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Serial Angiographic Follow-Up After Palmaz-Schatz Stent Implantation: Comparison With Conventional Balloon Angioplasty

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Objectives. Serial angiographic follow-up study was designed to evaluate the temporal mode of lumen diameter changes after Palmaz-Schatz stent implantation, and the results were compared with those from a cohort of patients undergoing balloon angioplasty.

Background. Restenosis remains a major limitation of balloon angioplasty. The Palmaz-Schatz balloon expandable coronary stent is now under clinical investigation to evaluate its efficacy in preventing restenosis.

Methods. Serial angiographic follow-up study was performed the day after stent implantation and at 1, 3 and 6 months after the procedure. The stent group consisted of 96 patients who had 97 lesions with a single stent. A cohort of 179 patients with 192 lesions were selected as the balloon group by the criteria of final balloon size ≥3 mm and lesion length <20 mm.

Results. A significantly larger lumen diameter was obtained immediately after stent implantation (2.9 ± 0.4 mm [mean ± SD] in the stent group vs. 2.1 ± 0.5 mm in the balloon group, $p < 0.001$). At 3 to 6 months of follow-up, a significantly larger lumen diameter was maintained in the stent group (2.2 ± 0.6 vs. 1.5 ± 0.7 mm, $p < 0.001$). The late restenosis rate according to a binary definition was significantly lower in the stent group (13% vs. 39%, $p < 0.001$). Stenosis exacerbation, frequently observed within 24 h after balloon angioplasty, was not found after stenting. Between the next day and 1 month, regression was dominant in the balloon group, whereas progression of stenosis was observed in the stent group. The greatest tendency to restenosis was observed in both groups between 1 and 3 months after the procedure. Between 3 and 6 months, significantly greater diameter loss was found in the stent group.

Conclusions. The Palmaz-Schatz stent was effective in reducing the restenosis rate in this highly selected cohort of patients. Reduction in restenosis rate was dependent on a larger lumen obtained immediately. Late loss of diameter was significantly greater after stenting. The restenosis rate after stenting should be evaluated by follow-up angiography at 6 months rather than at 3 months, which is adequate after conventional balloon angioplasty.

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Coronary balloon angioplasty constitutes a definitive advance in the treatment of coronary artery disease. Despite improvements in technique and equipment, restenosis remains a major limitation of coronary balloon angioplasty (1–3). Various new mechanical approaches have been undertaken to prevent restenosis but without proved efficacy. One such device, the Palmaz-Schatz stent, is reported to be effective in optimizing the initial lumen result after balloon angioplasty (4,5). Although its efficacy in preventing restenosis is not yet established, several observational reports (4,6–9) suggest a promising result in this respect. To evaluate the temporal mode of lumen diameter changes after stent placement, we performed serial angiographic follow-up on the next day and at 1, 3 and 6 months after Palmaz-Schatz stent implantation. The data were retrospectively compared with those from conventional balloon angioplasty to evaluate qualitative and quantitative differences in restenosis.

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Methods

Study patients. *Stent group.* From June 1990 to July 1991, Palmaz-Schatz stent implantation for native coronary arteries was attempted in 99 patients with 100 lesions. All were single-stent cases. Successful stent implantation was defined as successful delivery of stents and residual lumen diameter stenosis of <50%. Procedural success was achieved in 96 patients with 97 lesions (97%). Subacute stent thrombosis occurred in four lesions (4.1%). All four stents were successfully reopened by intracoronary thrombolysis or repeated balloon dilation, or both. Therefore, 97 lesions were patent at the time of hospital discharge. The Palmaz-Schatz stent group consisted of these 96 patients with 97 lesions.

Conventional balloon angioplasty group. Between January 1986 and August 1986, 546 patients underwent successful balloon angioplasty. Two-hundred twenty-nine patients with 288 lesions were prospectively enrolled in a serial angiographic follow-up study. The results from the total group have been published elsewhere (1). To eliminate small-sized arteries and diffuse disease unfavorable to stent implantation, a cohort of 179 patients with 192 lesions were selected by the criteria of final balloon size ≥3 mm and lesion length <20 mm. Patients and the responsible relatives gave in-

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formed consent to the interventional procedure and the follow-up protocol. The institutional ethical committee approved the study protocol.

