Restenosis After Placement of Palmaz-Schatz Stents in Native Coronary Arteries
Initial Results of a Multicenter Experience

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Background. Several metallic intracoronary stents are currently undergoing preliminary evaluation to ascertain potential benefit as means to reduce the 30–40% incidence of restenosis after balloon angioplasty.

Methods and Results. To determine the incidence and correlates of restenosis after placement of Palmaz-Schatz stents in native coronary arteries in the first group of patients selected for this procedure, clinical and quantitative angiographic data from 206 consecutive patients (221 stenoses) with successful stent placement (diameter stenosis <50%) were analyzed. Six patients (2.9%) had thrombosis-mediated stent closure within 1 month after stent placement and were excluded from long-term angiographic follow-up. One hundred eighty-one (91%) of the remaining 200 patients had angiography at 5.8±2.1 months. Patients with and without follow-up did not differ in any baseline characteristic; in particular, history of restenosis at the site stented (73% versus 65%), placement of multiple overlapping stents (17% versus 20%), and mean poststent diameter stenosis (16±12% versus 14±12%). The overall incidence of restenosis (diameter stenosis ≥50% at follow-up) in this group at high risk for restenosis was 36% (95% confidence interval, 29–43%) on a per-stentenasis basis. The incidence of restenosis when a single stent was placed was 30% (95% confidence interval, 23–37%). Risk was dependent upon a history of restenosis (present versus absent 36% versus 16%, p=0.02) and upon whether or not a poststenosis ≤5% was achieved (6% versus 33%, p=0.02). When multiple overlapping stents were placed, restenosis occurred at 64% of sites, and placement of multiple stents was discouraged during the later phases of this study as these results became apparent.

Conclusions. Although multiple stents appear to yield a poor long-term result, placement of single stents may offer a benefit compared with standard coronary angioplasty, particularly if an excellent angiographic result can be obtained in patients without prior restenosis. Further randomized trials in such patients are needed. (Circulation 1992;86:1836–1844)

KEY WORDS • angioplasty, coronary • stents • restenosis

In contrast to the dramatic increase in the primary success rate of coronary angioplasty,1–3 the long-term efficacy after a successful angioplasty has not improved since the first coronary procedure was performed in 1977.4,5 This has been almost entirely due to an unabated restenosis rate of 30–40%, which has persisted despite multiple attempted pharmacological6–13 and mechanical14,15 interventions.

The pathogenesis of restenosis is incompletely understood16 but is probably a multifactorial process that includes growth factor–stimulated smooth muscle cell proliferation,17–20 elastic recoil,21 and perhaps organization of thrombus adherent at the site of arterial injury.22 As early as 1969, Dotter23 introduced the concept of maintaining vascular patency by endoluminal stenting. Inadequate design prevented widespread clinical application of such devices for many years. More recently, however, improvements in the design and technology have allowed extensive clinical investigation of several intracoronary stents.24–27

One of these, the Palmaz-Schatz stent, a refinement based on the original design of Palmaz,28,29 has been

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undergoing clinical evaluation since 1988. The purpose of this report is to define the overall incidence of restenosis in the first group of patients selected to undergo this procedure and to explore the clinical and procedural variables that may modify the risk of restenosis after stent implantation.

Methods

Patient Population

Consecutive patients who underwent successful native coronary artery stent implantation and who were eligible for 6-month angiographic follow-up by August 1, 1990, are described in this report. The inclusion and exclusion criteria for patient enrollment into this trial in the United States as well as the early poststent results with this stent have been described. Patients who received an earlier prototype rigid stent (the Palmaz stent) or who did not receive warfarin after hospital discharge were excluded.

