

REVIEW ARTICLE

Balloon Angioplasty for the Treatment of Lesions in Saphenous Vein Bypass Grafts

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Objectives. The purpose of this review is to assess the value and limitations of balloon angioplasty for the treatment of saphenous vein bypass graft obstructions. The potential efficacy of new interventional techniques is discussed.

Background. Treatment of ischemia due to saphenous vein bypass graft obstructions poses a difficult problem that will be encountered more often as the pool of surgically treated patients continues to accumulate. Reoperation is technically demanding and is associated with high mortality and morbidity rates. Balloon angioplasty may provide a suitable alternative.

Methods. The review proposes a classification of patients with attempted saphenous vein graft angioplasty according to expected early and late outcome based on the data obtained from the relevant published data and personal experience.

Results. Angioplasty of a nonocclusive obstruction in a saphenous vein bypass graft has an initial success rate of approximately 90% and is a safe procedure (procedural death rate <1%, myocardial infarction rate <4%). The overall average restenosis rate is 42%. Surgical standby is limited and technically difficult. Angioplasty of chronic total occlusions in old grafts is associated

with poor initial and long-term results. The long-term clinical results are unfavorable because of the continuing progression of disease in nontreated vein graft segments and native coronary arteries, in addition to the high restenosis rate. New techniques, although promising, have shown neither better initial results nor reduction of restenosis. Stent placement may be useful in longer graft lesions containing friable material.

Conclusions. Patients may be classified into three groups according to expected early and late outcome on the basis of 1) unfavorable graft anatomy, 2) risk of cardiogenic shock in event of acute graft closure, and 3) age of grafts. The three groups are 1) those with an initial high success, low procedural risk and low restenosis rate; 2) those with an initial high success but high procedural risk and moderate to high restenosis rate; and 3) those with a low success, high risk and high restenosis rate. Balloon angioplasty to treat lesions in venous bypass grafts should be considered a palliative procedure, not a long-term solution, for ongoing progression of coronary artery and vein graft disease. The induced high restenosis rate remains a significant problem.

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The management of recurrent ischemia in patients who have had previous saphenous vein bypass graft surgery poses a serious, difficult problem. Recurrent ischemia occurs not only because of attrition of the saphenous vein grafts but also because of progression of coronary artery disease in the native coronary arteries. The attrition rate is 15% to 20% during the 1st year after operation; between 1 and 6 years after operation, it is 1% to 2%/year and between 6 to 10 years after surgery the rate is 4%/year (1-8). By 5 years, about 45% of the grafts are occluded (3,7). Progression of native coronary artery disease occurs in approximately 5% of the

patients/year after operation (8-11). Symptoms recur or progress in about 5% of patients/year, and it has been estimated that 10% to 15% of the patients will require a repeat operation within 10 years after the initial procedure (11-15). However, currently improved surgical techniques in combination with administration of aspirin and risk factor modification have improved the early graft attrition rate and may lessen long-term attrition (16). Despite improved surgical results, it may be expected that the number of patients with recurrence of ischemia will increase because the pool of surgically treated patients continues to accumulate.

Reoperation is technically more difficult to perform and is associated with a rather high mortality rate (3% to 6.5%) and a high perioperative myocardial infarction rate (3.4% to 11.5%), and the likelihood of complete relief of symptoms is less than with a first operation (11-16). These factors have stimulated the search for an alternative treatment.

Gruentzig et al. (17) reported as early as 1979 that vein graft angioplasty was successful in five of seven attempts. However, three of five grafts demonstrated restenosis during follow-up, and Gruentzig suggested that the "different kind

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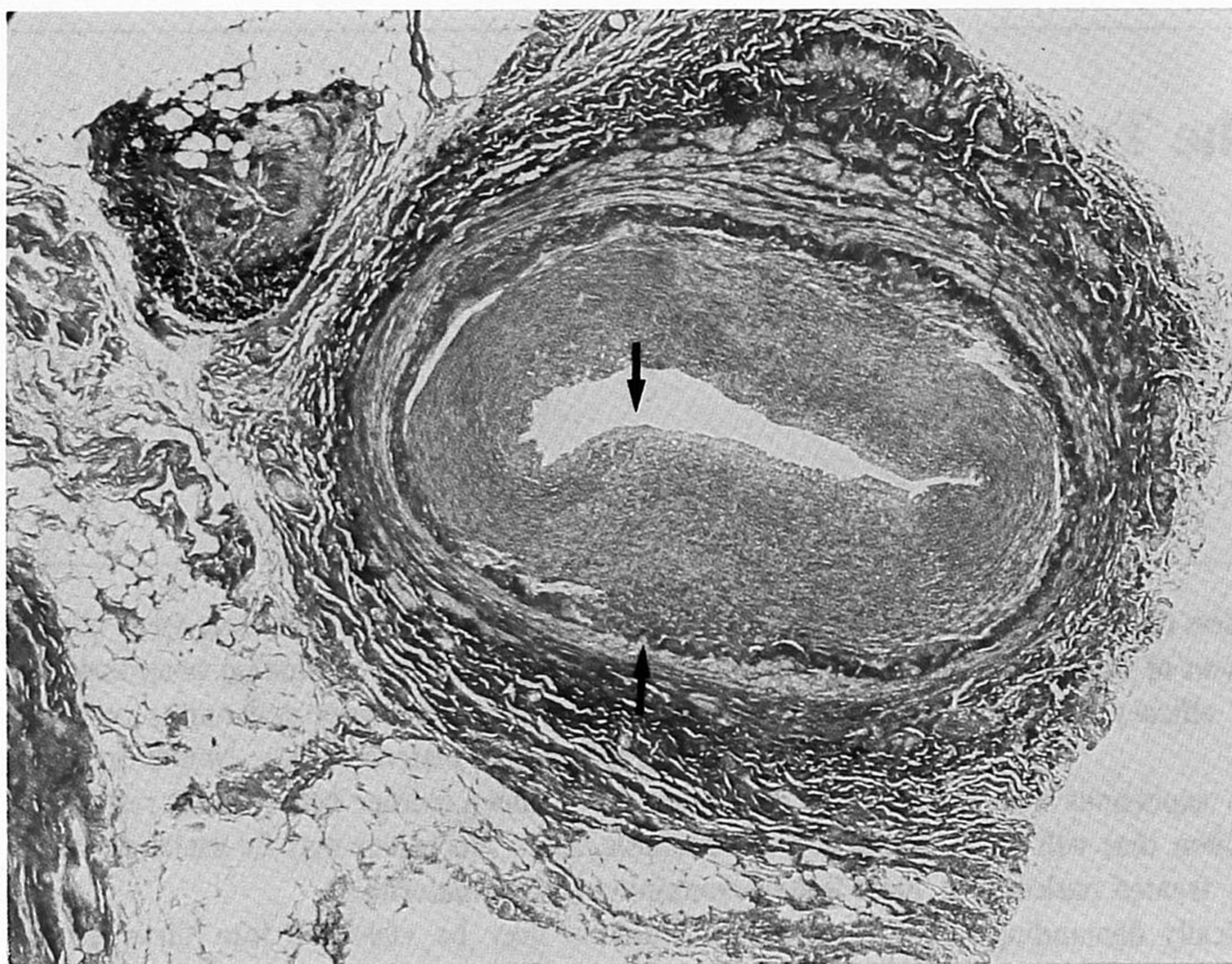


Figure 1. Histologic cross section of a 9-month old saphenous vein bypass graft showing severe lumen narrowing due to concentric intimal thickening (arrows) consisting of fibrocollagenous tissue. Elastic-van Gieson stain $\times 25$, reduced by 29%.

of disease'' in the bypass graft may have explained the high recurrence rate in graft stenosis. Since then, many studies concerning angioplasty of lesions in saphenous vein grafts have been reported.

In this article we review the reported data and discuss the indications and the initial and late results of angioplasty of saphenous vein bypass grafts. We also briefly discuss the role of new techniques including stents, directional and extraction atherectomy and laser angioplasty.

Pathoanatomy of saphenous venous grafts. The pathophysiologic mechanisms underlying graft failure can arbitrarily be distinguished into those occurring early, within 1 year and late after operation. In each period one assumes a specific predominant pathogenetic mechanism, although one must bear in mind that the different pathologic processes may occur in a continuous fashion and overlap in time (18,19).

