Angiographic and clinical predictors of acute closure after native vessel coronary angioplasty

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ABSTRACT To determine predictors of acute coronary closure after PTCA performed with steerable catheter systems, we compared 140 procedures complicated by acute closure and 311 representative successful attempts from 4772 procedures performed between April 1982 and March 1986. Sixteen clinical, 35 angiographic, and seven procedural variables were analyzed. Multivariate analysis found seven independent preprocedural factors related to closure: stenosis length of 2 or more luminal diameters, female gender, stenosis at a bend point of 45 degrees or more, stenosis at a branch point, stenosis-associated thrombus (filling defect or staining), other stenoses in the same vessel, and multivessel disease. In addition, four procedural factors were found to be associated with closure by univariate analysis: post-PTCA percent stenosis (p<.001), intimal tear or dissection (p<.001), use of prolonged heparin infusion (p<.001), and post-PTCA gradient of 20 mm Hg or more (p=.004). Multivariate analysis of both preprocedural and procedural variables found six factors independently related to closure: post-PTCA percent stenosis, dissection, prolonged post-PTCA use of heparin, branch point location, fixed bend point location, and other stenoses in the vessel dilated. The risk of coronary closure after PTCA has many determinants. While an estimation of risk can be made before performing PTCA, the most powerful predictors of closure can only be assessed during the procedure itself. 


WITH CURRENT TECHNIQUES of percutaneous transluminal coronary angioplasty (PTCA), acute closure after an initially successful dilatation occurs in only 4% of attempts, but it is the major cause of morbidity and mortality in this increasingly performed procedure. Female gender, unstable angina, and right coronary, severe, eccentric, and nondiscrete lesions were found by the National Heart, Lung, and Blood Institute (NHLBI)–PTCA Registry to be risk factors for closure. A more recent study analyzing over 3000 procedures performed between 1980 and 1984 emphasized, in addition, multivessel disease, lesion calcification, and lesion length as major risk factors for acute complications. The presence of thrombus at the site of dilatation also may increase the risk of the closure.

Recent interest has developed in the analysis of detailed coronary morphology to predict the occurrence of unstable angina and myocardial infarction. Such methods have not been applied to the prediction of risk of acute closure after PTCA. In addition, angioplasty equipment, indications, and primary success rates have changed considerably since the time of the Registry and other major analyses of risk. Previously reported risk factors may therefore no longer be as important.

This study was undertaken, therefore, to redefine coronary morphologic characteristics associated with acute closure during and after PTCA and relate these to other clinical and procedural descriptors of increased risk.

Methods

Patient population. From April 1, 1982, when steerable angioplasty catheter systems became available at Emory University Hospital, to March 31, 1986, 5046 PTCA procedures were undertaken. Sixty-one of these procedures were performed in the setting of a myocardial infarction (typical ischemic chest pain ≥ 60 min with associated electrocardiographic [ECG] changes) and 213 procedures were performed on coronary bypass grafts. These procedures were excluded from this analysis. The remaining 4772 attempts form the basis for this study.
PTCA procedure. The technique of angioplasty used has been described elsewhere. Briefly, arterial and venous sheaths with PTCA guiding and pacemaker catheters were inserted, by Seldinger’s technique, from the groin, followed by the administration of heparin (10,000 units iv bolus), nitroglycerin (0.4 mg sublingual), and/or nifedipine (10 mg sublingual). Heparin boluses (5000 units) were repeated hourly during the procedure. Preliminary angiography of the coronary artery to be dilated was performed in at least two projections. A dilatation balloon was chosen to have its inflated diameter approximate the normal luminal diameter at the site to be dilated. The balloon catheter system was inserted and selective intracoronary nitroglycerin (200 μg) was given. The balloon was positioned across the stenosis, a pressure gradient was recorded, and the balloon was inflated as many times as necessary to produce the optimal hemodynamic and angiographic results. At the end of the procedure, all catheters were removed but the sheaths were left in place. The sheaths were removed 2 to 3 hr later unless angiographic evidence of coronary dissection or thrombus was seen. If a nonocclusive tear or dissection or filling defect was apparent, the patient was usually maintained on heparin infusion overnight, with the removal of sheaths 2 hr after discontinuation of heparin. In 1985–1986, sheaths were usually removed before starting heparin infusions.

