Restenosis after successful coronary angioplasty in patients with single-vessel disease

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ABSTRACT To determine risk factors for restenosis, we studied 998 patients who underwent elective coronary angioplasty (PTCA) to native coronary arteries between July 1980 and July 1984. Restenosis, defined as a luminal narrowing of greater than 50% at follow-up, was present in 302 patients (30.2%). Univariate analysis of 29 factors revealed seven factors related to restenosis: vessel dilated (circumflex coronary artery 18%, right coronary artery 27%, left anterior descending artery 34%; p < .01), final gradient of 15 mm Hg or less compared with greater than 15 mm Hg (27% vs 38%, p < .01), duration of angina greater than 2 months compared with angina of shorter duration (27% vs 35%, p = .01), post-PTCA stenosis of 30% or less compared with 31% to 50% (28% vs 36%, p < .025), stable vs unstable angina (26% vs 34%, p < .05), presence vs absence of intimal dissection (26% vs 32%, p = .07), and female gender vs male gender (25% vs 32%, p = .08). Multivariate analysis revealed five factors independently related to increased risk of restenosis in the following order of importance: PTCA in the left anterior descending artery, absence of intimal dissection immediately after PTCA, final gradient greater than 15 mm Hg, a large residual stenosis after PTCA, and unstable angina. Restenosis after PTCA is a multifactorial problem. The hemodynamic and angiographic result at the time of PTCA significantly influences long-term outcome, but additional measures aimed at reducing the rate of recurrence of atherosclerotic plaque are required.

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PERCUTANEOUS transluminal coronary angioplasty (PTCA) has become an attractive therapeutic option in selected patients with coronary artery disease. Despite the undoubted initial and long-term efficacy of PTCA, the problem of restenosis remains. Reported rates of restenosis in various subsets of patients range from 17% to 47%,1–8 but there is limited information on factors that predispose to recurrence of lesions. Most reported studies include relatively few patients,1,3–8 and data from the National Heart, Lung, and Blood Institute (NHLBI) PTCA Registry,2 apart from representing early experience with the procedure, included results from many centers in which PTCA procedures and angiographic interpretations were not optimally standardized. This study was undertaken to determine the risk factors for restenosis in a large group of patients who underwent PTCA of a single lesion in a native coronary artery.

Material and methods

Patients. Between July 1980 and July 1984, elective first PTCA was attempted in 1995 patients with single-vessel disease (≥50% diameter stenosis). Not included in this study were patients who underwent PTCA of saphenous vein grafts or who underwent repeat PTCA for lesion recurrence or PTCA during the acute phase of myocardial infarction. PTCA was considered successful if it reduced the diameter stenosis to 50% or less and was not associated with a major complication (electrocardiographic or enzymatic evidence of myocardial infarction, need for bypass graft surgery during hospitalization, or in-hospital death). The procedure was angiographically and clinically successful in 1758 patients (88%). Of these patients, angiographic follow-up was available in 998 patients (57%) who form the study population.

Definitions

Restenosis. Restenosis was defined as a residual stenosis at the time of follow-up angiography of more than 50% of luminal diameter (definition 1). Since other definitions of restenosis have been reported,2 we compared this definition with restenosis defined as (2) a loss of greater than 50% of the gain in vessel diameter achieved at PTCA and (3) an increase of greater than 30% in the immediate post-PTCA stenosis.

Clinical and angiographic variables. Angina was graded according to the Canadian Cardiovascular Society classification.
Unstable angina was defined as angina of increasing severity including pain at rest or angina of new onset (≤2 months). Luminal diameter stenosis was measured by use of a previously validated digital electronic caliper.\(^5\) Diameter stenosis was expressed as the mean of measurements made in at least two different projections. The transstenotic pressure gradient was defined as the difference in mean arterial pressure recorded from the guiding catheter and the balloon catheter distal to the stenosis. Intimal dissection was defined in accordance with the NHLBI PTCA Registry definition.\(^1\) First-response pressure was defined as atmospheric balloon inflation pressure at the time of disappearance of the dumbbell shape of the balloon.

**Follow-up.** Angiographic follow-up was recommended 6 months after successful PCI but was performed earlier when clinically indicated. Angiograms performed elsewhere were forwarded to Emory University Hospital for analysis. When more than one angiogram was available, the one that provided the longest angiographic follow-up was evaluated. Mean angiographic follow-up time was 7 ± 5 months (±SD).

