Patency of Percutaneous Transluminal Coronary Angioplasty Sites at 6-Month Angiographic Follow-Up
A Key Determinant of Survival in Diabetics After Coronary Balloon Angioplasty

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Background—Several reports have demonstrated a high mortality rate in diabetic patients treated by standard coronary balloon angioplasty. No clear explanation has been provided for this finding.

Methods and Results—Consecutive diabetic patients successfully treated by standard coronary balloon angioplasty (n=604) were enrolled in a follow-up program including repeated angiography at 6 months and long-term clinical follow-up. Clinical follow-up was available in 603 patients (99.8%). Twelve patients died, 2 underwent bypass surgery before scheduled repeated angiography, and 76 declined angiography. Determinants of long-term mortality were analyzed in the 513 patients with angiography at 6 months and long-term clinical follow-up (mean follow-up, 6.5±2.4 years). On the basis of the results of repeated angiography, 3 groups of patients were defined: group 1, 162 patients without restenosis (32%); group 2, 257 patients with nonocclusive restenosis (50%); and group 3, 94 patients with coronary occlusion (18%). Overall actuarial 10-year mortality rate was 36%. Actuarial 10-year mortality was 24% in group 1, 35% in group 2, and 59% in group 3 (P<0.0001). Multivariate analysis demonstrated that coronary occlusion was a strong and independent correlate of long-term total mortality (hazard ratio, 2.16; 95% CI, 1.43 to 3.26; P=0.0003) and cardiac mortality (hazard ratio, 2.38; 95% CI, 1.48 to 3.85; P=0.0004).

Conclusions—This study demonstrates that restenosis, especially in its occlusive form, is a major determinant of long-term mortality in diabetic patients after coronary balloon angioplasty. (Circulation. 2001;103:1218-1224.)

Key Words: diabetes mellitus ■ survival ■ coronary disease ■ angioplasty ■ balloon

Diabetes, a major risk factor for atherosclerosis and cardiac death, is an increasing public health problem in western countries; indeed, diabetics represent 15% to 25% of patients referred for percutaneous or surgical treatment of coronary artery disease.1-5

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Percutaneous coronary revascularization (PCR) has gained acceptance as an alternative to CABG in selected patients. However, several reports that demonstrated poor long-term survival in diabetic patients treated by standard coronary balloon angioplasty2,3,6-8 have led to concerns regarding the use of PCR in this group of patients.9,10 It is of critical importance in the management of diabetic patients for physicians to understand the reason(s) for their poor outcome after PTCA. Although several hypotheses have been advanced,9-12 there is still no conclusive explanation for this unexpected “diabetes-PTCA” interaction.

We recently demonstrated that restenosis after standard coronary balloon angioplasty, the PTCA technique used in the above-mentioned studies2,3,6-8 not only was a frequent event in this population but also had a unique expression in that coronary occlusion (TIMI flow=0 or 1) was observed in ~14% of lesions4,13 compared with 3% in nondiabetic patients.13 From this observation, we hypothesized that restenosis might be one of the determinants of the poor outcome of diabetic patients after PTCA.

We report here the outcome of 637 consecutive diabetic patients included at the time of their first procedure and treated by standard balloon angioplasty. The determinants of long-term mortality, including the status of treated vessels at 6 months, were analyzed in this population.

Methods

Study Population and Follow-Up
Between January 1987 and December 1995, all consecutive patients with successful PTCA in our institution were enrolled in a follow-up program. We report here the outcome of 637 consecutive diabetic patients from the time of their first procedure in our institution during this period and treated by standard balloon angioplasty without...
adjunctive stent implantation. Patients undergoing primary or rescue balloon angioplasty for acute myocardial infarction (MI) were not included.

At the time of the procedure, patients were asked to return for follow-up angiography at 6 months regardless of symptomatic status. Angiography was performed earlier if clinically indicated.

Long-term clinical follow-up was accomplished by a questionnaire completed by the patient or by telephone contact. Review of hospital records and contact with the referring physician enabled us to complete some missing information. Finally, patients’ municipal records were checked for mortality status. The cause of death was recorded; sudden deaths or deaths of unknown cause were classified as cardiac deaths.