After PTCA, patients were taken to a postprocedure ward or coronary care unit where they were monitored by telemetry for at least 16 hr. A 12-lead electrocardiogram was obtained immediately after PTCA and daily for 2 days. Creatine kinase (CK) levels, including isoenzyme determinations, were performed immediately after PTCA and at 8 and 16 hr. All patients were medicated with cutaneous nitroglycerin (1 inch every 4 hr) or isosorbide dinitrate (5 mg sublingual every 4 hr), calcium-channel blocking agents, and aspirin (80 or 325 mg/day, orally). Patients were routinely discharged 2 days after PTCA.

Routine data collection and entry. Clinical information, angiographic measurements, and intra- and post-PTCA data were recorded prospectively on standard forms during hospitalization by a physician. Specific laboratory data including ECG results, CK levels, and other post-PTCA complication information was recorded by a physician at the time of hospital discharge. This information was audited for completeness and entered into a computerized data bank.

The following information was used for this analysis: age, sex, weight, history of insulin-dependent diabetes mellitus, hypertension, hyperlipidemia, plasma cholesterol on admission, smoking, presence of unstable angina (defined as either pain at rest, prolonged pain lasting more than 20 min, or worsening of angina status within 8 weeks), Canadian Heart Association angina class, history of prior myocardial infarction, single or multivessel disease, location of stenosis dilated, history of prior PTCA performed at the same coronary site, date of PTCA, pre- and postprocedure percent diameter stenosis, pre- and postdilatation translesional gradient, first and maximum balloon inflation pressure, the presence of a post-PTCA intimal tear or dissection, and use of a postprocedural heparin infusion (≥8 hr).

Definition of acute closure and myocardial infarction. Acute closure was defined as clinical or ECG evidence of myocardial ischemia and/or a critical reduction in blood flow in the vessel dilated leading to either emergency repeat cardiac catheterization, repeat PTCA, immediate coronary bypass surgery, or myocardial infarction (as defined by the Minnesota code, and/or total CK elevation greater than three times the upper limit of normal values, with an elevated MB fraction). Instances of side branch closure, in which the ischemic syndrome resulted solely from compromised blood flow to a side branch, were excluded.

Patient selection and angiographic review of morphologic detail. By use of these definitions, 210 procedures (4.4%) resulted in acute closure. Patients with acute closure were randomly matched 1:2 with patients whose procedures did not lead to acute closure by date of angioplasty (to the nearest week). In each case, the site that closed was matched with only one site that did not close, whether or not multisite PTCA was performed. Cineangiograms were available for review for 163 procedures complicated by acute closure (77.6%) and for 327 procedures not complicated by acute closure (77.9%). The remaining cineangiograms were not available for review at the time this study was undertaken. Each procedural cineangiogram was then reviewed by an experienced angiographer unaware of clinical outcome. After review, 23 patients from the acute closure group were eliminated from the analysis (site of closure could not be determined = 8; side branch closure only = 7; quality of cineangiogram insufficient to allow analysis of morphology = 5; index dilatation performed for acute closure = 2; no pre-PTCA angiograms available = 1) and 16 patients were eliminated from the control group (quality of cineangiogram insufficient to allow analysis of morphology = 11; no pre-PTCA angiograms available = 3; index dilatation performed for acute closure = 2).

Cines were reviewed for 17 prospectively defined morphologic variables, and the lesion dilated and its location in the coronary tree was described. The definitions of these variables were based on those used in the recently reported Coronary Artery Surgery Study on morphology, and were as follows: Ablupt proximal face. The stenosis was judged to have an “abrupt” proximal face if in any projection its proximal face formed an angle with a contiguous proximal lumen of less than 135 degrees.

Active kink point. An active kink point was judged to be present if in any projection it appeared that any part of the lesion was located in a portion of the vessel that bent by more than 15 degrees between end-diastole and end-systole.

Branch point. A branch point was considered present if any part of the lesion was adjacent to a branch vessel of diameter 25% or more of the diameter of the nondiseased native vessel.

