JACC Vol. 12, No. 2
August 1988:324–33

# Coronary Angioplasty for Unstable Angina: Immediate and Late Results in 200 Consecutive Patients With Identification of Risk Factors for Unfavorable Early and Late Outcome

PIM J. DE FEYTER, MD, HARRY SURYAPRANATA, MD, PATRICK W. SERRUYS, MD, FACC, KEVIN BEATT, MRCP, RON VAN DOMBURG, MSc, MARCEL VAN DEN BRAND, MD, JAN J. TIJSSEN, PhD, AIDA J. AZAR, MPH, PAUL G. HUGENHOLTZ, MD, FACC

*Rotterdam, The Netherlands*

**Two hundred patients (mean age 56 years, range 36 to 74) with unstable angina (chest pain at rest, associated with ST-T changes) underwent coronary angioplasty. In 65 patients with multivessel disease, only the "culprit" lesion was dilated. The initial success rate was 89.5% (179 of 200 patients). At least one major procedure-related complication occurred in 21 patients (10.5%): (death in 1, myocardial infarction in 16 and urgent surgery in 18).**

**All patients were followed up for 2 years. Five patients died late; 8 had a late nonfatal myocardial infarction and 52 had recurrence of angina pectoris. The restenosis rate was 32% (51 of 158) in the patients with initial successful angioplasty who had repeat angiography. At the 2 year follow-up, after attempted coronary angioplasty in all 200 patients, the total incidence rate of death was 3% (one procedure related; five late deaths), of nonfatal myocardial infarction 12% (16 procedure related and 8 late after angioplasty), and 13% (26 patients) were still symptomatic although they had improved in functional class.**

**Multivariate analysis showed that variables indicating an increased risk 1) for major procedure-related complications were: ST segment elevation, persistent negative T wave and stenosis ≥65% (odds ratio 3.7, 3.7 and 3.3, respectively); 2) for angiographic restenosis were: presence of collateral vessels, ST segment depression, multivessel disease, left anterior descending coronary artery stenosis and history of recent onset of symptoms (odds ratio: 2.2, 2.0, 1.9, 1.9 and 0.54, respectively); and 3) for late coronary events (recurrence of angina, late myocardial infarction or late death) were: multivessel disease, total occluded vessel and ST segment elevation (odds ratio 3.7, 2.8 and 0.44, respectively).**

**Thus, coronary angioplasty for unstable angina can be performed with a high initial success rate, but at an increased risk of major complications. The prognosis is favorable after initial successful coronary angioplasty.**

***(J Am Coll Cardiol 1988;12:324–33)***

The general term "unstable angina" is used to encompass patients who present with a wide variety of symptoms, electrocardiographic (ECG) changes, coronary anatomy and left ventricular function (1–3). This explains the wide divergence in prognosis reported by several studies (2,3). A subgroup of patients with unstable angina who have chest pain at rest associated with ECG changes have a poor short- and long-term prognosis (4–7). These patients must be considered as a different subgroup from patients with new onset or progressive angina, who have a prognosis that is only slightly worse than that of patients with chronic stable angina pectoris (8–10). Management of unstable angina has evolved progressively and, recently, coronary angioplasty has been shown to be a relatively safe and effective treatment for unstable angina (11–20).

In this study, we describe the immediate and 2 year follow-up results of coronary angioplasty in 200 consecutive patients with chest pain at rest associated with ECG changes. Furthermore, clinical, electrocardiographic, angiographic and angioplasty-related variables were analyzed to identify predictors for 1) major complications during attempted coronary angioplasty, or 2) restenosis, recurrence of angina, late myocardial infarction or late death after initial successful coronary angioplasty.

From the Thoraxcenter, University Hospital, Erasmus University, Rotterdam, The Netherlands.

Manuscript received October 5, 1987; revised manuscript February 29, 1988, accepted March 9, 1988.

Address for reprints: Pim J. de Feyter, MD, Catheterization Laboratory, Thoraxcenter, Bd 414, P.O. Box 1738, 3000 DR Rotterdam, The Netherlands.



0735-1097/88/$3.50

## Methods

**Study patients.** During the period from January 1983 to January 1985, a total of 2,887 patients were admitted to our coronary care unit. Of these, 442 patients were considered to have unstable angina pectoris, defined as chest pain at rest lasting for at least 15 min associated with documented electrocardiographic (ECG) ST-T segment changes and no subsequent signs of cardiac necrosis (cardiac enzyme increase less than twice normal and no development of an abnormal Q wave [≥0.03 ms]). Patients were treated with a combination of nitrates, beta-adrenergic blockers, calcium channel antagonists and heparin. All underwent coronary angiography. Patients with refractory unstable angina underwent either emergency coronary angioplasty or coronary bypass surgery. Elective angioplasty or surgery was performed in stabilized patients if they had persisting exertional angina detected clinically or with exercise testing.

Patients were selected for surgery if they had multivessel coronary disease with one or more critical stenoses supplying a large area of viable myocardium in addition to the ischemia-related vessel or if they had left main stem coronary disease. Selected for coronary angioplasty were patients with single or multivessel disease in whom the culprit lesion was technically suitable for angioplasty (21).

*The culprit lesion in patients with multivessel disease was identified* by the localization of ST-T segment changes during chest pain. Electrocardiographic changes in leads I and aVL and $V_1$ to $V_6$ were related to lesions of the left anterior descending coronary artery and to changes in leads II, III and aVF with either the right or the left circumflex coronary artery. Furthermore, certain angiographic characteristics (such as severity and morphologic features of the lesion, presence of an intracoronary thrombus and degree of anterograde filling) served as an aid in the detection of the culprit lesion.

Two hundred patients (164 men, 36 women) with a mean age of 56 years (range 36 to 74) underwent coronary angioplasty and comprise the study group. Single vessel coronary disease was present in 135 patients, and multivessel disease in 65. The mean global left ventricular ejection fraction was 0.59 ± 0.10. The extent of coronary artery disease and the details of the actual management of these patients are shown in Table 1. The characteristics of the undilated segments of the 65 patients with multivessel disease and balloon dilation of only the culprit lesion are shown in Table 2.

*Patients were categorized into three groups according to their history*: 1) recent onset unstable angina, defined as chest pain for the first time within 1 month before coronary angioplasty; 2) worsening angina, defined as chronic stable angina that had progressed to chest pain at rest, and 3) early postinfarction unstable angina, defined as occurrence of a myocardial infarction within 1 month before coronary angioplasty.