Graft occlusion early after operation is usually associated

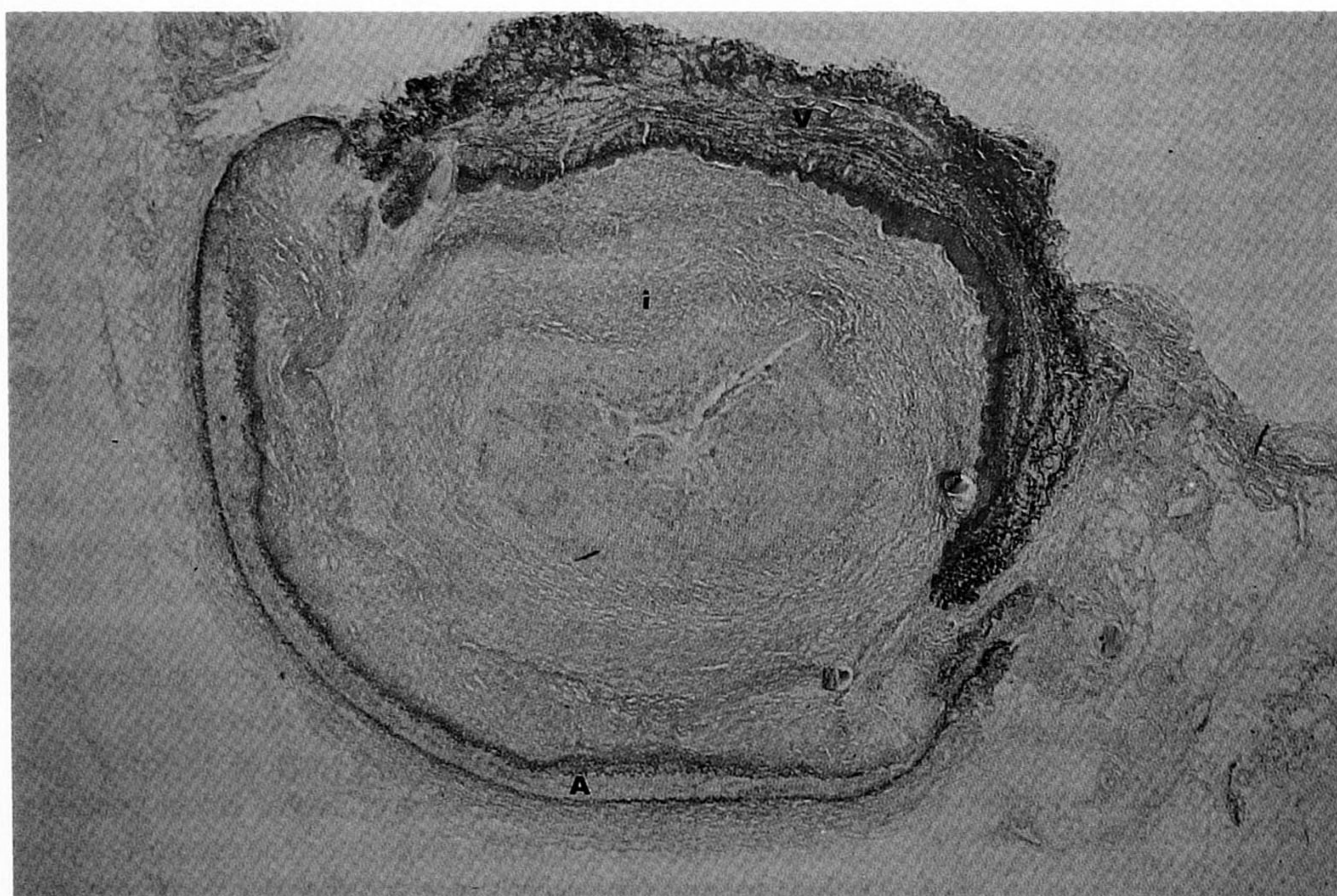
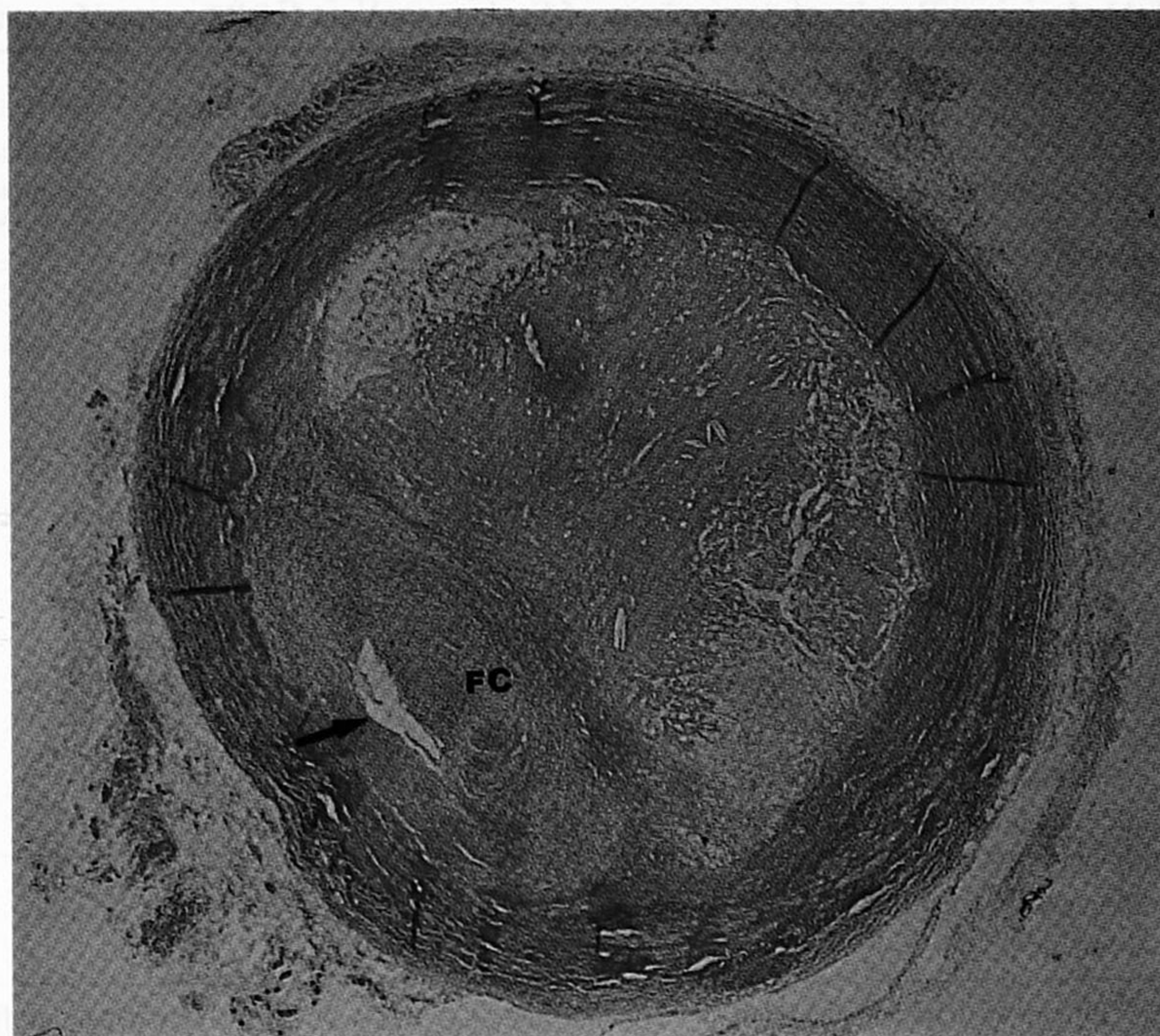


Figure 2. Saphenous vein (V)-coronary artery (A) anastomosis, 2 years after coronary bypass surgery, showing intimal thickening (I) and occlusive thrombus (yellow). Elastic-van Gieson stain $\times 25$, reduced by 29%.

Figure 3. Six-year old saphenous vein bypass graft showing severe lumen narrowing by a classical atherosclerotic plaque; fatty debris (yellow) is covered by a fibrous cap (FC) (lumen, arrow). Elastic-van Gieson stain $\times 25$, reduced by 29%.



with acute thrombosis (18,19), possibly attributable to harvesting and handling of the vein or to failure of surgical techniques at sites of anastomosis (20).

Fibrointimal hyperplasia is the dominant feature 1 to 12 months after operation (20-22). The cells of fibrointimal hyperplasia resemble smooth muscle cells and some cells may have a foamy cytoplasmic appearance (21). Focally stenotic lesions produced by this process appear particularly amenable to dilation.

In the late postoperative period, fibrointimal hyperplasia

at first remains the dominant feature, but gradually atherosclerotic lesions become more frequent (Fig. 1 and 2) (19). The fibrointimal lesion gradually diminishes its cellularity, and the smooth muscle cells are replaced by fibrous tissue and the matrix is increased. With time, there appears to be an increase in the number of foam cells within the intima. The development of atherosclerosis in the aortocoronary vein grafts is an important factor in late graft stenosis and occlusion (23-32). The atherosclerotic process proceeds to a fully developed complex atherosclerotic plaque (Fig. 3), and

Figure 4. Four-year old saphenous vein bypass graft showing a concentric fibrous intimal thickening and an eccentric atherosclerotic plaque with fatty debris separated from the lumen (L) by a very thin fibrous cap (arrows). Elastic-van Gieson stain $\times 25$, reduced by 29%.