Bend point. A bend point was considered present if in any angiographic projection it appeared that the balloon, in position to dilate, was located in a portion of the vessel that had a 45 degree or greater angulation at end-diastole. This degree of angulation had to be noted when the vessel was not appreciably foreshortened by the angiographic projection. This is a slight modification of the previous definition. Patients with stenosis at a bend point were further divided into those who had a bend point that was fixed (angulation between end-diastole and end-systole =< 15 degrees) in any angiographic projection and those who did not. After the initial data analysis, the 120 patients were randomly selected from the cohort of 451 procedures and the diastolic angle at the site of dilatation was measured to the nearest degree with a hand-held protractor.

Calculations. Calculations were present if fixed radiopaque densities having the appearance of calcification were noted in the area of the stenosis to be dilated.

Collaterals. Collaterals were present if any degree of collateral filling beyond the site to be dilated was noted on the predilatation angiograms.

Diffuse disease. Diffuse disease was defined as three or more 50% narrowings in the vessel to be dilated or as luminal irregularities present in one-third of the vessel to be dilated.

Distal ectasia. Distal ectasia was judged to be present if there was dilatation of the vessel beyond the normal luminal diameter immediately distal to the stenosis to be dilated.

Eccentricity. Eccentricity was defined as a stenosis asymmetrically positioned in the vessel in any angiographic projection.
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FIGURE 1. Method of measuring lesion length in terms of normal luminal diameter instead of absolute millimeters. Length was visually estimated to the nearest 0.25 luminal diameter.

Healed dissection. Healed dissection was considered present if on the predilatation angiograms there was a curvilinear intraluminal filling defect or luminal widening without contrast staining at the site to be dilated.

Intimal tear or dissection. This was considered present after PTCA if there was a curvilinear intraluminal filling defect or widening with contrast staining at the site of dilatation.

Lesion length of greater than two luminal diameters. This variable was described in terms of length of the stenosis exceeding 30%, 50%, or 70% narrowing (three separate determinations) compared with the adjacent normal luminal diameter (figure 1). This was described both in terms of luminal diameters and visually, approximated to the nearest millimeter by use of a guiding catheter of known dimension as a reference marker (total of six determinations).

Multivessel disease. Multivessel disease was defined as luminal compromise of 50% or more diameter of more than one of three major epicardial coronary arteries, or a large branch thereof.

Other stenoses greater than 50%. These were considered present if other stenoses in the vessel to be dilated equaled or exceeded a 50% narrowing on visual inspection.

Roughened lumen. The stenosis was judged to have a roughened lumen if the luminal edge at the site of the dilatation was irregular or had a "sawtooth" component. If there was any question as to the nature of the lesion, it was judged to be smooth.

Thrombus. Thrombus was judged to be present if a discrete luminal filling defect or an area of contrast staining was noted in the area of stenosis to be dilated.

Ulceration. Ulceration was judged to be present if a discrete luminal widening of the area of the stenosis in the form of a "crate" was present. If the widening exceeded the diameter of the normal lumen, it was judged to be an area of ectasia, not an ulcer.

Reproducibility of morphologic assessment. Forty randomly selected cineangiograms were independently reviewed by one of two additional experienced angiographers for each of the 17 morphologic variables noted above.

Statistical analysis. Analyses were performed to test the hypothesis that morphologic variables such as bend or branch point stenoses or irregular luminal edges, which at the time of the formulation of the study had not been tested as risk factors for acute closure after PTCA, would be important determinants of acute closure and also to better define the risk factors for closure.

The risk for each variable was expressed as an odds ratio:

\[
\begin{align*}
\text{Probability of closure, variable present} & \quad \text{Probability of no closure, variable present} \\
\text{Probability of closure, variable absent} & \quad \text{Probability of no closure, variable absent}
\end{align*}
\]

Statistical analysis with the chi-square significance test was used to assess differences in categorical variables and those with the unpaired Student's t test were used to assess differences in continuous variables. Unpaired Student's t tests were required because the incomplete cineangiogram retrieval left some patients unmatched. Univariate and multiple stepwise logistic regression analyses were performed to determine clinical, angiographic, and procedural predictors of acute closure. The multivariate analysis evaluating predictors with univariate correlation p ≤ .10 was performed with the use of data from the 291 patients for whom complete data were available (the most common variable not available in all patients was post-PTCA gradient, which for technical reasons was not always recorded during the procedure), and also with the use of data from the 347 patients for whom complete data were available, excluding the post-PTCA gradient. All analyses were performed with BMDP software (BMDP Statistical Software, Los Angeles).