All clinical, angiographic, and follow-up data were prospectively recorded on standard forms and stored in a relational database with the use of a VAX-750 computer. Questionnaires were used to record recurrence of anginal symptoms in patients who had follow-up angiograms from other hospitals. Routine discharge medications during the study period included 325 mg aspirin daily and a calcium antagonist.

**Statistical analysis.** All variables analyzed as possible risk factors for restenosis are listed in table 1. A chi-square test was used to assess differences in categorical variables. A one-way analysis of variance was used to assess differences in continuous variables between patients with restenosis and patients with continued success. A p (probability) value of <.05 was considered indicative of a significant difference. On data from a subset of patients for whom a complete set was available, multivariate analysis was performed with a stepwise logistic regression model. The following variables found to be significant or of borderline significance by univariate analysis were included in the multivariate analysis: age, sex, duration of angina (≥2 months vs <2 months), unstable angina, history of myocardial infarction, history of diabetes mellitus, insulin-dependent diabetes mellitus, vessel dilated at PCI, percent stenosis before and after PCI, uncomplicated intimal dissection on the immediate post-PCI angiogram, and transstenotic pressure gradient before and after PCI. Since patients with total or subtotal occlusions (≥95% diameter stenosis) were believed to be a special category unrepresentative of the total population, multivariate analysis was also done after excluding data from this group (n = 54).

Based on the results of this multivariate analysis (n = 787), an equation\(^2\) was developed that provided maximal power in classifying the chance of restenosis. This equation was then used to estimate the probability of restenosis by a stepwise procedure that includes and/or deletes potential risk factors based on a statistically significant gain or loss in classification power.

The probability of restenosis (%) = \(\frac{e^y}{1 + e^y} \times 100\), where

\[
e = 2.71828
\]

\[
Y = (V) + (A) + (D) + (G) + (0.015597 \times \% \text{ diameter stenosis after PCI}) - 1.2273
\]

V (PTCA vessel) = −0.91102 for circumflex artery

= −0.4275 for right coronary artery

= 0 for left anterior descending artery

A (angina) = 0.34463 for unstable angina

= 0.21474 if no angina

D (intimal dissection) = −0.48847 if present

= 0 if absent

G (final gradient) = 0.39049 if greater than 15 mm Hg

= 0 if 15 mm Hg or less

**Results**

Patient characteristics. Baseline characteristics of the 760 patients without follow-up angiograms and 998 patients in the study population are compared in table 2. Except for age, which was slightly greater in patients without follow-up angiograms, there were no significant differences in baseline characteristics of patients in the two groups.

**Rate of restenosis.** Restenosis defined as greater than 50% diameter stenosis at the time of follow-up angiography was present in 302 patients (30.3%). When restenosis was defined according to the traditional definition of a loss of 50% or more of the gain achieved at PCI (definition 2), restenosis occurred in 315 patients (31.6%). When defined as an increase in diameter stenosis of 30% or more (definition 3), restenosis was present in 272 patients (27.3%). Thus, when definitions 2 and 3 were used, as in the NHLBI PTCA Registry, restenosis was considered present in 36 patients who had residual narrowings of 50% or less. In another 12 patients PCI was considered, angiographically, a long-term success, even though the residual stenosis measured greater than 50% (figure 1).

**Clinical risk factors for restenosis (table 3).** Patients with unstable angina had a higher rate of restenosis than patients with stable angina (34% vs 26%, p < .05) and patients with a short history of angina (≤2 months).
TABLE 2
Characteristics of patients with and without follow-up angiograms