**Table 1.** Extent of Coronary Artery Disease and the Details of Management of 442 Patients With Unstable Angina*

| Unstable Angina | No. of Patients | Extent of Coronary Artery Disease | | | | |
|---|---|---|---|---|---|---|
| | | 0V | 1V | 2V | 3V | LM |
| Refractory to pharmacologic treatment | | | | | | |
| Emergency PTCA | 114 | 0 | 78 | 24 | 12 | 0 |
| Emergency CABG | 87 | 0 | 3 | 16 | 45 | 23 |
| Initially stabilized with pharmacologic treatment | | | | | | |
| Elective PTCA | 86 | 0 | 57 | 19 | 9 | 1 |
| Elective CABG | 73 | 0 | 4 | 13 | 49 | 7 |
| Pharmacologic treatment | 82 | 14 | 11 | 24 | 25 | 8 |
| Total no. of patients | 442 | 14 | 153 | 96 | 140 | 39 |

*The total number patients admitted to the coronary care unit from January 1, 1983 to July 1, 1985 was 2,887. CABG = coronary artery bypass grafting; LM = left main coronary artery; PTCA = percutaneous transluminal coronary angioplasty. 0, 1, 2, 3V = no, one, two, three vessel disease, respectively.

*The documented ECG ST-T segment changes associated with chest pain at rest were classified, in ranking order of severity, as follows*: 1) transient ST segment elevation (≥0.1 mV) during pain, with return to (nearly) normal or to the ST level that existed before the onset of chest pain at rest; 2) transient ST segment depression (≥0.1 mV) during pain, with return to (nearly) normal or to the level that existed before the onset of chest pain at rest; 3) development of permanent negative T waves (≥0.1 mV) during or after disappearance of chest pain at rest, without documented ST segment elevation or depression (≥0.1 mV); and 4) transient minimal ST-T changes: ST elevation or depression (1 mV), minimal T wave inversion (<0.1 mV), pseudonormalization of a negative T wave and T wave amplitude increase or decrease during pain.

**Coronary angiography.** Coronary angiograms were obtained in multiple views, including hemiaxial projections. A consensus of two angiographers was used to evaluate the

**Table 2.** Characteristics of Nondilated Coronary Stenosis in 65 Patients With Multivessel Disease and Angioplasty of the Culprit Lesion Only

| | Undilated Lesions per Patient | |
|---|---|---|
| | 1 Lesion | 2 Lesions |
| No. of patients | 44 | 21 |
| Technically suitable for CABG | 41 | 36 |
| Technically suitable for PTCA | 22 | 18 |
| Nondilated LAD | 6 | 2 |
| Nondilated RCA or LCx | 38 | 40 |
| Stenosis to infarcted myocardium | 15 | 6 |

CABG = coronary artery bypass grafting; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; RCA = right coronary artery; other abbreviations as in Table 1.

JACC Vol. 12, No. 2
August 1988:324–33

coronary angiograms. The degree of coronary obstruction was assessed with the use of a caliper system and was given in percent of luminal diameter. The length of the lesion was measured in relation to the length of the inserted balloon. A lesion was considered long if it was >1 cm. Lesions were categorized into concentric lesions (symmetric, hourglass coronary narrowing) and eccentric lesions (asymmetric narrowing). Collateral vessels were considered present when they were angiographically visible. For this study, only the presence of collateral circulation to the ischemia-related vessel was noted. An intracoronary thrombus was defined as 1) contrast medium staining at the site of an abrupt occlusion of the vessel, or 2) the presence of an intracoronary filling defect (22).

**Coronary angioplasty.** This was performed with a steerable dilation system. A 7F pacing electrode catheter was positioned in the right atrium. At the beginning of the procedure, heparin (100 mg) and aspirin (250 mg) were administered intravenously and low molecular weight dextran was infused slowly. The electrocardiogram and blood pressure were continuously monitored. To prevent coronary spasm, intracoronary nifedipine or isosorbide dinitrate was given (23). Initial balloon inflation pressure was 2.0 atm, with subsequent inflations ranging to 12 atm. Inflation was maintained according to the ECG changes, degree of decrease in blood pressure or induced pain; it never lasted >60 s. Balloon inflations were repeated until there was a significant reduction in the transstenotic pressure gradient and a reduction in the severity of the obstruction as judged from repeat angiograms obtained immediately after the dilation. Coronary angioplasty was considered to be successful if a reduction in the severity of the obstruction to <50% luminal diameter narrowing was obtained or if the transstenotic gradient index was reduced to <0.30 (according to the equation mean proximal pressure minus mean distal pressure divided by mean aortic pressure). Furthermore, it was required that acute ischemic symptoms be completely relieved and that no progression to myocardial infarction or death had occurred (24). All procedures were carried out with a cardiac surgical team on standby.

*Coronary intimal dissection was defined* according to the National Heart, Lung, and Blood Institute Percutaneous Transluminal Coronary Angioplasty Registry (25) by 1) the presence of angiographically evident intimal damage, producing an intraluminal filling defect; 2) extraluminal extravasation of contrast material; and 3) linear luminal density or luminal staining.

*The diagnosis of a (peri)procedural myocardial infarction was determined* by the development of a new pathologic Q wave (≥0.03 ms) or typical increase in serum cardiac enzymes (more than twice normal level). After bypass surgery, a myocardial infarction was determined by development of new Q waves.

*After the procedure*, all patients were monitored for 24 h in the coronary care unit, where the ECG and cardiac enzyme levels were monitored. They were discharged after 2 to 3 days, and treatment with nifedipine (40 to 60 mg daily) and aspirin (500 mg daily) was maintained for 6 months.

**Follow-up.** Clinical follow-up information was obtained by personal interview, data received from the referring physician or questionnaire. Patients were evaluated for the occurrence of death or myocardial infarction or recurrence of angina pectoris. The majority of the patients underwent exercise testing with thallium-201 scintigraphy and repeat coronary angiography.

Patients performed symptom-limited exercise on a bicycle ergometer with stepwise increments of 10 watts/min. The three orthogonal XYZ leads of the Frank lead system were recorded. An ischemic response was defined as ≥0.1 mV ST segment depression 0.08 s after the J point. The maximal work load achieved was expressed as a percent of the normal work load predicted for age, sex and height. Thallium-201 exertional scintigraphic imaging was performed in the anterior left anterior oblique 45° and 65° views, immediately after injection of 1.5 mCi of thallium-201 at peak stress, and postexercise images were obtained 4 h later. Images were obtained using a Searle Phogamma V camera and processed with computer interface as described previously (26). A defect with redistribution was considered to represent exercise-induced ischemia; a persistent defect without redistribution was considered to represent a scar.

*Repeat coronary angiograms* were obtained in multiple views, including hemiaxial views for the left coronary artery. Restenosis was defined as an increase in the luminal diameter stenosis of the dilated lesion of >50%.

**Data analysis.** The following four (ontoward) outcome events were defined: 1) major procedure-related complications (death, myocardial infarction, or urgent bypass surgery); 2) procedure-related death or myocardial infarction; 3) late coronary event (recurrence of angina pectoris, myocardial infarction or death within 1 year); and 4) restenosis (≥50% narrowing of the dilated artery at repeat angiography). The "late coronary event" outcome was defined only for patients with initial successful angioplasty. "Restenosis" was defined only for patients with initial successful angioplasty who subsequently underwent repeat angiography.