Results

Primary success and acute complications. Primary success in the 4772 patients, defined as a reduction in stenosis diameter to less than 50%, was 91%. Two hundred and ten patients (4.4%) had acute closure. Among 140 patients with cineangiograms that qualified for review, 74 (53%) patients developed closure in the catheterization laboratory and 66 patients (47%) had closure after they left the laboratory. The median time to closure was 10 min after completion of the procedure (range 0 min to 93 hr). Of these patients, 47 (34%) sustained a myocardial infarction without attempted revascularization, 15 (11%) underwent emergency PTCA without surgery, and 78 (55%) underwent bypass surgery. Of the patients whose closure was reopened by PTCA, six (40%) sustained a myocardial infarction. In the subset of patients requiring emergency bypass surgery, 60 (76%) had a Q wave or non-Q wave myocardial infarction. Overall, there were 56 Q wave and 57 non-Q wave infarctions. There were four deaths.

Clinical characteristics and predictors of acute closure.

The clinical characteristics and related univariate predictors of acute closure are listed in table 1. Women experienced closure significantly more often than men (odds ratio 1.66, p = .02). Age, history of diabetes, smoking, hyperlipidemia, unstable angina, and repeat PTCA did not influence the risk of acute closure, although there was a trend for those with hypertension to have a higher incidence of closure.

Angiographic characteristics and predictors of acute closure.

The angiographic characteristics and related univariate predictors of acute closure are listed in table 2. Three hundred sixty-six patients had single-vessel coronary artery disease and 85 patients had multivessel disease. Those with multivessel disease had acute closure somewhat more often on a per-site-dilated basis than those with single-vessel disease (p = .07).

There were 230 dilations in the left anterior descending (LAD) system (200 proximal, 30 mid/distal/
diagonal), 81 in the left circumflex (LCx) system (35 proximal, 46 mid/distal/obtuse marginal), and 140 in the right coronary artery (RCA) system (44 proximal, 96 mid/distal).

Although there was no significant difference in risk of closure between sites of dilatation, there was a trend toward higher closure rates with mid and distal LAD stenoses (odds ratio = 2.05), ostial LAD stenoses (odds ratio = 1.84), proximal LCx stenoses (odds ratio = 1.53), and proximal RCA stenoses (odds ratio = 1.45).

The most important angiographic univariate predictors of acute closure were: stenosis at a bend point (odds ratio = 2.03, p = .003), stenosis at a branch point (odds ratio = 1.87, p=.002), stenosis length (≥50% stenosis of ≥ two luminal diameters) (odds ratio = 1.80, p = .01), other stenoses of 50% or more in the vessel dilated (odds ratio = 2.05, p = .03), and eccentric stenosis (odds ratio = 1.68, p = .05) (figure 2). When the end-diastolic angle was measured to the nearest degree in 120 randomly selected patients, risk appeared to increase with increasing angle such that for angles of 0 to 30 degrees, the odds ratio was 0.51; for those of 31 to 45 degrees, the odds ratio was 0.89; and for those greater than 45 degrees, the odds ratio was 2.00 (p<.01). Stenoses located at end-diastolic bends of 45 degrees or more that also were active had a somewhat lesser likelihood of closure (odds ratio=1.56, p = .07 vs not active). One other morphologic variable, thrombus (odds ratio = 1.68, p = .09), approached statistical significance as a univariate predictor of closure. Pre-dilatation percent stenosis, the presence of collaterals, lesion calcification, abrupt proximal face, and absolute lesion length in millimeters by the 30% or more and 50% or more stenosis definitions and the other morphologic variables were not predictors of the acute closure (p≥.10).

Procedural predictors of acute closure. Four procedural variables, gradient after PTCA (measured before evidence of acute closure), the presence of an intimal tear or dissection after PTCA, prolonged heparin infusion after PTCA, and the measured percent stenosis after PTCA, were highly predictive of acute closure (table 3). The initial and maximal balloon inflation pressures and the translesional gradient measured before dilatation were not correlated with the risk of acute closure.

Preprocedural predictors of acute closure (multivariate analysis). The variables that would be available before attempted PTCA that demonstrated an independent effect on risk of closure are listed in table 4. In order of entry into the multivariate equation they were: lesion length, female gender, bend point location, branch point location, thrombus in situ, other stenoses of 50% or more in the vessel dilated, and multivessel disease. Only 6% of all acute closures occurred in the absence of the above-mentioned predictors.