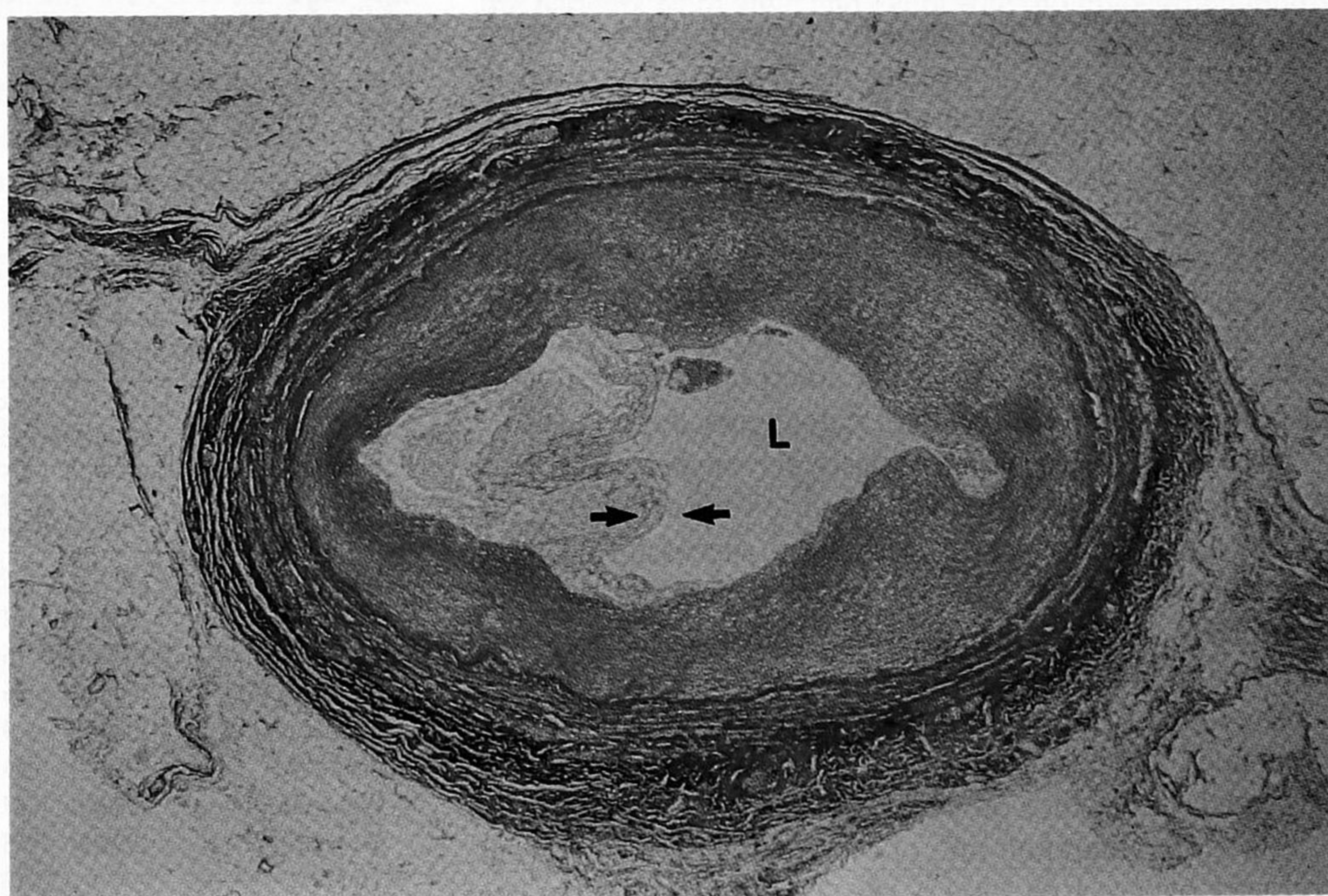


Table 1. Initial Results of Angioplasty of Saphenous Vein Grafts

| Reference | | Pts (no.) | Clinical Success | | Procedural Complications | | | |
|--------------------|------|--------------|------------------|----|--------------------------|-----------|---------------------|--------------------|
| First Author (no.) | Year | | No. | % | Death (%) | MI (%) | Embolization (%) | Urgent CABG (%) |
| Douglas (34) | 1983 | 62 | 58 | 94 | 0 | 2 | 0 | 2 |
| El Gamal (35) | 1984 | 44 | 41 | 93 | 0 | 5 | 0 | 0 |
| Block (36) | 1984 | 40 | 31 | 78 | 0 | 0 | 0 | 2.5 |
| Corbelli (37) | 1985 | 35 | 31 | 89 | 0 | 0 | 0 | 3.0 |
| Reeder (38) | 1986 | 19 | 16 | 84 | 5 | 5 | 0 | 0 |
| Douglas (39) | 1986 | 235 | 216 | 92 | 0 | 7* | 3 | 1.3 |
| Cote (40) | 1987 | 82 | 70 | 85 | 0 | 1 | 2 | 1 |
| Ernst (41) | 1987 | 33 | 32 | 97 | 0 | 3 | NR | 0 |
| Dorros (42) | 1988 | 53 | 44 | 83 | 2 | 2 | 6 | 2 |
| Reed (43) | 1989 | 54 | 47 | 90 | 0 | 0 | 0 | 2 |
| Cooper (44) | 1989 | 24 | 18 | 75 | 4 | 0 | NR | 0 |
| Platko (45) | 1989 | 101 | 90 | 90 | 2 | 6 | 3 | 2 |
| Webb (46) | 1990 | 140 | 119 | 85 | 0 | 4 | NR | 1 |
| Meester (47) | 1991 | 84 | 69 | 82 | 1 | 8 | NR | 2.5 |
| Plokker (48) | 1991 | 454 | 408 | 90 | 0.7 | 2.8 | NR | 1.3 |
| Reeves (49) | 1991 | 57 | 47 | 83 | 2 | 9 | 7 | 2 |
| Total | | 1,571 | 1,337 | 88 | <1 | <4 | <3 | <2 |

*Fifteen of 16 patients had non-Q wave infarction. CABG = coronary artery bypass grafting; MI = myocardial infarction; NR = not reported; Pts = Patients.

rupture of the plaque leads to a superimposed thrombotic occlusion (23,24). The plaques are often large, fragile and ulcerated and the graft may show aneurysmal dilation (25,26).

There is still some controversy as to whether venous graft atherosclerosis differs from coronary atherosclerosis. Some investigators (28,29) suggest that vein graft lesions contain more foam cells, and that they exhibit an inflammatory reaction with foreign body giant cells. This process undermines the thickened intima, so that the fibrous cap is weakened (Fig. 4) (28). One study (29) demonstrated lack of a fibrous cap. This thinning and weakening of the fibrous cap may explain the greater propensity of venous plaque rupture and thrombosis. The propensity of thrombus formation in vein grafts is enlarged because the lack of side branches, the large diameter of vessels and consequently low flow velocities may contribute to platelet aggregation and thrombus formation. These factors may explain the frequent occurrence of thrombotic complications or embolization of material during balloon angioplasty. However, other investigators (18,19,26,30-32) believe that atherosclerosis vein graft disease is not different from arterial atherosclerosis.

Immediate results of angioplasty for saphenous venous bypass grafts. Ford et al. (33) reported a small series of seven patients of whom six underwent successful dilation. Since then, many centers have reported their initial results of angioplasty of saphenous vein grafts. Only the updated latest reports for each center are presented in Table 1.

The initial success rate varies from 75% to 94%, with a combined overall success rate of 88%. The major complication rate is low, with a procedure-related death rate of <1%, a myocardial infarction rate of ≈4% and a need for urgent coronary bypass graft surgery of <2%. The remarkably low

tendency to abrupt occlusion and the relatively high success rate are probably due to the absence of side branches and tortuosity. The risk of embolization of friable, thrombotic material into the native circulation is <3%. Embolism usually occurs during attempted angioplasty in older grafts, with long diseased segments containing friable, thrombotic lesions. The initial success rate depends on the site of dilation (Table 2). The overall combined initial results of dilation of the proximal site is 87%, of the graft body 94% and of the distal site 90%. These rates appear to be similar except for a slightly lower success rate for dilation at the proximal site. The high success rate and low complication rate reflect the careful selection of patients. Difficult lesions with potential high risk, such as long diffuse lesions or ulcerated, thrombotic, friable lesions, were probably excluded from these series.

Table 2. Initial Success Rate of Dilation of Saphenous Vein Grafts at Different Sites

| Reference | | Site of Graft Dilation (no. of lesions) | | | | | |
|--------------------|------|---|----|------|-----|--------|----|
| First Author (no.) | Year | Proximal | | Body | | Distal | |
| | | No. | % | No. | % | No. | % |
| Douglas (34) | 1983 | 5 | 80 | 23 | 96 | 34 | 94 |
| Dorros (50) | 1984 | 12 | 84 | 8 | 88 | 13 | 69 |
| Corbelli (37) | 1985 | 26 | 88 | 7 | 100 | 14 | 93 |
| Pinkerton (51) | 1988 | 36 | 94 | 29 | 90 | 35 | 94 |
| Cooper (44) | 1989 | 9 | 56 | 3 | 67 | 12 | 92 |
| Platko (45) | 1989 | 53 | 89 | 24 | 100 | 30 | 93 |
| Webb (46) | 1990 | 47 | 80 | 39 | 86 | 56 | 89 |
| Meester (47) | 1991 | 28 | 86 | 33 | 97 | 32 | 81 |
| Total | | 216 | 86 | 162 | 93 | 226 | 90 |

Table 3. Restenosis After Successful Angioplasty of Saphenous Venous Grafts

| Reference | | Pts (no.) | Definition of Restenosis (% lumen narrowing) | Completeness of Angiographic Follow-Up (%) | Angiographic Restenosis by Graft Site (no. [%] of lesions) | | | |
|--------------------|------|--------------|---|--|---|----------------|----------------|-----------------|
| First Author (no.) | Year | | | | Proximal | Body | Distal | All Sites |
| Douglas (34) | 1983 | 41 | NR | NR | 1/2 | 9/17 | 4/22 | 14/41 [34] |
| El Gamal (35) | 1984 | 16 | >50 | 61 | 1/2 | 5/12 | 2/3 | 7/17 [41] |
| Block (36) | 1984 | 22 | >50 | 71 | NR | NR | NR | 12/22 [55] |
| Dorros (50) | 1984 | 26 | NR | NR | 8/10 | 2/7 | 2/9 | 12/26 [46] |
| Reeder (38) | 1986 | 16 | >30 | 100 | 2/3 | 3/5 | 3/8 | 8/16 [50] |
| Douglas (39) | 1986 | 130 | NR | NR | 11/14 | 40/65 | 10/51 | 61/130 [47] |
| Cote (40) | 1987 | 26 | >50 | 70 | 3/9 | 5/21 | 2/13 | 10/43 [23] |
| Dorros (42) | 1988 | 25 | NR | 57 | 3/5 | 3/7 | 4/13 | 10/25 [40] |
| Pinkerton (51) | 1988 | 23 | NR | 92 | 1/3 | 3/5 | 6/15 | 10/23 [44] |
| Platko (45) | 1989 | 49 | >50 | 56 | 21/42 | 9/20 | 10/24 | 40/86 [47] |
| Meester (47) | 1991 | 59 | >50 | NR | NR | NR | NR | 13/59 [22] |
| Reeves (49) | 1991 | 45 | >50 | 93 | 7/11 | 20/32 | 5/14 | 32/57 [56] |
| Total | | 478 | | | 58/101 [58] | 99/191 [52] | 48/172 [28] | 229/545 [42] |

NR = not reported; Pts = Patients.