**Patient Definitions**

At the time of the procedure, patients were classified as diabetic if they were treated by oral hypoglycemic drugs or insulin or if they had a history of elevated (>140 mg/dL) fasting blood glucose on ≥2 separate occasions in conjunction with ongoing dietary measures. They were classified into 3 categories—(1) diet alone, (2) oral hypoglycemic drugs (diet and oral hypoglycemic drugs but no insulin), and (3) insulin (regardless of other therapy)—at the time of the initial procedure. In patients treated with oral hypoglycemic drugs, the use of sulfonylureas was recorded.

Baseline blood glucose and creatinine levels, as well as the presence of end-organ damage (retinopathy, nephropathy, neuropathy) as documented in the medical records, were recorded. Clinical status was recorded at the time of the initial procedure and at angiographic follow-up.

**Angioplasty Procedure**

Balloon angioplasty without adjunctive stent implantation was performed according to the standard technique in our laboratory. A vascular segment was considered successfully treated when the residual luminal narrowing in the dilated segment immediately after angioplasty was <50%. The procedure was considered successful when ≥1 vascular segment was successfully treated and when no major complication (ECG or enzymatic evidence of MI, the need for bypass surgery during hospitalization, or in-hospital death) occurred.

<table>
<thead>
<tr>
<th>TABLE 1. Baseline Characteristics of the Study Population According to Target Vessel Patency at Follow-Up Angiography</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No Restenosis</strong> (n=162)</td>
</tr>
<tr>
<td>Age, y</td>
</tr>
<tr>
<td>Males, n (%)</td>
</tr>
<tr>
<td>Smokers, n (%)</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
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<tr>
<td>Hypercholesterolemia, n (%)</td>
</tr>
<tr>
<td>&lt;250 mg/dL without treatment</td>
</tr>
<tr>
<td>&lt;250 mg/dL with treatment</td>
</tr>
<tr>
<td>≥250 mg/dL</td>
</tr>
<tr>
<td>Family history of CAD, n (%)</td>
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<tr>
<td>Glucose, mmol/L</td>
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<tr>
<td>Creatinine, µmol/L</td>
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<tr>
<td>End-organ damage, n (%)</td>
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<tr>
<td>Retinopathy</td>
</tr>
<tr>
<td>Nephropathy</td>
</tr>
<tr>
<td>Neuropathy</td>
</tr>
<tr>
<td>≥1 of the above</td>
</tr>
<tr>
<td>Antidiabetic regimen, n (%)</td>
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<td>Diet</td>
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<td>Oral hypoglycemic drugs</td>
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<td>Nonsulfonylureas</td>
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<tr>
<td>Sulfonylureas</td>
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<tr>
<td>Insulin</td>
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<td>Previous PTCA, n (%)</td>
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<tr>
<td>Previous CABG, n (%)</td>
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<tr>
<td>Recent (&lt;1 mo) MI, n (%)</td>
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<tr>
<td>Unstable angina, n (%)</td>
</tr>
<tr>
<td>Diseased vessels, n</td>
</tr>
<tr>
<td>Treated lesions, n</td>
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<tr>
<td>LVEF, %</td>
</tr>
</tbody>
</table>

CAD indicates coronary artery disease.

*No restenosis versus restenosis (restenosis = nonocclusive restenosis + coronary occlusion).
†Coronary occlusion versus no occlusion (no occlusion = no restenosis + nonocclusive restenosis).
Angiographic Analyses
Quantitative computer-assisted angiographic measurements were performed as previously described on end-diastolic frames of angiograms performed at maximal dilatation with the Computer-Assisted Evaluation of Stenosis and Restenosis system.\(^4\) The antegrade blood flow was graded with the classification of the TIMI study group.\(^14\) Restenosis was defined as a $>50\%$ diameter stenosis at follow-up, and complete vessel occlusion was defined as a TIMI grade flow $<2$.