Overall predictors of acute closure (multivariate analysis). When all factors, including those available only during or after the procedure, were analyzed, seven factors were found to be independent predictors of acute closure (table 5). Listed in order of entry into the regression equation, they were: post-PTCA percent stenosis (best point of dichotomization = 35%), intimal tear or dissection, post-PTCA heparin infusion, branch point stenosis, fixed bend point stenosis, and other stenoses of 50% or more in the vessel to be dilated. The presence of multivessel disease was also of some importance as an independent predictor of acute closure (p = .09). These seven factors remained the only independent variables associated with closure when data from patients in whom post-PTCA gradient was not available were allowed to be entered into the regression analysis.

Effect of presence of multiple preprocedural risk factors on risk of closure. The importance of the presence of multiple preprocedural risk factors is demonstrated by the finding that, in this study sample containing a 31% mix of patients with acute closure, the risk of closure on average was 14% in the presence of no risk factors, 26% with one risk factor, 35% with two risk factors, 58% with three risk factors, and 100% with four risk factors (chi-square = 32.6, p<.0001). Specific combinations of risk factors, such as lesion length and branch point stenosis (risk of

### TABLE 1

<table>
<thead>
<tr>
<th>Clinical characteristics and predictors of acute closure</th>
<th>% closure group (n=140)</th>
<th>% nonclosure group (n=311)</th>
<th>Odds ratio</th>
<th>Univariate value^a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female gender</td>
<td>32.8</td>
<td>22.1</td>
<td>1.66</td>
<td>.02</td>
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<tr>
<td>Hypertension</td>
<td>43.5</td>
<td>34.2</td>
<td>1.48</td>
<td>.06</td>
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<tr>
<td>Unstable angina</td>
<td>57.1</td>
<td>49.0</td>
<td>1.38</td>
<td>NS</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>20.1</td>
<td>15.1</td>
<td>1.41</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes</td>
<td>12.9</td>
<td>9.5</td>
<td>1.41</td>
<td>NS</td>
</tr>
<tr>
<td>Canadian Heart Assoc. angina class III–IV</td>
<td>57.0</td>
<td>53.3</td>
<td>1.17</td>
<td>NS</td>
</tr>
<tr>
<td>Smoking</td>
<td>52.2</td>
<td>49.5</td>
<td>1.11</td>
<td>NS</td>
</tr>
<tr>
<td>Age ≥60 years</td>
<td>15.7</td>
<td>19.0</td>
<td>0.87</td>
<td>NS</td>
</tr>
<tr>
<td>Repeat angioplasty (same site)</td>
<td>11.4</td>
<td>15.4</td>
<td>0.71</td>
<td>NS</td>
</tr>
</tbody>
</table>

^aOnly p values ≤.10 listed specifically.

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FIGURE 2. Pre- (A, B) and post-PTCA (C, D) angiograms from a 64-year-old woman with single-vessel LAD disease. The lesion is at a bend and branch point. Post-PTCA angiograms show an intimal tear in the left anterior oblique projection. Nine hours after PTCA, the patients developed severe pain and ST segment elevation requiring emergency return to the catheterization laboratory.

closure = 42%) appeared to convey higher risk than other combinations, but there were in general too few patients having many of the various possible combinations to accurately access the risk for each combination.

Interobserver variability and assessment of morphology of stenosis. Assessment of morphology of stenosis is subjective. Interobserver discordance between two highly trained angiographers for the five variables found to be risk factors for acute closure were as follows: lesion length (within one luminal diameter) = 22%; branch point location = 20%; bend point location = 12%; other stenosis of 50% or more in the vessel dilated = 12%; a lesion eccentricity = 24%.
### Discussion

Between 1977 and 1982, the NHLBI PTCA Registry collected data on the early angioplasty experience from 105 clinical centers. The majority of the attempts were performed with double-lumen catheters with a fixed guidewire at the tip, or moveable systems with stiff guidewires. 9, 12 The Registry reported major complications in 9.1% of attempts, and acute complications related to female gender, unstable angina pectoris, RCA dilatation, multivessel disease, and severe, nondiscrete, tubular, or eccentric lesions. 2 A more recent comprehensive review of complications found major complications in 4.1% and emphasized the importance of multivessel disease, lesion eccentricity, lesion calcification, and female gender. 1 In that analysis, RCA site and severity of stenosis were not found to be independent predictors of major complications.

