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Coronary Bypass Graft Fate: Long-Term Angiographic Study

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In 222 patients, 741 venous coronary bypass grafts were studied angiographically early, at 1 year and at a late examination at >6.5 years (mean 9.6) after operation; 565 of these grafts were also examined 5 years postoperatively. Grafts were graded for patency and disease considered to be atherosclerotic and for both extent and profile of lesions.

Graft occlusion rates increased steadily from 8% early to 20% at 5, 41% at 10 and 45% at >11.5 years after operation. All grafts were considered free of atherosclerosis early, but disease appeared in 8% at 1 year, increasing to 38% at 5 and 75% at 10 years postoperatively. Increasing involvement of vessel wall area was associated with greater protrusion of lesions into the graft lumen. Diseased grafts became more so at subsequent examinations, with occlusion occurring in many. However, absence of disease had little prognostic significance because diseased and abruptly occluded grafts were generated in those with healthy appearance at earlier examinations. For instance, 82% of very diseased grafts at the 5 year study originated from normal grafts at 1 year and 73% of occluded grafts at 1 year had appeared normal early postoperatively.

Of 590 patent grafts free of disease at 1 year, 30% were occluded at the late examination, 76% of those patent were diseased, 55% of these were diffusely diseased and 35% were >50% narrowed. Only 17% of the original 590 patent grafts were healthy at this time. Bypass graft atherosclerosis severely limits the long-term utility of these grafts. It is suggested that the solution may lie in some powerful drug regimen.

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It is well known that the long-term fate of coronary bypass vein grafts is principally determined by graft atherosclerosis. We (1) previously reported angiographic features of this disease up to 5 years after operation. We now describe its appearance and progression in coronary bypass vein grafts from 6.5 to >11.5 years postoperatively. As in our previous study, we used a grading system to define patency and atherosclerosis of 741 vein grafts studied consecutively.

Methods

Study patients and grafts. In this study, we included only those patients who had follow-up angiograms early and at 1 year and >6.5 years after operation. One hundred sixty-four patients (565 grafts) were studied early (mean 0.96 months) and at 1 year (mean 12.84 months), 5 years (mean 60 months) and >6.5 years after operation. In addition, there were 51 patients (176 grafts) studied early, at 1 year and at a remote time after operation. Two hundred thirty-seven grafts were studied at 7.5 years (mean 7.4), 403 at 10 years (mean 9.9) 61 at 12.5 years (mean 12.7) and 40 later (mean 14.2 years) after operation. Thus, 741 grafts in 222 patients were studied early, after 1 year and in the course of a late examination ≥6.5 years after operation; the majority of grafts were also examined 5 years after operation. The interval from operation to the late examination ranged from 80 to 187 months (mean 116) (9.6 years). The study included angiographic examination of 25% of all grafts in patients surviving 6.5 years after operation. The subjects were mainly military personnel, all men, ranging in age from 31 to 67 years (mean 45.7).

Operations. Preoperative and follow-up evaluation of the patients was done by the Cardio-Pulmonary Unit of the National Defence Medical Centre, Ottawa, Ontario, Canada, but operations were undertaken by five surgeons at the University of Ottawa Heart Institute, where patients usually remained for ≤48 h after operation. All operations entailing coronary bypass surgery were included in the study (for example, bypass operations also involving other procedures such as valve replacement or ventricular aneurysm repair). There were 3.3 vein grafts/operation. Internal mammary grafts were excluded. As background information, we report a perioperative mortality rate of 1.7% for all 1,202 first coronary bypass operations and of 5.4% for all 149 reoperations. In the larger group from which study patients were derived, 92.7% (824 of 889) of patients had survived 6.5 years after operation. We (2) previously reported a perioperative myocardial infarction rate of 7.8% (transmural 3.2%).