We used a composite logistic prediction function to determine which pretreatment characteristics were independently related to the risk for each of the described untoward outcome events. Of the clinical characteristics, we considered gender, age, history of angina, presence of previous myocardial infarction, and the presence of refractory versus stabilized symptoms. The ECG data included the presence of ST segment displacement of ≥0.1 mV, T wave inversion of ≥0.1 mV and minor ST segment displacement or T wave inversion. The angiographic data included site and severity of the lesion, length and eccentricity of the lesion, the presence of multivessel disease, the extensiveness of collat-

**Table 3.** Initial and 1 Year Results of Coronary Angioplasty for Unstable Angina in Different Subsets of 200 Patients

| | | | | | | | | Cumulative Incidence of Coronary Events After Successful Angioplasty | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Major Complication | | | Success Rate | | Death | | MI | | AP | |
| | No. of Patients | Total | Death | MI | Acute Surgery | % | (n) | At 6 Months | At 1 Year | At 6 Months | At 1 Year | At 6 Months | At 1 Year |
| All patients | 200 | 21 | 1 | 16 | 18 | 90 | (179) | 2 | 2 | 3 | 5 | 37 | 47 |
| AP status | | | | | | | | | | | | | |
| Refractory | 114 | 11 | 1 | 8 | 9 | 90 | (103) | 1 | 1 | 2 | 4 | 20 | 24 |
| Stabilized | 86 | 10 | 0 | 8 | 9 | 88 | (76) | 1 | 1 | 1 | 1 | 17 | 23 |
| Recent onset AP | 85 | 10 | 0 | 7 | 9 | 88 | (75) | 0 | 0 | 0 | 0 | 10 | 16 |
| Changing AP pattern | 81 | 9 | 1 | 7 | 7 | 89 | (72) | 2 | 2 | 2 | 4 | 17 | 20 |
| Post MI AP | 34 | 2 | 0 | 2 | 2 | 94 | (32) | 0 | 0 | 1 | 1 | 10 | 11 |
| ECG findings | | | | | | | | | | | | | |
| ST elevation | 58 | 8 | 0 | 7 | 6 | 86 | (50) | 0 | 0 | 1 | 1 | 9 | 9 |
| ST depression | 33 | 2 | 1 | 1 | 1 | 94 | (31) | 0 | 0 | 1 | 2 | 7 | 8 |
| Neg T wave | 55 | 8 | 0 | 6 | 8 | 85 | (47) | 0 | 0 | 1 | 1 | 11 | 14 |
| Minor ST-T | 54 | 3 | 0 | 2 | 3 | 94 | (51) | 2 | 2 | 0 | 1 | 10 | 16 |
| CAD | | | | | | | | | | | | | |
| Single vessel | 135 | 15 | 0 | 11 | 12 | 89 | (120) | 0 | 0 | 2 | 3 | 17 | 24 |
| Multivessel | 65 | 6 | 1 | 5 | 6 | 91 | (59) | 2 | 2 | 1 | 2 | 20 | 23 |
| LAD or LM | 135 | 12 | 0 | 9 | 10 | 91 | (123) | 2 | 2 | 1 | 2 | 27 | 34 |
| RCA or LCx | 65 | 9 | 1 | 7 | 8 | 86 | (56) | 0 | 0 | 2 | 3 | 10 | 13 |

AP = angina pectoris; CAD = coronary artery disease; MI = myocardial infarction; Neg = negative. Other abbreviations as before.

eral flow, the presence of an intracoronary thrombus and the global ejection fraction. Procedural characteristics were also involved in the construction of the risk model for "late coronary event" and in that for "restenosis"; these included duration of balloon inflation, maximal inflation pressure, the occurrence of a dissection and the postdilation transstenotic pressure gradient.

*In univariate analysis*, the relation between predictors (for example, gender) and outcome was expressed as a relative risk (or risk ratio) (that is, as the ratio of the rate of the respective outcome event observed in patients belonging to one category relative to that observed in the other category). For instance, the risk associated with gender is the rate of the outcome event in men divided by that in women. Continuous variables were dichotomized. The 95% confidence limits of the relative risk estimates are also given (27). If the 95% confidence interval does not contain the value 1, the association of the predictor and outcome is statistically significant at the 0.05 level.

*The objective of multivariate logistics analysis* was to find the combination of characteristics that predicted (an unfavorable) outcome as accurately as possible. As a general principle, indicator variables were used. These are variables that assume the value of 1 if the property considered is present and 0 if absent. The BMDP package was used, which selects stepwise predictor variables based on the maximal likelihood ratio. This provides a measure of significance and has an asymptotic chi-square distribution. Thus, variables were included into the model if they led to a substantial improvement of the log-likelihood ($p < 0.10$) or if their removal led to a substantially significant decrease ($p < 0.15$). Forward selection and backward elimination of the variables into the model yielded the same models.

*The regression coefficients have a direct epidemiologic implication.* Each coefficient represents the log odds of unfavorable outcome controlling for the other variables in the model. Its antilogarithm is the relative risk for the property considered. As an example, if the regression coefficient for ST elevation is 1, its antilogarithm ($e^1$) is 2.7. This means that the risk of unfavorable outcome within 1 year for patients with ST elevation is 2.7 times as high as that for patients who had no ST elevation. In fact, the coefficients concern relative odds, which are a good approximation for the relative risk because the frequency of unfavorable outcome is relatively low.

## Results

**Initial success rate and major procedure-related complications.** The overall initial success rate was 89.5% (179 of 200 patients). A major complication (death, myocardial infarction or urgent surgery) occurred in 10.5% (21 of 200 patients). The initial success rate and major complication rate for the relevant clinical subsets are listed in Table 3. The results of univariate analysis are presented in Table 4. The majority of the analyzed variables were insignificant. The significant risk factors using multivariate analysis of variables predictive for an increased risk of a major complication

JACC Vol. 12, No. 2
August 1988:324–33

**Table 4.** Univariate Analysis of Variables to Predict Immediate and Late Unfavorable Outcome After Coronary Angioplasty

| Unfavorable Outcome | Variable* | n | Risk Ratio | 95% Confidence Interval |
|---|---|---|---|---|
| Procedural major complication (death, MI, acute surgery) | Stenosis 65% | 110 | 2.6 | 1.0 to 6.7 |
| Procedural death or MI | None | | | |
| Coronary events at 1 year (death, MI, recurrent angina) | Worsening angina | 73 | 1.4 | 0.9 to 2.2 |
| | Previous MI | 67 | 1.5 | 1.0 to 2.4 |
| | ST elevation | 53 | 0.6 | 0.3 to 1.0 |
| | ST depression | 30 | 1.4 | 0.8 to 2.2 |
| | Stenosis ≤65% | 93 | 1.4 | 0.9 to 2.3 |
| | Total occlusion | 16 | 1.9 | 1.0 to 3.0 |
| | Multivessel CAD | 58 | 2.3 | 1.5 to 3.7 |
| | Collateral vessels | 30 | 1.5 | 0.9 to 2.5 |
| Angiographic restenosis | ST depression | 28 | 1.5 | 0.9 to 2.3 |
| | Total occlusion | 14 | 1.7 | 0.9 to 2.7 |
| | Multivessel CAD | 51 | 1.6 | 1.0 to 2.5 |
| | Collateral vessels | 29 | 1.6 | 1.0 to 2.4 |
| | Dissection | 40 | 1.4 | 0.9 to 2.1 |

*Only the significant variables are tabulated. Abbreviations as in Tables 1 and 2.

and procedure-related myocardial infarction are shown in Table 5.