Stent placement. All stent implantation procedures were performed using the Johnson & Johnson stent delivery system and standard techniques (9). When further expansion of the stent was considered necessary after initial stent deployment, a larger balloon catheter was used to overexpand the stent.

Pharmacologic management. Routine antianginal medications consisting of long-acting nitrates and calcium channel antagonists were continued. At least 1 day before the procedure, aspirin, 240 mg, and dipyridamole, 75 mg, were initiated as antiplatelet therapy. Low molecular weight dextran was administered by intravenous infusion starting 2 h before the procedure and continuing for a total dose of 500 ml. At the time of arterial access, heparin, 10,000 U, was injected by way of the femoral artery sheath. Intracoronary isosorbide dinitrate, 2.5 mg, was administered before control angiography and after the procedure. The same protocol with regard to isosorbide dinitrate was used at all subsequent follow-up studies. Immediately after stent implantation, intravenous heparin infusion, 200 U/kg per day was started, and after sheath removal on the next morning the heparin dose was adjusted to keep activated partial thromboplastin time at 60 to 80 s. Intravenous heparin infusion was continued until oral anticoagulant (Coumadin) therapy achieved therapeutic effect (prothrombin time 16 to 18 s). Coumadin was continued for 3 months. In patients undergoing conventional balloon angioplasty, intravenous heparin infusion was performed only when angiographic evidence of dissection was noted. Coumadin was not administered after conventional balloon angioplasty.

Serial coronary angiography. Follow-up angiography was performed on the next day and at 1, 3 and 6 months after the procedure regardless of patients' symptomatic status. Follow-up angiography was performed on an outpatient basis with use of a 5F femoral or a 6F brachial diagnostic catheter. The restudy rate of each cohort is shown in Table 1. The lower restudy rate on the next day and at 1 month in the stent group was due to unequivocal results obtained from earlier patients. The protocol of angiography on the next day and at 1 month was discarded in later patients. Follow-up angiograms at 3 and 6 months were available in >90% of patients in both groups.

Measurement of coronary stenosis. Severity of coronary stenotic lesions in the balloon group was assessed by quantitative coronary angiography with cinevideodensitometric analysis using the Vanguard XR-70 coronary analyzer. Details of measurement have been reported previously (1). Stenosis measurement in the stent group was performed by calipers. A worst view after stent implantation was selected from multiprojections. Stenosis measurement of control and follow-up angiograms was performed in nearly identical views. *Absolute lumen diameter* was calculated from the known diameter of the guiding and diagnostic catheters. *The reference diameter* was measured at the proximal or distal segment that was apparently free of disease. The stenotic segment was selected from both the stented portion and the nearby nonstented area. *Angiographic restenosis* was defined as ≥50% diameter stenosis anywhere within or adjacent to the stented segment. The *acute gain* in lumen diameter was defined as the difference in minimal stenosis diameter before and after the procedure. The *late loss* was defined as the difference between stenosis diameter after the procedure and that at follow-up.

Table 1. Follow-Up Angiographic Protocol

	Balloon Angioplasty Group		Stent Group	
	Interval (days)	No. (% eligible)	Interval (days)	No. (% eligible)
Post	—	192	—	97
Day 1	1	155 (81)	1	69 (71)
1 month	34 ± 6	192 (100)	35 ± 6	79 (81)
3 months	94 ± 9	183 (99)	97 ± 14	95 (98)
6 months	186 ± 13	129 (91)	194 ± 18	85 (93)

Unless otherwise indicated, data are expressed as mean value ± SD. Post = immediately after the procedure.

Statistical analysis. Values are expressed as mean value ± 1 SD. Categoric variables were analyzed by the chi-square test. Continuous variables were analyzed with a two-factor analysis of variance; $p < 0.05$ was regarded as significant.