Patients were considered to have restenosis if $\geq 1$ of the successfully dilated lesion(s) had restenosis at follow-up and to have coronary occlusion if $\geq 1$ of the successfully dilated lesion(s) had restenosis and TIMI flow $<2$ at follow-up angiography. The presence of new occlusion(s) (TIMI flow $<2$) at nontreated sites was also recorded. Left ventricular ejection fraction (LVEF) was calculated on ventriculograms obtained before angioplasty and at follow-up.

Statistical Analysis
Data are presented as mean±SD. Comparisons between groups for continuous data were made with paired or unpaired Student's $t$ test or ANOVA followed by Scheffé's $F$ test as appropriate. Differences between proportions were assessed by $\chi^2$ analysis.

Late survival was estimated with the Kaplan-Meier method. Differences were tested with a log-rank test. Multivariate correlates of survival were analyzed with Cox's proportional-hazard model. For verification purposes, variables obtained by Cox analysis were entered into logistic models designed to predict mortality at 5 years, and concordance indexes were calculated. A 2-sided value of $P<0.05$ was considered to indicate statistical difference. Analyses were performed with SAS software (version 6.12, SAS Institute).

Results

Patients Characteristics and Follow-Up
PTCA was successful in 604 of the 637 patients ($95\%$). Clinical follow-up was available in 603 of the 604 patients ($99.8\%$). Of the 604 patients with successful PTCA, 12 had died at 6 months, and 2 underwent CABG without repeated coronary angiography. Of the remaining 590 patients, 514 ($87\%$) underwent repeated angiography at a mean of 6.7±2.6 months after PTCA. Seventy-six patients declined angiography. One patient was lost to follow-up after repeated angiography. There were no differences in baseline characteristics between patients who were and who were not restudied. We present the results of the 513 patients with 6-month repeated angiography and clinical follow-up.

In the 513 patients, 426 ($63\%$) of the 680 successfully treated lesions had a diameter stenosis $>50\%$ at repeated angiography, including 99 lesions ($14\%$) that had occluded (TIMI flow $<2$). On the basis of these results, 3 groups of patients were defined: group 1, 162 patients without restenosis ($32\%$); group 2, 257 patients with nonocclusive restenosis ($50\%$); and group 3, 94 patients with coronary occlusion ($18\%$). Occlusion at an untreated site occurred in 18 patients ($3\%$).

Baseline study population characteristics are shown in Table 1. As expected, restenosis, both occlusive and nonocclusive, was more frequently observed in patients who underwent multsite PTCA ($P=0.0001$). Patients with end-organ damage had a higher risk of restenosis ($P<0.05$), whereas patients treated with insulin or those with a recent MI had a higher risk of coronary occlusion at target sites ($P<0.05$).

Changes in LVEF and Clinical Status at Follow-Up Angiography
Changes in LVEF are reported in Table 2. There were no differences in LVEF among groups before PTCA. During follow-up, there was a decrease in LVEF in patients with occlusion at follow-up angiography, whereas no significant change was observed in the other 2 groups. These changes resulted in a significantly lower LVEF at follow-up angiography in the group of patients with occlusion compared with the other 2 groups.

The clinical status of patients at follow-up angiography is presented in Table 2. Although patients with nonocclusive or occlusive restenosis were more likely to have angina than patients without restenosis ($P<0.0001$), 30% to 35% of patients with restenosis had no recurrence of angina at follow-up angiography. In addition, $<40\%$ of patients with coronary occlusion had clinical and/or ECG evidence of unstable angina or acute MI. Among these patients, 8 had a documented Q-wave MI, 10 had a documented non–Q-wave MI, and 19 were classified as having unstable angina. The onset of the ischemic episode was documented within the first 2 months in 12 patients, between months 2 and 4 in 13 patients, and after month 4 in 12 patients. Clinically driven repeated angiography within 4 months of the initial procedure was performed in 22 of 94 patients ($23\%$) with coronary occlusion.