**Clinical follow-up and angiographic stenosis.** The data from clinical follow-up after successful and unsuccessful coronary angioplasty are shown in Table 6. The majority of coronary events occurred within 6 months after the procedure. The total need for bypass surgery (acute and elective) was 14% at 1 year and 15% at 2 years (Table 7). The total incidence of myocardial infarction (either procedure-related or late, or both) was 11% at 1 year and 12% at 2 years. The total incidence of death was 2% at 1 year and 3% at 2 years. At 1 year follow-up, 85% of all the patients were in New York Heart Association functional class 1, 11% were in class 2 and 2% were in class 3 (Table 7).

*Exercise testing and thallium-201 scintigraphy were performed after successful angioplasty* in 157 and 146 patients, respectively, 1.7 ± 3.8 months after the procedure (Fig. 1). Twenty-two patients did not perform an exercise test because of a physical handicap in 5, death in 2, refusal in 1 and an unknown cause in 14. The majority of the patients achieved a work load of >80% of the predicted value and experienced no angina during the test. A reversible thallium-201 perfusion defect could be induced in 29% of the patients.

*A repeat coronary angiogram* was performed in 158 patients 5.1 ± 4.8 months after successful angioplasty. Recatheterization was not performed in 21 patients because of death in 2, late myocardial infarction in 1, relative contraindication in 3 and refusal in 15. Of the latter group, 4 patients had recurrent angina. Angiographic restenosis was present in 32% (51 of 158 patients). Nine patients (18%) with restenosis did not experience angina. The results of univariate analysis are presented in Table 4. The majority of the analyzed variables were insignificant.

*The significant risk factors using multivariate analysis of variables* predictive for late coronary events or angiographic restenosis after a successful procedure are shown in Table 5. Multivessel disease was shown to be a rather strong predictor for late coronary events. This was mainly a result of the persistence of undilated lesions.

**Dilation of only the culprit lesion in multivessel disease.** The initial success rate and major complication rate were comparable in patients with single vessel disease and dilation of that particular lesion (89 and 11%, respectively) and patients with multivessel disease and dilation of the culprit lesion only (91 and 9%, respectively). The angiographic restenosis rate was comparable in patients with culprit lesion dilation and multivessel disease with that in patients with single vessel disease (18 [35%] of 51 versus 33 [31%] of 107).

*Persistent or recurrent angina* was significantly more frequent in patients with incomplete revascularization than in those with complete revascularization at 6 months and 1 year (17 [13%] of 135 versus 20 [31%] of 65 at 6 months and 24 [18%] of 135 versus 23 [35%] of 65 at 1 year) (Tables 3 and 6). In patients with dilation of the culprit lesion only and multivessel disease, the maximal work capacity achieved during stress testing was less, while the occurrence of exercise-induced ischemic ST segment depression and reversible perfusion defect was more frequent than in patients with single vessel disease and dilation of that particular lesion (Fig. 2).

**Table 5.** Risk Factors to Predict Procedure-Related Major Complications, Restenosis and Coronary Events at 1 Year After Angioplasty

| Risk Factor | Coefficient | SE | OR | 95% CI |
|---|---|---|---|---|
| A. Procedure-Related Major Complication (death, MI and urgent surgery) | | | | |
| ST elevation | 1.3 | 0.63 | 3.7 | 1.1 to 12.6 |
| Persistent neg T wave | 1.3 | 0.63 | 3.7 | 1.0 to 13.1 |
| % Stenosis ≥65% | 1.2 | 0.54 | 3.3 | 0.93 to 6.9 |
| Constant | −3.7 | 0.67 | — | — |
| B. Procedure-Related Death or Myocardial Infarction | | | | |
| ST elevation | 1.2 | 0.71 | 3.3 | 0.84 to 13.4 |
| Persistent neg T wave | 1.2 | 0.73 | 3.3 | 0.84 to 13.4 |
| Constant | −3.3 | 0.59 | — | — |
| C. Angiographic Restenosis | | | | |
| Collateral vessels | 0.77 | 0.44 | 2.2 | 0.92 to 5.1 |
| ST depression | 0.68 | 0.45 | 2.0 | 0.82 to 4.8 |
| Multivessel disease | 0.66 | 0.37 | 1.9 | 0.94 to 4.0 |
| LAD stenosis | 0.64 | 0.41 | 1.9 | 0.86 to 4.2 |
| Worsening AP or post-MI AP | +0.62 | 0.38 | 1.6 | 1.3 to 5.7 |
| Constant | −2.0 | 0.49 | — | — |
| D. Coronary Events at 1 Year (recurrent AP, MI and late death) | | | | |
| Multivessel disease | 1.3 | 0.36 | 3.7 | 1.8 to 7.5 |
| Total occlusion | 1.0 | 0.56 | 2.8 | 0.94 to 8.4 |
| ST elevation | −0.82 | 0.42 | 0.44 | 0.2 to 1.0 |
| Constant | −1.3 | 0.26 | — | — |

Abbreviations as in Tables 1 to 4.

## Discussion

**Immediate results of coronary angioplasty for unstable angina.** Patients described as having unstable angina as originally defined by Conti et al. (1) can be divided into three subgroups: 1) recent onset angina; 2) progressive effort angina with a deteriorating clinical pattern superimposed on chronic stable angina; and 3) episodes of prolonged ischemic pain at rest. Patients belonging to subgroups 1 and 2 have a reasonably good prognosis, with most patients having a satisfactorily initial response to medical therapy (8–10). These patients can usually be evaluated for myocardial revascularization on an elective basis. In this study, we reserved the term "unstable angina" for patients in subgroup 3 (that is, patients with prolonged episodes of chest pain at rest). The prognosis for this subgroup is worse, especially if ischemic symptoms persist (4–7). We believe that immediate control of ischemia with stepwise intensification of pharmacologic treatment is the cornerstone in managing these patients. If, despite this tailored pharmacologic approach, patients continue to have attacks of ischemia, prolongation of what must be regarded as ineffective treatment should be avoided and an attempt at myocardial revascularization, either acute coronary bypass surgery or