Technical background. Saphenous vein was harvested with minimal manipulation and, pending placement, was kept in normal saline solution containing 60 mg of papaver-

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Table 1. Graft Patency Grading on Follow-Up Angiography

	Early	1 Year	5 Years	7.5 Years	10 Years	>11.5 Years
Total grafts	741	741	565	237	403	101
Graft grade						
A	631 (85)	608 (82)	428 (76)	109 (46)	211 (52)	40 (40)
B	52 (7)	34 (5)	24 (4)	31 (13)	27 (7)	15 (15)
A + B (patent)	683 (92)	642 (87)	452 (80)	140 (59)	238 (59)	55 (55)
O	58 (8)	99 (13)	113 (20)	97 (41)	165 (41)	46 (45)

Numbers in parentheses are percent. A = good patency; B = graft narrowed at some point to <50% of *grafted artery*; O = graft occluded.

ine/100 ml. Most bypass grafts had a single distal anastomosis, but some were of the sequential type, our experience with these having been described elsewhere (3). Twenty-eight percent of the grafts were to the anterior descending coronary artery, 20% to its diagonal branches, 28% to branches of the marginocircumflex trunk and 24% to the trunk or branches of the right coronary artery. At angiography, grafts were selectively opacified, usually with a Judkins size 4 right coronary catheter, in at least four vertical planes ranging from 60° to the right of the mid-line to 90° to the left of the mid-line, sometimes with axial views. Catheterization was facilitated by graft loop markers placed on the aortic wall at operation. Grafts were usually demonstrated to be occluded by opacification of stumps, sometimes supplemented by collateral evidence in native selective coronary angiograms; rarely, proximal aortic-flood examinations were required. High resolution radiographic equipment was used for recording on 35 mm cinefilm at 60 frames/s.

Graft patency grading. We used the grading system previously reported (4) to define bypass graft patency. The proximal and distal anastomoses of the graft and the trunk were assessed separately and each assigned a letter: A (good), B (fair) or O (occluded). Grade B indicated stenosis *reducing the lumen to <50% of the grafted artery*. The grade for the entire graft was the lowest of the three site gradings. This was essentially a hemodynamic grading system. We used another method, previously described (1), to classify angiographic appearances we believed due to atherosclerosis. This provided more purely morphologic information. Category I indicated that the graft outline was completely smooth without any irregularity that might be due to disease; in category II grafts, <50% of the estimated surface area of the graft intima was irregular; category III grafts had >50% of the intima involved. To define this finding more precisely and perhaps add more prognostic value, we also classified the lesions into high profile or low profile types (that is, high or low rise, elevation or relief) depending on whether they *encroached >50% or <50% on what was considered to be the normal graft lumen* at that point. These grading systems, all based on the worst aspects of four-plane views, entailed use of an "eyeballing" technique. Great care was taken, however, to classify grafts as accurately as possible within the framework outlined.

Results

Graft occlusion. Graft occlusion rates early, at 1, 5, 7.5, 10 and >11.5 years after operation (Table 1) were 8%, 13%, 20%, 41%, 41% and 45%, respectively. Grafts to the marginocircumflex and right coronary arteries had a significantly lower patency rate at the late examination than did grafts to the anterior descending coronary artery and its diagonal branches (Table 2). Most of the grafts were graded A, with a small core of grade B grafts. It is of surgical importance that in the early postoperative studies, most (46 [89%] of 52) of the B gradings were assigned because of narrowing at the distal anastomosis. After 5 years, an increasing number of grade B grafts were so graded because of trunk stenosis associated with atherosclerosis.

The 7.5 year phenomenon. The striking increase in B grades at 7.5 years, followed by a decrease at 10 years, prompts an explanation applicable to other values in the 7.5 year columns of Tables 1 and 3. We (5) have drawn attention to this phenomenon elsewhere. Our ideal follow-up practice is to perform angiography early and at 1 and 5 years and then every 5 years after coronary bypass operations. Practically all our patients have early angiograms and the majority are restudied at 1 year (1,4–6), but military/civilian career, geographic and other factors reduce the number examined at 5 and 10 years after operation. Findings in a group of regularly examined, compliant and readily available patients are probably well represented in the 5 and 10 year columns of Tables 1 and 3. However, at 7.5 years (between 79 and 102 months postoperatively), we examined patients presenting for a "clinical" rather than a "routine" reason, including a number of patients who presented ostensibly "late" for their

Table 2. Vessels Grafted and Late Graft Occlusion

	Grafted (%)	Late Occlusion (%)
Left anterior descending (LAD)	28	34
Diagonal branches of LAD	20	38
Marginocircumflex	28	49
Right coronary	24	45

Right and marginocircumflex occlusion rates significantly different ($p < 0.05$) from rates in left anterior descending and diagonal grafts.