| TABLE 2. Changes in LVEF and Clinical Status at Follow-Up Angiography According to Late Vessel Patency |
|--------------------------------------------------|---------------------------------|------------------|------------------|------------------|--------|
| LVEF, %                                          | No Restenosis                  | Nonocclusive Restenosis | Coronary Occlusion | $P$   |
| Baseline (n=495)                                 | 60.3±14.3                      | 58.5±13.4            | 57.2±12.5         | 0.13  |
| Follow-up angiography (n=492)                    | 60.5±13.6                      | 59.3±14.2            | 52.5±13.8         | 0.0001|
| Changes between baseline and follow-up angiography (n=484) | 0.2±10.5                      | 0.8±9.8              | −4.8±12.6         | 0.0001|
| Clinical status at follow-up angiography, n      | 162                            | 257                 | 94               |       |
| No angina, n (%)                                 | 106 (65)                       | 84 (33)              | 29 (31)           | 0.0001|
| Stable angina, n (%)                             | 46 (29)                        | 108 (42)             | 28 (30)           |       |
| Unstable angina or acute MI, n (%)               | 10 (6)                         | 65 (25)              | 37 (39)           |       |
Long-Term Clinical Follow-Up

Long-term clinical follow-up was obtained in the 513 patients a mean of 6.5±2.4 years after follow-up angiography (7.0±2.4 years after the initial PTCA). One hundred thirty-six patients (27%) died during follow-up. This rate was similar to the rate observed in the 76 patients with initially successful procedures who did not undergo repeated angiography (20 deaths; 26%). The Figure shows the Kaplan-Meier survival curves as a function of late vessel patency documented at repeated angiography. At the end of follow-up, there were 28 deaths among the 162 patients without restenosis (17%), 66 deaths among the 257 patients with nonocclusive restenosis (26%), and 42 deaths among the 94 patients with coronary occlusion (45%; P<0.0001). Actuarial 5- and 10-year mortality rates were 13% and 24% in patients without restenosis, 23% and 35% in patients with nonocclusive restenosis, and 38% and 59% in patients with coronary occlusion (P<0.0001; the Figure, bottom). Actuarial 5- and 10-year cardiac mortality rates were 10% and 17% in patients without restenosis, 18% and 27% in patients with nonocclusive restenosis, and 31% and 51% in patients with coronary occlusion (P<0.0001; the Figure, bottom).

When imbalances in baseline characteristics were controlled for by a Cox’s proportional-hazard model, the hazard ratios (HRs) for nonocclusive restenosis versus no restenosis were 1.63 (95% CI, 1.05 to 2.55; P=0.03) for total mortality and 1.72 (95% CI, 1.01 to 2.87; P=0.04) for cardiac mortality, and the HRs for coronary occlusion versus no restenosis were 3.12 (95% CI, 1.90 to 5.13; P=0.0001) for total mortality and 3.40 (95% CI, 1.93 to 5.99; P=0.0001) for cardiac mortality.

Univariate predictors of long-term mortality among characteristics at baseline and at follow-up angiography are listed in Table 3. Multivariate analysis including variables at baseline and at follow-up angiography are listed in Table 3. Multivariate analysis including variables at baseline and at follow-up angiography showed that occlusive but not nonocclusive restenosis was an independent predictor of long-term mortality (Table 4). Six variables were independently associated with total mortality: coronary occlusion, age at initial procedure, decrease in LVEF, end-organ damage, a lower LVEF at baseline, and hypertension (Table 4). Eight variables were independently associated with cardiac mortality: coronary occlusion, decrease in LVEF, end-organ damage, age at initial procedure, multivessel coronary dis-
ease, a lower LVEF at baseline, hypertension, and the occurrence of an occlusion at an untreated site (Table 4). When variables obtained by Cox analysis were entered into logistic regression models designed to determine 5-year total and cardiac mortality, concordance indexes for the 2 models were 0.736 and 0.758, respectively. After exclusion of patients with a recent (<1 month) MI at baseline, coronary occlusion remained an independent predictor of total (HR, 1.88; 95% CI, 1.16 to 3.09; P = 0.01) and cardiac mortality (HR, 2.33; 95% CI, 1.33 to 4.09; P = 0.003) by multivariate analysis.

