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Restenosis After Directional Coronary Atherectomy: Differences Between Primary Atheromatous and Restenosis Lesions and Influence of Subintimal Tissue Resection

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Rates of restenosis were evaluated in 70 patients (74 lesions) after successful directional coronary atherectomy. The extent of vascular tissue resection was correlated with restenosis rates for coronary (n = 59) and vein bypass graft (n = 15) lesions.

After 6 months, the overall restenosis rate was 50% (37 of 74 lesions); it was 42% (15 of 36 lesions) when intima alone was resected, 50% (7 of 14 lesions) when media was resected and 63% (15 of 24 lesions) when adventitia was resected. Subintimal tissue resection increased the restenosis rate for vein grafts (43% with intimal resection versus 100% with subintimal resection, p = 0.01) but not for coronary arteries (50% versus 48%). There was no overall difference in restenosis rates after atherectomy between primary lesions and restenosis lesions that occurred after balloon angioplasty (46% versus 54%). Among postballoon angioplasty restenosis lesions, a higher rate of restenosis after atherectomy was found with subintimal than with intimal resection (78% versus 32%, p = 0.01).

Tissues from patients undergoing a second atherectomy for restenosis after initial atherectomy (n = 8) demonstrated neointimal hyperplasia that appeared histologically identical to restenotic tissue developing after balloon angioplasty (n = 37).

These data suggest that the cellular response to directional coronary atherectomy is characterized by neointimal proliferation similar to that which may develop after balloon angioplasty. The extent of fibrous hyperplasia appears to be related to the depth of tissue resection in vein graft lesions and coronary artery restenosis lesions that occur after balloon angioplasty but not in primary atheromatous coronary artery lesions.

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Although percutaneous coronary angioplasty is of proved utility in treating ischemic coronary syndromes (1–3), restenosis occurs in up to 45% of patients after successful balloon dilation (4). Specific restenosis rates may be influenced by patient selection (5,6) and procedural variables (7,8).

Directional coronary atherectomy is under investigation as an alternative to conventional balloon angioplasty for the treatment of coronary artery disease. Because atherectomy devices resect obstructive lesions rather than remodel them, the rate of restenosis may be less than that associated with balloon angioplasty. Several potential mechanisms have been suggested (9) to be of importance: 1) removal of atheroma may provide a luminal geometry able to accommodate subsequent cellular hyperplasia without appreciable impediment to blood flow; 2) the radial stretch injury that is caused by high pressure balloon inflation may be reduced with atherectomy; and 3) removal of smooth muscle cells from within or beneath the atheroma or neointima may reduce the number of cells available for replication and thereby reduce the substrate for restenosis. Although resection of medial smooth muscle cells occurs frequently with directional atherectomy and does not appear to be associated with an increased risk of acute complications (10,11), the effect of subintimal (media or adventitia) tissue resection by atherectomy on subsequent restenosis rates is unknown.

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The present study was undertaken to evaluate the incidence of restenosis after 6 months of follow-up in patients who underwent directional coronary atherectomy. Overall rates of restenosis, the influence of subintimal tissue resection and differences between primary atheromatous and postangioplasty restenosis lesions were assessed.

Methods

Patient selection. Patients referred to the Mayo Clinic cardiac catheterization laboratory between October 1988 and November 1989 were eligible for directional coronary atherectomy if they had evidence of myocardial ischemia with either a positive exercise test or classic anginal symptoms and if the luminal cross-sectional diameter was reduced by ≥70% in a segment of a coronary artery or saphenous vein bypass graft that was accessible to the device. This study was approved by the Institutional Review Board, and all patients gave informed consent. Only the outcome after a first atherectomy was considered in determining the rates of restenosis.

Coronary angiography. Angiography was performed with use of the Judkins technique (12) and standard cardiac catheterization equipment. All treated lesions were viewed from at least two orthogonal views and images were recorded on cineangiographic film. Severity of stenosis was determined visually by at least two experienced observers.

Atherectomy. Atherectomy was performed with the directional AtheroCath device (Devices for Vascular Intervention) as previously described (13,14). The size of the device used was determined by the size of the least diseased vascular segment adjacent to the lesion to be treated: vessels that were 2 to 2.5 mm in diameter were treated with a 6F atherectomy catheter, those >2.5 mm in diameter were treated with a 7F catheter and those <2 mm in diameter were treated with a 5.5F device.