Stent Design and Implantation

The Palmaz-Schatz stent is composed of two 7-mm-long slotted stainless steel tubes connected by a 1-mm bridging strut to allow for longitudinal flexibility. The individual strut diameter is 0.075 mm. When balloon-expanded, the stent slots take on a diamond configuration (see Figure 1) that resists compression and has very limited recoil. In animal models, the stent becomes covered with platelets and fibrin within minutes to hours after implantation and is fully endothelialized at 4 weeks. The technique of stent implantation has been described. Throughout most of this experience, operators attempted to reduce the final diameter stenosis to <20%. As data suggesting that an even better result might lower the likelihood of restenosis became available, further balloon expansion of the stent was often performed. Before stent implantation, all patients received aspirin (325 mg p.o. q.d.), dipiridamole (75 mg p.o. t.i.d.), a calcium channel antagonist, dextran-40 (100 ml/hr for 2–4 hours), heparin (10,000–15,000 units intravenous bolus followed by 2,000–5,000 units/hr during the procedure), and intravenous nitroglycerin (20–100 mg/min titrated to a systolic blood pressure of 100–110 mm Hg). Patients were continued on dextran for 12–24 hours and heparin until therapeutic on warfarin, which was continued and titrated to a prothrombin time of 16–18 seconds (INR [international normalized ratio]=1.8–2.4), for 1–3 months. Aspirin, dipiridamole, and the calcium channel antagonist were continued for 3 months, after which time the latter two were often discontinued.

Initial and Follow-up Angiography

Predilatation, postdilatation, and immediate poststent angiography of the stenosis treated was performed in a minimum of two orthogonal views best documenting the stenosis and after intracoronary administration of nitroglycerin. Follow-up angiography was performed for clinical indications and also by protocol ≥4 months after stent implantation. At follow-up angiography, care was taken to replicate the angiographic angles and nitroglycerin usage of the first angiograms. Follow-up angiograms obtained less than 4 months after stenting were excluded from this analysis unless they demonstrated restenosis at the stent site.

Angiographic Analysis

All angiograms were forwarded to a core angiographic laboratory at Thomas Jefferson University Hospital (Philadelphia, Pa.) for quantitative analysis. Relative and absolute dimensional measurements were obtained using a validated computer-assisted ADAC system (ADAC Laboratories, Edenvale, Calif.). Mean variability for repeated measurements performed on separate days is ±0.10 mm for minimal lumen diameter and ±4.2% for percent diameter stenosis. The angiographic catheter was used to calibrate the dimensions for analysis. Results reported herein were recorded as the mean value from orthogonal projections when nonoverlapped and nonforeshortened orthogonal views were available. A small number of angiograms could not be processed by this system, usually because they had been obtained using digital acquisition on tape format and were analyzed by digital calipers. The similarity between the results obtained by these techniques has been described.

Definitions

Definitions are as follows: adequate angiographic follow-up: angiographic follow-up ≥4 months after stent implantation or >1 month after stenting if restenosis could be documented; optimal angiographic result: immediate poststent percent diameter stenosis ≤0 compared with adjacent reference diameter; restenosis: ≥50% diameter stenosis at follow-up.

Statistical Analysis

All data are expressed as mean±SD unless otherwise indicated. Group differences between continuous and categorical outcomes for normally distributed variables were assessed using the Student’s t test and χ² analysis with Yates’s correction where applicable, respectively. For variables with non-Gaussian distribution (e.g., poststent percent diameter stenosis), Kruskal-Wallis one-way ANOVA testing was used to assess differences in outcome. Effects were considered significant when the null hypothesis could be rejected with 95% confidence (two-sided testing). Independent correlates of restenosis and significant luminal renarrowing were determined by multivariate logistic regression analysis and multivariate linear regression analyses, respectively. Only stenoses that had been quantitatively analyzed by using the ADAC system were used to determine the correlates of significant luminal renarrowing. The variables tested as possible correlates of these end points were limited to those previously shown from multiple studies to be correlates of postangioplasty restenosis and those shown in prior preliminary studies to correlate with restenosis after stenting in order to minimize the likelihood of a type I statistical error. The variables evaluated were chronic total occlusion, current smoking at the time of stent implantation, diabetes mellitus, sex, left anterior descending coronary artery stenosis, multiple stent implantation, normal arterial diameter adjacent to the segment stented, poststent percent diameter stenosis, prior restenosis at the site stented, optimal angiographic result, proximal vessel stenosis stented, and unstable angina pectoris. All analyses were performed using SYSTAT software.
Results