**Possible Mechanisms for the Diabetes-PTCA Interaction**

It is now well established that long-term survival of diabetic patients with multivessel coronary artery disease treated by standard coronary balloon angioplasty is significantly lower than after bypass surgery,2,7,8 a trend that is not seen in the nondiabetic population.2 This unique behavior suggests a specific interaction between diabetes mellitus and the “response” of coronary vessels to balloon angioplasty. Although several possible mechanisms have been suggested for this diabetes-PTCA interaction,9–12 no conclusive explanation has been provided.

Early procedural complications, progression of atherosclerosis, or high restenosis rates are generally cited as potential explanations for the poor outcome of diabetic patients treated by standard balloon angioplasty. However, one of the most intriguing findings in several different studies was the fact that diabetics had a relatively low rate of in-hospital complications and that the detrimental effects of angioplasty became apparent only at a distance from the procedure.2,6,8 Considering this observation, as well as biological and epidemiolog-

### Table 3. Predictors of Mortality by Univariate Analysis

<table>
<thead>
<tr>
<th>Total Mortality</th>
<th>Cardiac Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Event (n=136)</td>
<td>Event (n=101)</td>
</tr>
<tr>
<td>No Event (n=377)</td>
<td>No Event (n=412)</td>
</tr>
<tr>
<td><strong>P</strong></td>
<td><strong>P</strong></td>
</tr>
</tbody>
</table>

**Baseline patient characteristics**

- **Age, y**
  - Event: 63 ± 9, No Event: 60 ± 9
  - P = 0.004
- **Hypertension, n (%)**
  - Event: 93 (68), No Event: 229 (61)
  - P = 0.11
- **Creatinine, µmol/L**
  - Event: 124.5 ± 122.30, No Event: 95.13 ± 20.62
  - P = 0.0001

**End-organ damage, n (%)**

- **Retinopathy**
  - Event: 34 (25), No Event: 65 (17)
  - P = 0.06
- **Nephropathy**
  - Event: 50 (36), No Event: 59 (16)
  - P = 0.0001
- **Neuropathy**
  - Event: 19 (14), No Event: 30 (8)
  - P = 0.06
- **≥1 of the above**
  - Event: 61 (44), No Event: 91 (24)
  - P = 0.0001
- **Unstable angina**
  - Event: 54 (40), No Event: 139 (37)
  - P = 0.05
- **Diseased vessels, n**
  - Event: 1.96 ± 0.77, No Event: 1.65 ± 0.70
  - P = 0.0001
- **Treated lesions, n**
  - Event: 1.39 ± 0.63, No Event: 1.30 ± 0.55
  - P = 0.11
- **LVEF at baseline, %**
  - Event: 56.6 ± 14.9, No Event: 59.5 ± 13.2
  - P = 0.03

**Patient characteristics at follow-up angiography**

- **Clinical status, n (%)**
  - No angina: 47 (35), Stable angina: 50 (37)
  - P = 0.03
  - Unstable angina or acute MI: 39 (28)
  - P = 0.03
- **Vessel patency, n (%)**
  - No restenosis: 28 (20), Nonocclusive restenosis: 66 (49)
  - P = 0.0001
  - Coronary occlusion: 42 (31)
  - P = 0.0001
- **Occlusion at an untreated site, n (%)**
  - Event: 8 (6), No Event: 10 (3)
  - P = 0.08
- **LVEF at follow-up angiography, %**
  - Event: 53.7 ± 16.8, No Event: 60.0 ± 13.2
  - P = 0.0001
- **Change in LVEF, %**
  - Event: −2.9 ± 12.2, No Event: 0.5 ± 10.0
  - P = 0.002

Statistical analysis was performed using baseline and follow-up characteristics presented in Tables 1 and 2. Variables with P > 0.10 are not presented.
tical perspectives, the possibility that atherosclerosis progression may contribute to adverse outcome in this population is a plausible hypothesis. However, data on this subject are sparse. Although the present study was not specifically designed to investigate this issue, it is interesting to note that the rate of spontaneous occlusion(s) at untreated sites was relatively high in our population, 3% during a 6-month period, and that this parameter was an independent predictor of cardiac mortality.