<table>
<thead>
<tr>
<th></th>
<th>Follow-up angiogram available (n = 998)</th>
<th>No follow-up angiogram (n = 760)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>769 (77%)</td>
<td>558 (73%)</td>
<td>NS</td>
</tr>
<tr>
<td>Female</td>
<td>229 (23%)</td>
<td>202 (27%)</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>54 ±10</td>
<td>56 ±10</td>
<td>&lt;.025</td>
</tr>
<tr>
<td>Unstable anginaa</td>
<td>507/911 (56%)</td>
<td>390/697 (56%)</td>
<td>NS</td>
</tr>
<tr>
<td>History of previous myocardial infarction</td>
<td>218 (22%)</td>
<td>184 (24%)</td>
<td>NS</td>
</tr>
<tr>
<td>History of diabetes mellitus</td>
<td>85 (9%)</td>
<td>62 (8%)</td>
<td>NS</td>
</tr>
<tr>
<td>Vessel dilated at PTCA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td>613 (62%)</td>
<td>474 (62%)</td>
<td></td>
</tr>
<tr>
<td>LCX</td>
<td>123 (12%)</td>
<td>98 (13%)</td>
<td>NS</td>
</tr>
<tr>
<td>RCA</td>
<td>262 (26%)</td>
<td>188 (25%)</td>
<td></td>
</tr>
<tr>
<td>Mean % stenosis Before PTCA</td>
<td>74 ±12</td>
<td>73 ±12</td>
<td>NS</td>
</tr>
<tr>
<td>After PTCA</td>
<td>24 ±11</td>
<td>24 ±11</td>
<td>NS</td>
</tr>
<tr>
<td>Transstenotic gradient (mm Hg)b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before PTCA</td>
<td>52 ±14 (n = 938)</td>
<td>51 ±15 (n = 720)</td>
<td>NS</td>
</tr>
<tr>
<td>After PTCA</td>
<td>12 ±7 (n = 899)</td>
<td>12 ±8 (n = 682)</td>
<td>NS</td>
</tr>
<tr>
<td>Presence of uncomplicated intimal dissection</td>
<td>263 (26%)</td>
<td>222 (29%)</td>
<td>NS</td>
</tr>
<tr>
<td>Maximal inflation pressure (atmospheres)</td>
<td>8.5±2.1</td>
<td>8.3±2.1</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Plus or minus* values are mean ± SD.
LAD = left anterior descending artery; LCX = left circumflex artery; RCA = right coronary artery.

aOf 1608 patients with typical angina.
bWhen available.

had a higher rate of restenosis than those without angina (35% vs 27%, p = .01). Women had a lower rate of restenosis than men (25% vs 32%), but the difference did not quite reach statistical significance (p = .08). Other clinical risk factors analyzed (table 1) had no significant influence on restenosis.

Angiographic variables (table 3). Rate of restenosis was significantly related to the vessel in which PTCA was performed (figure 2). The rate was 34% when PTCA was done in the left anterior descending coronary distribution, 27% when it was done in the right coronary artery, and 18%, the lowest rate, when the

![FIGURE 1. Patients with documented restenosis according to the three different definitions. With the use of a combination of definition 2 and 3, 12 patients had a follow-up stenosis of greater than 50%, but their PTCA procedures were considered long-term successes. According to definition 1, 36 patients had stenosis of 50% or less but would have been considered to have restenosis by definition 2 or 3.](image1)

![FIGURE 2. Restenosis and distribution of dilated vessels. The highest rate of restenosis was seen in the left anterior descending artery (LAD; 34%) and the lowest was in the left circumflex (LCX; 18%). Follow-up angiograms were available in 56% of patients in whom the LAD was dilated. 58% of those in whom the RCA was dilated, and 57% of those in whom the LCX was dilated (p = NS).](image2)
procedure was done in the left circumflex artery and its branches (p < .01). The proportion of patients who underwent angiographic restudy in each group was similar (figure 2). Restenosis was related to severity of stenosis before PTCA only when data from patients with totally occluded vessels before PTCA were included in the analysis. The rate of restenosis in patients with total occlusions (n = 33) was 48%. No other pre-PTCA angiographic variables listed in table 1 were found to have a significant influence on restenosis.

**Procedural variables (table 3).** A post-PTCA stenosis of 30% or less was associated with a lower rate of restenosis than a post-PTCA stenosis greater than 30% (28% vs 36%, p < .025). Similarly, a transstenotic pressure gradient of 15 mm Hg or less was associated with a lower rate than a gradient greater than 15 mm Hg (27% vs 38%, p < .01) (figure 3). The presence of an uncomplicated intimal dissection on the immediate post-PTCA angiogram was also associated with a lower rate of restenosis (26%) compared with when no intimal dissection was visible (32%), but by univariate analysis the difference did not quite reach statistical significance (p = .07). The immediate gain in luminal diameter in the stenotic left circumflex artery of 53 ± 15% was higher than that achieved in the left anterior descending artery (50% ± 15%, p < .05). At follow-up angiography, the loss in luminal diameter of the stenotic vessel was also lowest in the left circumflex (11 ± 22%) and right coronary arteries (13 ± 25%), and both were significantly less than the loss in the left anterior descending artery (18 ± 26%, p < .001). Thus, the long-term gain in diameter was significantly higher in the left circumflex and right coronary arteries.