Baseline Patient and Stenosis Characteristics and Procedural Outcome

The characteristics of the patients and stenoses with and without angiographic follow-up are enumerated in Table 1. There were no significant differences between patients with and without angiographic follow-up with respect to baseline characteristics or initial results.

Absence of Late Angiographic Follow-up

Six patients (2.9%) of the total cohort of 206 patients had stent closure 2–21 days after implantation and were not considered eligible for this analysis. Nineteen of the 200 patients eligible for late (≥4 months) follow-up did not have angiography performed. Four patients underwent angiography 2–3 months after stent implantation and were not found to have restenosis at that time but were not restudied later and were asymptomatic at latest follow-up. Eleven patients refused angiographic restudy (all currently with angina class 0 or 1), three patients had early noncardiac death, one patient had an arrhythmia death without evidence of myocardial ischemia, and no patients were lost to clinical follow-up.

Overall Incidence of Restenosis

For the 90.5% of patients with angiographic follow-up, the overall incidence of restenosis was 36.4%. Expressed on a per-stenosis basis, there was a 90.6% follow-up and a 35.7% (95% confidence intervals, 28.7–42.7%) incidence of restenosis. Based on equations predicting the likelihood of having restenosis developed by the M-HEART group,36,37 and excluding the influence of prior restenosis present in many of these patients that might be expected to increase their risk of restenosis,38,39 the restenosis rate for this group might be expected to be about 34%. Mean angiographic follow-up occurred at 5.8±2.1 months (range, 2–12 months). The average change in luminal dimension of the region stented from the immediate procedural result to follow-up was 0.99±0.73 mm, and in the vast majority of stenoses the regrowth occurred diffusely throughout the stented area.

Correlates of Restenosis and Luminal Renarrowing

As shown in Tables 2 and 3 and Figures 2 and 3, the placement of multiple overlapping stents was associated with a much higher incidence of restenosis and a greater luminal renarrowing than placement of a single stent (both multivariate p<0.001) and was the only significant independent correlate of restenosis. Figure 2 illustrates the apparent importance of stent overlap in the pathogenesis of luminal renarrowing. Patients with prior restenosis (p=0.01) or with chronic total occlusion (p=0.06) also had a higher likelihood of restenosis. The degree of luminal renarrowing was independently correlated with poststent percent diameter stenosis (greater displacement of the angiographically apparent stenosis) (p<0.001), the placement of multiple stents (p=0.002), and after angioplasty and stent placement in a previously chronically occluded artery (p=0.03).

Incidence and Correlates of Restenosis After Single-Stent Implantation

The incidence of restenosis after single-stent implantation was 30.2% (95% confidence interval, 23.1–37.3%). The estimated likelihood of developing reste-
TABLE 1. Baseline Characteristics

<table>
<thead>
<tr>
<th>Patients</th>
<th>Angiographic follow-up</th>
<th>No angiographic follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>181</td>
<td>19</td>
</tr>
<tr>
<td>Sex (% male patients)</td>
<td>76.2</td>
<td>68.4</td>
</tr>
<tr>
<td>Age (years)</td>
<td>57±13</td>
<td>56±14</td>
</tr>
<tr>
<td>Diabetes mellitus (%)</td>
<td>18.8</td>
<td>31.6</td>
</tr>
<tr>
<td>Current smoking (%)</td>
<td>12.7</td>
<td>10.5</td>
</tr>
<tr>
<td>Unstable angina pectoris (%)</td>
<td>39.2</td>
<td>31.6</td>
</tr>
</tbody>
</table>