Restenosis after successful dilation of saphenous vein grafts. The restenosis rate (defined as >50% lumen diameter narrowing in the majority of the reported studies) after initially successful angioplasty depends highly on the site of dilation within the graft (Table 3). Ostial or very proximal graft lesions tend to have a very high restenosis rate (58% on average). The restenosis rate of the body of the graft is 52% and the restenosis rate in the distal, anastomotic part of the graft is 28%. The overall combined restenosis rate is 42%. These data probably overestimate the true incidence of restenosis because not all asymptomatic patients are restudied. The angiographic completeness of follow-up varied from 56% to 100% in the reported studies (Table 3).

In native coronary arteries, the restenosis process takes place in the majority of the patients within 6 months after angioplasty. It is not known whether the restenosis process in venous bypass grafts is similar, but data reported by Douglas et al. (52) suggest that the interval is longer. In follow-up studies of 599 patients with successful angioplasty, restenosis was found in 32% of dilated lesions within 6 months of operation in 43% by 6 months to 1 year, in 61% by 1 to 5 years and in 64% after 5 years.

Long-term follow-up after angioplasty of saphenous vein bypass grafts. The long-term outcome of patients selected for angioplasty of a saphenous vein bypass graft is influenced not only by the restenosis rate and rate of progression of disease in the native arteries but also by the extent of the left ventricular dysfunction. The frequency of late death, myocardial infarction and recurrence of angina is listed in Table 4. The wide range of rates reflects the differences in patient selection and duration of follow-up. It appears that the clinical event rate is significantly higher if the graft is older. The clinical event rate was 64% in grafts >36 months old and 33% in grafts <36 months old (45).

Plokker et al. (48) reported that, after a follow-up period of 5 years, 74% of 454 patients were alive and only 26% of the patients were alive and event free (no myocardial infarction, no repeat bypass surgery or repeat angioplasty) (Fig. 5). The interval between angioplasty and bypass surgery was a significant predictor for 5-year event-free survival. The event-free survival rates for patients who had bypass surgery 1 year before, between 1 and 5 years and 5 years after bypass surgery were 45%, 25% and 19% respectively. The late mortality and late myocardial infarction rates are expected

Table 4. Long-Term Results After Immediate Successful Graft Lesion Angioplasty

| Reference | | | Mortality (%) | Myocardial Infarction (%) | Recurrence of Angina (%) | Follow-Up Months (mean \pm SD) |
|--------------------|------|-----------|------------------|---------------------------------|--------------------------------|-------------------------------------|
| First Author (no.) | Year | Pts (no.) | | | | |
| Cote (40) | 1987 | 82 | 2.5 | 0 | 29 | 21 \pm 2 |
| Reed (43) | 1989 | 50 | 2 | 2 | 52 | 23 \pm 11 |
| Platko (45) | 1989 | 87 | 11.5 | 20 | 47 | 17 \pm 14 |
| Webb (46) | 1990 | 119 | 7.6 | 11 | 19* | 33 \pm 26 |
| Reeves (49) | 1991 | 50 | 4 | 4 | 54 | 32 |
| Meester (47) | 1991 | 69 | 11.5 | 10 | 42* | 25 |
| Plokker (48) | 1991 | 454 | 22 | NR | NR | 60 |

*These patients underwent a second operation or a second angioplasty procedure. Abbreviations as in Table 3.

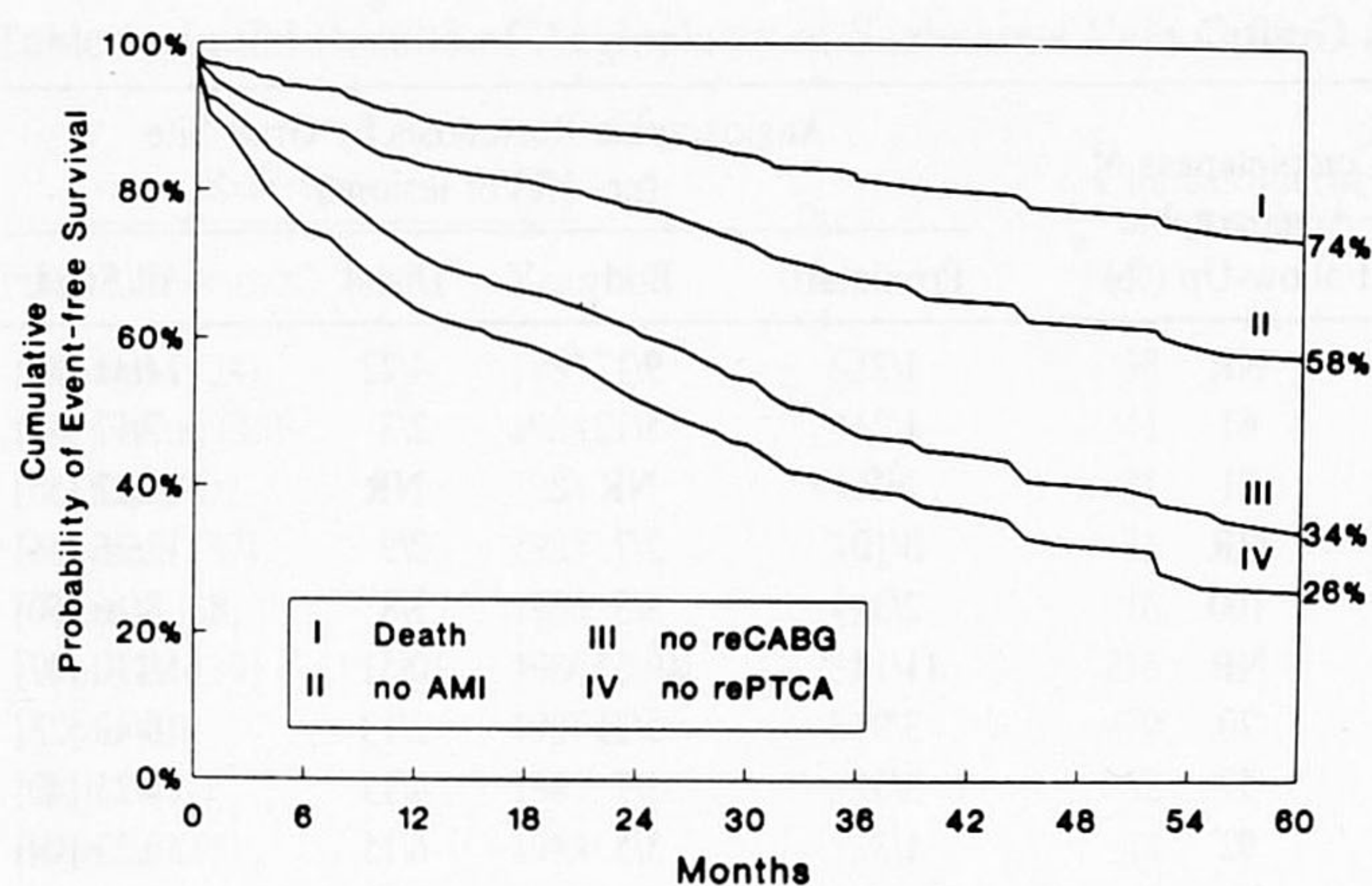


Figure 5. The cumulative probability of survival of 454 patients followed up for 5 years. I = survival; II = survival without acute myocardial infarction (AMI); III = survival without acute myocardial infarction and without repeat coronary artery bypass grafting (reCABG), and IV = survival without any cardiac event (death, acute myocardial infarction, coronary artery bypass grafting or repeat coronary balloon angioplasty (rePTCA). Reproduced, with permission, from Plokker et al. (48).

to be high because often these patients present with end-stage coronary artery disease. In these patients, angioplasty should be considered a palliative treatment and one should not expect a beneficial effect on late mortality.

Balloon angioplasty of early (within 1 year) occlusion of saphenous vein bypass grafts. Graft occlusion occurring within 1 month after operation is almost always associated with graft thrombosis (18–20,24,27). Technical factors, such as stenosis at surgical anastomotic sites or intraoperative vein trauma or poor distal runoff due to severely diseased native arteries, play a role and limit the possibilities of immediate and sustained beneficial effect of angioplasty.