Advances in catheter technology and increased operator experience have allowed a rapid growth in apparent indications and in the application of PTCA. In 1980, it was estimated that 1000 dilatation procedures were performed in this country. Since that time, flexible guidewires and low-profile catheter systems have been introduced, and dilatation equipment continues to be improved. 9, 12, 14 In 1985, approximately 100,000 dilatation procedures were performed.

This analysis was limited to procedures performed after April 1982, when steerable catheter systems became widely available at this institution. Between April 1982 and March 1986, 4772 elective, native vessel angioplasty procedures were performed with a 4.4% incidence of acute closure.

In view of these changes in operator experience and equipment, it is not surprising that risk factors for acute closure may be changing. In addition, there have been recent advances in methods of assessment of morphology of coronary stenosis. 5, 6 It is appropriate, therefore, to attempt to redefine the risk factors for acute closure.

Since acute closure after PTCA is a relatively uncommon event, the present study used a design in which patients with acute closure were randomly matched by date of PTCA on a 1:2 basis with patients without acute closure. Since by definition, the incidence of closure in the case-matched, study population was artificially elevated to 31%, the absolute risk associated with the presence of one or more of these morphologic factors cannot be easily extrapolated to the general population of PTCA candidates. The study, however, emphasizes the additive effect of these identified risk factors on acute closure associated with PTCA.

Multivariate analysis found seven independent preprocedural risk factors for acute closure: (1) lesion

<table>
<thead>
<tr>
<th>TABLE 3</th>
<th>Procedural predictors of acute closure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% closure</td>
</tr>
<tr>
<td>Intimal tear or dissection</td>
<td>72.8</td>
</tr>
<tr>
<td>Gradient ≥20 mm Hg after PTCA</td>
<td>37.1</td>
</tr>
<tr>
<td>Prolonged heparin infusion after PTCA</td>
<td>24.3</td>
</tr>
<tr>
<td>Stenosis after PTCA ≥35%</td>
<td>47.9</td>
</tr>
<tr>
<td>Gradient ≥50 mm Hg before PTCA</td>
<td>67.1</td>
</tr>
<tr>
<td>Initial inflation pressure</td>
<td>5.8 ± 2.0</td>
</tr>
<tr>
<td>Maximum inflation pressure</td>
<td>9.0 ± 1.7</td>
</tr>
</tbody>
</table>

*Mean values are ± SD.*
TABLE 4
Independent procedural predictors of acute closure

<table>
<thead>
<tr>
<th>Variable (descending order)</th>
<th>Coefficient</th>
<th>p value to add</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lesion length ≥2 luminal diameters</td>
<td>-.36</td>
<td>.004</td>
</tr>
<tr>
<td>Female gender</td>
<td>-.46</td>
<td>.003</td>
</tr>
<tr>
<td>Bend point ≥45 degrees</td>
<td>-.40</td>
<td>.01</td>
</tr>
<tr>
<td>Branch point</td>
<td>-.41</td>
<td>.01</td>
</tr>
<tr>
<td>Thrombus</td>
<td>-.165</td>
<td>.003</td>
</tr>
<tr>
<td>Other stenoses ≥50% in same vessel</td>
<td>-.22</td>
<td>.03</td>
</tr>
<tr>
<td>Multivessel disease</td>
<td>-.28</td>
<td>.09</td>
</tr>
</tbody>
</table>

Constant = -.637.

length, (2) female gender, (3) bend point location, (4) branch point location, (5) thrombus, (6) other stenoses of 50% or more in the vessel dilated, and (7) multivessel disease (table 4). Three of the four most important predictors were not described in the NHLBI Registry and only lesion length and thrombus have been emphasized in other large series of patients undergoing PTCA.1, 4, 18

Meier et al.19 previously noted an increase in complications associated with angioplasty of long and eccentric lesions. Although closure is occasionally caused by thrombosis20 or spasm,21 it is usually caused by coronary dissection.1, 14, 20–22 In experimental preparations, dissections have been found most often in areas containing thick and multiple atherosclerotic plaques.23 It should not be surprising that acute closure may be related to lesion length, which appears to correlate with the amount of atherosclerotic material at the site of balloon inflation.