Table 3. Bypass Graft Atherosclerosis on Follow-Up Angiography

	Early	1 Year	5 Years	7.5 Years	10 Years	>11.5 Years
Patent grafts	683	642	452	140	238	55
Not diseased (I)	683 (100)	590 (92)	278 (62)	25 (18)	60 (25)	11 (20)
Diseased						
II	0	46 (7)	98 (22)	53 (38)	79 (33)	12 (22)
III	0	6 (1)	76 (16)	62 (44)	99 (42)	32 (58)
II + III	0	52 (8)	174 (38)	115 (82)	178 (75)	44 (80)
Proportion of diseased grafts high profile	0	3 (6)	24 (14)	43 (37)	62 (35)	21 (48)
Proportion of all patent grafts high profile	0	3 (0.5)	24 (5)	43 (31)	62 (26)	21 (38)

Numbers in parentheses are percent. II = graft with <50% of the intima estimated diseased; III = graft with >50% of the intima estimated diseased; high profile = atheroma encroaching >50% on graft lumen.

5 year studies but on close questioning were found to have new angina. The values in the 7.5 year column in Tables 1 and 3 are thus weighted toward more extensive graft disease and a higher rate of graft occlusion.

Graft disease. Early after operation, all grafts were considered to be free of atherosclerosis (Table 3). At 1 year, 92% of the grafts appeared smooth walled and the remainder had some irregularity of outline, involving >50% of the graft surface area in only a few cases. By 5 years, however, only 62% of grafts had normal-appearing intima and almost 50% of the remaining grafts were considered to have atherosclerosis involving >50% of the graft surface area. Five percent of the diseased grafts had high profile lesions reducing the size of the graft lumen by >50%. At 10 years, only 25% of the patent grafts were considered healthy, >50% of the diseased grafts were in category III and 35% of the diseased grafts had high profile lesions. Further deterioration was seen >11.5 years after operation. Table 3 demonstrates proliferation of the lesions we believe to be due to atherosclerosis in the plane of the vessel wall and at the same time progressively rising from the intima to obstruct blood flow. At >11.5 years after operation, the atherosclerotic process produced >50% luminal obstruction in almost 50% of the diseased grafts; at this time, slightly >50% of the grafts remained patent and 80% of these were diseased.

Diseased graft prognosis. Our data confirm that the presence of atherosclerosis in a coronary bypass vein graft presages increasing disease in the future. In 12 of the 31 instances, category II grafts in the 1 year study became category III grafts at 5 years, and 3 other grafts became occluded. Similarly, of 33 low profile lesions at the 1 year study 7 became high profile lesions at 5 years and 4 others were associated with graft occlusion. Between the 5 year study and the late examination, 55 of 98 category II grafts became category III grafts and 27 became occluded. Likewise, of 76 category III grafts at the 5 year study, 41 were occluded at the late examination. Of 150 low profile lesions at the 5 year examination, 52 became high profile and 49 showed graft occlusion at the late examination. Of 24 high profile lesions at the 5 year examination, 19 showed graft occlusion at the late examination.

Healthy graft prognosis. The absence of disease in the course of any examination did not guarantee a similar state at the next study. For instance, 62 (82%) of 76 category III grafts at the 5 year examination were category I grafts at the 1 year study. Sixty-eight (44%) of 155 category III grafts at the late examination were classified in category I at the 5 year study. Of 193 category III grafts at the late examination, 176 (91%) were classified as category I at the early study. Similarly, 30 (73%) of 41 occluded grafts at the 1 year study were classified as grade A category I grafts at the early examination and 26 (70%) of 37 occluded grafts at the 5 year study originated from grade A category I grafts at the 1 year examination. Ninety-one (53%) of 172 occluded grafts at the late examination originated from grade A category I grafts at the 1 year study.