Results

Clinical results of stent placement. Palmaz-Schatz stent implantation was attempted in 99 patients with 100 lesions. Seventy-four stents were attempted electively and 26 stents in the emergency setting of occlusion or dissection after balloon angioplasty, or both. In three lesions, the stent could not be delivered owing to proximal vessel tortuosity. Therefore, procedural success was achieved in 96 patients with 97 lesions (97%). Procedural success was obtained in all 74 lesions for elective stenting, and in 23 of 26 lesions (88%) for emergency procedures. There were no acute vessel closures within 24 h after successful stent implantation. There were four subacute thrombotic closures (4.1%) between 5 and 7 days after the procedure. The subacute thrombosis rate was 1.4% in elective stenting in contrast to 13.0% in emergency procedures. Stent-related myocardial infarction occurred in six patients (6.2%). The causes of stent-related myocardial infarction were subacute thrombosis in three patients, side branch occlusion in two patients and vessel closure after unsuccessful stent delivery in one patient. There were no emergency bypass operations or in-hospital deaths. Bleeding complications requiring blood transfusion or surgical repair occurred in two patients (2.1%).

Baseline patient and lesion characteristics (Table 2). Baseline patient and lesion characteristics differed in several aspects in the two groups. Mean age was significantly higher in the stent group, and this group included significantly more patients with previous coronary angioplasty and fewer patients with acute myocardial infarction. The stent group included significantly more left main coronary artery lesions, fewer left circumflex artery lesions and more restenotic

Table 2. Baseline Patient and Lesion Characteristics

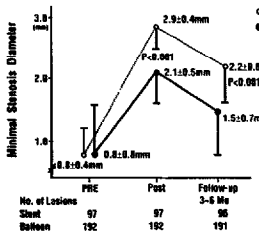
	Balloon Angioplasty Group	Stent Group	p Value
Patients			
No.	179	96	
Age (yr)	60 ± 10	63 ± 9	< 0.05
Mean range	34 to 78	34 to 80	
M/F	130/49	74/22	NS
Prior PTCA	55 (31)	66 (69)	< 0.001
Prior MI	92 (51)	51 (53)	NS
Acute MI	17 (9)	1 (1)	< 0.05
Extent of CAD			
1-VD	106 (58)	50 (52)	NS
2-VD	43 (24)	26 (27)	NS
3-VD	21 (12)	9 (9)	NS
LMCA	2 (1)	2 (2)	NS
Post CABG	7 (4)	9 (9)	NS
Lesions			
No.	192	97	
Location			
LAD	108 (56)	49 (51)	NS
RCA	49 (26)	33 (34)	NS
LCx	33 (17)	7 (7)	< 0.05
LMCA	2 (1)	8 (8)	< 0.01
Restenotic lesion	53 (28)	52 (54)	< 0.001
Lesion length (mm)	<20	9.0 ± 4.0	
Reference diameter (mm)	3.0 ± 0.6	3.4 ± 0.6	< 0.001
Final balloon size (mm)	3.1 ± 0.3	3.5 ± 0.4	< 0.001

Data are expressed as mean value ± SD or number (%) of patients or lesions. CABG = coronary artery bypass graft; CAD = coronary artery disease; F = female; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; LMCA = left main coronary artery; M = male; MI = myocardial infarction; Post = after; PTCA = percutaneous transluminal coronary angioplasty; RCA = right coronary artery; VD = vessel disease.

lesions. Mean reference diameter and final balloon size were significantly greater in the stent group.

Minimal stenosis diameter before, immediately after stent implantation and at 3 to 6 months of follow-up (Fig. 1). Minimal stenosis diameter before the procedure was not

Figure 1. Changes in minimal stenosis diameter before (Pre), after (Post) and at 3 to 6 months of follow-up after Palmaz-Schatz stent implantation (**open circles**) and balloon angioplasty (**solid circles**).