All patients received aspirin (325 mg orally) and heparin (15,000 U intravenously) before atherectomy. Patients also received nifedipine and nitroglycerin during atherectomy. Aspirin was continued throughout the follow-up period.

Atherectomy was considered successful if tissue was obtained, the luminal diameter was increased by >30% with a residual stenosis <50% and no complication occurred that required a cardiac surgical intervention.

Histopathology. Tissue specimens were fixed in 10% neutral buffered formalin and routinely prepared for light microscopy. Tissues were stained with hematoxylin-eosin and elastic-van Gieson, the latter to define medial and adventitial borders. All specimens were reviewed by an experienced cardiovascular pathologist (W.D.E.) who did not know the clinical history. The presence of atheromatous intima, restenotic neointima and medial and adventitial elements was recorded. Subintimal resection was considered to have occurred if internal elastic membrane, media or adventitia was present in the tissue specimen. Immunohistochemical staining was performed in a subset of patients to document that intimal hyperplasia was composed chiefly of smooth muscle cells (unpublished data).

Follow-up. Patients returned at 3 and 6 months after atherectomy for follow-up evaluation. At the 6 month visit, patients underwent coronary angiography. When patients could not return to the Mayo Clinic, follow-up angiograms were performed at an outside hospital and the results forwarded to the Mayo Clinic.

Follow-up angiography was performed earlier than 6 months if symptoms suggesting myocardial ischemia developed. Restenosis was considered present if the treated vascular segment contained a lesion of >50% severity or if >50% of the initial gain had been lost.

Statistical analysis. Results are expressed as events per lesion unless otherwise noted. Continuous data are expressed as mean values ± SD. To preserve independence of observations, data pertaining to the outcome of repeat atherectomy within the same vascular segment previously treated with atherectomy were not included in the assessment of restenosis rates. Differences between data groups were assessed with use of a two-tailed unpaired Student's t test for continuous variables and chi-square contingency table analysis for discrete variables, assuming a standard normal probability distribution. Differences were considered significant at $p \leq 0.05$. Rates of restenosis were determined by assessing the percent of lesions developing restenosis within the 6 month follow-up period.

Results

Study patients. Between October 6, 1988 and November 11, 1989, directional atherectomy was successful and follow-up completed in 76 patients (80 lesions) at Mayo Clinic. One patient was lost to follow-up because of late (noncardiac) death and four patients refused repeat angiography. Tissue from one patient was lost. Therefore, complete clinical, angiographic and histopathologic data were available for 70 patients (74 lesions) undergoing a successful first atherectomy procedure. Of these, six patients had follow-up coronary angiography at other institutions.

Restenosis rates (Table 1). Among the patients studied, the mean severity of luminal stenosis of 86 ± 9% before treatment was reduced to 16 ± 12% by atherectomy. At follow-up angiography the mean vessel stenosis severity was 55 ± 30%. Restenosis occurred in 37 (50%) of 74 treated lesions and 36 (51%) of 70 patients. Patients developing restenosis were somewhat older and more likely to have diabetes mellitus than were those who did not develop restenosis. Patients who developed restenosis required earlier follow-up angiography (mean interval 143 ± 50 days) compared with patients who did not develop restenosis (mean interval 181 ± 17 days, $p = 0.0005$).

Table 1. Clinical and Angiographic Characteristics of 70 Patients With and Without Restenosis After Directional Coronary Atherectomy

	No Restenosis (n = 34)	Restenosis (n = 36)	p Value
Clinical characteristics			
Age (yr)	59 ± 11	64 ± 12	0.05
Men	27 (79%)	26 (72%)	NS
Symptoms			
None or minimal	13 (37%)	13 (36%)	NS
Moderate to severe	21 (62%)	23 (64%)	NS
Acute MI	0	1 (3%)	NS
Smoker (current or former)	20 (57%)	23 (64%)	NS
Diabetes mellitus	1 (3%)	7 (19%)	0.05
Prior CABG	10 (29%)	10 (28%)	NS
Angiographic characteristics			
Lesions (no.)	37	37	NS
Multivessel disease	24 (71%)	22 (61%)	NS
Vessel treated			
LAD	23	20	NS
LCx	4	1	NS
RCA	6	5	NS
SVG	4	11	0.05
Stenosis severity (%)*			
Before atherectomy	85 ± 9	87 ± 9	NS
After atherectomy	16 ± 13	16 ± 11	NS
Follow-up	29 ± 14	82 ± 15	0.0005

*Refers to percent luminal cross-sectional narrowing visually referenced to the least diseased adjacent vascular segment. CABG = coronary artery bypass graft surgery; LAD = left anterior descending artery or diagonal branch; LCx = left circumflex artery or obtuse marginal branch; MI = myocardial infarction; RCA = right coronary artery; SVG = saphenous vein bypass graft.

