

## Clinical, Histologic and Quantitative Angiographic Predictors of Restenosis After Directional Coronary Atherectomy: A Multivariate Analysis of the Renarrowing Process and Late Outcome

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**Objectives.** To characterize predictors of restenosis after successful directional atherectomy, we reviewed the clinical, angiographic and procedural data obtained during 132 consecutive procedures.

**Methods.** Clinical and angiographic follow-up data were obtained in a prospectively collected and consecutive series of 125 patients who underwent 132 atherectomy procedures for de novo (89%) or restenotic (11%) lesions in native coronary arteries. Restenosis was assessed clinically and by quantitative coronary angiography. A dual approach to data analysis was taken to gain insight into factors affecting the clinical outcome and vessel wall healing response. Therefore, multivariate analysis was performed to 1) determine the correlates of residual lumen diameter at follow-up (angiographic outcome), and 2) characterize the determinants of the late lumen loss (renarrowing process).

**Results.** Clinical and angiographic follow-up data after successful atherectomy were obtained in 100% and 95%, respectively. Atherectomy achieved an acute lumen gain of  $1.28 \pm 0.48$  mm (mean  $\pm$  SD), resulting in a minimal lumen diameter of  $2.44 \pm 0.47$  mm. At follow-up, the minimal lumen diameter decreased to

$1.78 \pm 0.64$  mm. The angiographic restenosis rate was 28% if the traditional 50% stenosis cutoff criterion was applied. Larger vessel size and postatherectomy minimal lumen diameter and right coronary or left circumflex artery lesions were independent predictors of a larger minimal lumen diameter (angiographic outcome). Lumen loss during follow-up (renarrowing process) was independently predicted by relative lumen gain and preprocedural minimal lumen diameter.

**Conclusions.** In analyzing the long-term results of new interventional techniques such as directional atherectomy, the late lumen loss during follow-up (renarrowing process), which is characterized by the vessel wall healing response after an intervention, should be considered together with the residual lumen diameter at follow-up (clinical outcome). It is clear that whereas improved clinical outcome is associated with larger vessel size and postprocedural lumen diameter and non-left anterior descending artery location, greater relative gain at intervention is predictive of more extensive lumen renarrowing.

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Directional coronary atherectomy is now accepted as a feasible alternative to conventional balloon angioplasty for the treatment of coronary artery disease (1-8). When examining the long-term results of intracoronary interventions, two aspects must be considered: 1) the residual minimal lumen diameter at follow-up, which determines the angiographic outcome, and 2) the renarrowing process that can be

characterized by the late lumen loss during follow-up, which is initiated by the injury inflicted to the vessel wall during intervention. From a clinical point of view, Kuntz et al. (9) demonstrated that a large postprocedural lumen was the principal determinant for the best outcome at 6 months (that is, a large lumen at follow-up) and advocated the motto that "bigger is better." Although this may be a valid finding, the analysis was based on the relation of the minimal lumen diameter after the intervention and at follow-up without taking the vessel size and proportional gain into account. The influence of these two variables should be considered for two reasons. First, the range of vessels treated in interventional experience is 2 to 5 mm. Second, the restenosis rate has been reported (10,11) to vary with vessel size. Furthermore, greater lumen increase at intervention has been shown to be associated with a greater risk of coronary ectasia after atherectomy (12), acute complications (13) and increased lumen loss after angioplasty (14). Studies (15-17)

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have demonstrated that procedural lumen gain is the greatest single determinant of subsequent lumen loss.

Our group (15-17) has focused our attention on the renarrowing process and have reported the relation between relative lumen gain and relative lumen loss (that is, gain and loss normalized for the vessel size) as correlates of the biologic response of the vessel wall after an intervention. This "biologic approach" has unveiled the general biologic law relating healing process to vessel wall injury and has been encapsulated in the motto "the more you gain, the more you lose." The purpose of this study was to attempt to reconcile these apparently opposite viewpoints into a coherent methodologic approach by assessing the determinants of the angiographic outcome and renarrowing process in a consecutive series of patients treated by atherectomy.

### Methods

**Patients.** One hundred thirty-one patients underwent 138 successful consecutive directional coronary atherectomy procedures at the Thoraxcenter (n = 97) and University of Louvain Hospital (n = 41). Although all patients completed clinical follow-up, six patients (4%) did not undergo a 6-month angiographic follow-up and were excluded from the study.

**Atherectomy procedure.** The procedure was performed as described previously (5,6,8,17). On average,  $5.9 \pm 2.8$  cuts (range 2 to 14) in selected directions were performed across a stenosis. Although an optimal angiographic result was sought for each lesion treated, the procedure was considered angiographically successful when the residual diameter stenosis was <50% after tissue retrieval. Patients were monitored for 24 h and electrocardiograms and cardiac enzyme levels were obtained twice a day. A calcium channel antagonist was given every 2 h for 24 h after the procedure, and patients were kept on aspirin therapy for 6 months.

**Quantitative coronary angiography.** Quantitative analysis of the coronary segments was performed with the computer-based Coronary Angiography Analysis System, previously described in detail (8,16-21). In essence, boundaries of a selected coronary artery segment were detected automatically from optically magnified and video-digitized regions of interest ( $512 \times 512$  pixels) of a cineframe. The absolute diameter of the stenosis (in mm) was determined using the guiding catheter. The computer estimation of the original dimension of the artery at the site of the obstruction allowed us to define the interpolated reference diameter. The percent diameter stenosis was then calculated. Intracoronary isosorbide dinitrate (1 to 3 mg) was given before and after atherectomy. At follow-up catheterization, the administration of intracoronary nitrates was recommended before angiography. To standardize the method of data acquisition and data analysis and to ensure reproducibility of post-atherectomy and follow-up angiograms, measures were taken as previously described (17-19,21).