In summary, of 590 patent grafts free of disease at the 1 year examination, 177 (30%) were occluded at the late examination; of the 413 patent grafts, 314 (76%) were diseased; 174 (55%) of the unhealthy grafts were diffusely diseased and 111 (35%) of these were >50% narrowed; only 99 (17%) of the original 590 patent grafts were healthy. Similarly, of 278 patent grafts free of disease at 5 years, 55 (20%) were occluded at the late examination, 153 (69%) of the 223 patent grafts were diseased, 69 (45%) of unhealthy grafts were diffusely diseased and 43 (28%) of these were >50% narrowed; only 70 (25%) of the original 278 patent grafts free of disease at the 5 year study were healthy at the late examination.

Discussion

Graft occlusion. Early after operation, coronary bypass grafts may become occluded by thrombus, frequently forming at the distal graft-coronary anastomosis. It is probably highly significant that 89% of our early postoperative B

grades were assigned to grafts with distal anastomosis defects. However, these technical factors are not alone of importance. Almost 75% of grafts found occluded at the 1 year study had been angiographically normal early after operation. Intimal disruption, perhaps associated with myointimal hyperplasia, and localized platelet dysfunction may lead to the formation of an occlusive thrombus. These events are poorly understood. There is no doubt, however, that later in their course coronary bypass grafts become subject to atherosclerosis (7–15). There are proliferation of smooth muscle cells, intimal damage, the complex interaction of endothelium and locally deposited platelets and the accumulation of lipids in "foam cells" (11–13). Plaques form and assume different compositions depending on the degree of fibrosis, the extent of lipid deposit, the addition of thrombus and the occurrence of calcification. Localized aneurysm formation may take place, invariably associated with advanced atherosclerosis. The degree of disease in the bypass grafts does not appear to parallel the progress of atherosclerosis in the native coronary arteries (15). Thrombus may accumulate slowly or after an abrupt "plaque accident" similar to that occurring in a coronary artery (16). Atheroembolism may occur spontaneously or lethally at reoperation (17). The final event is graft occlusion.

Autopsy studies and examination of grafts removed at coronary bypass reoperations attest that with the passage of time atherosclerosis becomes very common in these grafts. Neitzel et al. (12) observed the disease process in 71% of grafts removed between 6 and 12 years after operation. We believe that we are observing this process at various stages in our follow-up angiograms.

Graft patency grading. Our A, B and O patency grading seems noncontroversial. The A and O grades can be assigned easily; a B grade presents some problems. Grafts are graded B because of stenosis of the proximal or distal anastomosis or in the trunk of the graft *reducing the size of the lumen to <50% of the grafted artery*. We believe that we have validated the honesty of the B grading (graft lumen <50% of the grafted artery). In a large series, we (4) demonstrated that 24% of early grade B grafts were occluded at 1 year and that 39% remained the same; however, only 6% of grade A grafts went on to occlusion and 4% became grade B ($p < 0.0005$).

Graft disease grading. Diagnosing and grading atherosclerosis in coronary bypass graft angiograms involve some speculation. We recognize that we have no systematic pathologic corroboration of our angiographic findings. Lytle et al. (10) indicated that myointimal hyperplasia and thrombus present diagnostic pitfalls. Solymoss et al. (13) described late thrombosis in vein grafts associated with nonatherosclerotic intimal hyperplasia, although this most often accompanies atherosclerosis. Nevertheless, we believe that ascription of atherosclerosis to coronary bypass graft angiograms showing what we have described as category I, II or III grafts with high or low profile lesions is in accord with pathologic and angiographic opinion (7–31). Grondin et al. (26) described these angiographic findings, including use of the terms "irregular wall," "plaque," "conventional stenosis," "spur diaphragm" and "cauliflower." These features may be seen in native vessel coronary angiograms.

In our sequential studies, as might be expected to occur with atherosclerotic disease, proliferation of lesions in the graft's mural plane is progressively associated with their elevation from the vessel wall to produce luminal obstruction. We believe that our grading system is justifiable in describing what we consider to be atherosclerosis in coronary bypass vein grafts.

Effects of drugs. For >20 years, we have given to our patients with coronary disease medications affecting the behavior of platelets for reasons arising from Duguid's encrustation theory (32) of the development of atherosclerosis. These drugs have usually been aspirin and dipyridamole, sulfinpyrazone occasionally being substituted for the former. Administration of aspirin but not dipyridamole was discontinued for 1 week before operation, sulfinpyrazone being substituted, but treatment with both aspirin and dipyridamole was started again 1 to 3 days after operation. The beneficial effect of these two agents on early and late coronary bypass graft patency has been demonstrated by Chesebro et al. (33,34). We have not had a control series. Use of these medications is not the topic of this report, but attention must nevertheless be drawn to their administration.