Restenosis and depth of vascular resection. The rate of restenosis was assessed for each lesion as it related to the depth of tissue resection (Fig. 1). Overall, restenosis occurred in 15 (42%) of 36 lesions with intimal resection only, 7 (50%) of 14 lesions with medial resection and 15 (63%) of 24 lesions with adventitial resection. Although subintimal resection was associated with a tendency toward a higher rate of restenosis than that associated with intimal resection, the difference was not significant (22 [58%] of 38 versus 15 [42%] of 36, p = 0.2). When subintimal resection occurred in coronary arteries, restenosis developed in 14 (48%) of 29; this rate was not different from the restenosis rate for coronary arteries with intimal resection only (15 [50%] of 30, p = NS). In contrast, subintimal resection in saphenous vein bypass grafts was associated with a significantly higher rate of restenosis than that associated with intimal resection only (8 [100%] of 8 versus 4 [57%] of 7, p = 0.05).

Restenosis in primary and postballoon angioplasty lesions. Restenosis rates were assessed for patients with primary atherometous lesions (n = 37) and with restenosis lesions that occurred after balloon angioplasty (n = 37) (Fig. 2). There was no difference overall in restenosis rates between

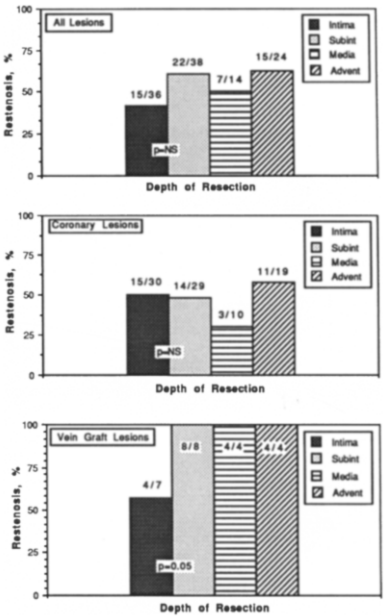

Figure 1. Six month restenosis rates after coronary atherectomy. Restenosis rates are shown for all lesions, coronary artery lesions only and saphenous vein bypass graft lesions only in the **top, middle and bottom panels**, respectively. Rates are displayed according to the depth of tissue resection. Subintimal (Subint) resection refers to resection of media or adventitia (Advent).

these two groups (17 [46%] of 37 primary lesions versus 20 [54%] of 37 restenosis lesions, p = NS).

For primary atheromatous lesions, intimal resection and subintimal resection resulted in similar restenosis rates (9 [50%] of 18 versus 8 [42%] of 19, p = NS). However, for postballoon angioplasty restenosis lesions, subintimal resection resulted in a significantly higher restenosis rate than that associated with intimal resection only (6 [32%] of 19 versus 14 [78%] of 18, p = 0.01). Adventitial resection was associated with restenosis in 10 (83%) of 12 postangioplasty restenosis lesions studied.

Restenosis and location of treated lesion (Table 2). Restenosis was assessed as it related to the vessel treated.

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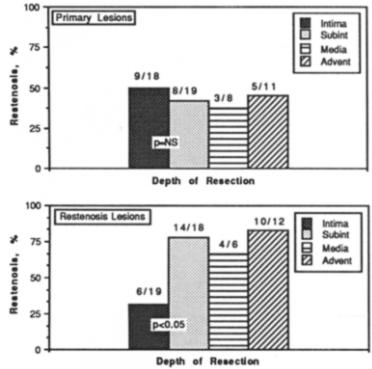

Figure 2. Six month restenosis rates for primary atheromatous and restenosis lesions displayed according to depth of tissue resection. Note the increased restenosis rate for subintimal resection in postangioplasty restenosis lesions but not for primary lesions. Abbreviations as in Figure 1.