**Table 6.** Clincial Follow-Up After Attempted Coronary Angioplasty in 200 Patients With Unstable Angina

| Follow-Up | <6 Months | 6 to 12 Months | 12 to 24 Months |
|---|---|---|---|
| Successful PTCA (n = 179) | | | |
| Late death | 2 | 0 | 2 |
| New myocardial infarction | 3 | 2 | 2 |
| Recurrent angina | 37 (20) | 10 (3) | 3 (1) |
| Repeat PTCA | 18 (11) (3*) | 3 | 2 (1) (1*) |
| Bypass surgery | 8 (5) | 3 (1) | 1 (1*) |
| Pharmacologic therapy | 11 (4) | 7 (2) | 0 |
| Unsuccessful PTCA (n = 21) | | | |
| Late death | 0 | 1 | 0 |
| New myocardial infarction | 0 | 1 | 0 |
| Recurrent angina pectoris | 2 | 0 | 0 |

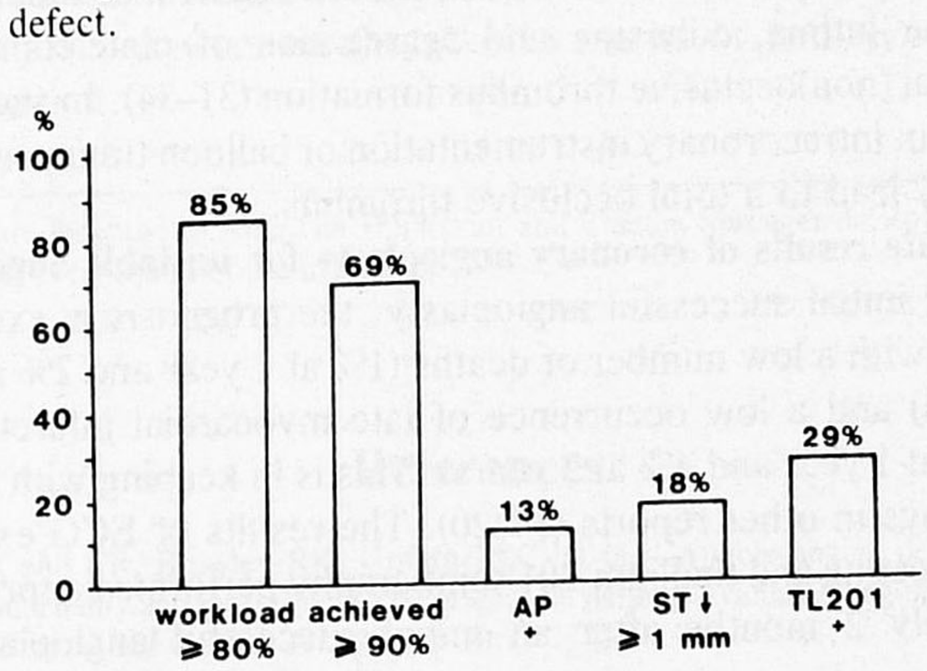
*New stenosis at nondilated site; ( ) = patients with multivessel disease; other abbreviations as before.

**Table 7.** Cumulative Incidence of Major Coronary Events (death, myocardial infarction), Bypass Surgery, Repeat Angioplasty and Functional Class at Initial Attempt and at 6, 12, and 24 Months Follow-Up

| | Initial Attempt | 6 Months | 12 Months | 24 Months |
|---|---|---|---|---|
| No. | 200 | 199 | 197 | 196 |
| Death | 1 | 3 | 4 | 6 |
| MI | 16 | 19 | 22 | 24 |
| CABG | 18 | 26 | 29 | 30 |
| Repeat PTCA | — | 18 | 21 | 23 |
| Functional class* | | | | |
| I | 0 | 178 | 170 | 168 |
| II | 0 | 17 | 22 | 22 |
| III | 86 | 2 | 4 | 4 |
| IV | 114 | 0 | 0 | 0 |

*New York Heart Association. Abbreviations as before.

**Figure 1.** Exercise thallium-201 scintigraphy after successful coronary angioplasty in patients with unstable angina. Work load achieved: ≥80 or ≥90% predicted for age, gender and height. AP+ = exercise-induced angina pectoris; ST↓ = exercise-induced ischemic ST segment depression; TL201+ = reversible thallium perfusion defect.



JACC Vol. 12, No. 2
August 1988:324–33

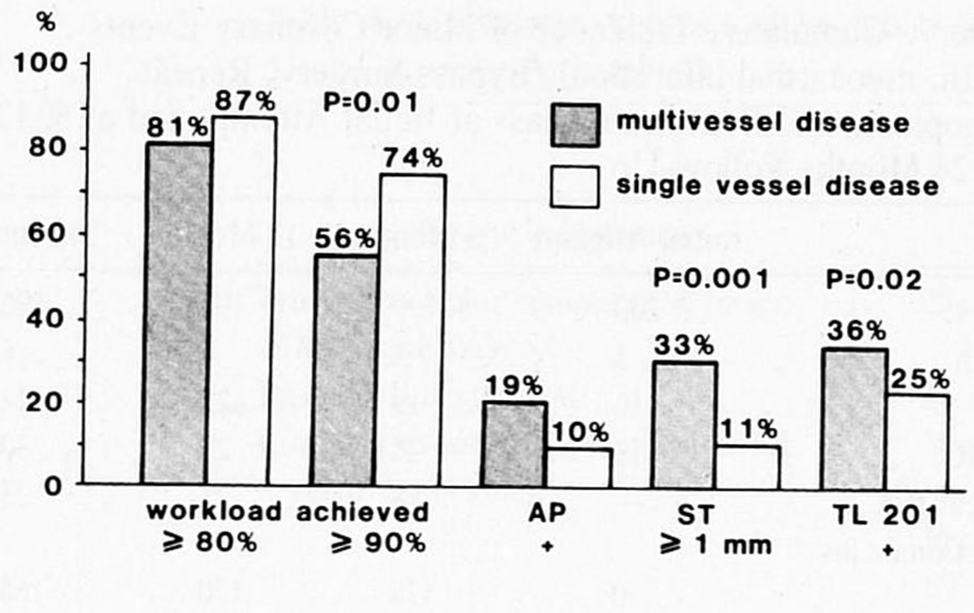



**Figure 2.** Comparison of exercise thallium-201 scintigraphy after angioplasty of the culprit lesion in 51 patients with multivessel disease and 101 patients with single vessel disease. Abbreviations as in Figure 1.

acute angioplasty, should be instituted without delay in an attempt to prevent progression to myocardial infarction and to improve prognosis.