The importance of the location of stenosis at a bend or branch point in the genesis of acute closure has not been previously emphasized in other large series. The mechanism of vessel angulation is perhaps more easily understood, since the high-pressure balloon must necessarily tear an atherosclerotic fixed and rigid bend lesion as it straightens and opens it. The risk of acute closure is related to the degree of angulation at the stenosis, with a much increased risk as the end-diastolic angulation exceeds 45 degrees. The mechanisms of closure at branch and also at bend points may also be due to what in materials science is termed stress concentration, wherein the maximal stress is several times greater when there is a geometric discontinuity in the object to which this stress is applied.24

Female gender has previously been noted as a risk factor for closure,1, 2 but it has been suggested that 3.0 mm balloons, commonly used in the early PTCA experience, might have been too large for women and hence the cause of the increased complication rate.25 The continued increased occurrence of complications in women during this time period when more appropriately sized balloons were used suggests a special propensity toward acute closure in women, but the influence of balloon-artery size mismatch was not addressed in the present study.

Sugrue et al.4 have suggested that the presence of an angiographically visible thrombus at the dilatation site increases the risk of acute closure. In this larger series, thrombus was again found to be an independent predictor of acute closure, despite the frequent use of intracoronary streptokinase when a filling defect was noted angiographically, which was not done in the Mayo Clinic series.4 In fact, for the small number of procedures in which a 2 mm or larger filling defect was observed before PTCA, acute closure occurred with an odds ratio of 3.86. The presence of a thrombus in situ may likely lead to clot propagation and/or the release of vasoactive substances,26 both of which might cause acute closure.

The presence of other stenoses in the vessel dilated was also a major independent predictor of closure and it has not been previously emphasized.1, 2 Other stenoses may reflect diffuse atherosclerotic involvement of the vessel dilated, and like lesion length, correlate with the amount of atheroma present and hence risk of closure. Interestingly, however, the presence of diffuse intimal irregularities, as determined angiographically, did not correlate with the risk of acute closure.

It appears then that the risk profile for acute complications after elective coronary angioplasty is changing. Several factors found to be important in the NHLBI Registry: lesion severity, lesion eccentricity, RCA location, and the presence of unstable angina, seem no longer to be major factors that might enable one to predict the likelihood of acute closure.

Initial morphologic indications for PTCA included proximal, “discrete,” concentric, and noncalcified stenoses. This study does not address the question of primary angiographic success, but in considering the risks of ischemic complications, these indications need redefinition. Only the relative requirement for

TABLE 5
Independent predictors of acute closure

<table>
<thead>
<tr>
<th></th>
<th>Coefficient</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-PTCA percent stenosis</td>
<td>-.597e-01</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Intimal tear or dissection</td>
<td>-.909</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Post-PTCA heparin infusion</td>
<td>-.738</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Branch point stenosis</td>
<td>-.514</td>
<td>.002</td>
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<tr>
<td>Fixed bend point</td>
<td>-.702</td>
<td>.018</td>
</tr>
<tr>
<td>Other stenoses ≥50% in the same vessel</td>
<td>-.883</td>
<td>.030</td>
</tr>
</tbody>
</table>

Constant = 3.39.
discrete stenoses appears to be relevant to current practice.

Four procedural variables, post-PTCA percent stenosis, the presence of an intimal tear or dissection, use of a prolonged heparin infusion after PTCA, and post-PTCA gradient, were found to be powerful predictors of closure, but all are inherently related to the event itself and cannot be used to predict complications before the procedure is undertaken.

Finally, in considering a patient for angioplasty, one would like to be able to estimate the likelihood of acute closure, and hence the risk of potential emergency bypass surgery or myocardial infarction. In this analysis, reflecting our experience in over 4700 elective coronary angioplasties since 1982, we were able to define seven major independent factors whose presence beforehand increased the likelihood of closure. In the absence of any of these risk factors, the risk of closure is very small, while if three or more factors are present, the risk of closure may be such that alternative forms of revascularization need seriously be considered, especially if the vessel to be dilated serves a large amount of myocardium or if multiple such lesions are in need of dilatation. Identification of these factors and their associated risk should improve patient selection and better define the role of PTCA in the management of patients with coronary artery disease.

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