Stenoses

| Number | 193 | 20 |
| Artery site | | |
| Left main (%) | 0.0 | 5.0 |
| LAD (%) | 36.3 | 45.0 |
| LCx (%) | 10.9 | 10.0 |
| RCA (%) | 52.8 | 40.0 |
| Normal artery diameter (mm) | 3.2±0.5 | 3.0±0.6 |
| Pre-PTCA % diameter stenosis | 72±16 | 72±8 |
| Bend stenosis (%) | 10.9 | 5.0 |
| Calcification (%) | 17.1 | 30.0 |
| Chronic total occlusion (%) | 6.2 | 5.0 |
| Diffuse disease (%) | 29.5 | 10.0 |
| Eccentric stenosis (%) | 49.2 | 40.0 |
| Prior restenosis (%) | 72.5 | 65.0 |
| >2 | 11.4 | 10.0 |

Stenosis length (mm) | 9±7 | 6±2 |

Initial results

| Multiple stents placed (%) | 17.1 | 20.0 |
| Poststent % diameter stenosis (25th/50th/75th percentiles) | 7/15/26 | 5/17/24 |
| Poststent minimal lumen diameter (mm) | 2.7±0.5 | 2.6±0.5 |
| Optimal angiographic result (%) | 13.7 | 10.0 |
| ADAC computer-assisted analysis (93%) | 9.3 | . . . |

LAD, left anterior descending coronary artery; LCx, left circumflex coronary artery; RCA, right coronary artery; PTCA, percutaneous transluminal coronary angioplasty.

Discussion

Recent placebo-controlled randomized trials with high rates of angiographic follow-up have reported a 28–41% incidence of restenosis after coronary angioplasty.7,9,10,13 The annual cost of revascularizing patients with restenosis after coronary angioplasty in the United States has been estimated to be 3.8 billion dollars.34 To date, no pharmacological16–13 or mechanical14,15,40 intervention has consistently decreased the likelihood of restenosis after successful coronary angioplasty.

Many different definitions of restenosis have been used to assess the long-term results of coronary angioplasty.41–45 Serruys and colleagues41 have emphasized that different definitions of restenosis will yield incongruous results. To assess the clinical importance of luminal renarrowing, we used the definition ≥50% diameter stenosis at follow-up on the basis of the clinical standard of care. The incidence of restenosis by this definition, however, is strongly influenced not only by the amount of serial recoil46 or luminal renarrowing but also by the initial mechanical result or platform upon which myointimal regrowth takes place. To better understand the effect of stenting on late dimensional change per se, we compared the absolute change in luminal dimensions after stenting and at follow-up with those reported after standard balloon angioplasty and also analyzed the factors affecting luminal renarrowing after stenting.

Several intracoronary stents are currently undergoing clinical or late preclinical evaluation.24–27,47,48 The stainless steel Palmaz-Schatz stent appears to have good biological compatibility based on comparison of reported neointimal thicknesses and rates of thrombosis with other stents in animal models, with the acknowledged caveat that comparison between different animal models must be done cautiously. Conversely, however, it is minimally radio-opaque, which makes its precise placement difficult and may lead to inadvertent stent overlap when two stents are to be placed adjacent. This stent is thrombogenic, as are all other stents currently under evaluation, and patients are necessarily placed at some risk of bleeding by the systemic anticoagulation required. Recently, Serruys et al49 reported a lower restenosis rate (14%) but a much higher early abrupt reclosure rate (24%) compared with 3%25 with the initial Wallstent experience. This stent is self-expanding, longer, and covers a greater surface area per unit length than does the Palmaz-Schatz stent.50 Restenosis data with other stents are not available from a large series of patients.