**Restenosis.** Two different approaches (categoric vs. continuous) were used to define restenosis. Using the categoric approach, the criterion chosen was an increase of the diameter stenosis from <50% after the intervention to  $\geq 50\%$  at follow-up, as is generally applied in clinical practice. Using a continuous approach, minimal lumen diameter at follow-up, lumen loss during follow-up and relative loss (normalized loss for vessel size) were determined.

**Lumen changes at intervention and during follow-up.** These are calculated as follows:

$$\text{Gain} = \text{MLD post} - \text{MLD pre}$$

$$\text{Loss} = \text{MLD post} - \text{MLD F-UP}$$

$$\text{Relative gain} = (\text{MLD post} - \text{MLD pre}) / \text{vessel size}$$

$$\text{Relative loss} = (\text{MLD post} - \text{MLD F-UP}) / \text{vessel size,}$$

where MLD = minimal lumen diameter, F-UP = at follow-up; post = after atherectomy and pre = before atherectomy. Absolute lumen changes for the individual vessel size were normalized, thereby eliminating the bias of vessel size as previously described (15-17).

**Multivariate analysis approach.** The long-term angiographic lumen changes after successful directional atherectomy were thus evaluated using two separate multiple linear regression analyses with minimal lumen diameter or lumen loss at follow-up, as the dependent variables. Variables potentially predictive of restenosis were divided into three general categories. Patient-related variables included age, gender, diabetes, hypertension, hypercholesterolemia (defined as elevated levels of serum cholesterol  $>6.5$  mmol/liter, requiring treatment with lipid-lowering drugs [22]) and unstable angina (defined as pain at rest requiring treatment with intravenous nitrates and intravenous heparin). Lesion-related factors included characteristics unique to each lesion. The following factors were assessed: vessel size, preatherectomy minimal lumen diameter, postatherectomy minimal lumen diameter, diameter stenosis before and after atherectomy, absolute gain and relative gain in minimal lumen diameter, treated vessel (left anterior descending coronary artery, left circumflex artery or right coronary artery) and de novo versus restenotic lesion. Procedure-related factors assessed included the center (Rotterdam or Louvain), number of atherectomy cuts, device size, device/artery ratio (defined as device size divided by the interpolated reference diameter) and the presence of media or adventitia in the excised specimens.

**Statistics.** All continuous variables are expressed as mean value  $\pm 1$  SD. A p value  $< 0.05$  was considered as significant. Differences between variables measured before atherectomy, after atherectomy and at follow-up were assessed using one-way analysis of variance for repeated measurements. When the result was significant, paired *t* tests were performed to determine the significant differences. Selected angiographic and procedural variables were evaluated by univariate regression analysis for their correlation with absolute loss in lumen diameter during follow-up and for their correlation with minimal lumen diameter at

**Table 1. Clinical Demographics of 125 Patients With 132 Stenoses Undergoing Coronary Atherectomy**

Age (yr)	58 ± 10
Male (%)	82
Angina status (%)	
Stable	60
Unstable	40
Multivessel disease (%)	23
Restenotic lesion (%)	11
Angiographic follow-up (%)	93

Values presented are mean value ± SD or percent of patients.

follow-up. To avoid arbitrary subdivision of continuous variables, cut points were derived by dividing the data in two groups, each containing roughly 50% of the total data base. The groups were compared with the use of two-group *t* tests. Two-group *t* tests for continuous variables and chi-square analysis for categorical variables were also used to compare the results from the two centers. The independent contribution of variables was assessed using a multivariate stepwise regression analysis with F to enter tests based on the mean square error criterion (23). All analyses were performed using BMDPC 90 statistical software.

## Results

**Patient characteristics and procedural results (Table 1).** The study group consisted of 125 consecutive patients who underwent 132 coronary atherectomy procedures for symptomatic de novo (*n* = 117) and restenotic (*n* = 15) native coronary artery disease. The mean age was 58 ± 10 years and the majority of the patients were men with single-vessel disease. The target stenosis (*n* = 132) in these 125 patients was located in the left anterior descending artery in 89 cases, the left circumflex artery in 14 cases and the right coronary artery in 29 cases. The clinical and immediate angiographic success rates as well as the complication rate for both centers have been described in detail elsewhere (5). The long-term results of the initial patients treated at the Rotterdam center with a primary lesion were previously reported in a comparative study (17) with balloon angioplasty. All but 23 patients were treated with a 6F atherotome, 21 were treated with a 7F atherotome and 2 patients with a 5F atherotome. The angiographic follow-up rate in the present study group was 95%. Of the six patients who did not undergo repeat angiography, one died 3 days after successful atherectomy (24), one had bypass surgery 7 days after the procedure for presumed tamponade and four asymptomatic patients refused angiography. At 6 months, 38 patients (31%) had recurrence of their anginal symptoms. Fifteen patients underwent either balloon angioplasty, repeat atherectomy (*n* = 3) or stent implantation (*n* = 1) for symptomatic restenosis of the previously treated segment. During the follow-up period, three patients were referred for elective coronary bypass surgery.