Graft occlusion occurring between 1 month and 1 year after operation is characterized by lesions consisting of fibrointimal hyperplasia with superimposed occlusive thrombosis (18–21,24,27,28,30). These occlusions are predominantly focal, not associated with diffuse vein graft disease, and usually the thrombotic component of the occlusion is not

extensive. They appear to be amenable to successful perforation and dilation.

Balloon angioplasty of chronic totally occluded saphenous vein bypass grafts >1 year after operation. The underlying pathoanatomy plays an important role in the expected success rate of angioplasty of a chronic totally occluded graft. Late occlusions (1 to 3 years postoperatively) are usually associated with focal atherosclerosis with occlusive thrombosis (18–21,24,27,28,30). These lesions may be amenable to successful perforation and dilation without increased risk of thrombotic embolization. Older occlusive obstructions are often composed of large, ulcerated plaques containing friable thrombotic material. The chronic total occlusions are often extended over a long segment of the graft and are often associated with diffuse graft disease. Obviously, perforation and dilation of these chronic occlusions may be unsuccessful or even harmful because of dislodgment of material into the native coronary circulation with ensuing myocardial infarction.

The safety and results of angioplasty in occluded grafts are controversial. de Feyter et al. (53) reported in 13 patients with chronically totally occluded old degenerated grafts that attempts to recanalize the graft expose the patient to a high risk of embolization and, even if it is possible to reopen the graft, it frequently reoccludes. In their study only 1 of 13 patients had a long-term success, and the procedure was complicated by a myocardial infarction in 5 patients (Table 5). Apparently, the presence of large amounts of thrombotic material and its dislodgment are the main causes of low success and high complications with balloon angioplasty. These results contrast highly with a recent report of Kahn et al. (54), who reported a 83% success rate (64 of 82 patients) with a 1.5% in-hospital death rate, 3% myocardial infarction rate and no urgent bypass surgery. However, at 3-year follow-up study, only 33% of the patients had had no repeat angioplasty or bypass surgery. The difference in immediate results between the two reports may be explained by differences in patient selection. Results in short occluded segments of grafts with a reasonably normal angiographic

Table 5. Coronary Angioplasty of Chronic Totally Occluded Vein Grafts

| Reference | | Duration of Occlusion (mo) | Mean Age of Grafts (yr) | All Pts | Initial Clinical Success | Procedural Complications | | Long-Term Success | Late Death | Late MI |
|-------------------------|------|-------------------------------|----------------------------|------------|--------------------------------|-----------------------------|----|----------------------|---------------|------------|
| First Author (no.) | Year | | | | | Death | MI | | | |
| Angioplasty Alone | | | | | | | | | | |
| de Feyter (53) | 1989 | 3 to 6 | 6 ± 4 | 13 | 7 | 0 | 5 | 1 | 0 | 0 |
| Urokinase + Angioplasty | | | | | | | | | | |
| Sievert (55) | 1988 | 0.5 to 4 | 0.2 to 3.6 | 7 | 6 | 0 | 0 | 4 | 0 | 0 |
| Hartmann (56) | 1991 | 0.5 to 6 | 7 (range 1 to 13) | 46* | 36† | 0 | 2 | 22 | 4 | 0 |
| Levine (57) | 1992 | NR | NR | 10 | 6 | 2 | 2 | 0 | 0 | 1 |

*Eight patients had acute non-Q wave infarction. †Twenty patients had a repeat angiogram; seven had an occluded graft. Unless otherwise indicated, data are expressed as number of patients. Abbreviations as in Table 1.

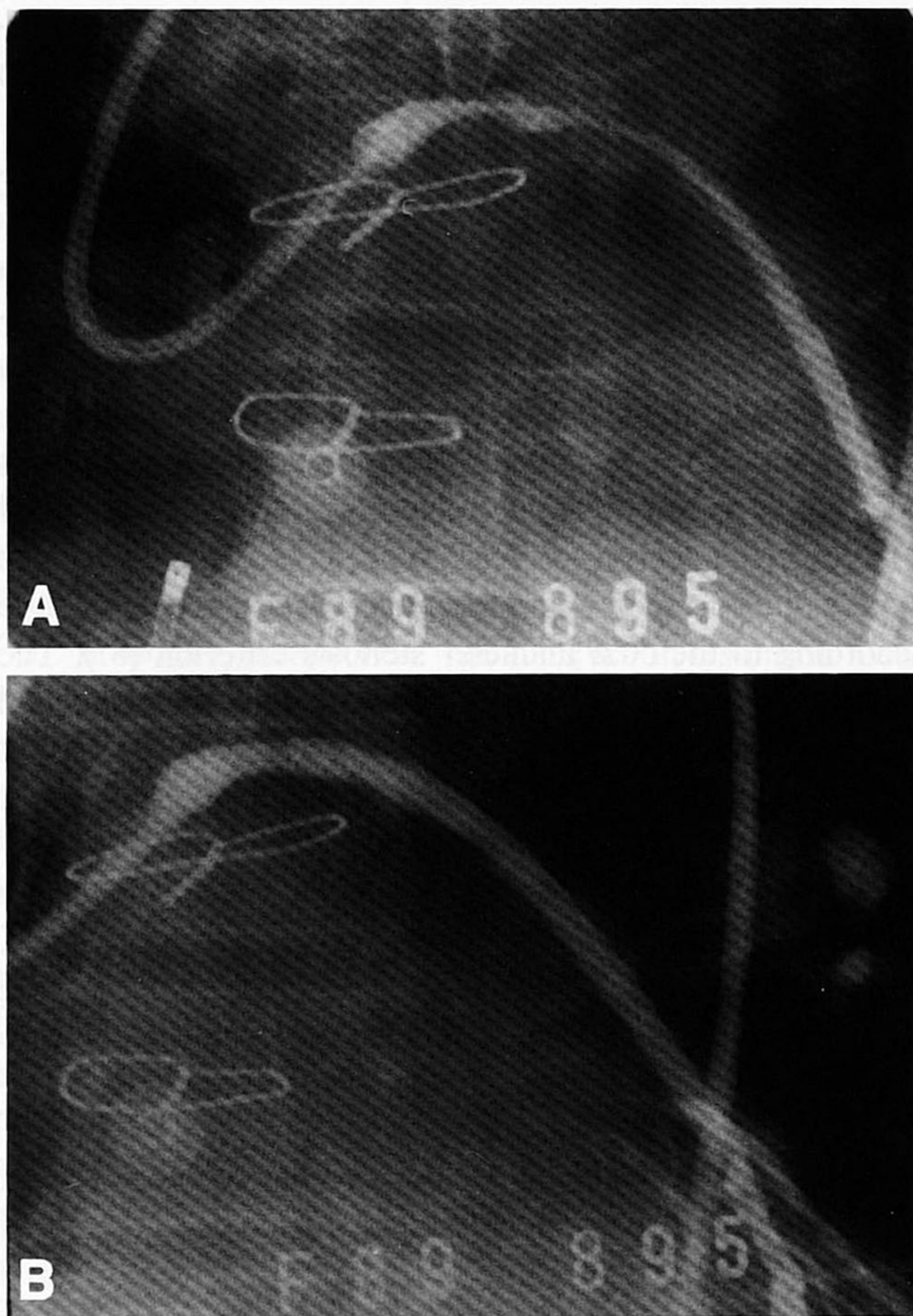


Figure 6. A, Diffuse disease 7 years after bypass graft implantation in the left anterior descending artery. B, Immediate result after implantation of two overlapping stents (Wall stent).

appearance are probably better than those in long occluded segments of degenerated vein grafts.

Pretreatment of chronic total occlusion with urokinase followed by angioplasty. Dissolving thrombus in chronically occluded grafts with a short infusion of urokinase dramatically improved the short- and long-term results of angioplasty (Table 4). Sievert et al. (55) showed that, after

pretreatment with urokinase, six of seven patients had successful recanalization, and four patients had long-term success. Hartmann et al. (56) also showed good results with long-term urokinase infusion (range 7.5 h to 77 h [mean 31]) followed by angioplasty. Recanalization was achieved more easily in patients with a short estimated duration of occlusion. The price for this treatment was the occurrence of a significant hematoma in 22% of the patients and a long stay in a coronary care unit. Unfortunately, the reocclusion/restenosis rate was rather high, and success was sustained in 48% (22 of 46) of the patients during 1 to 24 (mean 11) months of follow-up. Levine et al. (57) demonstrated that patency was achieved in 8 of 10 patients. However, two patients died and two had embolic myocardial infarction, and no patient was free of reocclusion, myocardial infarction or death during a follow-up period of an average of 13 ± 6 months.

Identification of risk factors for unfavorable outcome.
Variables predictive of unfavorable initial results. Factors that predict a poor initial result included 1) diffuseness of saphenous vein graft disease (40); 2) attempted angioplasty of stenoses in grafts >4 to 6 years old (45,46); 3) chronic totally occluded grafts (13); and 4) the presence of intravein graft thrombus (49). The presence of one or more of these variables is associated with a high frequency of major complications (death, myocardial infarction and need for urgent bypass surgery), often due to embolization of friable material into the coronary circulation or the occurrence of abrupt occlusion with thrombosis formation.

Variables predictive of late restenosis. These include 1) lesions in old (>36 months) grafts (restenosis rate 83% vs. 42%) (45); 2) multiple lesions, diffuse graft disease and total occlusion (100% vs. 38%) (49); 3) small diameter (<2.2 mm) of the grafted coronary artery (78% vs. 27%) (58); 4) length of stenosis >10 mm (62% vs. 12%) (58); and 5) dilation of lesion at the proximal site and body of the graft (Table 3).