Graft disease and bypass grafting. An incidence of late coronary venous bypass graft atherosclerotic stenosis and occlusion generally similar to ours has been reported by others, notably in major series from the Montreal Heart Institute (8,15,31) and the Cleveland Clinic (10); our results confirm theirs. We demonstrated a relentless progression of disease in coronary bypass vein grafts with increasing large areas of intimal involvement and steady growth in the volume of lesions, graft occlusion being the end result. At 10 years after operation, 41% of our bypass grafts were occluded and 75% of those that were not occluded were diseased. Furthermore, we demonstrated in our angiograms that graft atherosclerosis behaves in a capricious manner. Bypass graft disease surely begets disease—there is nothing as atherogenic as atheroma—but a "healthy" appearance does not perpetuate itself. Graft occlusion, the final insult, is sometimes the end stage of atherosclerosis and its complications and at other times occurs for no obvious reason.

What then is the continuing long-term utility of the saphenous vein coronary bypass graft? Our limited experience with other large bore conduits, arm vein, umbilical vein and synthetic material, has been disappointing, although a single Gore-Tex right coronary bypass graft remained patent at 1 year but was occluded at 5 years after operation. Our patency rates are somewhat better for internal mammary artery grafts than for vein grafts. In a recent unpublished series of 169 consecutive internal mammary artery grafts opacified early after operation, the patency rate was 96% but there was a 10% incidence of grade B grafts, almost all

associated with distal anastomotic defects. In the long term, however, the internal mammary artery remains free of atherosclerosis and the effect of its use on late mortality has been demonstrated (35). Nevertheless, there are only two internal mammary arteries and the right vessel is anatomically limited in its reach. The right gastroepiploic artery has been used successfully for coronary bypass grafting (36,37), but its dissection complicates the coronary bypass procedure and it has not yet become widely used. Certainly, if it were not for graft atherosclerosis, the saphenous vein, which is easily accessible and plentiful, would be the ideal conduit for coronary bypass grafting.

Causes of vein graft atherosclerosis. Campeau et al. (31) demonstrated an association between elevated levels of certain blood lipoproteins and atherosclerosis in coronary bypass grafts; others (38) have narrowed the field of search. We (39) demonstrated a statistically significant relation between atherosclerosis of coronary venous bypass grafts and smoking. There are undoubtedly many other factors, ranging from intimal damage done at the time of operation to the long-term effects of "arterializing" a vein, to say nothing of possible autoimmune or even more remote genetic considerations. The reader is referred to the excellent editorial review by Grondin (11).

The future. Meticulous handling of the venous conduit at operation and exacting surgical technique are essential, particularly for early success, but these are probably not sufficient factors to prevent atherosclerosis in the long term. The high cost in lives and money of coronary bypass reoperations militates against their use in dealing with the problem of saphenous vein graft atherosclerosis. Control of conventional risk factors, particularly diet and smoking, is mandated by the available evidence. Yet this may not be enough. We have assiduously prescribed agents modifying the behavior of platelets from the beginning of our experience with coronary bypass grafting, but we doubt that our long-term results can be considered confirmation of their efficacy in preventing or retarding atherosclerosis. Perhaps our results would have been much worse if we had not used these drugs. Newer medications may save the day. These include drugs to normalize dyslipidemias (40) and the calcium channel blockers (41) reportedly used with some success. We await the advent of the agent or the technique that will restore to the saphenous vein coronary bypass graft its once great promise.