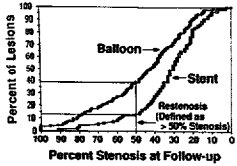
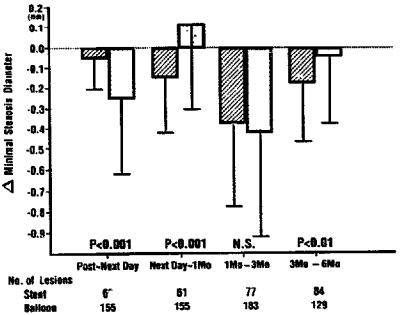


Figure 2. Cumulative distribution function curve of minimal lumen diameter at follow-up. Restenosis was defined as ≥50% diameter stenosis at follow-up. Any angiograms showing restenosis and angiograms taken after 3 months without showing restenosis were regarded as valid for evaluation of restenosis rate.

different between the balloon and stent groups. A significantly larger lumen diameter was obtained immediately after stent implantation (2.9 ± 0.4 mm in the stent group vs. 2.1 ± 0.5 mm in the balloon group; $p < 0.001$). At 3 to 6 months of follow-up, a significantly larger lumen was maintained in the stent group (2.2 ± 0.6 vs. 1.5 ± 0.7 mm, $p < 0.001$). Acute gain in lumen diameter was significantly larger in the stent group (2.2 ± 0.5 vs. 1.3 ± 0.6 mm; $p < 0.001$). Late loss was also significantly greater in the stent group (0.7 ± 0.6 vs. 0.5 ± 0.7 mm, $p < 0.01$). Late restenosis rate according to a binary definition was significantly lower in the stent group (12 [13%] of 96 vs. 74 [39%] of 191, $p < 0.001$) (Fig. 2).

Changes in minimal stenosis diameter in each follow-up interval (Fig. 3). Between the period immediately after balloon angioplasty and the next day, minimal stenosis

Figure 3. Changes (Δ) in minimal stenosis diameter at each follow-up interval in the balloon angioplasty (**gray bars**) and stent (**hatched bars**) groups. Positive values for changes in minimal stenosis diameter signify interval regression of stenosis; negative values represent interval progression. Numbers of paired angiograms available are shown at **bottom**.



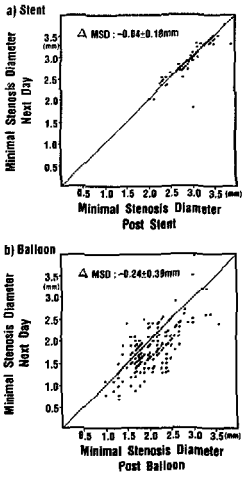


Figure 4. Changes in minimal stenosis diameter (Δ MSD) between the period immediately after (Post) the procedure and the next day. a, Stent group. b, Balloon angioplasty group.

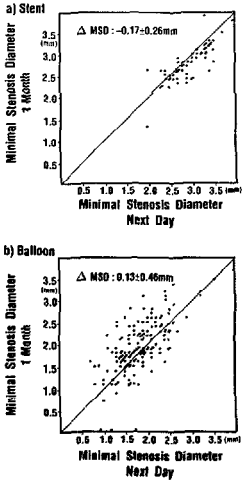


Figure 5. Changes in minimal stenosis diameter between the next day and 1 month after the procedure. a, Stent group. b, Balloon angioplasty group. Abbreviations as in Figure 4.

diameter tended to decrease (−0.24 ± 0.39 mm) (Fig. 4). In contrast to this tendency toward stenosis exacerbation in the balloon group, stenosis diameter in the stent group was remarkably constant during this interval (−0.04 ± 0.18 mm). Significant loss of diameter in two cases was observed, not inside the stents but at the junction of the stented and nonstented portion of the artery. Between the next day and 1 month follow-up, stenosis diameter tended to increase in the balloon group and decrease in the stent group (0.13 ± 0.46 vs. −0.17 ± 0.3 mm) (Fig. 5). Between 1 and 3 months, the greatest tendency to restenosis was observed in both groups (−0.42 ± 0.51 vs. −0.37 ± 0.41 mm) (Fig. 6). Despite this diameter loss, most lesions in the stent group sustained good patency. Between 3 and 6 months, stenosis diameter remained relatively constant in both groups (Fig. 7). However, mean loss of diameter was significantly greater in the stent group (−0.03 ± 0.36 vs. −0.16 ± 0.32 mm).