Limitations of surgical backup. In many centers the availability of immediate surgical backup is considered a prerequisite for performing angioplasty. Although emergency operation for acute ischemia may not totally eliminate the development of myocardial infarction, there is evidence that

Table 6. Stent Implantation in Venous Bypass Grafts

| Reference | | Pts (no.) | Implantation Success | Clinical Success | In-Hospital Complications | | | | Restenosis |
|---------------------|------|--------------|-------------------------|---------------------|---------------------------|-------|-----------------------|---------------------|------------|
| First Author (no.) | Year | | | | Death | MI | Embolization/ CABG | Serious Bleeding | |
| Wall stent | | | | | | | | | |
| Urban (62) | 1989 | 13 | 95 | 95 | 0 | 0 | 0 | 15 | 36 |
| de Scheerder (64) | 1992 | 69 | 100 | 87 | 4* | 7 | 6 | 33 | 47 |
| PalmaZ-Schatz stent | | | | | | | | | |
| Pomerantz (65) | 1991 | 54 | 100 | 100 | 0 | 0 | 0 | NR | 21 |
| Leon (66) | 1991 | 192 | 98 | 97 | 1.6 | 1.0 | 1.6 | NR | 26 |
| Flexible coil stent | | | | | | | | | |
| Bilodeau (61) | 1992 | 37 | 100 | 86.5 | 0 | 13.5† | 0 | 21.6 | 35 |

*Some patients who died are also listed in other groups. †All acute myocardial infarctions were seen in patients treated with stenting for dissection or threatened closure. Unless otherwise indicated, all data are expressed as percent of patients. Abbreviations as in Table 1.

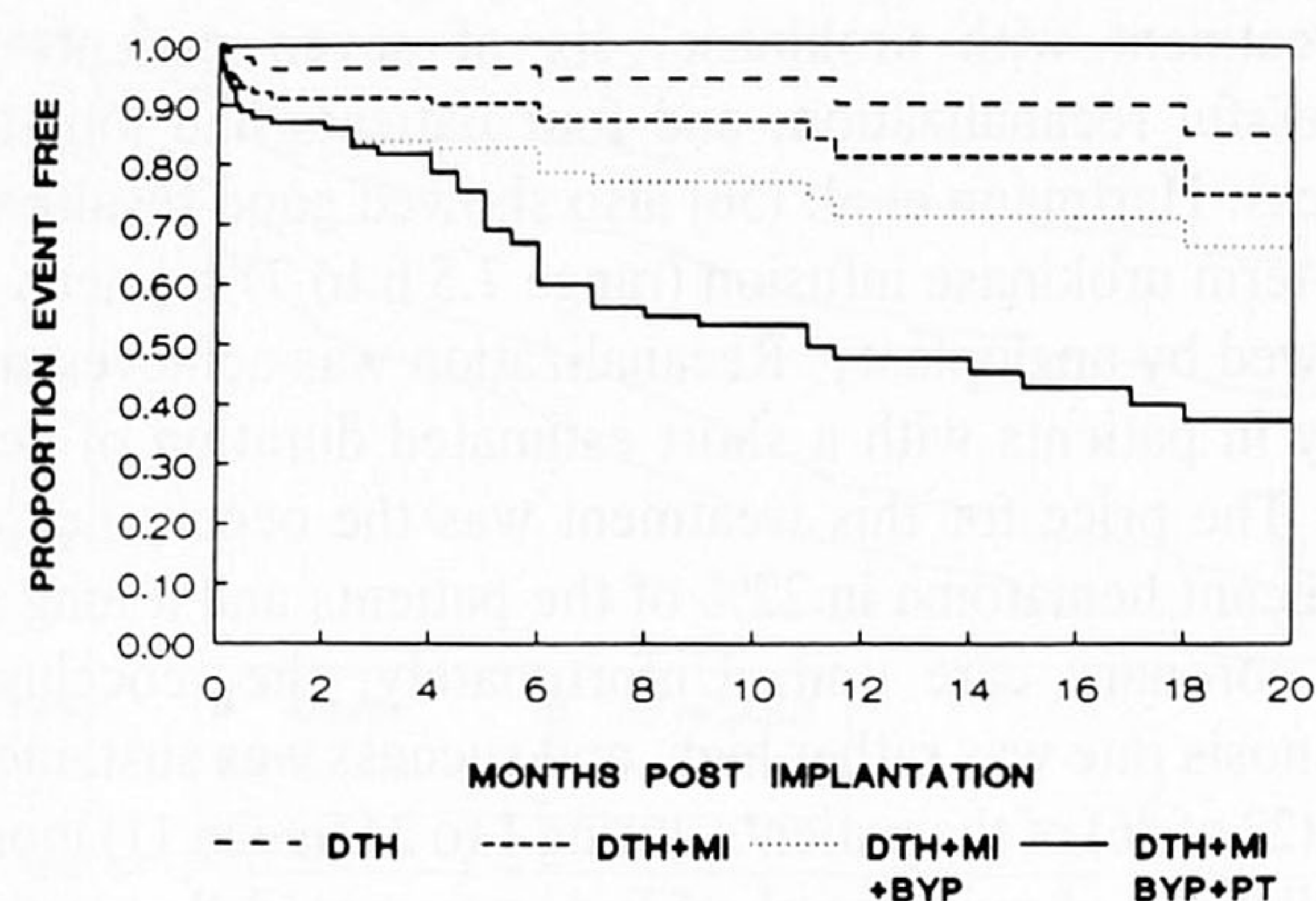


Figure 7. Actuarial event-free survival of 145 patients followed up for up to 20 months after stent implantation in a bypass graft. The curves (from upper to lower) represent freedom from death (DTH) alone; death plus myocardial infarction (MI); death, myocardial infarction plus coronary bypass surgery (BYP); and death, myocardial infarction, coronary bypass surgery plus angioplasty or atherectomy (PT). Reproduced, with permission, from Strauss et al. (67).

prompt revascularization does limit the damage (59). The time between the onset of ischemia and revascularization determines the outcome (60). The ischemic period during reoperation is considerably prolonged because immediate access to the heart is hampered by the fibrosis of the previous operation, which may require considerable time to dissect free the heart. This inevitable delay limits the potential of revascularization to reduce myocardial damage. If possible, bailout techniques, such as use of autoperfusion catheters, should always be used in these situations. Stent implantation for threatened closure has produced satisfactory results (61).

This expected time delay should be considered when counseling a patient. It would be prudent to refer a patient for reoperation if, for instance, abrupt closure of a lesion would lead to acute heart failure due to the large area of myocardium at risk.

New interventional techniques for treatment of saphenous vein bypass grafts. Stent implantation. The intracoronary stent was the first of the new interventional techniques to be

applied in bypass graft angioplasty. Shortly after the introduction into the native coronary artery system, stents were also implanted in bypass grafts (Fig. 6) (62-64). The immediate angiographic and implantation success rate is extremely high ($\geq 95\%$, Table 6) (61,62,64-66). However, early experience was associated with an unacceptable high incidence of subacute thrombosis and serious bleeding complications. Increased operator experience and meticulous anticoagulation resulted in a substantial decrease in complications (64-66). Unfortunately, the incidence of restenosis does not seem to have been reduced (Table 6). One detailed study on restenosis after Wall stent implantation in venous bypass grafts reports an incidence of restenosis of 39% according to the 50% diameter stenosis criterion (67). The restenosis rate was 35% after implantation of a flexible coil (61) and $<28\%$ after implantation of a Palmaz-Schatz stent (68). Restenosis occurred more frequently after Wallstent implantation in bypass grafts than in stented native coronary arteries (69). The rates were similar in grafts and native arteries after Palmaz-Schatz stent implantation (68). Whether the better results obtained with the Palmaz-Schatz were due to the more favorable features of this stent or to improved periprocedural pharmacologic management or different patient selection is unknown.

The long-term clinical follow-up results of Wallstent implantation in bypass grafts collected from 145 patients of six European centers is shown in Figure 7 (67). The actuarial event-free survival (freedom from death, myocardial infarction, bypass surgery or angioplasty) for bypass graft patients was 37% at 20 months with an overall mortality rate of 9%. About 30% of the adverse events were unrelated to the stented lesion and were due to worsening of different lesions or to development of new lesions (64,67).

Atherectomy: directional and extractional. The use of directional atherectomy in saphenous venous bypass grafts was feasible and successful in $>90\%$ of patients (Table 7) (65,70,71). The complication rate was acceptable, but one can imagine that the use of such a bulky device in case of

Table 7. Initial Results of New Devices for Treatment of Lesions of Saphenous Vein Bypass Grafts

| Reference | | Pts (no.) | Clinical Success | Complications | | | | |
|--------------------------|------|--------------|---------------------|---------------|-----|----------------|--------------|------------|
| First Author (no.) | Year | | | Death | MI | Emergency CABG | Embolization | Restenosis |
| Directional Atherectomy | | | | | | | | |
| Kaufmann (70) | 1990 | 14 | 93 | 7* | 1 | 0 | 7 | 63 |
| Pomerantz (65) | 1991 | 29 | 93 | 0 | 0 | 0 | 7 | 31 |
| Selmon (71) | 1991 | 76 | 91 | 0 | 9.2 | 1.3 | 11.5 | 60 |
| Extractional Atherectomy | | | | | | | | |
| Meany (72) | 1992 | 278 | 89 | 0.3 | 0.3 | 0.7 | 3.5 | 53 |
| Excimer Laser | | | | | | | | |
| Untereker (73) | 1991 | 225 | 97 | 0.4 | 4.4 | 0.8 | 4.4 | 61 |

*One patient crossed over to coronary angioplasty. The procedure was complicated by abrupt closure, necessitating emergency coronary artery bypass grafting; eventually the patient died.

friable, thrombotic lesions easily embolizes material. Preliminary data suggest that the restenosis rate is also high.