**Table 2. Quantitative Angiographic Analysis of the Immediate and Late Effects of Directional Coronary**

		p Value
Reference diameter (mm)		
Pre	3.29 ± 0.64	
Post	3.30 ± 0.50	NS
Follow-up	3.02 ± 0.60	< 0.001
Minimal lumen diameter (mm)		
Pre	1.16 ± 0.39	
Post	2.44 ± 0.47	< 0.001
Follow-up	1.78 ± 0.64	< 0.001
% diameter stenosis		
Pre	65 ± 11	
Post	26 ± 11	< 0.001
Follow-up	41 ± 18	< 0.001
Gain in lumen diameter		
Absolute (mm)	1.28 ± 0.48	
Relative	0.41 ± 0.19	
Loss in lumen diameter		
Absolute (mm)	0.65 ± 0.64	
Relative	0.20 ± 0.19	

Values presented are mean value ± SD. Post = after atherectomy; Pre = before atherectomy.

**Quantitative angiographic analysis (Tables 2 and 3).** The reference diameter did not change from before to after the procedure. The minimal lumen diameter increased from 1.16 ± 0.39 mm by 1.28 ± 0.48 mm, resulting in a minimal lumen diameter of 2.44 ± 0.47 mm after the procedure. At follow-up, the minimal lumen diameter was 1.78 ± 0.64 mm (Fig. 1). Thus, the late loss was 0.65 ± 0.64 mm. Likewise, percent diameter stenosis decreased from 65 ± 11% before atherectomy to 26 ± 11% after atherectomy and increased during follow-up to 41 ± 18% (*p* < 0.001). The restenosis rate was 28% if the 50% diameter stenosis criterion was applied. Although no statistical difference was found in lumen loss or minimal lumen diameter at follow-up between patients with stable and unstable angina, a trend toward a larger minimal lumen diameter at follow-up was observed in the stable group. "Restenotic" lesions did not differ significantly from primary lesions with respect to lumen loss during follow-up or retrieval of subintimal tissue. Subintimal tissue was excised (media [*n* = 19] or adventitia [*n* = 3]) and found to be related to the number of atherectomy cuts (5.7 ± 3.0 vs. 7.5 ± 2.8; *p* = 0.04), but not to the other procedural or angiographic variables.

**Univariate and multivariate analysis of residual lumen at follow-up: clinical outcome (Table 3).** A greater minimal lumen diameter at follow-up was associated with 1) vessel size >3.25 mm, 2) minimal lumen diameter after atherectomy >2.42 mm, 3) device/artery ratio ≤1.09, 4) preprocedural minimal lumen diameter >1.11 mm, 5) device size >6F and 6) lesion located in a vessel other than the left anterior descending artery. Multivariate stepwise regression analysis revealed that 1) vessel size, 2) minimal lumen diameter after atherectomy, and 3) non-left anterior descending artery lesions were independently predictive of minimal lumen diameter at follow-up. The multivariate model can be de-

Table 3. Categorical Approach to Assess Correlations for Renarrowing After Directional Coronary Atherectomy by Quantitative Angiography

