) and native coronary artery (below). The arrow indicates a surgically created artefact affecting both intima and media of the posterior wall of the artery; such a defect may lead to a dissecting aneurysm. (Elastic van Gieson, $\times 20$.)

Discussion

Fifty three per cent of non-grafted arteries showed grade 4 luminal narrowing and grade 3 narrowing was present in a further 26%—that is, 92% of non-grafted coronary arteries showed at least one lesion that narrowed the arterial lumen by 51% or more. This appears to be a reflection of the diffuse nature of the atherosclerotic process in the coronary arteries of the patients referred for coronary bypass surgery. Angiography may have failed to show some important foci of obstruction. Furthermore, 29% of grafted coronary arteries showed grade 4 narrowing distal to the anastomosis and grade 3 narrowing was present in 39% of such distal arterial segments—that is, 68% of grafted arteries showed 51% or more luminal narrowing distal to the site of vein graft anastomosis. Possible reasons for this include poor angiographic evaluation as suggested above and failure of the surgeon to carry the graft distal to the significant arterial obstruction. Others have also reported appreciable obstruction in coronary arterial segments, both in non-grafted arteries^{14 24} and in arterial segments beyond grafts.^{14 23 24} Progression of atherosclerosis in non-bypassed coronary arteries appears to be the major cause of recurrence of myocardial ischaemia some time after coronary bypass surgery.^{24 26} Early post-operative mortality rates and the frequency of post-operative myocardial infarction are lower and the fre-

quency of disappearance of angina pectoris and improvement in exercise tolerance are greater in patients having more than one vein graft.²⁴

Six patients (11%) were found to have coronary arterial dissection at the level of the vein graft anastomosis. This is slightly less than the 17% incidence of this complication reported by Spray and Roberts.²³ As in their patients, the length of dissection was usually short and the occurrence of this complication appeared to result from detachment of atherosclerotic plaque from the coronary arterial wall; it was seen most commonly when the anastomosis was created at a site showing appreciable coronary arterial atherosclerosis. In two patients, however, surgical damage to the posterior wall intima, which appeared to have occurred at the time of incision into the native coronary artery at the anastomosis site, seemed to be the cause of the dissection. The idea that this is an alternative mode of initiating dissection is supported by the finding of occasional similar surgical artefacts in coronary vessels at anastomotic sites unassociated with dissection (fig 4). Aortic dissection has been reported as a complication of coronary bypass,²⁷ usually being related to trauma during surgical cannulation with retrograde perfusion through atherosclerotic femoral or iliac arteries or secondary to damage produced by aortic cross clamping.

About half of the vein grafts (49%) showed microscopical evidence of thrombosis; this was recent in 16 grafts (29%) studied in the early postoperative period. Vein grafts examined up to two weeks after implantation were devoid of endothelial lining cells. Chesebro *et al*^{28 29} believe that graft occlusion begins during the operation with endothelial cell damage to the vein graft and is followed by thrombosis. Thus drug treatment to oppose this process must start before the operation to have maximal effect. Intimal fibroplasia, mostly of limited extent, was noted in eight grafts (15%) in this study. A graft that had undergone occlusive thrombosis in the early post-operative period was represented by a fibrous cord. The two dehiscence vein grafts appeared to have developed this complication owing to the inclusion of too little aortic tissue in the sutures. This allowed very good apposition of venous intima to aortic intima, but meant that even slight cutting of the suture material through the aortic tissues led to dehiscence of the anastomosis. Both grafts appeared to be of an adequate length. Several reports document the pathology of autogenous saphenous veins used as conduits for aortocoronary bypass grafts.^{30–42} In addition to complications similar to those noted above, the following changes have been documented: atherosclerotic aneurysm in aortocoronary vein graft,³⁵ late vein graft thrombosis due to rupture of an atherosclerotic plaque in a vein graft,³⁸ early focal narrowing

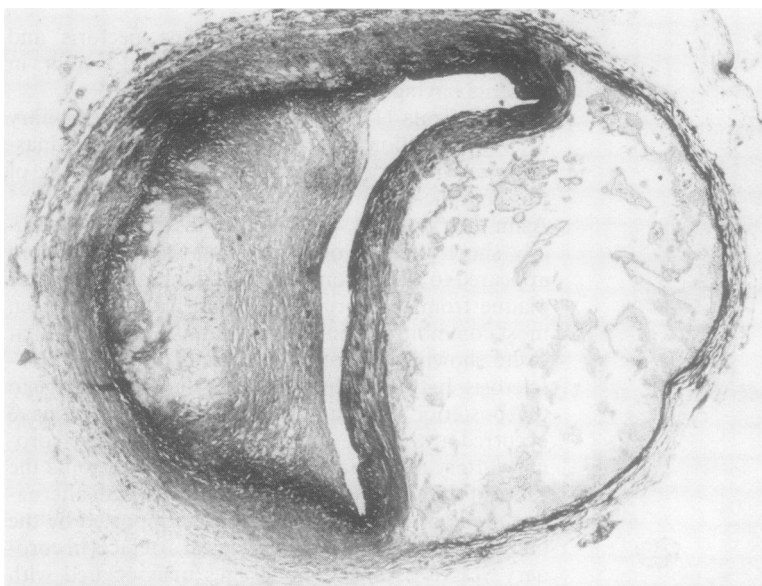


Fig 5 Section of coronary artery distal to graft anastomosis shows dissection between media and adventitia resulting in severe luminal narrowing. (Elastic van Gieson, $\times 13$.)