Extractional atherectomy of vein graft lesions was successful in 89% of patients (Table 7) (72). The complication rate was low and the restenosis rate was 53%. It is conceivable that the use of this device in vein grafts containing much material is safe and is associated with a higher success rate and decreased risk of embolization due to the suction, extraction and removal of material with this device, although embolization occurred in 3.5% of patients.

Excimer laser angioplasty. In an initial experience including 225 patients (Table 7), it was shown that excimer laser angioplasty can be performed safely and effectively (73). A success rate of 97% was achieved in lesions in older saphenous vein grafts; however, the preliminary reported restenosis rate of 61% was rather high.

Conclusions. Currently sufficient data are lacking to establish the merits of reoperation and balloon angioplasty to treat obstructions in venous bypass grafts. The published results on reoperation and balloon angioplasty should not be compared because of differences in patient selection, and firm conclusions about the superiority of one treatment above the other should not be drawn. A review of the published data indicates that in selected patients balloon angioplasty may be the preferred strategy and, in case of inoperability, it is the only strategy.

Angioplasty of nonocclusive obstructions in venous bypass grafts is safe and the success rate is high. The high restenosis rate adversely affects the long-term results. The immediate and long-term results of angioplasty for chronic total occlusion in old grafts are poor.

Patients considered for saphenous vein graft angioplasty may be classified into three groups according to expected

early and late outcome: 1) those with an initial high success, low procedural risk and low restenosis rate; 2) those with an initial high success but high procedural risk and moderate to high restenosis rate; and 3) those with a low success, high risk and high restenosis rate (Table 8).

New techniques have shown to be promising and stent placement, in particular, may be useful to "tack" friable material. However, definite conclusions concerning the merits of new techniques must await finalization of ongoing randomized trials. Balloon angioplasty is a palliative procedure, not a long-term solution in patients previously operated on, who often present with late-stage coronary artery disease. The high restenosis rate is a serious limitation of balloon angioplasty.

References

1. Lawrie GM, Lie JT, Morris GC, Beazley HL. Vein graft patency and intimal proliferation after aortocoronary bypass: early and long-term angiopathologic correlations. *Am J Cardiol* 1976;38:856-62.
2. 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.
3. Hamby RI, Aintablian A, Handler M, et al. Aortocoronary saphenous vein bypass grafts: long-term patency, morphology and blood flow in patients with patent grafts early after surgery. *Circulation* 1979;60:901-9.
4. Bourassa MG, Enjalbert M, Campeau L, Lesperance J. Progression of atherosclerosis in coronary arteries and bypass grafts: ten years later. *Am J Cardiol* 1984;53:102C-7C.
5. Bourassa MG, Fisher LD, Campeau L, Gillespie MJ, McConney M, Lesperance J. Long-term fate of bypass grafts: the Coronary Artery Surgery Study (CASS) and Montreal Heart Institute experiences. *Circulation* 1985;72(suppl V):V-71-8.
6. Seides SF, Borer JS, Kent KM, Rosing DR, McIntosh CL, Epstein SE. Long-term anatomic fate of coronary artery bypass grafts and functional status of patients five years after operation. *N Engl J Med* 1978;298:1213-7.
7. Virmani R, Atkinson JB, Forman MB. Aortocoronary saphenous vein bypass grafts. *Cardiovasc Clin* 1988;18:41-59.
8. Campeau L, Enjalbert M, Lesperance 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.
9. Frick MH, Valle M, Harjola PT. Progression of coronary artery disease in randomized medical and surgical patients over a 5-year angiographic follow-up. *Am J Cardiol* 1983;52:681-5.
10. Palac RT, Hwang MH, Meadows WR, et al. Progression of coronary artery disease in medically and surgically treated patients 5 years after randomization. *Circulation* 1981;64(suppl II):II-17-21.
11. de Feyter PJ, Serruys PW, Brower RW, van den Brand M, ten Katen HJ, Hugenholtz PG. Comparison of pre-operative, operative and post-operative variables in asymptomatic or minimally symptomatic patients to severely symptomatic patients three years after coronary artery bypass grafting: analysis of 423 patients. *Am J Cardiol* 1985;55:362-6.
12. Loop FD, Cosgrove DM. Repeat coronary bypass surgery: selection of cases, surgical risks, and long-term outlook. *Mod Concepts Cardiovasc Dis* 1986;55:31-6.
13. Schaff HV, Orzulak TA, Gersh BJ, et al. The morbidity and mortality of re-operation for coronary artery disease and analysis of late results with use of actuarial estimate of event-free interval. *J Thorac Cardiovasc Surg* 1983;85:508-15.
14. Cameron A, Kemp HG, Green GE. Re-operation for coronary artery disease. *Circulation* 1988;78(suppl I):I-158-62.
15. Laird-Meeter K, van Domburg R, van den Brand M, Lubsen J, Bos E, Hugenholtz PG. Incidence, risk and outcome of reintervention after aorta coronary bypass surgery. *Br Heart J* 1987;57:427-35.

Table 8. Classification of Patients With Attempted Saphenous Vein Graft Angioplasty According to Expected Early and Late Outcome

| A. Success Rate >90%, Complication Rate <2%, Restenosis Rate 30% | |
|---|--|
| Focal, short lesion | |
| Graft <4 to 6 years old | |
| Single graft | |
| Distal part sequential graft | |
| Lesion at distal site | |
| B. Success Rate >90%, Complication Rate <5%, Restenosis Rate 45% to 50% | |
| Long lesion | |
| Graft >4 to 6 years old | |
| Diffuse vein graft disease | |
| Intragraft thrombus | |
| Proximal part sequential graft | |
| Lesion at proximal site | |
| Lesion at body | |
| C. Success Rate <50%, Complication Rate >10%, Restenosis Rate >60% | |
| Chronic totally occluded old vein graft | |