	Median	No.	Vessel Size (mm)	MLD pre (mm)	Abs Gain (mm)	Rel Gain	MLD post (mm)	Abs Loss (mm)	Rel Loss	MLD FUP (mm)	DS FUP (%)
Vessel size	≤3.25 mm	67	2.80 ± 0.38	1.03 ± 0.34	1.30 ± 0.53	0.48 ± 0.21	2.34 ± 0.49	0.78 ± 0.60	0.25 ± 0.20	1.56 ± 0.58	43 ± 19
	>3.25 mm	65	3.79 ± 0.42*	1.28 ± 0.40*	1.26 ± 0.43	0.33 ± 0.12*	2.54 ± 0.43†	0.53 ± 0.66†	0.15 ± 0.18†	2.01 ± 0.63*	39 ± 16
MLD pre	≤1.11 mm	68	3.03 ± 0.61	0.86 ± 0.16	1.45 ± 0.48	0.49 ± 0.20	2.31 ± 0.47	0.70 ± 0.66	0.23 ± 0.21	1.62 ± 0.66	45 ± 19
	>1.11 mm	64	3.56 ± 0.53*	1.48 ± 0.30*	1.10 ± 0.41*	0.31 ± 0.12*	2.57 ± 0.43*	0.61 ± 0.61	0.17 ± 0.17	1.96 ± 0.57†	36 ± 16†
MLD post	≤2.42 mm	66	3.11 ± 0.57	1.04 ± 0.32	1.02 ± 0.38	0.34 ± 0.15	2.06 ± 0.28	0.44 ± 0.55	0.15 ± 0.19	1.62 ± 0.62	45 ± 18
	>2.42 mm	66	3.47 ± 0.66†	1.27 ± 0.43*	1.54 ± 0.43*	0.47 ± 0.20*	2.82 ± 0.27*	0.88 ± 0.65*	0.25 ± 0.19†	1.94 ± 0.63†	37 ± 17†
DS pre	>65%	62	3.39 ± 0.58	0.91 ± 0.26	1.47 ± 0.46	0.44 ± 0.17	2.38 ± 0.45	0.66 ± 0.69	0.20 ± 0.20	1.72 ± 0.68	45 ± 18
	≤65%	70	3.20 ± 0.68	1.38 ± 0.36*	1.11 ± 0.44†	0.33 ± 0.14†	2.49 ± 0.49	0.65 ± 0.60	0.20 ± 0.19	1.84 ± 0.60	37 ± 17†
DS post	>26%	65	3.43 ± 0.59	1.10 ± 0.43	1.11 ± 0.48	0.32 ± 0.14	2.20 ± 0.42	0.50 ± 0.63	0.14 ± 0.19	1.71 ± 0.68	46 ± 17
	≤26%	67	3.15 ± 0.66†	1.22 ± 0.43	1.45 ± 0.43*	0.48 ± 0.20*	2.67 ± 0.40*	0.81 ± 0.61†	0.25 ± 0.19†	1.86 ± 0.60*	36 ± 17*
Abs gain	≤1.29 mm	67	3.35 ± 0.59	1.30 ± 0.40	0.90 ± 0.27	0.27 ± 0.10	2.20 ± 0.45	0.47 ± 0.59	0.15 ± 0.19	1.73 ± 0.69	43 ± 19
	>1.29 mm	65	3.23 ± 0.68	1.00 ± 0.32	1.67 ± 0.30*	0.54 ± 0.16*	2.68 ± 0.35†	0.84 ± 0.63*	0.25 ± 0.13†	1.84 ± 0.54	38 ± 16
Rel gain	≤0.38	66	3.53 ± 0.56	1.37 ± 0.41	0.93 ± 0.31	0.26 ± 0.07	2.30 ± 0.52	0.50 ± 0.61	0.15 ± 0.18	1.80 ± 0.70	42 ± 19
	>0.38	66	3.05 ± 0.63*	0.94 ± 0.24*	1.63 ± 0.34*	0.55 ± 0.15*	2.58 ± 0.36*	0.81 ± 0.63†	0.25 ± 0.19†	1.77 ± 0.58	40 ± 16
Lesion	Restenosis	15	3.07 ± 0.68	1.17 ± 0.40	1.28 ± 0.31	0.43 ± 0.21	2.45 ± 0.45	0.79 ± 0.64	0.20 ± 0.20	1.65 ± 0.54	41 ± 15
	Primary	117	3.32 ± 0.63	1.16 ± 0.40	1.28 ± 0.48	0.40 ± 0.18	2.44 ± 0.47	0.64 ± 0.64	0.20 ± 0.20	1.80 ± 0.65	41 ± 18
	LAD	89	3.19 ± 0.64	1.12 ± 0.37	1.29 ± 0.49	0.42 ± 0.20	2.41 ± 0.46	0.75 ± 0.66	0.23 ± 0.20	1.67 ± 0.63	42 ± 18
	Not LAD	43	3.48 ± 0.60†	1.23 ± 0.44	1.26 ± 0.46	0.38 ± 0.16	2.49 ± 0.50	0.47 ± 0.55†	0.14 ± 0.17†	2.02 ± 0.61†	38 ± 16
Dev size	≤6F	111	3.22 ± 0.63	1.14 ± 0.38	1.24 ± 0.48	0.40 ± 0.19	2.38 ± 0.45	0.67 ± 0.63	0.21 ± 0.20	1.71 ± 0.63	42 ± 18
	>6F	21	3.67 ± 0.52	1.26 ± 0.44	1.47 ± 0.46†	0.42 ± 0.17	2.75 ± 0.44†	0.56 ± 0.67	0.15 ± 0.19	2.19 ± 0.56†	37 ± 17
Dev/art	>1.09	69	2.82 ± 0.40	1.03 ± 0.35	1.30 ± 0.52	0.33 ± 0.12	2.34 ± 0.48	0.74 ± 0.61	0.24 ± 0.20	1.60 ± 0.58	43 ± 19
	≤1.09	63	3.80 ± 0.42*	1.29 ± 0.39*	1.26 ± 0.44	0.47 ± 0.21*	2.55 ± 0.43†	0.56 ± 0.66	0.16 ± 0.18†	1.99 ± 0.65*	39 ± 16
Cuts	>5	64	3.36 ± 0.67	1.12 ± 0.36	1.29 ± 0.45	0.40 ± 0.19	2.41 ± 0.47	0.63 ± 0.68	0.19 ± 0.21	1.79 ± 0.67	43 ± 17
	≤5	68	3.22 ± 0.61	1.19 ± 0.42	1.28 ± 0.51	0.40 ± 0.19	2.46 ± 0.47	0.68 ± 0.60	0.21 ± 0.21	1.78 ± 0.61	39 ± 18
Histology	Media/adv	22	3.08 ± 0.65	1.23 ± 0.48	1.19 ± 0.42	0.41 ± 0.20	2.43 ± 0.42	0.71 ± 0.60	0.22 ± 0.20	1.73 ± 0.73	43 ± 21
	Intima	110	3.33 ± 0.63	1.14 ± 0.38	1.30 ± 0.49	0.40 ± 0.18	2.44 ± 0.48	0.65 ± 0.65	0.20 ± 0.20	1.79 ± 0.62	41 ± 17
Age	>58.9 yr	66	3.27 ± 0.58	1.07 ± 0.39	1.38 ± 0.45	0.43 ± 0.16	2.46 ± 0.49	0.67 ± 0.55	0.21 ± 0.17	1.79 ± 0.63	41 ± 18
	≤58.9 yr	66	3.31 ± 0.70	1.24 ± 0.38†	1.18 ± 0.48†	0.38 ± 0.20	2.42 ± 0.44	0.64 ± 0.72	0.20 ± 0.22	1.77 ± 0.66	41 ± 17
Gender	Female	24	3.27 ± 0.74	1.14 ± 0.39	1.31 ± 0.53	0.43 ± 0.22	2.44 ± 0.49	0.65 ± 0.71	0.19 ± 0.23	1.80 ± 0.68	38 ± 20
	Male	108	3.29 ± 0.62	1.16 ± 0.40	1.28 ± 0.47	0.40 ± 0.18	2.44 ± 0.49	0.66 ± 0.62	0.20 ± 0.19	1.78 ± 0.64	42 ± 17
Angina	Stable	80	3.23 ± 0.63	1.14 ± 0.41	1.33 ± 0.46	0.43 ± 0.18	2.46 ± 0.46	0.55 ± 0.60	0.20 ± 0.18	1.82 ± 0.63	39 ± 18
	Unstable	52	3.39 ± 0.63	1.19 ± 0.39	1.21 ± 0.51	0.37 ± 0.19	2.40 ± 0.46	0.67 ± 0.70	0.20 ± 0.22	1.73 ± 0.66	44 ± 18
Center	Rotterdam	91	3.18 ± 0.57	1.19 ± 0.39	1.24 ± 0.47	0.41 ± 0.20	2.42 ± 0.52	0.71 ± 0.60	0.22 ± 0.18	1.71 ± 0.55	41 ± 17
	Brussels	41	3.52 ± 0.64†	1.09 ± 0.40	1.37 ± 0.50	0.39 ± 0.15	2.47 ± 0.45	0.53 ± 0.70	0.17 ± 0.22	1.94 ± 0.79	41 ± 17