**TABLE 3**

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>(%)</th>
<th>Difference</th>
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</thead>
<tbody>
<tr>
<td>Sex</td>
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<td></td>
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</tr>
<tr>
<td>Female</td>
<td>229</td>
<td>25</td>
<td>p = .08</td>
</tr>
<tr>
<td>Male</td>
<td>769</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>Angina</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Stable</td>
<td>404</td>
<td>26</td>
<td>p &lt; .05</td>
</tr>
<tr>
<td>Unstable</td>
<td>507</td>
<td>34</td>
<td></td>
</tr>
<tr>
<td>Duration of angina</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 2 months</td>
<td>529</td>
<td>27</td>
<td>p &lt; .01</td>
</tr>
<tr>
<td>≤ 2 months</td>
<td>344</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>Vessel dilated at PTCA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LCX</td>
<td>123</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>RCA</td>
<td>262</td>
<td>27</td>
<td>p &lt; .01A</td>
</tr>
<tr>
<td>LAD</td>
<td>613</td>
<td>34</td>
<td></td>
</tr>
<tr>
<td>% Stenosis after PTCA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 30%</td>
<td>830</td>
<td>28</td>
<td>p &lt; .025</td>
</tr>
<tr>
<td>31%–50%</td>
<td>268</td>
<td>36</td>
<td></td>
</tr>
<tr>
<td>Final gradient&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 15 mm Hg</td>
<td>652</td>
<td>27</td>
<td>p &lt; .01</td>
</tr>
<tr>
<td>&gt; 15 mm Hg</td>
<td>247</td>
<td>38</td>
<td></td>
</tr>
<tr>
<td>Intimal dissection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>263</td>
<td>26</td>
<td>p = .07</td>
</tr>
<tr>
<td>Absent</td>
<td>735</td>
<td>32</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations are as in table 2.

<sup>a</sup>χ<sup>2</sup> for linear trend.

<sup>b</sup>Transstenotic pressure gradient at the conclusion of PTCA.

**FIGURE 3.** Restenosis and transstenotic pressure gradient at the conclusion of PTCA (final gradient). The rate of restenosis in patients with a final gradient of 15 mm Hg or less (n = 652) was significantly lower (27%) than in patients with a final gradient greater than 15 mm Hg (n = 247; rate of restenosis, 38%).

**Multivariate analysis.** Multivariate analysis revealed six variables independently related to restenosis. In order of significance these factors were (1) vessel dilated at PTCA, (2) pre-PTCA diameter stenosis, (3) absence of uncomplicated intimal dissection on the immediate post-PTCA angiogram, (4) transstenotic pressure gradient greater than 15 mm Hg at the conclusion of PTCA, (5) a high residual (>30%) diameter stenosis immediately after PTCA, and (6) unstable angina before PTCA. When data from patients with total or subtotal occlusions were excluded from the analysis, pre-PTCA diameter stenosis was unrelated to risk of restenosis (table 4).

**Time between PTCA and follow-up angiography.** The relationship between restenosis and timing of the follow-up angiogram is shown in figure 4. The highest rate of restenosis was observed in patients who underwent follow-up angiography in the first 4 months after PTCA (53%). Restenosis was infrequently noted when the follow-up angiogram was obtained after 12 months. Clinical information regarding recurrence of symptoms was available at the time of follow-up an-
giography in 821 patients. The majority of patients (84%) who had repeat angiograms in the first 4 months after PTCA reported chest discomfort. In contrast, 44% of patients who had repeat angiograms 12 months or more after PTCA reported some chest discomfort. In neither group was there a close relationship between the reporting of symptoms and angiographically detected restenosis.

**Probability of restenosis.** The estimated probability of restenosis for each of the three coronary arteries dilated is shown in figure 5. Depending on the presence or absence of angiographically evident intimal tear or dissection, an optimal result (final gradient ≤15 mm Hg and diameter stenosis ≤30%), and unstable angina, the probability of restenosis was 20% to 53% for the left anterior descending artery, 14% to 43% for the right coronary artery, and 9% to 31% for the left circumflex artery.