16. Goldman S, Copeland J, Mortiz T, et al. Improvement in early saphenous vein graft patency after coronary artery surgery with antiplatelet therapy: results of a Veterans Administration Cooperative Study. *Circulation* 1988;6:1324-32.
17. Gruentzig AR, Senning A, Siengenthaler WE. Nonoperative dilatation of coronary-artery stenosis: percutaneous transluminal coronary angioplasty. *N Engl J Med* 1979;301:61-8.
18. Waller BF, Gorfinkel HJ, Dillon JC, Girod DA, Rothbaum DA. Morphologic observations in coronary arteries, aortocoronary saphenous vein bypass grafts and infant aortae following balloon angioplasty procedures. *Cardiol Clin* 1984;2:593-619.
19. Cox JL, Chiasson DA, Gottlieb AI. Stranger in a strange land: the pathogenesis of saphenous vein graft stenosis with emphasis on structural and functional differences between veins and arteries. *Prog Cardiovasc Dis* 1991;34:45-68.
20. Vlodaver Z, Edwards JE. Pathologic analysis in fatal cases following saphenous vein coronary arterial bypass. *Chest* 1973;64:555-63.
21. Vlodaver Z, Edwards JE. Pathologic changes in aortic-coronary arterial saphenous vein grafts. *Circulation* 1971;44:719-28.
22. Unni KK, Kottke BA, Titus JL, et al. Pathologic changes in aorta coronary saphenous vein grafts. *Am J Cardiol* 1974;34:526-32.
23. Kern WH, Dermer GB, Lindesmith GG. The intimal proliferation in aortic-coronary saphenous vein grafts: light and electron microscopic studies. *Am Heart J* 1972;84:771-7.
24. Spray TL, Roberts WC. Changes in saphenous vein used as aorto-coronary bypass grafts. *Am Heart J* 1977;94:500-16.
25. Kalan JM, Roberts WC. Morphologic findings in saphenous veins used as coronary arterial bypass conduits for longer than one year: necropsy analysis of 53 patients, 123 saphenous veins and 1865 five millimeter segments of veins. *Am Heart J* 1990;119:1164-84.
26. Walts AE, Fischbein MC, Matloff JM. Thrombosed, ruptured atheromatous plaques in saphenous vein coronary artery bypass grafts: ten years experience. *Am Heart J* 1987;114:718-23.
27. Lie JT, Lawrie GM, Morris GC. Aortocoronary bypass saphenous vein graft atherosclerosis. *Am J Cardiol* 1977;40:906-14.
28. Smith SH, Geer JC. Morphology of saphenous vein coronary artery bypass grafts. *Arch Pathol Lab Med* 1983;107:13-8.
29. Ratliff NB, Myles JL. Rapidly progressively atherosclerosis in aorto-coronary saphenous vein grafts: possible immune-mediated disease. *Arch Pathol Lab Med* 1989;113:772-6.
30. Bulkley BH, Hutchins GM. Accelerated "atherosclerosis": a morphologic study of 97 saphenous vein coronary artery bypass grafts. *Circulation* 1977;55:163-9.
31. Barboriak JJ, Pintar K, Korn ME. Atherosclerosis in aortocoronary vein grafts. *Lancet* 1974;2:621-4.
32. Walton KW, Slaney G, Ashton F. Atherosclerosis in vascular grafts for peripheral vascular disease. *Atherosclerosis* 1985;54:49-64.
33. Ford WB, Wholey MH, Zikria EA, Somadani SR, Sullivan ME. Percutaneous transluminal dilation of aortocoronary saphenous vein bypass grafts. *Chest* 1981;5:529-35.
34. Douglas JS Jr, Gruentzig AR, King SB III, et al. Percutaneous transluminal coronary angioplasty in patients with prior coronary bypass surgery. *J Am Coll Cardiol* 1983;2:745-54.
35. El Gamal M, Bonnier H, Michels R, Heijman J, Stassen E. Percutaneous transluminal angioplasty of stenosed aortocoronary bypass grafts. *Br Heart J* 1984;52:617-20.
36. Block PC, Cowley MJ, Kaltenbach M, Kent KM, Simpson J. Percutaneous angioplasty of stenoses of bypass grafts or of bypass graft anastomotic sites. *Am J Cardiol* 1984;53:666-8.
37. Corbelli J, Franco I, Hollman J, Simpfendorfer C, Galan K. Percutaneous transluminal coronary angioplasty after previous coronary artery bypass surgery. *Am J Cardiol* 1985;56:398-403.
38. Reeder GS, Bresnahan JF, Holmes DR Jr, et al. Angioplasty for aorto-coronary bypass graft stenosis. *Mayo Clin Proc* 1986;61:14-9.
39. Douglas JS Jr. Angioplasty of saphenous vein and internal mammary artery bypass grafts. In: Topol EJ, ed. *Textbook of Interventional Cardiology*. Philadelphia: WB Saunders, 1990:327-43.
40. Cote G, Myler RK, Stertz SH, et al. Percutaneous transluminal angioplasty of stenotic coronary artery bypass grafts: 5 years' experience. *J Am Coll Cardiol* 1987;9:8-17.
41. Ernst SMPG, van der Feltz TA, Ascoop CAPL, et al. Percutaneous transluminal coronary angioplasty in patients with prior coronary artery bypass grafting. *J Thorac Cardiovasc Surg* 1987;93:268-75.
42. Dorros G, Lewin RF, Mathiak LM, et al. Percutaneous transluminal coronary angioplasty in patients with two or more previous coronary artery bypass grafting operations. *Am J Cardiol* 1988;61:1243-7.
43. Reed DC, Beller GA, Nygaard TW, Tedesco C, Watson DD, Burwell LR. The clinical efficacy and scintigraphic evaluation of post-coronary bypass patients undergoing percutaneous transluminal coronary angioplasty for recurrent angina pectoris. *Am Heart J* 1989;117:60-71.
44. Cooper I, Ineson N, Demirtas E, Coltart J, Jenkins S, Webb-Peploe M. Role of angioplasty in patients with previous coronary artery bypass surgery. *Cathet Cardiovasc Diagn* 1989;16:81-6.
45. Platko WP, Hollman J, Whitlow PL, Franco J. Percutaneous transluminal coronary angioplasty of saphenous vein graft stenosis: long-term follow-up. *J Am Coll Cardiol* 1989;14:1645-50.
46. Webb JG, Myler RK, Shaw RE, et al. Coronary angioplasty after coronary bypass surgery: initial results and late outcome in 422 patients. *J Am Coll Cardiol* 1990;16:812-20.
47. Meester BH, Samson M, Suryapranata H, et al. Long-term follow-up after attempted angioplasty of saphenous vein grafts: the Thoraxcenter experience 1981-1988. *Eur Heart J* 1991;12:648-53.
48. Plokker HWT, Meester BH, Serruys PW. The Dutch experience in percutaneous transluminal angioplasty of narrowed saphenous veins used for aortocoronary arterial bypass. *Am J Cardiol* 1991;67:361-6.
49. Reeves F, Bonan R, Cote H, et al. Long-term angiographic follow-up after angioplasty or venous coronary bypass grafts. *Am Heart J* 1991;122:620-7.
50. Dorros G, Johnson WD, Tector AJ, Schmahl TM, Kalush SL, Janke L. Percutaneous transluminal coronary angioplasty in patients with prior coronary artery bypass grafting. *J Thorac Cardiovasc Surg* 1984;87:17-26.
51. Pinkerton CA, Slack JD, Orr CM, van Tassel JW, Smith ML. Percutaneous transluminal angioplasty in patients with prior myocardial revascularization surgery. *Am J Cardiol* 1988;61:15G-22G.
52. Douglas JS, Weintraub WS, Liberman HA, Jenkins M, Cohen CL, Morris DC. Update of saphenous graft (SVG) angioplasty: restenosis and long-term outcome (abstr). *Circulation* 1991;84(suppl II):II-249.
53. de Feyter PJ, Serruys PW, van den Brand M, Meester H, Beatt K, Suryapranata H. Percutaneous transluminal angioplasty of a totally occluded venous bypass graft: a challenge that should be resisted. *Am J Cardiol* 1989;64:88-90.
54. Kahn JK, Rutherford BD, McConahay DR, Johnson WL, Ligon R, Hartzler GO. PTCA of totally occluded saphenous vein grafts: safety and success (abstr). *J Am Coll Cardiol* 1992;19:350A.
55. Sievert H, Köhler KP, Kaltenbach M, Kober G. Wiedereröffnung langstreckig verschlossener aortakoronarer venen-bypasses. *Dtsch Med Wochenschr* 1988;113:637-40.
56. Hartmann JR, McKeever LS, Stamato NJ, et al. Recanalization of chronically occluded aortocoronary saphenous vein bypass grafts by extended infusion of urokinase: initial results and short-term clinical follow-up. *J Am Coll Cardiol* 1991;18:1517-23.
57. Levine DJ, Sharaf BL, Williams DO. Late follow-up of patients with totally occluded saphenous vein bypass grafts treated by prolonged selective urokinase infusion (abstr). *J Am Coll Cardiol* 1992;19:292A.
58. Jost S, Gulba D, Daniel WG, et al. Percutaneous transluminal angioplasty of aortocoronary venous bypass grafts and effect of the caliber on the grafted coronary artery on graft stenosis. *Am J Cardiol* 1991;68:27-30.
59. Kux A, Höpp HW, Hombach V, Hannekum A, Arnold G, Hügel W. Global and regional left ventricular function following acute coronary artery occlusion and emergency bypass grafting. *Z Kardiol* 1988;77:165-71.
60. Klepzig H, Kober G, Satter P, Kaltenbach M. Analysis of 100 emergency aortocoronary bypass operations after percutaneous transluminal coronary angioplasty: which patients are at risk for large infarctions? *Eur Heart J* 1991;12:946-51.
61. Bilodeau L, Iyer S, Cannon AD, et al. Flexible coil stent (Cook Inc.) in saphenous vein grafts: clinical and angiographic follow-up (abstr). *J Am Coll Cardiol* 1992;19:264A.

62. Urban P, Sigwart U, Golf S, Kaufmann U, Sadeghi H, Kappenberger L. Intravascular stenting for stenosis of aortocoronary venous bypass grafts. *J Am Coll Cardiol* 1989;13:1085-91.
63. Serruys PW, Strauss BH, Beatt KJ, et al. Angiographic follow-up after placement of a self-expanding coronary artery stent. *N Engl J Med* 1991;324:13-7.
64. de Scheerder IK, Strauss BH, de Feyter PJ, et al. Stenting of venous bypass grafts: a new treatment modality for patients who are poor candidates for reintervention. *Am Heart J* 1992;123:1046-54.
65. Pomerantz R, Kuntz R, Carrozza J, Fishman R, Safian R, Baim D. Treatment of vein graft stenoses by stents or directional atherectomy (abstr). *Circulation* 1991;84(suppl II):II-249.
66. Leon MB, Ellis SG, Pickard AD, Baim DS, Heuser RR, Schatz RA. Stents may be the preferred treatment for focal aortocoronary vein graft disease (abstr). *Circulation* 1991;84(suppl II):II-249.
67. Strauss BH, Serruys PW, Bertrand ME, et al. Quantitative angiographic follow-up of the coronary Wall-stent in native vessels and bypass grafts. *Am J Cardiol* 1992;69:475-81.
68. Leon MB, Kent KM, Baim DS, et al. Comparison of stent implantation in native coronaries and saphenous vein grafts (abstr). *J Am Coll Cardiol* 1992;19(suppl A):263A.
69. Strauss BH, Serruys PW, de Scheerder IK, et al. Relative risk analysis of angiographic predictors of restenosis within the coronary Wall-stent. *Circulation* 1991;84:1636-43.
70. Kaufmann UP, Garratt KN, Vlietstra RE, Holmes DR. Transluminal atherectomy of saphenous vein aortocoronary bypass grafts. *Am J Cardiol* 1990;65:1430-3.
71. Selmon MR, Hinohara T, Robertson GC, et al. Directional coronary atherectomy for saphenous vein graft stenoses (abstr). *J Am Coll Cardiol* 1991;17(suppl A):23A.
72. Meany T, Kramer B, Knopf W, et al. Multicenter experience of atherectomy of saphenous vein grafts: immediate results and follow-up (abstr). *J Am Coll Cardiol* 1992;19(suppl A):262A.
73. Untereker WJ, Litvack F, Margolis JR, et al. Excimer laser coronary angioplasty of saphenous vein grafts (abstr). *Circulation* 1991;84(suppl II):II-249.