\*p < 0.001, †p < 0.05, ‡p < 0.01, all by Student t test. Abs gain = absolute lumen gain; Abs loss = absolute lumen loss; adv = adventitia; Dev/art = device/artery ratio; Dev size = device size; DS = diameter stenosis; F = French; FUP = follow-up; LAD = left anterior descending coronary artery; MLD = minimal lumen diameter; post = after atherectomy; pre = before atherectomy; Rel gain = relative lumen gain; Rel loss = relative lumen loss.

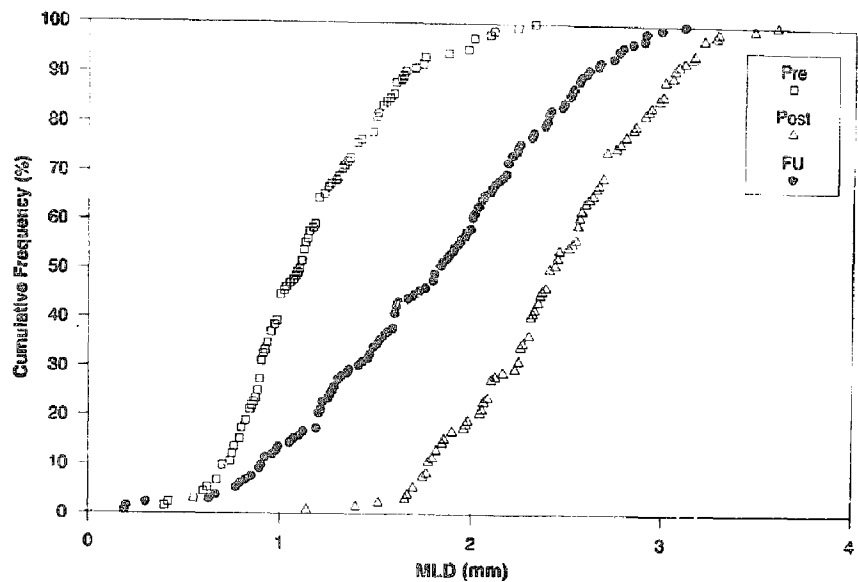


Figure 1. Cumulative frequency curves to illustrate the immediate and follow-up (FU) effects on minimal lumen diameter (MLD) of directional coronary atherectomy as assessed by quantitative coronary angiography. Post = after atherectomy; Pre = before atherectomy.

scribed by the following equation: minimal lumen diameter (MLD) at follow-up =  $0.21 + 0.25 \times \text{vessel size} + 0.37 \text{ MLD post} - 0.25 \times \text{LAD}$ , where a left anterior descending artery (LAD) lesion = 1 and a non-left anterior descending artery lesion = 0.

Univariate and multivariate analysis of late lumen loss: biologic approach (Table 3, Fig. 2 and 3). Relative gain  $>0.38$ , absolute gain  $>1.29$  mm, postatherectomy minimal lumen diameter  $>2.42$  mm, postatherectomy diameter stenosis  $\leq 26\%$ , lesion located in the left anterior descending artery and device/artery ratio  $>1.09$  were univariate predictors of a large absolute lumen loss during follow-up. The stepwise multiple regression analysis showed that 1) relative gain in lumen, and 2) preprocedural minimal lumen diameter were the only independent predictors of lumen loss during follow-up, calculated as: absolute loss =  $-0.59 + 2 \times \text{relative gain} + 0.399 \times \text{MLD pre}$  (Fig. 2A). Similarly, if lumen loss was normalized for individual vessel size, multivariate analysis revealed that relative gain is the strongest independent predictor of relative loss (Fig. 2B). It is readily appreciated that the wide scatter in the correlation plots implies that factors other than lumen dimensions (that is, biologic factors such as diabetes or ultrastructural constituents such as stellate cells and nonmuscular myosin) clearly play a considerable part in the process of restenosis.

In the univariate analysis, the relation between absolute loss and preprocedural minimal lumen diameter is negative (absolute loss =  $0.76 - 0.094 \text{ MLD pre}$ ); however because of the confounding effect of relative gain ((MLD post - MLD pre)/vessel size), the mathematic sign becomes positive in the multivariate analysis (absolute loss =  $-0.59 + 2 \text{ relative gain} + 0.399 \text{ MLD pre}$ ) (Fig. 2A).

The reconciliation of outcome and process. As seen in Figure 2, a linear relation exists between absolute gain and absolute loss and between relative gain and relative loss. Although some lesions show further lumen improvement

during follow-up, the observed linear relation imply that a greater lumen gain achieved at atherectomy is associated with a greater lumen loss during follow-up. Conversely, a satisfactory atherectomy result (large postatherectomy minimal lumen diameter) is predictive of a better lumen diameter at follow-up. Although these results appear contradictory, the slope of the gain/loss relation is clearly less than and divergent from the identity line, so that greater lumen gain is not fully offset by the subsequent loss. Thus, a beneficial long-term angiographic outcome (a large minimal lumen diameter at follow-up) will be achieved despite an augmented biologic renarrowing process (a greater lumen loss).