**Discussion**

Restenosis after PTCA remains an important limitation to the long-term benefit of the procedure. The mechanisms underlying recurrence of lesions remain unclear and controlled trials aimed at reducing restenosis have, in general, yielded disappointing results.
Nonetheless, there is a perceptible trend toward lower rates of restenosis in some recent reports\(^4\) and a need to closely examine factors whose modification may reduce the problem.

Reported rates of restenosis\(^1\) vary greatly and may be in part influenced by definition and rates of angiographic restudy. In the present study, we chose to define restenosis as a lesion causing greater than 50% luminal diameter narrowing at restudy. There are sound physiologic\(^13\) grounds for the use of such a definition. This study only included those patients in whom the initial post-PTCA diameter stenosis was 50% or less (mean 24 ± 11%) and on average, a significant decrease in luminal diameter had to occur before restenosis was considered to be present. Restenosis occurred in 30.3% of patients who underwent repeat angiography. Of importance was the finding that, when the more conventional definition\(^2\) of restenosis (loss of ≥50% of the initial gain) was used, it was only recorded in an additional 1.3% of patients. The rate of restenosis was least (27.3%) when the third definition (increase in diameter stenosis of ≥30%) was used.

The rate of angiographic restudy might be expected to influence the incidence of restenosis since more symptomatic patients are submitted to repeat coronary arteriography. In the present study the restudy rate was 57%, which is considerably less than that of 84% in the NHLBI PTCA Registry.\(^2\) Despite the difference, the incidence of restenosis was similar and this does not appear to be explained by a difference in the definition used. The lowest incidence of angiographic restenosis thus far reported (17%)\(^4\) comes from a study in which angiographic follow-up was 94% complete. However, in another relatively large study\(^5\) with similar excellent follow-up data, the rate of restenosis was 29%. In the present study 760 patients (43%) did not undergo angiographic follow-up. In terms of the risk factors studied the 998 patients with follow-up angiograms were characteristic of all 1758 patients in whom PTCA was successful. Thus, it appears that the results of this study are representative but that the incidence of restenosis might have been lower if all patients had undergone repeat arteriography. However, the conclusions from this study must be interpreted as strictly applying only to patients who undergo angiographic follow-up.

As expected, patients restudied early had symptoms and the highest rate of restenosis. These findings once again confirm that if restenosis occurs it is most likely to do so in the first 4 months. In spite of the general relationship between recurrence of symptoms and restenosis, the incidence of chest discomfort reported by patients always exceeded the incidence of angiographic restenosis. Information on symptoms was not obtained by physician interview and therefore may have been somewhat unreliable.

Univariate analysis identified two clinical factors — unstable angina and duration of symptoms — as predictors of restenosis and this agrees with results from the smaller NHLBI Registry study group.\(^2\) Similarly, two important procedure-related variables — post-PTCA diameter stenosis and final transstenotic pressure gradient — were identified as univariate predictors of restenosis. Two angiographic variables were also related to restenosis. The first, “pre-PTCA diameter stenosis,” was also noted in the NHLBI PTCA Registry,\(^2\) but the second, “vessel undergoing PTCA,” has not been previously reported. The pre-PTCA diameter stenosis only influenced lesion recurrence when patients with totally or subtotally (≥95% diameter stenosis) occluded vessels were included. In the relatively small subset of 33 patients with totally occluded vessels who underwent angiographic restudy, the restenosis rate was 48%. The high rate of restenosis in patients with total or near-total occlusions before PTCA may relate to the incidence of unstable angina in such patients. However, when included in the multivariate analysis the effect of post-PTCA diameter stenosis was independent of a history of unstable angina. The competitive flow effect of collaterals in such vessels may play some role.

In contrast to the NHLBI study,\(^2\) male gender and history of myocardial infarction were not selected as independent predictors of restenosis. The pre-PTCA factors responsible for the relatively high rate of restenosis in the left anterior descending artery remain unclear. First, the proximal left anterior descending artery is most often the coronary artery with the largest diameter. Since over this time period a large majority of lesions were dilated with 3.0 mm diameter balloons, it is possible that balloon catheters were relatively undersized for the left anterior descending artery and oversized for the right coronary and left circumflex arteries. Preliminary reports\(^15\) have associated a larger balloon/artery diameter ratio to a lower initial post-PTCA diameter stenosis and reduced rates of restenosis, but this remains to be confirmed. Second, lesions in the proximal left anterior descending artery often involve the origin of the vessel and branch points,\(^17\) and this has been related to increased risk of restenosis.\(^18\) Balloon dilatation of stenoses at the ostium of the renal artery are known to lead to unsatisfactory initial and long-term results.\(^20\) Finally, prox-
eral segments of the left anterior descending artery are well recognized as showing an increased tendency to develop localized stenoses.\textsuperscript{17, 21, 22} Whether the same underlying mechanisms may predispose to recurrence of lesions is unknown.