Fig 6 Site of dehiscence (indicated by the probe) of a vein graft at its aortic anastomosis.

at the anastomoses or in the vein graft due to inappropriate surgical techniques, and endocardial lesions.⁴³ Both homologous⁴⁴ and heterologous⁴⁵ saphenous vein grafts give poor results. Although the search for a suitable prosthetic aortocoronary bypass graft continues,^{46 47} nothing better than the autologous vein has been introduced. Some authors recommend the internal mammary artery as an alternative graft.⁴⁸

Other unusual complications of coronary bypass surgery include a continuous murmur due to anastomosis of a vein graft to a coronary vein,^{49 50} coronary arterial spasm induced by coronary bypass,⁵¹ false aneurysm of the ascending aorta,⁵² constrictive pericarditis,^{53 54} postoperative phlebitis of a graft,⁵⁵ twisting of a grafted vein,⁵⁶ aortic aspergilloma resulting in supravalvular aortic stenosis after coronary bypass surgery,⁵⁷ and a dissecting aneurysm of

Table 3 Incidence of myocardial necrosis in distribution according to time after operation among 55 patients with coronary arterial bypass vein grafts (%)

Days survived after surgery	No of cases of necrosis	Types of myocardial necrosis		
		Coagulative	Contraction band	Myocytolysis
0-6 (n = 39)	24 (71)	10* (26)	7 (18)	7 (18)
7-30 (n = 7)	4 (57)	3 (43)	1 (14)	1 (14)
> 30 (n = 9)	4 (44)	2 (22)	1 (11)	1 (11)
Total (n = 55)	32 (58)	15 (27)	9 (16)	10 (18)

*Two patients had haemorrhagic, reperfusion type infarcts.

the grafted saphenous vein itself.⁵⁸ Reoperation may be necessary and it has been shown to have relieved severe symptoms in more than 60% of patients five years later.⁵⁹ Percutaneous transluminal coronary angioplasty has also been performed in patients who have previously had coronary bypass surgery.⁶⁰

Seventy one per cent of patients who died less than six days after operation showed evidence of acute myocardial necrosis and two of these patients had haemorrhagic, reperfusion type infarcts. Roughly half of the patients who died later than this showed recent myocardial necrosis. These findings are similar to those of Vlodaver and Edwards,¹⁴ who encountered acute myocardial infarction in 14 out of 15 patients dying one to six days after coronary bypass surgery and in nine out of 12 patients surviving seven to 30 days. Lie *et al*⁶¹ reported haemorrhagic myocardial infarcts in 38% of patients one to 14 days after operation. In a clinical study, Langou *et al*⁶² found that perioperative myocardial infarction occurred in 23% of all patients undergoing coronary bypass surgery. A similar study by Guiteras *et al*⁶³ found perioperative

myocardial infarction in 13.4% of patients having this operation. Schaff *et al*⁶⁴ state that perioperative myocardial infarction has an important independent influence on late survival after coronary bypass and is surpassed only by left ventricular function, age, and number of associated medical diseases.

Bulkley and Hutchins⁶⁵ report that coronary artery reflow through widely patent grafts after the period of operative non-perfusion (rather than graft or intrinsic coronary artery occlusion) accounts for most of the operation related myocardial "infarcts" associated with coronary bypass surgery. Robinson *et al*⁶⁶ observed myocardial necrosis with contraction bands or haemorrhagic myocardial necrosis (or both) in the hearts of 44 out of 50 patients. They attribute sub-endocardial haemorrhagic myocardial necrosis to hypoperfusion of the myocardium, whereas contraction band necrosis had a multifactorial aetiology—for example, anoxic arrest, cardiotoxic drugs, electrical defibrillation, electrolyte imbalance, microthrombi, and hypoperfusion. Keon *et al*⁶⁷ observed perioperative myocardial infarction caused by atheroembolism: the emboli originated from vein graft ostia in ulcerated atherosclerotic aortas, from coronary endarterectomy sites, or from operative disruption of plaques in coronary arteries having bypass surgery. Others have studied the ultrastructural features produced by reperfusion of ischaemic myocardium.^{68 69} Reperfusion of ischaemic myocardium induces slow structural recuperation after reversible injury, and accelerated cellular destruction after irreversible ischaemic injury. Willerson *et al*⁷⁰ believe that the frequency of perioperative infarction depends on the clinical condition of the patient, the technical skill of the operating team, and the type of myocardial protection used, as well as the diligence with which this diagnosis is sought.

Important findings to emerge from the present study include a high incidence of severe coronary arterial disease in non-grafted major coronary arteries as well as in the distal segments of grafted arteries. These

Table 4 Principal cause of death in 55 patients with coronary bypass vein grafts undergoing postmortem examination

	No (%) of cases
Primary vein graft thrombosis	7 (13)
Operative technique	
Dehiscence of anastomosis	2 (3)
Vein graft too short	2 (3)
Coronary artery dissection	7 (13)
Grade 4 distal narrowing	6 (11)
General postoperative complications	7 (13)
Fresh myocardial necrosis	7 (13)
Miscellaneous complications	
Prosthetic valve endocarditis	1 (2)
Bleeding from right atrium	1 (2)
Brain haemorrhage (anticoagulant)	1 (2)
Septicaemia	1 (2)
Unknown	13 (23)

fatal cases also showed the potential for reducing the mortality rate of coronary bypass by improving surgical techniques since problems related to the latter accounted for 30% of the deaths in this series.

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