Factors Predisposing to Restenosis

These results highlight the difference in restenosis incidence between stenoses requiring one stent and multiple overlapping stents (30% versus 64%, p<0.001) that had previously been suggested in analyses of smaller numbers of stenoses.31,51 Greater metal density probably increases surface thrombogenicity and by inference, possible myointimal regrowth.16,50–54 It has
TABLE 2. Correlates of Restenosis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Risk ratio</th>
<th>Univariate p</th>
<th>Multivariate coefficient estimate±SE</th>
<th>Multivariate p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiple stents</td>
<td>2.11</td>
<td>&lt;0.001</td>
<td>0.99±0.41</td>
<td>0.007*</td>
</tr>
<tr>
<td>History of restenosis</td>
<td>1.74</td>
<td>0.03</td>
<td>0.85±0.37</td>
<td>0.01*</td>
</tr>
<tr>
<td>Chronic total occlusion</td>
<td>2.50</td>
<td>0.002</td>
<td>0.93±0.60</td>
<td>0.06*</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.45</td>
<td>0.14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single stent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of restenosis</td>
<td>2.23</td>
<td>0.015</td>
<td>0.73±0.42</td>
<td>0.04†</td>
</tr>
<tr>
<td>Optimal angiographic result</td>
<td>0.17</td>
<td>0.018</td>
<td>-0.67±0.41</td>
<td>0.05†</td>
</tr>
</tbody>
</table>

All variables with univariate \( p < 0.15 \) are shown.
*Log likelihood = -119.22; constant = -1.50. †Log likelihood = -92.77; constant = -1.33.

been suggested that the passivation of stainless steel or other surfaces early after contact with blood elements may decrease thrombogenicity.\(^{55}\) Repeated stent-on-stent trauma with overlapping stents might impair passivation, promote thrombus formation, and thereby increase the risk of restenosis (see Figure 2).

A history of restenosis was also associated with a greater likelihood of restenosis after placement of a single Palmaz-Schatz stent (multivariate \( p = 0.01 \)). Restenosis, particularly soon after\(^{58}\) or multiple times after coronary angioplasty,\(^{30}\) appears to heighten the risk of restenosis after PTCA. These factors also increase the risk of restenosis after other coronary interventions such as atherectomy.\(^{56}\) Specimens retrieved and analyzed after atherectomy or at autopsy suggest that arterial trauma incites a change in the smooth muscle cell phenotype from contractile to synthetic,\(^{57,58}\) and it has been suggested that trauma to an area containing many phenotypically synthetic smooth muscle cells may heighten the risk of restenosis.

The extent of residual stenosis after stenting was highly correlated with the likelihood of subsequent restenosis. A stent dimension nearly approximating the adjacent vessel would provide a larger scaffold such that more myointimal hyperplasia would be required to encroach upon the normal lumen until a \( \geq 50\% \) diameter stenosis results. Importantly, however, a small residual stenosis was also correlated with increased myointimal regrowth, perhaps implying that at some dimension the effect of increased vessel wall stretch or trauma outweighed a possible benefit of lessened blood turbulence with its attendant tendency for less platelet dep-

TABLE 3. Correlates of Luminal Renarrowing

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate correlation coefficient</th>
<th>Univariate p</th>
<th>Multivariate coefficient</th>
<th>Multivariate p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall results</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poststent % diameter stenosis</td>
<td>-0.017±0.005</td>
<td>&lt;0.001</td>
<td>-0.018±0.004*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Multiple stents</td>
<td>0.522±0.153</td>
<td>0.001</td>
<td>0.491±0.156*</td>
<td>0.002</td>
</tr>
<tr>
<td>Chronic total occlusion</td>
<td>0.775±0.245</td>
<td>0.002</td>
<td>0.534±0.241*</td>
<td>0.03</td>
</tr>
<tr>
<td>History of restenosis</td>
<td>0.225±0.134</td>
<td>0.10</td>
<td></td>
<td>NS</td>
</tr>
<tr>
<td>Single stents only</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poststent % diameter stenosis</td>
<td>-0.013±0.005</td>
<td>0.008</td>
<td>-0.013±0.005†</td>
<td>0.008</td>
</tr>
</tbody>
</table>