Restenosis rates were similar for lesions in the left anterior descending coronary artery (20 [47%] of 43), right coronary artery (5 [45%] of 11) and left circumflex artery (1 [20%] of 5). Four of the five left circumflex artery lesions were resected deeply, although the restenosis rate was not increased by this factor. Restenosis occurred in 11 (73%) of 15 saphenous vein bypass grafts treated; as stated earlier, subintimal tissue resection was associated with a high rate of restenosis in vein grafts.

Histopathology of postatherectomy restenosis. Eight patients underwent repeat atherectomy of lesions in which stenosis recurred after a successful first atherectomy. Microscopic analysis of the tissue recovered from these patients indicated that loosely arranged hyperplastic smooth muscle cells constituted the neointimal layer that had formed after directional atherectomy (Fig. 3).

Table 2. Restenosis Rates in 74 Coronary Artery Segments and Saphenous Vein Grafts With Intimal and Subintimal Resection

	Intimal Resection (n = 37)	Subintimal Resection (n = 37)*	All Lesions (n = 74)†
LAD	9/22 (41%)	11/21 (52%)	20/43 (47%)
LCx	0/1	1/4 (25%)	1/5 (20%)
RCA	3/7 (43%)	2/4 (50%)	5/11 (45%)
SVG	3/7 (43%)	8/8 (100%)	11/15 (73%)

*Subintimal resection refers to resection of media or adventitia. †No significant difference found between values. Abbreviations as in Table 1.

These tissues had histopathologic features that were indistinguishable by light microscopy from those of tissues obtained from restenosis lesions that developed after balloon angioplasty.

Discussion

Mechanism of restenosis. The process of vascular restenosis appears to involve several cell populations that respond to direct and mediated influences. Smooth muscle cells from within atheroma and underlying media are stimulated to proliferate and migrate when subjected to radial stretching forces by balloon angioplasty (15–17). This process appears to be facilitated by the presence of mural thrombus (18), which presumably involves platelet-derived growth factor and other smooth muscle cell mitogens (18,19). Smooth muscle cell hyperplasia may be inhibited by the presence of an intact endothelial layer (9,20), in part by the release of heparin sulfate by confluent endothelial cells (9), which is known have a regulating effect on vascular smooth muscle cell replication (21). Balloon angioplasty is known to disrupt endothelial cell continuity and to cause injury to subintimal tissue (18), which may promote restenosis (18,20,22,23). Deep vascular injury is associated with mural thrombosis, even when a therapeutic dose of heparin sulfate is administered (24,25).

Directional atherectomy and restenosis. Initial experience with the directional atherectomy catheter in peripheral blood vessels (26,27) suggested that the rate of restenosis might be lower than that associated with balloon angioplasty, especially if a residual stenosis of <30% was obtained. Several possible contributors to this outcome were postulated. Removal of tissue should produce a greater luminal area that could accommodate subsequent neointimal growth without appreciably impairing blood flow (27). Improved laminar blood flow should reduce wall shear stress fluctuations at the lesion site and thereby reduce platelet deposition (18), which has been implicated in the promotion of neointimal hyperplasia after angioplasty (19,20). Laminar blood flow should also enhance endothelial recovery (9). Additionally, reducing the radial distending pressure applied to the treated vessel might limit the stimulus to smooth muscle cell proliferation caused by stretch of the arterial wall.

Nevertheless, neointimal proliferation has been observed after directional coronary atherectomy. Only preliminary data on the frequency of restenosis are available (28–30). In the present study, restenosis developed in 50% of treated lesions and was not related to baseline clinical variables other than age and presence of diabetes mellitus. Restenosis occurred as often in patients undergoing treatment of primary atheromatous lesions as in those undergoing treatment of restenosis lesions that occurred after balloon angioplasty. The rate of restenosis was high (73%) in