## Discussion

Late angiographic renarrowing as assessed by coronary angiography remains the major limitation of any coronary intervention. Neither pharmacologic (21,25-27) nor alternative interventional techniques including atherectomy (19,28-33) have been shown to abate the restenosis rate. Accepting that restenosis as a healing response to vessel wall injury is inevitable after atherectomy, it is appropriate to investigate the possibility of detecting patient, lesion and procedural factors that might be associated with a favorable or unfavorable influence. In this study, we used a well validated quantitative angiography analysis system to objectively assess immediate and long-term angiographic outcome after atherectomy. The angiographic follow-up rate of 95% further enhances the validity of the conclusions. Furthermore, we considered the lumen renarrowing process and the late lumen diameter (angiographic outcome) as variables of equal importance to resolve currently conflicting views.

Pathophysiologic considerations. In experimental studies, Schwartz et al. (34-36) observed a strong positive correlation between vessel wall injury (rupture of the internal elastic lamina) and the subsequent neointimal hyperplastic

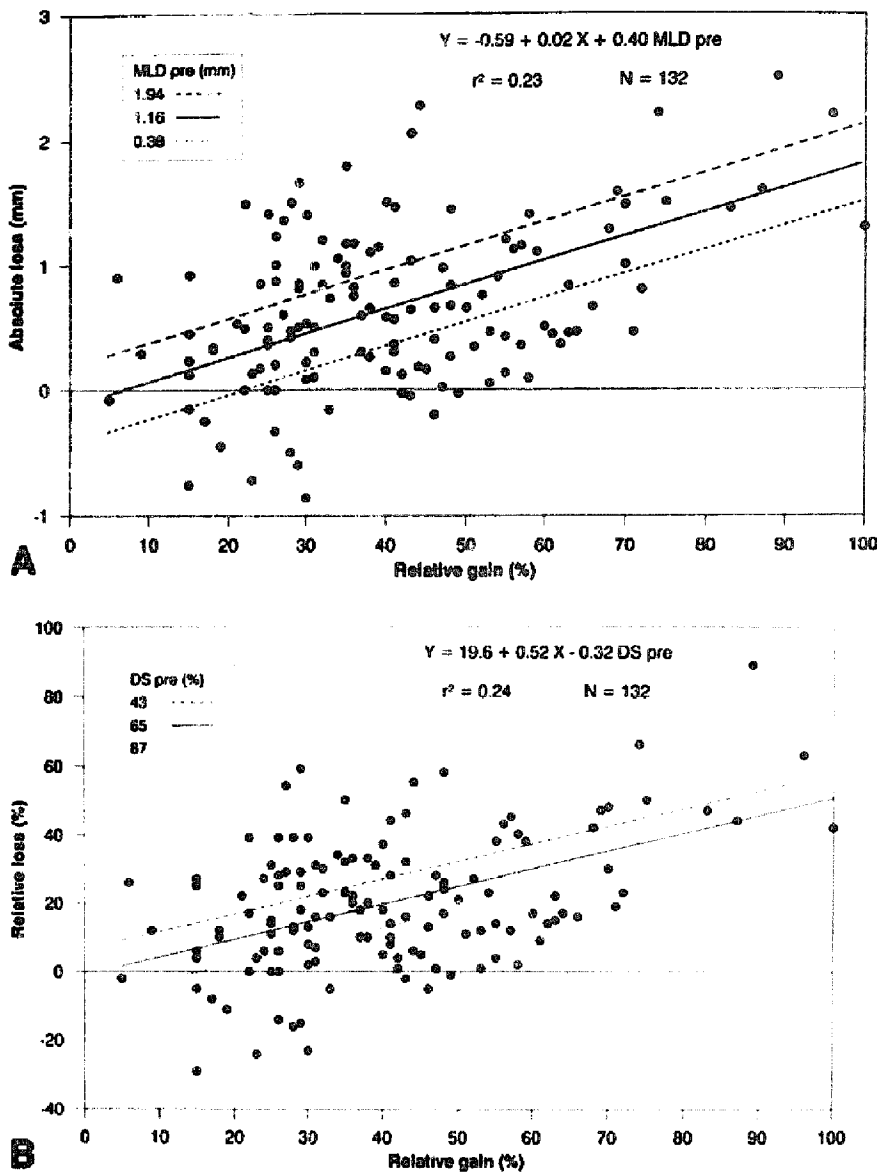


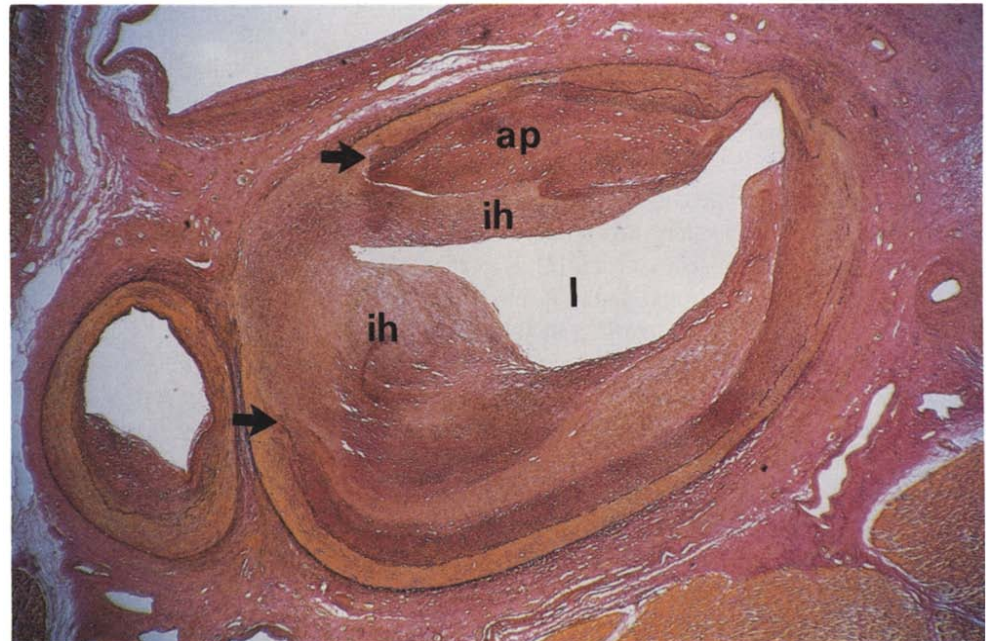
Figure 2. A, Scattergram of values obtained for relative gain achieved at directional atherectomy and absolute loss during follow-up in 132 procedures. The three lines are projections of the two-variate linear regression before atherectomy when minimal lumen diameter (MLD pre) equals 0.38 mm (mean - 2 SD), 1.16 mm (mean) and 1.94 mm (mean + 2 SD), respectively. B, Plot of the relative gain in lumen achieved at atherectomy versus the relative lumen loss during follow-up for 132 procedures. The three straight lines are projections of the two-variate linear regression when diameter stenosis before atherectomy (DS pre) equals 43% (mean - SD), 65% (mean) and 87% (mean + SD), respectively.

response during follow-up. To test this hypothesis in a clinical setting, we substituted the concept of "injury score" and "neointimal hyperplasia" used by Schwartz et al. (34-36) with the angiographically derived variables of relative gain and relative loss (15-17), so that the biologic relation between wall injury and the healing response could be more appropriately analyzed. It is crucial to elucidate whether the atherectomy procedure can to some extent escape the implacable consequences of the fundamental biologic laws governing the healing response to wall injury. The scientific value of the relation between relative gain and relative loss lies in the fact that this relation constitutes a unifying approach that may characterize the intrinsic efficacy of a device independently from the vessel size in which it is operational.