In our study, the initial success rate of coronary angioplasty was 90% and the major complication rate was 10%. This is comparable with the recently reported (14–20) initial success rates of 70 to 93% and major complication rates of 3 to 12%. The procedure-related death rate was low (0.5%) and compared favorably with reported (14–20) death rates of 0 to 5.4%. The procedure-related myocardial infarction rate was 8% and the need for acute surgery was 9%, which are within the range of 4 to 12% and 1.5 to 12%, respectively, reported by others (14–21). Our results and those reported (14–21) reveal a high level of major complications of coronary angioplasty in patients with unstable angina. This major complication rate is definitely higher than the rate of 3 to 4% reported (28,29) for coronary angioplasty in patients with stable angina. The reasons for this high complication rate may be related to the clinical instability of these patients and, more specifically, to the higher frequency of complicated lesions (severe eccentric lesions) due to different underlying pathologic features and an increased risk of abrupt closure due to the formation of an acute occlusive thrombus (30). A thrombus may form more readily because unstable angina is related to plaque fissuring with denudation of the intima, adhesion and aggregation of platelets and partial (non)occlusive thrombus formation (31–34). In such a milieu, intracoronary instrumentation or balloon trauma may easily lead to a total occlusive thrombus.

**Late results of coronary angioplasty for unstable angina.** After initial successful angioplasty, the prognosis is excellent, with a low number of deaths (1% at 1 year and 2% at 2 years) and a low occurrence of late myocardial infarction (3% at 1 year and 4% at 2 years). This is in keeping with the findings in other reports (14–20). The results of ECG exercise testing and thallium-201 scintigraphy performed approximately 2 months after an initial successful angioplasty indicated good functional recovery and, in the majority of patients, an absence of evidence of ischemia. Angiographic restenosis occurred in 32% of the patients within 6 months of the procedure. Angiography was repeated in 88% of all patients with successful coronary angioplasty, so that this study fairly accurately reflects the actual restenosis rate and is only minimally biased by the refusal of asymptomatic patients to undergo a second angiogram. This rate does not appear to be higher than the restenosis rate of 25 to 30% for stable angina (35,36), although it has been suggested (12,35,36) that the restenosis rate in unstable angina is somewhat higher. At 1 year follow-up study, the recurrence rate of angina after an initial successful angioplasty was 26%, the majority occurring within 6 months after the procedure.

*Thus, at 1 year follow-up study of all 200 patients who had an attempted coronary angioplasty*, 4 patients (2%) died (1 procedure-related and 3 late deaths); 22 patients (11%) sustained a nonfatal myocardial infarction (16 procedure-related and 6 late); 26 patients (13%), although they had experienced some improvement, were still symptomatic (functional classes II and III) and 170 patients (85%) were symptom-free. However, to achieve this result, coronary bypass surgery was necessary in 29 patients (15%) (18 acute surgery and 11 late, elective surgery) and 21 patients (11%) underwent repeat coronary angioplasty.

**Predictors of early and late unfavorable outcome.** In an attempt to improve patient selection for coronary angioplasty and identify high risk patients, univariate and multivariate analysis was performed on potential clinical and angiographic predictors of a major complication. It has been suggested that unstable angina itself is a risk factor for early (37) and (12,35,36) late unfavorable outcome. In patients undergoing elective angioplasty, major complications were shown to be associated with 1) patient-related variables (female gender [38–41], patients >60 years old [40], multivessel disease [38], angina duration >6 months [40], prior coronary bypass surgery [40]); and 2) lesion-related variables (eccentricity [38–40,42], calcium in lesion [38–40], lesion length [38–40,42], "complicated" lesion [43], intracoronary thrombus [44] and vein graft lesion [40]). In our study, which differs from the studies just cited in that only patients with unstable angina were analyzed, we found that the ECG changes during an ischemic attack (ST segment elevation or persistent negative T waves) and the severity of the stenosis were associated with an increased risk of a major procedure-related complication. Transient ST segment elevation is thought to reflect transmural ischemia and is associated with more severe ischemia than is ST segment depression that represents subendocardial ischemia (45). The development of new T wave inversion that persists may be related to minimal cell necrosis not detected by current enzyme measurement techniques and represents severe coronary artery stenosis (46). Patients with this finding are known to have a poor overall prognosis (46,47). We could not establish a

relation between the presence of intracoronary thrombus detected angiographically and an increased risk of major complications as have others (44). However, this is not surprising because angiography is a very insensitive method for detecting intracoronary thrombus. Careful postmortem sectioning (30–32) and angioscopic examination (34) of the coronary arteries have shown that plaque rupture and thrombus formation almost invariably occur in patients with unstable angina and have established the role of thrombus in the pathogenesis of this condition.

*Angiographic restenosis after angioplasty has been shown to be associated with* 1) patient-related variables (male gender [35], diabetes [48–50], duration of angina <2 months [35,36,49,50], variant angina [51], multivessel disease [52], smoking [49,50] and hypercholesterolemia [49,50]); 2) lesion-related variables (total or >90% occlusion [35,36,50,52,53], left anterior descending coronary lesion [36,52] and vein graft stenosis [35,54,55]); and 3) procedure-related variables: no dissection [36], >30% residual stenosis [36] and >15 mm Hg residual pressure gradient [35,36]).

*In our study*, we found more frequent angiographic restenosis in patients with angiographically visible collateral vessels to the dilated vessel, multivessel disease, left anterior descending coronary stenosis and transient ST segment depression during an ischemic attack. Coronary events at 1 year, including recurrent angina pectoris (late myocardial infarction or late death), were associated with multivessel disease and total occlusion. Multivessel disease was a risk factor because of the persistence of undilated coronary artery lesions.

**Dilation of only the culprit lesion in multivessel disease.** The foremost consideration in the immediate treatment of unstable angina must be the preservation of myocardial function. Coronary angioplasty of only the "culprit lesion" in patients with refractory unstable angina and multivessel disease has been shown (21,56) to be an effective therapeutic alternative to coronary bypass surgery. Improvement in myocardial function has been demonstrated (57) using this strategy. This approach was used because multiple dilations in these clinically unstable patients may increase the risk of major complications and there is a risk of performing unnecessary dilations because of the difficulty in assessing the significance of any additional stenosis in the acute setting. The initial success rate and major complication rate of coronary angioplasty in patients with single vessel disease and dilation of that particular vessel were comparable with those of patients with multivessel disease and dilation of only the culprit lesion. The angiographic restenosis rate was higher, although not statistically significant, in patients with multivessel disease compared with those with single vessel disease. However, recurrent or persistent angina pectoris at 6 month follow-up was significantly higher in patients with multivessel disease (31%) than in patients with single vessel disease (13%). Incomplete dilation was also associated with increased occurrence of stress-induced angina pectoris, significant ST segment depression, reversible perfusion defect and reduced exercise tolerance.

We believe that angioplasty of the culprit lesion in patients with unstable angina and multivessel disease should be regarded as an initial treatment strategy in those whose symptoms do not respond adequately to pharmacologic treatment. In most patients, this approach will have a long-term success, but in some, further balloon dilations or bypass surgery will be required, so that this strategy does not provide a definitive long-term treatment in all patients. However, the subsequent interventions can be performed on a more elective basis with less risk. This strategy warrants further evaluation in a randomized controlled study.