Finally, although diabetes mellitus is a well-established risk factor for restenosis after balloon angioplasty, restenosis is generally not considered as the explanation for the high mortality rate observed in this population. Indeed, studies investigating the effect of restenosis on long-term follow-up in unselected populations did not demonstrate a significant impact of restenosis on survival. The assumption that restenosis could not be the explanation for the poor outcome of diabetic patients treated by PTCA was recently called into question by some new findings.

**Coronary Occlusion and Long-Term Mortality**

We recently reported that restenosis in diabetic patients treated by balloon angioplasty had a unique feature, namely the frequent occurrence of coronary occlusion (TIMI flow = 0 or 1) at dilated sites, that was observed in ~14% of lesions compared with 3% in nondiabetic patients. From this observation, we hypothesized that restenosis might be one of the determinants of the poor outcome of diabetic patients after PTCA. The present study involving a population of 604 consecutive diabetic patients successfully treated by balloon angioplasty at ≥1 sites, with 6-month angiographic follow-up in 87% of patients and long-term clinical follow-up (6.5 years) in 99.8% of patients, was specifically designed to investigate this issue. The results of the study confirmed our previous observation in that coronary occlusion was observed in 15% of treated lesions. This led to a higher risk of occlusion in patients with multisite angioplasty and to an overall rate of coronary occlusion of 18% in our population. More importantly, our study demonstrated that restenosis, especially in its occlusive form, was a strong predictor of poor long-term survival in diabetic patients.

Multivariate analysis demonstrated that the effect of coronary occlusion was independent of other risk factors previously described in this population (age, end-organ damage, hypertension, baseline LVEF, and multivessel disease). It demonstrated also that the deleterious effect of coronary occlusion on survival was not exclusively related to the decrease in LVEF observed at the time of angiographic follow-up. This finding is consistent with previous observations and suggests that additional mechanisms, including late ventricular remodeling, improvement in electrical stability, and provision of collaterals, may play a role.

It is also interesting to point out that the number of treated lesions per patient, in addition to being a predictor of coronary occlusion (Table 1), was a predictor of mortality by univariate analysis (Table 3). However, because this parameter was also strongly correlated with the severity of coronary artery disease and the risk of occlusion, we were unable to demonstrate its effect on late outcome by multivariate analysis.

It is important to emphasize that our results suggest that the status (patent/occluded) of dilated segments at 6 months may allow a better assessment of long-term prognosis than clinical symptoms in diabetic patients. Indeed, coronary occlusion was associated with clinical instability in <40% of cases, and clinical symptoms at the time of restudy were not a correlate of survival by multivariate analysis.

**Clinical Implications**

The present demonstration of the deleterious effect of restenosis on long-term survival is potentially of major clinical importance. Diabetics represent a large proportion of patients referred for coronary revascularization, and it will be very difficult from a practical viewpoint to refer all diabetics for