**Residual lumen at follow-up.** Of all directly acquired measurements by quantitative angiography, the absolute value of the minimal lumen diameter has been shown to be

the greatest single determinant of the hemodynamic consequences of a stenosis because this variable affects blood flow by the fourth power term (37). Moreover, the minimal lumen diameter at follow-up may have some functional component; we found that a minimal lumen diameter at follow-up of 1.45 mm correlates with the freedom from recurrence of angina (38). Thus, from a clinical point of view, the largest minimal lumen diameter at follow-up is the goal for which to strive when performing intracoronary interventions (9). Our study shows that a large reference diameter, a nonsevere preprocedural lesion, a large postprocedural diameter and presumably but not necessarily a greater absolute gain at atherectomy are associated with a large minimal lumen diameter at follow-up. Thus, previous findings of Kuntz et al. (9) are confirmed that a greater lumen after atherectomy provides greater late residual lumen. However, in addition, we found that a larger vessel itself is predictive of a greater follow-up lumen. It is also noteworthy that the greatest acute

**Figure 3.** Example of the *biologic* process after directional coronary atherectomy. Histologic cross section of the circumflex branch of the left coronary artery at the site of directional coronary atherectomy 9 months before death. The site of the previous atherectomy shows that the initial underlying atherosclerotic plaque (ap) has been excised. The internal elastic membrane and media are disrupted (arrows), indicating that subintimal resection has occurred at atherectomy. The fibrocellular proliferation that developed after the procedure is histologically distinct from the underlying plaque and has the typical appearance of intimal hyperplasia (ih). This proliferative process is limited not to the section where the plaque has been excised, but also occurs in the area that was exposed to the support balloon. At the site of excision and subintimal disruption, the proliferative response is substantially larger; however, balloon inflation, even with low pressures, also provokes a proliferative response. l = lumen. Verhoeff-van Gieson stain  $\times 4$ , reduced by 31%.



procedural results were achieved in larger vessels in this as well as in other series (10,11,39). In addition, a large relative lumen loss was observed in smaller vessels, in which greater relative gain had been achieved. This indicates that atherectomy appears more traumatizing and would, in our view, infallibly be associated with a poor long-term outcome (that is, a small minimal lumen diameter at follow-up). We surmise that this general type of response to the atherectomy procedure may be unveiled in the recently completed Coronary Angioplasty Versus Excisional Atherectomy (CAVEAT) Trial (32) comparing balloon angioplasty and atherectomy.

**The "restenosis" paradox.** The apparent paradox of greater lumen increase at intervention associated with greater lumen renarrowing during follow-up has now been demonstrated in several clinical studies (16,40,41). In this study, the greatest determinant of lumen loss or relative loss was the relative lumen gain achieved at atherectomy. This finding is in agreement with published findings (15,16) in studies of balloon angioplasty. On the basis of these findings, it would appear appropriate to use the relative gain/relative loss relation as angiographic correlates for the injury/hyperplasia phenomenon described in experimental models (34-36) and clinical research (4,6-9,11,13,14,18-20).