**Conclusions.** The initial success rate for coronary angioplasty in patients with unstable angina is 90%. The hazards of dilation in patients with unstable angina are high and clearly exceed the hazards of dilation in those with stable angina. The total major complication rate in patients with unstable angina is about twice that in patients with stable angina. No strong single predictor or set of predictors of unfavorable outcome has been identified, but the procedure appears to be more hazardous in patients who have chest pain at rest associated with transient ST segment elevation or who develop persistent negative T waves.

After an initially successful procedure, the prognosis is excellent, with a low rate of late death and myocardial infarction. The rate of angiographic restenosis or angina at 6 months is approximately 30% and, therefore, appears comparable with that obtained in patients with stable angina. However, these results apply to selected patients with predominantly single vessel disease and well preserved left ventricular function.

At present, it seems reasonable to consider such patients for coronary angioplasty, although the risks of the procedure in this acute setting are relatively high. However, the alternative, coronary bypass surgery, is also associated with a rather high complication rate in these patients (58–62). Currently, there are no randomized trials comparing the results of coronary angioplasty and surgery in patients with unstable angina. There is a compelling need for randomized trials to compare the results of both strategies and to provide definitive answers on the relative merits of both types of treatment.

We are indebted to Anja van Huuksloot and Claudia Sprenger de Rover for expert preparation of the manuscript.

## References

1. Conti CR, Brawley RK, Griffith LSC, et al. Unstable angina pectoris: morbidity and mortality in 57 consecutive patients evaluated angiographically. Am J Cardiol 1973;32:745–50.

2. Cairns JA, Fantus JG, Klassen GA. Unstable angina pectoris. Am Heart J 1976;92:373–86.
3. Scanlon PJ. The intermediate coronary syndrome. Prog Cardiovasc Dis 1981;23:351–64.
4. Krauss KR, Hutter AM, De Sanctis RW. Acute coronary insufficiency: course and follow-up. Arch Intern Med 1972;129:808–13.
5. Gazes PC, Mobley EM, Faris HM, Duncan RC, Humphries CB. Preinfarctional (unstable) angina: a prospective ten year follow-up. Circulation 1973;48:331–7.
6. Bertolasi CA, Trong CJE, Riccitelli MA, Villamayor RM, Zuffardi E. Natural history of unstable angina with medical or surgical therapy. Chest 1976;70:596–605.
7. Olson HG, Lyons KP, Aronow WS, Stinson RJ, Kuperus J, Waters HJ. The high-risk angina patients. Circulation 1981;64:674–84.
8. Harris PH, Harrell FE, Lee KL, Behar VS, Rosati RA. Survival in medically treated coronary artery disease. Circulation 1979;60:1259–69.
9. Duncan B, Fulton M, Morrison SL, et al. Prognosis of new and worsening angina pectoris. Br Med J 1976;1:981–5.
10. Roberts KB, Califf RM, Harrell FE, Lee KL, Pryor DB, Rosati RA. The prognosis for patients with new onset angina who have undergone cardiac catheterisation. Circulation 1983;68:970–8.
11. Williams DO, Riley RS, Singh AK, Gewirtz H, Most AS. Evaluation of the role of coronary angioplasty in patients with unstable angina pectoris. Am Heart J 1981;102:1–9.
12. Meyer J, Schmitz HJ, Kiesslich T, et al. Percutaneous transluminal coronary angioplasty in patients with stable and unstable angina pectoris: analysis of early and late results. Am Heart J 1983;106:973–80.
13. Faxon DP, Detre KM, McGabe CH, et al. Role of percutaneous transluminal coronary angioplasty in the treatment of unstable angina: report from the National Heart, Lung, and Blood Institute Percutaneous Transluminal Coronary Angioplasty and Coronary Artery Surgery Study Registries. Am J Cardiol 1983;53:131C–35C.
14. de Feyter PJ, Serruys PW, van den Brand M, et al. Emergency coronary angioplasty in refractory unstable angina. N Engl J Med 1985;313:342–7.
15. Quigley PJ, Erwin J, Maurer BJ, Walsh MJ, Gearty GF. Percutaneous transluminal coronary angioplasty in unstable angina: comparison with stable angina. Br Heart J 1986;55:227–30.
16. de Feyter PJ, Serruys PW, Soward A, van den Brand M, Bos E, Hugenholtz PG. Coronary angioplasty for early postinfarction unstable angina. Circulation 1986;74:1365–70.
17. Safian RD, Snyder D, Synder BA, et al. Usefulness of PTCA for unstable angina pectoris after non Q-wave acute myocardial infarction. Am J Cardiol 1987;59:263–6.
18. Timmis AD, Griffin B, Crick JCP, Sowton E. Early percutaneous transluminal coronary angioplasty in the management of unstable angina. Int J Cardiol 1987;14:25–31.
19. Steffenino G, Meier B, Finci L, Rutishauser W. Follow-up results of treatment of unstable angina by coronary angioplasty. Br Heart J 1987;57:416–9.
20. de Feyter PJ, Serruys PW, Suryapranata H, Beatt K, van den Brand M. Coronary angioplasty early after the diagnosis of unstable angina. Am Heart J 1987;114:48–54.
21. de Feyter PJ, Serruys PW, Arnold A, et al. Coronary angioplasty of the unstable angina related vessel in patients with multivessel disease. Eur Heart J 1986;7:460–7.
22. Vetrovec GW, Cowley MJ, Overton H, Richardson DW. Intracoronary thrombus in syndromes of unstable myocardial ischemia. Am Heart J 1981;102:1202–8.
23. Serruys PW, van den Brand M, Brower RW, Hugenholtz PG. Regional cardioplegia and cardioprotection during transluminal angioplasty, which role for nifedipine? Eur Heart J 1983;4:115–9.
24. Wijns W, Serruys PW, Reiber JHC, et al. Quantitative angiography of the left anterior descending coronary artery: Correlations with pressure gradient and exercise thallium scintigraphy. Circulation 1985;71:239–49.
25. Cowley MJ, Dorros G, Kelsey F, van Raden M, Detre KM. Acute coronary events associated with percutaneous transluminal coronary angioplasty. Am J Cardiol 1984;53:12C–6C.
26. Reiber JHC, Lie SP, Simoons ML, Wijns W, Gerbrandts JJ. Computer quantification of location, extent and type of thallium-201 myocardial perfusion abnormalities. In: Proceedings of the 1st International Symposium on Medical Imaging and Image Interpretation. New York: ISM III 1982. IEEE Cat. No. 82 CH1084-4; 1982:123–8.
27. Miettinen O, Nurminen M. Comparative analysis of two rates. Stat Med 1985;4:213–26.
28. Anderson HV, Roubin GS, Leimgruber PP, Douglas JS, King SB, Gruentzig AR. Primary angiographic success rates of percutaneous transluminal coronary angioplasty. Am J Cardiol 1985;56:712–7.
29. Block PC. Percutaneous transluminal coronary angioplasty: role in the treatment of coronary artery disease. Circulation 1985;72(suppl V):V-161–5.
30. MacDonald RG, Feldman RL, Conti RC, Pepine CJ. Tromboembolic complications of coronary angioplasty. Am J Cardiol 1984;54:916–7.
31. Falk E. Plaque rupture with severe pre-existing stenosis precipitating coronary thrombosis: characteristics of coronary atherosclerotic plaques underlying fatal occlusive thrombi. Br Heart J 1983;50:127–34.
32. Davies MJ, Thomas AC. Plaque fissuring—the cause of acute myocardial infarction, sudden ischemic death and crescendo angina. Br Heart J 1985;53:363–73.
33. Gorlin R, Fuster V, Ambrose JA. Anatomic-physiologic link between acute coronary syndromes. Circulation 1986;74:6–9.
34. Sherman CT, Litvack F, Grundfest W, et al. Coronary angioscopy in patients with unstable angina pectoris. N Engl J Med 1986;315:913–9.
35. Holmes DR, Vlietstra RE, Smith HC, et al. Restenosis after percutaneous transluminal coronary angioplasty: a report from the PTCA registry of the NHLBI. Am J Cardiol 1984;53:77C–81C.
36. Leimgruber PP, Roubin GS, Hollman J, et al. Restenosis after successful coronary angioplasty in patients with single vessel disease. Circulation 1986;73:710–7.
37. Cowley MJ, Dorros G, Kelsey SF, van Raden M, Detre KM. Emergency coronary bypass surgery after coronary angioplasty: the NHLBI PTCA registry experience. Am J Cardiol 1984;53:22C–6C.
38. Bredlau CE, Roubin GS, Leimgruber PP, Douglas JS, King SB, Gruentzig AR. In-hospital morbidity and mortality in patients undergoing elective coronary angioplasty. Circulation 1985;72:1044–52.
39. Cowley MJ, Dorros G, Kelsey SF, van Raden M, Detre KM. Acute coronary events associated with percutaneous transluminal coronary angioplasty. Am J Cardiol 1984;53:12C–6C.
40. Dorros G, Cowley MJ, Janke L, Kelsey SF, Mullin SM, van Raden M. In-hospital mortality rate in the NHLBI PTCA registry. Am J Cardiol 1984;53:17C–21C.
41. Cowley MJ, Mullin S, Kelsey SF, et al. Sex differences in early and longterm results of coronary angioplasty in the NHBLI PTCA Registry. Circulation 1985;71:90–7.
42. Meier B, Gruentzig AR, Hollman J, Ischinger T, Bradford JM. Does length or eccentricity of coronary stenoses influence the outcome of transluminal dilatation? Circulation 1983;67:497–9.
43. Ischinger T, Gruentzig AR, Meier B, Galan K. Coronary dissection and total coronary occlusion associated with percutaneous transluminal coronary angioplasty: significance of initial angiographic morphology of coronary stenoses. Circulation 1986;72:1371–8.
44. Mabin TA, Holmes DR, Smith HC, et al. Intracoronary thrombus: role in coronary occlusion complicating percutaneous transluminal coronary angioplasty. J Am Coll Cardiol 1985;5:198–202.
45. Plotnick GD, Conti R. Transient ST-segment elevation in unstable angina: clinical and hemodynamic significance. Circulation 1975;51:1015–9.
46. Haines DE, Raabe DS, Gundel WD, Wackers FJ. Anatomic and prognostic significance of new T wave inversion in unstable angina. Am J Cardiol 1983;52:14–8.