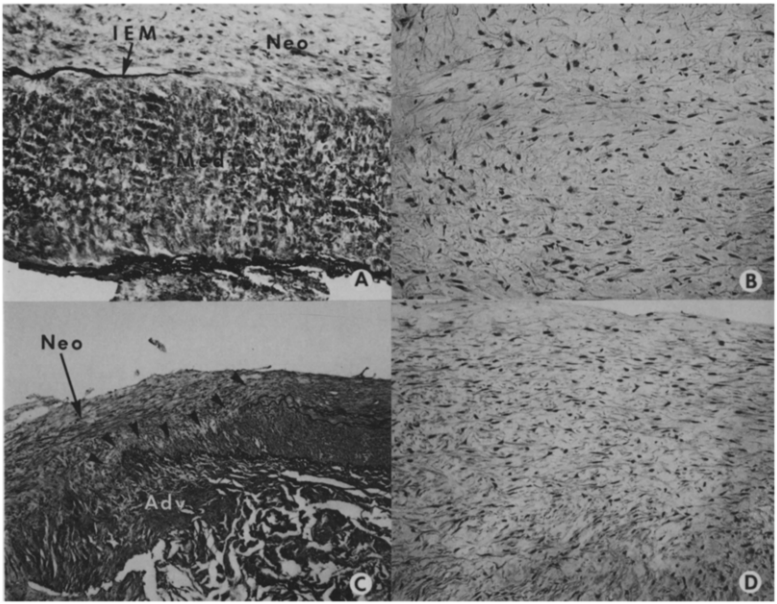

Figure 3. Photomicrographs of tissue resected with atherectomy. A, Tissue from the right coronary artery in a 48 year old man 3 months after balloon angioplasty. Internal elastic membrane (IEM) is disrupted with development of neointima (Neo) confluent with underlying media (Med). Verhoeff-van Gieson, magnification ×50. B, Restenosis tissue from the left anterior descending artery in a 70 year old woman 6 months after balloon angioplasty. Tissue consists of loosely arranged cells having characteristics of smooth muscle and myofibroblasts. Hematoxylin-eosin, magnification ×50. C, Tissue from the left anterior descending artery of a 65 year old woman 4 months after directional atherectomy. All vascular tissue layers are present in the specimen. The atherectomy resection border is visible (**arrowheads**) and extends into the adventitia (Adv). Verhoeff-van Gieson, ×25. D, Restenosis tissue from the same patient as in C, showing the same characteristics as seen in postballoon angioplasty restenosis lesions. Hematoxylin-eosin, ×50. All panels reduced by 30%.

the small number of saphenous vein bypass graft lesions treated.

Restenosis and depth of tissue resection. Although resection of media and adventitia was associated with only a small overall increase in the rate of restenosis over that associated with resection of intima alone (58% versus 42%), the relation between depth of tissue resection and restenosis appeared to depend on the nature of the lesion treated. Subintimal resection of primary atheromatous lesions was not associated with an increased rate of restenosis. However, subintimal resection of postangioplasty restenosis lesions was associated with a significantly higher rate of restenosis (78%) than occurred with resection of intima only (32%). It is therefore possible that the greater vascular injury associated with subintimal resection may be a more potent stimulus of smooth muscle cell proliferation than is the limited injury that occurs when resection is confined to the atheromatous intima. This hypothesis would be consistent with the view that the degree of injury and the development of intimal hyperplasia are related and that more severe injury (both direct and mediated through hemorrheologic factors) may transform a larger number of medial smooth muscle cells from a quiescent state to a synthetic state than would a lesser injury (9,15,16). The result would be greater cellular prolif-

eration, migration and production of extracellular connective tissue matrix (9,15,16). These data also support the hypothesis that repeated vascular trauma results in recruitment of additional quiescent medial smooth muscle cells to participate in the replicative process, which has been suggested (12) as the cause of an accentuated proliferative response after early repeat balloon angioplasty of a restenosis lesion.

Restenosis and location of treated lesion. Although the subgroups were small, restenosis rates did not appear to vary significantly among different coronary artery segments. Subintimal resection did not appear to correlate with increased rates of restenosis when specific coronary artery segments were analyzed. These observations must be considered as preliminary because of the small number of observations involved; a larger experience is needed to confirm these data.

Histopathology of postatherectomy restenosis. Evaluation of tissues resected from patients who underwent a second atherectomy when restenosis developed after the first atherectomy confirmed that postatherectomy restenosis involves the same proliferative process as that seen after balloon angioplasty. Thus, the cellular response to the two mechanisms of vascular injury is similar, although the extent of proliferation may be greater with subintimal atherectomy injury.

Conclusions. This study demonstrates that intimal hyperplasia occurs after percutaneous directional atherectomy of coronary artery and vein graft lesions in a manner similar to that observed after balloon angioplasty. Careful patient selection and modification of atherectomy instrumentation and technique to provide better control over the depth of tissue resection may improve the long-term results after coronary artery and vein graft atherectomy.

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