Although others have focused on the angiographic outcome (that is, final minimal lumen diameter) and found a reduced restenosis rate with increased lumen gain achieved

with newer devices (9), our group is focusing in clinical studies mainly on the degree of renarrowing as a measure of the extent of the "biologic" renarrowing process (that is, the development of intimal hyperplasia). This is the difference as has been expressed by Schwartz et al. (35) between the "doughnut and the doughnut hole." There is little doubt that a larger lumen at follow-up is clinically "better" for the patient and this variable is of great importance in assessing the long-term outcome of therapy. However, in large clinical trials directed at the prevention of renarrowing, the effect of therapy must be measured by its restricting effect on the thickness of the "doughnut," which we believe is best encapsulated angiographically by the relative lumen loss during follow-up. As described in the present report, we believe that application of both approaches (residual lumen and renarrowing process) to the same population yield equal findings. The apparently conflicting viewpoints arise not from differences in therapeutic results, but from differences in focus and approach. The coherent double approach to restenosis reveals that the clinician may achieve the best final outcome (large lumen at follow-up) by aiming for an optimal procedural result (large postprocedural lumen), particularly in large vessels. On the contrary, a large (relative) lumen loss is observed in small vessels in which a large relative gain is seen. This indicates that the renarrowing

process (lumen loss during follow-up) is augmented when a severe lesion in a small vessel is treated by atherectomy.

Whether subintimal tissue retrieval leads to an increased incidence in restenosis remains an unresolved issue with conflicting published reports (42,43). In this observational study, medial or adventitial tissue retrieval was not an independent variable related to more extensive lumen renarrowing, although the frequency of retrieval of media and adventitia was only 20% compared with >50% in other studies (2,43).

In the present study, no clinical and procedural variables were found to be independent predictors of restenosis. In two recent multicenter restenosis trials (21,27), diabetes was the only patient-related variable found to be independently related to the amount of renarrowing at follow-up. In our study, <10 patients with diabetes or hypercholesterolemia underwent atherectomy. Therefore, the predictive value of this variable cannot be evaluated in this study. Using univariate analysis, the device/artery ratio was found to be correlated with lumen loss; however, this was not retained in the multivariate analysis. This observation underscores the necessity to strive for an optimal selection of the atherotome. With the clinical implementation of quantitative angiography, proper device selection (device/artery ratio 1 to 1.1) can be performed and the final result can be guided by these on-line measurements.

Compared with previously published data (9,11,32,33) on lumen gain and loss after atherectomy, the acute lumen gain in this patient cohort seems low. These differences may be secondary to the applied method of quantitative angiographic analysis. Specifically, it has been observed that measurements obtained by visual assessment tend to overestimate the severity of tight stenoses and underestimate the degree of milder ones (44-46), whereas the opposite has been reported (47) of automated contour detection using well known phantom diameters. Therefore, visual or caliper measurements will yield higher values for lumen gain achieved at intervention compared with quantitatively assessed measurements. Nevertheless, the relation between gain and loss is maintained and is similar to that in other reports (9). Furthermore, a discrepancy between reference diameters will arise when comparing reports (9,39) in which the average of the diameter of the vessel proximal and distal to the stenosis is used as the reference. To avoid the bias introduced by the arbitrary selection of the user-defined reference in the proximal or distal segment of the stenosis, many years ago we (8,16-21) implemented an interpolated technique that is not user defined to determine the reference diameter at the actual stenosis site.

**Clinical implications.** Lumen renarrowing after successful atherectomy is a process that cannot be accurately predicted by simple clinical and angiographic variables. In analyzing the long-term results of new interventional techniques such as directional atherectomy, the renarrowing process (lumen loss during follow-up) characterized by the vessel wall healing response is of equal importance as the

angiographic outcome (minimal lumen diameter at follow-up), which conveys some index of the clinical outcome in the long-term. It is clear that whereas improved clinical outcome is associated with larger vessel size and postprocedural lumen diameter, greater relative gain at intervention is predictive of more extensive lumen renarrowing.

**Limitations.** Several limitations of this study are acknowledged. 1) It is an uncontrolled observational study limited to a subset of patients with a successful coronary atherectomy. 2) Although angiography may detect lumen changes after intervention, it may not be the most reliable method to analyze the (biologic) process taking place in the vessel wall itself. Because intravascular ultrasound provides an *in vivo* assessment of morphologic changes in the vessel wall, this technique may provide more precise information, although reliable quantitative measures cannot yet be routinely obtained (48). 3) It could be claimed that an acute gain of 1.28 mm represents a cautious approach to atherectomy, leading to a modest angiographic result. However, the postprocedural lumen diameter in this series is comparable to that observed by other groups (32,33,49) although smaller than that in the series of Kuntz et al. (9). This observation does not influence the conclusions of the present study because the linear relation between (relative) gain and (relative) loss is maintained at all levels of (relative) gain. 4) Lumen loss during follow-up may not only result from the biologic proliferative response but also may be due to elastic recoil. From a methodologic aspect, we recommend a 15-min recovery time after balloon deflation before proceeding with the administration of intracoronary nitrates and assessment of the final angiographic result. Furthermore, it has been the experience of our group (50) and others (51) that no further deterioration occurs in the 24 h after balloon deflation if this methodologic premise is respected. Although the occurrence of elastic recoil was not studied, previous reports (6,52) demonstrated minimal if any elastic recoil after coronary atherectomy. Finally, it should be appreciated that the predictive values of the models are weak because of a wide scatter of correlation plots. From a statistical viewpoint, the large standard error of the estimate found implies that other major biologic determinants of the late angiographic outcome and renarrowing process have not yet been unraveled. Because the restenosis process appears inherently uncontrollable, pharmacologic control of the proliferative response appears more mandatory than ever.

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