An optimal initial result, manifest by a gradient of 15 mm Hg or less and residual stenosis of 30\% or less, predicted a relatively low likelihood of restenosis, as has been previously reported.\textsuperscript{2, 5, 18, 23} It is therefore apparent that all reasonable efforts should be made at the time of PTCA to achieve optimal results. Methods that allow the PTCA operator to accurately measure results during the PTCA procedure, particularly before guidewire removal, need improvement. The favorable association between an uncomplicated intimal dissection, providing the final gradient is 15 mm Hg or less, and continued success has also been reported.\textsuperscript{5, 24, 25} Since the extent of dissection induced by dilatation cannot be controlled by the operator, attempts to achieve intimal dissection cannot be recommended. Angiographically evident intimal dissection increases the chance of a major complication sixfold.\textsuperscript{26}

Even allowing for an optimal gradient and diameter stenosis immediately after PTCA, in this group, the estimated risk of restenosis remained from 9\% to 29\% for patients with stable symptoms and 12\% to 36\% for patients with unstable symptoms, depending on the vessel dilated (figure 5). It is clear, therefore, that in spite of optimal initial results, occlusive plaques can recur rapidly after PTCA. The histologic appearance of some plaques may be indistinguishable from that of disease in nondilated segments,\textsuperscript{27} but a recent report\textsuperscript{28} suggests early restenosis may represent intimal proliferation of smooth muscle cells. The present study confirms previous findings\textsuperscript{2} that have suggested that an “active disease state,” unstable angina, and a short history of symptoms are predictors of restenosis. Pharmacologic approaches that suppress the underlying disease process or inhibit the proliferation of cellular elements after PTCA probably hold the greatest promise in solving the problem of restenosis. Since almost all patients in this study were discharged on aspirin, the influence of this agent on restenosis cannot be determined. Also included in the present study were patients in two prospective, randomized trials\textsuperscript{5, 30} examining the effects of coumadin\textsuperscript{1} and the calcium antagonist nifedipine.\textsuperscript{30} Since neither of these agents showed any beneficial effect over that of aspirin alone, their inclusion in the therapeutic regimens of this study population would have been unlikely to influence the results.

A potential limitation of any study based on diameter stenosis is the difficulty in accurately measuring the severity of stenosis immediately after PTCA. To minimize this problem, we expressed the stenosis as a mean of that measured in several projections. Furthermore, restenosis occurred in only 12 patients (1.2\%) in whom the diameter stenosis increased less than 20\%. It is therefore unlikely that inaccuracy of measurements could have significantly influenced the results.

Clinical implications. The data presented may be useful in identifying patients at high and low risk of restenosis after PTCA. A typical patient at high risk of restenosis in our series presented with unstable angina and a lesion in the left anterior descending artery (figure 5, A). If the dilated lesion did not show some visible evidence of intimal dissection and had a final gradient greater than 15 mm Hg and residual diameter stenosis greater than 30\%, the probability of restenosis was as high as 53\%. Such a patient should be followed closely after PTCA and would be an ideal candidate for interventions aimed at reducing restenosis.

In contrast, a typical patient at low risk for restenosis had stable angina and presented with a lesion in the left circumflex artery or one of its branches (figure 5, C). If such a dilated lesion showed some angiographic evidence of intimal disruption and had a final gradient of less than 15 mm Hg and a residual diameter stenosis of less than 30\%, the probability of restenosis was as low as 9\%.

This study confirms that the risk of restenosis is reduced if initial angiographic and hemodynamic results are optimal. Although the presence of an angiographically evident intimal tear or dissection favored long-term success, it needs to be emphasized that the extent of arterial dissection cannot be controlled by the PTCA operator. Accordingly, the dilatation technique should not be aimed at producing evidence of intimal disruption. Alternative methods of reducing restenosis are required. Pharmacologic interventions aimed at reducing the rate of recurrence of obstructive plaque probably hold the greatest hope for reducing the rate of restenosis after PTCA.

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