All variables with univariate \( p \leq 0.15 \) are shown.
*Constant=1.033; †constant=1.108.
late loss" was first reported by Kuntz et al. This conclusion derives from a comparison of the results of the absolute dimensions after stenting reported herein with available dimensions reported after coronary angioplasty. A composite of results from Serruys et al. and Popma and Dehmer finds a postangioplasty minimum luminal diameter of 2.1 mm in their carefully studied postangioplasty series. Final poststent lumen dimension was 2.7 mm in this series. That this is not entirely the result of treatment stenoses in larger vessels with stents is suggested by a comparison of the postangioplasty versus poststent diameter stenoses, 26±13% versus 16±12%. Importantly, the late loss described in these reports after angioplasty appears to be less than that seen after stenting in this series: 0.4±0.7 mm versus 0.9±0.7 mm.

**Limitations**

Several limitations inherent to this study should be considered in its interpretation. First, this report summarizes the initial results with this device, and the technique of stent implantation continues to evolve. Second, as with all clinical studies, complete angiographic follow-up was not obtained. Analyses of the effect of incomplete angiographic follow-up suggest that estimates assuming that patients without follow-up do not have restenosis, based upon the presumption that asymptomatic patients will be less likely to have restenosis and also will be less likely to return for follow-up, underestimate the true risk of restenosis, whereas assuming that the incidence of restenosis within the group returning for follow-up is the same as the entire patient population overestimates the restenosis rate. Uncertainty increases with diminishing follow-up. Nonetheless, the 90% follow-up in this series is similar or better than that reported in most other recent restenosis series. Third, patients chosen for stenting are not at all representative of all patients undergoing coronary angioplasty; hence, comparison with reports after balloon angioplasty should be made with extreme caution. The incidences of current smoking and unstable angina, factors sometimes linked to resteno-

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**Figure 2.** Photomicrograph of necropsy specimen from a 63-year-old man who had undergone left circumflex coronary artery stent placement 2 months before death from an intracranial hemorrhage. Note the area of enhanced neointimal thickening in the area of stent overlap (arrows).

**Figure 3.** Bar graph shows incidence of observed restenosis in lesion subsets of interest. 1, Single stent; 2, single stent, left anterior descending coronary artery (LAD) only; 3, single stent and no prior restenosis; 4, single stent and prior restenosis; 5, single stent and final stenosis >0%; 6, single stent and final stenosis ≤0%; 7, multiple overlapping stents.
Implications

The implications of these results are multiple and must be understood in the context of the relatively high safety but finite risk of the procedure:\(2^{4}\): 1) There probably is no role for Palma-Schatz stent placement to prevent restenosis when multiple overlapping stents will be required, 2) randomized trials will be required to ascertain if the restenosis results achieved after single-stent placement are superior to those after coronary angioplasty alone and can be achieved with a low enough risk to be justifiable, 3) angioplasty operators should attempt to approximate the adjacent “normal” lumen diameter with stent placement, but given the increase in luminal narrowing when a small final diameter stenosis was achieved noted in this study and experimental results linking myointimal proliferative response to stent-induced arterial trauma,\(^5\) caution must be urged not to vigorously overdilate stented arteries, and 4) given the apparent lack of reduction of luminal regrowth after stenting compared with PTCA alone, it appears that pharmacological means of limiting the proliferative response to injury will be required to further diminish the incidence of restenosis after balloon angioplasty. Finally, improvements in stent design that may markedly affect thrombogenicity and possibly restenosis are expected,\(^6\) and these results should not be generalized to all coronary stents.

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References


Restenosis After Intracoronary Stent Implantation


34. europas: A randomized clinical trial of patients with unstable angina pectoris: Analysis of early and late results. *Am Heart J* 1983;106:973–980


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S G Ellis, M Savage, D Fischman, D S Baim, M Leon, S Goldberg, J W Hirshfeld, M W Cleman, P S Teirstein and C Walker

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