References

1. FitzGibbon GM, Leach AJ, Keon WJ, Burton JR, Kafka HP. Coronary bypass graft fate: angiographic study of 1,179 vein grafts early, one year, and five years after operation. J Thorac Cardiovasc Surg 1986;91:773–8.
2. Burton JR, FitzGibbon GM, Keon WJ, Leach AJ. Perioperative myocardial infarction complicating coronary bypass. J Thorac Cardiovasc Surg 1981;82:758–64.
3. Kieser TM, FitzGibbon GM, Keon WJ. Sequential coronary bypass grafts: long term follow-up. J Thorac Cardiovasc Surg 1986;91:767–72.
4. FitzGibbon GM, Burton JR, Leach AJ. Coronary bypass graft fate: angiographic grading of 1400 consecutive grafts early after operation and of 1132 after one year. Circulation 1978;57:1070–4.
5. FitzGibbon GM, Hamilton MG, Leach AJ, Kafka HP, Markle HV, Keon WJ. Coronary artery disease and coronary bypass grafting in young men: experience with 138 subjects 39 years of age and younger. J Am Coll Cardiol 1987;9:977–88.
6. FitzGibbon GM, Keon WJ, Burton JR. Aorta-coronary bypass in patients with coronary artery disease who do not have angina. J Thorac Cardiovasc Surg 1984;87:717–24.
7. Campeau L, Lespérance J, Hermann J, Corbara F, Grondin CM, Bourassa MG. Loss of the improvement of angina between 1 and 7 years after aortocoronary bypass surgery: correlations with changes in vein grafts and in coronary arteries. Circulation 1979;60(suppl I):I-1–5.
8. Campeau L, Enjalbert M, Lespérance J, Vaislic C, Grondin CM, Bourassa MG. Atherosclerosis and late closure of aortocoronary saphenous vein grafts: sequential angiographic studies at 2 weeks, 1 year, 5 to 7 years, and 10 to 12 years after surgery. Circulation 1983;68(suppl II):II-1–7.
9. Grondin CM. Late results of coronary artery grafting: is there a flag on the field? J Thorac Cardiovasc Surg 1984;87:161–6.
10. Lytle BW, Loop FD, Cosgrove DM, Ratliff NB, Easley K, Taylor PC. Long term (5 to 12 years) serial studies of internal mammary artery and saphenous vein coronary bypass grafts. J Thorac Cardiovasc Surg 1985; 89:248–58.
11. Grondin CM. Graft disease in patients with coronary bypass grafting: why does it start? Where do we stop? J Thorac Cardiovasc Surg 1986;92: 323–9.
12. Neitzel GF, Barboriak KP, Qureshi I. Atherosclerosis in aortocoronary bypass grafts: morphologic study and risk factor analysis 6 to 12 years after surgery. Arteriosclerosis 1986;6:594–600.
13. Solymoss BC, Nadeau P, Millette D, Campeau L. Late thrombosis of saphenous vein coronary bypass grafts related to risk factors. Circulation 1988;78(suppl I):I-140–3.
14. Lambert M, Kouz S, Campeau L. Preoperative and operative predictive variables of late clinical events following saphenous vein coronary artery bypass graft surgery. Can J Cardiol 1989;5:87–92.
15. Grondin CM, Campeau L, Thornton JC, Engle JC, Cross FS, Schreiber H. Coronary artery bypass grafting with saphenous vein. Circulation 1989;79(suppl I):I-24–9.
16. Walts AE, Fishbein MC, Sustaita H, Matloff JM. Ruptured atheromatous plaques in saphenous vein coronary artery bypass grafts: a mechanism of acute, thrombotic, late graft occlusion. Circulation 1982;65:197–201.
17. FitzGibbon GM, Keon WJ. Atheroembolic perioperative infarction during repeat coronary bypass surgery: angiographic documentation in a survivor. Ann Thorac Surg 1986;43:218–9.
18. Kennedy JH, Wieting DW, Hwang NHC, et al. Hydraulic and morphologic study of fibrous intimal hyperplasia in autogenous saphenous vein bypass grafts. J Thorac Cardiovasc Surg 1974;67:805–13.
19. Barboriak JJ, Batayias GE, Pintar K, Korns ME. Pathological changes in surgically removed aortocoronary vein grafts. Ann Thorac Surg 1976;21: 524–7.
20. Barboriak JJ, Pintar K, Korns ME. Atherosclerosis in aortocoronary vein grafts. Lancet 1974;2:621–4.
21. Batayias GE, Barboriak JJ, Korns ME, Pintar K. The spectrum of pathologic changes in aortocoronary saphenous vein grafts. Circulation 1977;56(suppl II):II-18–22.
22. Barboriak JJ, Batayias GE, Pintar K, Tieu TM, Van Horn DL, Korns ME. Late lesions in aorta-coronary artery vein grafts. J Thorac Cardiovasc Surg 1977;73:596–601.
23. Campeau L, Lespérance J, Corbara F, Hermann J, Grondin C, Bourassa MG. Patency of aortocoronary saphenous vein bypass grafts 5 to 7 years after surgery. Can J Surg 1978;21:118–20.
24. Kouchoukos NT, Karp RB, Oberman A, Russell RO, Alison HW, Holt JH Jr. Long-term patency of saphenous veins for coronary bypass grafting. Circulation 1978;58(suppl I):I-96–9.
25. Campeau L, Lespérance J, Corbara F, Hermann J, Grondin CM, Bourassa MG. Aortocoronary saphenous vein bypass graft changes 5 to 7 years after surgery. Circulation 1978;58(suppl I):I-170–5.
26. Grondin CM, Campeau L, Lespérance J, et al. Atherosclerotic changes in coronary vein grafts six years after operation. J Thorac Cardiovasc Surg 1979;77:24–31.
27. Loop FD, Cosgrove DM, Kramer JR, et al. Late clinical and arterio-