Discussion

Prevention of restenosis by the Palmaz-Schatz stent. The idea of preventing restenosis by implanting a stent was introduced by Dotter and Judkins in 1964 (10). Currently, several stent designs are under clinical investigation. Sigwart et al. (11) reported the first implantation of self-expanding stents in the coronary circulation of humans. Serruys et al. (12) reported angiographic follow-up results of placement of a self-expanding stent. Despite an apparently promising late restenosis rate (14%), an unacceptably high stent thrombosis rate (24%) makes these data difficult to interpret. Roubin et al. (13) reported a late restenosis rate of 41%, with 76% angiographic follow-up after balloon-expandable coil stent implantation in bailout situations. With regard to the Palmaz-Schatz stent, long-term angiographic follow-up results in large patient series are not yet fully available; however, the data to date suggest that the Palmaz-Schatz stent is promising in preventing restenosis. Carrozza et al. (9) reported a 25% restenosis rate in 117 lesions. Schatz et al. (7) reported a 20% restenosis rate in 103 patients with single-stent implantation. Haude et al. (8) reported a 15% restenosis rate in 33 patients. Our data of a 13% restenosis rate in 109 lesions with 98% angiographic follow-up are comparable to those restenosis figures. Of course, it is premature to conclude that the Palmaz-Schatz stent really reduces the late restenosis rate after balloon angioplasty. In the present study, there are several important differences in the baseline characteristics between the stent and balloon groups. The stents were implanted preferentially in discrete regions in larger arteries. Lesion length and arterial diameter are reported to be significant predictors of restenosis (3), whereas a 55% inci-

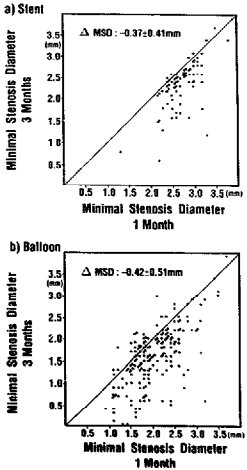


Figure 6. Changes in minimal stenosis diameter between 1 and 3 months after the procedure. **a**, Stent group. **b**, Balloon angioplasty group. Abbreviations as in Figure 4.

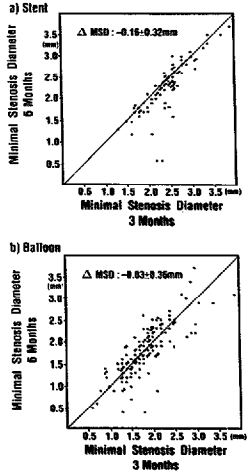


Figure 7. Changes in minimal stenosis diameter between 3 and 6 months after the procedure. **a**, Stent group. **b**, Balloon angioplasty group. Abbreviations as in Figure 4.

dence of restenotic lesions in the stent group is considered to be predisposing to an increased incidence of restenosis. In the future, carefully designed randomized trials will clarify the true restenosis rate of the Palmaz-Schatz stent in a large patient cohort comparable to balloon angioplasty. At this point, however, a restenosis rate of <20% in a highly selected cohort of patients seems to be a remarkable step toward prevention of restenosis.

The mechanisms of restenosis prevention, if it occurs at all, after Palmaz-Schatz stent implantation are not fully understood. Schatz (14) postulated in his early report that nonflexing rigid stents may create a localized region of reduced wall stress, thus inhibiting the cascade of events producing atherosclerosis. Levine et al. (4), however, reported a 0.40- to 0.68-mm thickness of the neointimal layer within even those stents free of restenosis. Recently, Kuntz et al. (15) introduced the terms "acute gain" and "late loss." They compared the apparent restenosis rates among the three interventions (19% for stents, 31% for atherectomy and 50% for laser balloon angioplasty). They concluded that the observed differences in restenosis rate could be explained by the differences in acute gain rather than late loss. Also in the present study, late lumen loss was significantly greater in the stent group than in the balloon group. Acute lumen gain, in contrast, was markedly greater in the stent group. Therefore, the predominant mechanism of preventing restenosis was considered to be dependent on its efficacy in optimizing the initial lumen geometry. Significantly greater late loss in the stent group was compensated by much greater acute gain. Beatt et al. (16) reported paradoxic increase in restenosis rate by a criterion of loss of an absolute lumen diameter ≥0.72 mm when a large improvement in the minimal lumen diameter was obtained at the time of balloon angioplasty. A larger acute gain is considered to be associated with more extensive vascular injury, resulting in greater late intimal hyperplasia. Kuntz et al. (17) reported that late loss is related to acute gain by a factor (the loss index) whose value for both stenting and balloon angioplasty is approximately 0.4. This relation correlated well with the observed late losses seen in our study.