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>( \beta ) Coefficient</th>
<th>SE</th>
<th>( \chi^2 )</th>
<th>( P )</th>
<th>HR</th>
<th>95% CI</th>
</tr>
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<tbody>
<tr>
<td><strong>Total mortality</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occlusive restenosis</td>
<td>0.77</td>
<td>0.21</td>
<td>14.60</td>
<td>0.0003</td>
<td>2.16</td>
<td>1.43–3.26</td>
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<tr>
<td>Age (10-y increase)</td>
<td>0.37</td>
<td>0.11</td>
<td>11.21</td>
<td>0.0008</td>
<td>1.44</td>
<td>1.17–1.79</td>
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<tr>
<td>Change in LVEF (5% fall)</td>
<td>0.14</td>
<td>0.04</td>
<td>11.09</td>
<td>0.001</td>
<td>1.15</td>
<td>1.06–1.25</td>
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<tr>
<td>End-organ damage</td>
<td>0.51</td>
<td>0.18</td>
<td>7.53</td>
<td>0.006</td>
<td>1.66</td>
<td>1.16–2.38</td>
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<tr>
<td>Baseline LVEF (5% fall)</td>
<td>0.10</td>
<td>0.03</td>
<td>7.21</td>
<td>0.007</td>
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<td>0.42</td>
<td>0.19</td>
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<td>0.04</td>
<td>1.51</td>
<td>1.03–2.23</td>
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<td><strong>Cardiac mortality</strong></td>
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<tr>
<td>Occlusive restenosis</td>
<td>0.87</td>
<td>0.24</td>
<td>13.09</td>
<td>0.0004</td>
<td>2.38</td>
<td>1.48–3.85</td>
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<tr>
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<td>0.05</td>
<td>9.81</td>
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<tr>
<td>End-organ damage</td>
<td>0.58</td>
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<td>6.75</td>
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<td>Multivessel disease</td>
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<td>0.23</td>
<td>6.63</td>
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<td>1.84</td>
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<td>Baseline LVEF (5% fall)</td>
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<td>0.04</td>
<td>6.58</td>
<td>0.01</td>
<td>1.11</td>
<td>1.03–1.21</td>
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<td>Hypertension</td>
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<td>0.24</td>
<td>6.40</td>
<td>0.01</td>
<td>1.87</td>
<td>1.16–3.00</td>
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<td>Occlusion at untreated site</td>
<td>0.90</td>
<td>0.40</td>
<td>5.67</td>
<td>0.03</td>
<td>2.45</td>
<td>1.11–5.39</td>
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bypass surgery. Better insight into the causes of their poor outcome might help to specifically design and test new therapeutic approaches that could make PCR a safer technique in these patients. Although the results of this study do not prove that occlusion of target sites is the only reason for the poor outcome of diabetic patients, they strongly suggest that it is a key part of the problem. Our findings also raise the possibility that strategies designed to minimize late coronary occlusion after angioplasty may improve the outcome of these patients. Several recent observations lend support to this hypothesis. It is encouraging to see that the use of stents in diabetic patients has been associated with a more acceptable risk of late occlusion (≤ 5% per lesion) than after standard balloon angioplasty. Similarly, subgroup analyses of studies that investigated the effects of the potent antiplatelet agent abciximab suggest that the beneficial effect of this drug in reducing major adverse cardiac events after PCR is even more potent in diabetics than in the population at large. Our previous demonstration that some angiographic characteristics, including angioplasty at saphenous vein graft, thrombus, or TIMI flow < 3 before angioplasty and the degree of residual stenosis after angioplasty, were found to predict restenosis and late vessel occlusion in diabetic patients is also consistent with a potential benefit of these 2 strategies. In the light of our observation, we may even suggest that the apparent reduction in major cardiac events at 1 year observed with the combined use of stent and abciximab in diabetic patients, as reported in the EPISTENT trial, was probably related to an improved midterm patency of treated vessels.

Study Limitations
Because this is a single-center retrospective study and because patient referral, the technique of PTCA, or medical management may have influenced the outcome, our results warrant confirmation by other studies. However, because the rates of angiographic and long-term clinical follow-up were very high in our population, our results are likely to reflect the true rate of coronary occlusion and long-term mortality in our study population. In addition, the technique of PTCA is identical to the technique used in previous studies in which the poor outcome of diabetic patients has been demonstrated. Similarly, the characteristics of our study population, including baseline patient characteristics, revascularization strategy (number of lesions treated per patient), and mortality rate, are in the range of what has been previously reported.

Conclusions
Our results demonstrate that restenosis, especially in its occlusive form, is a key determinant of long-term survival in diabetic patients after standard balloon angioplasty. Strategies designed to target the problem of coronary occlusion might significantly affect long-term prognosis in diabetic patients treated by PCR.

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Patency of Percutaneous Transluminal Coronary Angioplasty Sites at 6-Month Angiographic Follow-Up: A Key Determinant of Survival in Diabetics After Coronary Balloon Angioplasty

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