graphic results in 500 coronary artery re-operations. J Thorac Cardiovasc Surg 1981;81:675–85.

28. Bourassa MG, Campeau L, Lespérance J, Grondin CM. Changes in grafts and coronary arteries after saphenous vein aortocoronary bypass surgery: results at repeat angiography. Circulation 1982;65(suppl II):II-90–7.
29. Palac RT, Meadows WR, Hwang MH, Loeb HS, Pifarré R, Gunnar RM. Risk factors related to progressive narrowing in aortocoronary vein grafts studied 1 and 5 years after surgery. Circulation 1982;66(suppl I):I-40–4.
30. Singh RN, Sosa JA, Green GE. Long term fate of the internal mammary artery and saphenous vein grafts. J Thorac Cardiovasc Surg 1983;86:359–63.
31. Campeau L, Enjalbert M, Lespérance J, et al. The relation of risk factors to the development of atherosclerosis in saphenous-vein bypass grafts and the progression of disease in the native circulation. N Engl J Med 1984;311:1329–32.
32. Duguid JB. Pathogenesis of atherosclerosis. Lancet 1949;257:925–7.
33. Chesebro JH, Clements IP, Fuster V, et al. A platelet inhibitor drug trial in coronary bypass operations: benefit of perioperative dipyridamole and aspirin therapy on early postoperative vein-graft patency. N Engl J Med 1982;307:73–8.
34. Chesebro JH, Clements IP, Fuster V, et al. Effect of dipyridamole and aspirin on late vein-graft patency after coronary bypass operations. N Engl J Med 1984;310:209–14.
35. Loop FD, Lytle BW, Cosgrove DM, et al. Influence of the internal mammary artery graft on 10 year survival and other cardiac events. N Engl J Med 1986;314:1–6.
36. Pym J, Brown PM, Charrette EJP, Parker JO, West RO. Gastroepiploic-coronary anastomosis: a viable alternative bypass graft. J Thorac Cardiovasc Surg 1987;94:256–9.
37. Lytle BW, Cosgrove DM, Ratliff NB, Loop FD. Coronary artery bypass grafting with the right gastroepiploic artery. J Thorac Cardiovasc Surg 1989;97:826–31.
38. Blankenhorn DH, Alaupovic P, Wickham E, Chin HP, Azen SP. Prediction of angiographic change in native human coronary arteries and aortocoronary bypass grafts: lipid and nonlipid factors. Circulation 1990; 81:470–6.
39. FitzGibbon GM, Leach AJ, Kafka HP. Atherosclerosis of coronary artery bypass grafts and smoking. Can Med Assoc J 1987;136:45–7.
40. Blankenhorn DH, Nesim SA, Johnson RD, Sanmarco ME, Azen SP, Cashin-Hemphill L. Beneficial effects of combined colestipol-niacin therapy on coronary atherosclerosis and coronary venous bypass grafts. JAMA 1987;257:3233–40.
41. Waters D, Lespérance J, Théroux P, Francetich M, Reitman M, Havel RJ. Prevention of progression of minimal coronary lesions with a calcium channel blocker (abstr). J Am Coll Cardiol 1990;15:116A.