47. Granborg J. Diagnostic and prognostic implications of transient isolated negative T waves in suspected acute myocardial infarction. Am J Cardiol 1986;57:203–7.

48. Margolis JR, Krieger R, Glemser E. Coronary angioplasty: increased restenosis rate in insulin dependent diabetics (abstr). Circulation 1984;70(suppl II):II-175.

49. Shaw RE, Myler RK, Fishman-Rosen J, Murphy MC, Stertzer SH, Topol EJ. Clinical and morphologic factors in prediction of restenosis after multivessel angioplasty (abstr). J Am Coll Cardiol 1986;7:63A.

50. Myler RK, Topol EJ, Shaw RE, et al. Multiple vessel coronary angioplasty: classification, results and patterns of restenosis in 494 consecutive patients. Cathet Cardiovasc Diagn 1987;13:1–15.

51. David PR, Waters DD, Scholl JM. Percutaneous transluminal coronary angioplasty in patients with variant angina. Circulation 1982;66:695–702.

52. Mata LA, Bosch X, David PR, Rapold HJ, Corcos T, Bourassa MG. Clinical and angiographic assessment 6 months after double vessel percutaneous coronary angioplasty. J Am Coll Cardiol 1985;6:1239–44.

53. Serruys PW, Umans V, Heyndrickx GR, et al. Elective PTCA of totally occluded coronary arteries not associated with acute myocardial infarction: short term and long term results. Eur Heart J 1985;6:2–12.

54. Douglas JS, Gruentzig AR, King SB. Percutaneous transluminal coronary angioplasty in patients with prior coronary bypass surgery. J Am Coll Cardiol 1983;2:745–54.

55. Block PC, Cowley MJ, Kaltenbach M, Kent KM, Simpson J. Percutaneous angioplasty of stenoses of bypass grafts or of bypass graft anastomotic sites. Am J Cardiol 1984;53:666–8.

56. Wohlgelernter D, Cleman M, Highman HA, Zaret BL. Percutaneous transluminal coronary angioplasty of the "culprit" lesion for management of unstable angina pectoris in patients with multivessel coronary artery disease. Am J Cardiol 1986;58:460–4.

57. de Feyter PJ, Surypranata H, Serruys PW, Beatt K, van den Brand M, Hugenholtz PG. Effects of successful percutaneous transluminal coronary angioplasty on global and regional left ventricular function in unstable angina pectoris. Am J Cardiol 1987;60:993–7.

58. Rankin JS, Newton JR, Califf RM, et al. Clinical characteristics and current management of medically refractory unstable angina. Ann Surg 1984;200:457–64.

59. Luchi RJ, Scott SM, Dupree RH. Comparison of medical and surgical treatment for unstable angina pectoris. Results of a Veterans Administration Cooperative Study. N Engl J Med 1987;316:977–84.

60. Williams DB, Ivey TD, Bailey WW, Irey SJ, Rideout JT, Stewart D. Postinfarction angina: results of early revascularization. J Am Coll Cardiol 1983;2:859–64.

61. Gertler JP, Elefteriades JA, Kopf GS, Hashim SW, Hammond GL, Geha AS. Predictors of outcome in early revascularization after acute myocardial infarction. Am J Surg 1985;149:441–4.

62. Singh AK, Rivera R, Cooper GN, Karlson KE. Early myocardial revascularization for postinfarction angina: results and longterm follow-up. J Am Coll Cardiol 1985;6:1121–5.