Serial angiographic follow-up. Serial angiographic follow-up after Palmaz-Schatz stent implantation and balloon angioplasty revealed both different and common temporal features of lumen diameter changes. Immediately after the procedure, a significantly larger lumen was obtained in the stent group with use of a larger final balloon size. Percent recoil comparing final balloon size and minimal lumen diameter after the procedure was greater in the balloon group than in the stent group (30% vs. 15%, respectively). It is apparently curious to find elastic recoil after implantation of this rigid, plastically deformable

stent. The Palmaz-Schatz stent, however, has an articulation point, and significant recoil is usually found at this point after overdilation. Bonner et al. (18) also reported 16% recoil in the acute period after Palmaz-Schatz stent implantation.

From immediately after the procedure to the next day. During this period, minimal stenosis diameter tended to decrease after balloon angioplasty. Postulated mechanisms of this early exacerbation of stenosis are elastic recoil, thrombus formation and propagation of dissection. This early progression of stenosis was not observed after stent implantation. A scaffolding effect of the implanted stent is considered to prevent recoil and propagation of dissection. Also, thrombus formation within 24 h might well be limited to such a small extent that it is undetectable by angiography.

From the next day to 1 month. During this interval, stenosis diameter tended to improve after balloon angioplasty. Pathologic studies (19,20) have shown intimal proliferation of smooth muscle cells to be active during this interval. Stenosis improvement during this interval could possibly be explained by the favorable remodeling of the disrupted intima or resolution of thrombus. In contrast to balloon angioplasty, significant progression of stenosis was observed in this interval after stent implantation. Schatz et al. (21) reported that after experimental stent implantation in canine coronary arteries, myofibroblastic cells replaced thrombus at 3 weeks. Morphometrically measured neointimal thickness at 3 weeks approximated 0.08 mm. This neointimal thickness correlated well with our mean lumen diameter loss of 0.22 mm at 1 month.

Between 1 and 3 months. The greatest tendency to restenosis was observed both after balloon angioplasty and Palmaz-Schatz stent implantation during this period. Histopathologic findings of stent restenosis in human coronary arteries are not yet well described. Bonner et al. (18) suggested that chronic stent compression rather than intimal hyperplasia is the principal cause of stent restenosis. Stent diameter was not measured in the present study because the Palmaz-Schatz stent is only faintly radiopaque, and measurement of stent diameter is not reliable with current radiographic equipment. However, in an experimental study, Schatz et al. (21) reported intense fibroblast proliferation at 8 weeks after stent implantation. Progression of stenosis during this interval suggests that the same mechanism of restenosis as that with balloon angioplasty (proliferation of smooth muscle cells) is responsible for stent restenosis.

Between 3 and 6 months. During this period, stenosis diameter was relatively constant in both groups, but mean loss of diameter was significantly greater in the stent group. Schatz et al. (21) reported sclerotic change and marked regression of neointima between 8 and 32 weeks after experimental stent implantation. This regression was not observed in human atherosclerotic coronary arteries, at least up to 6 months. Greater loss of diameter during this interval after stent implantation is difficult to interpret. In the balloon group, most patients with lesions with a high proliferative potential dropped out at 3 months owing to angiographic restenosis. In the stent group, even those highly proliferative lesions might remain patent owing to a larger lumen obtained immediately after the procedure. Inclusion of these lesions predisposing to restenosis might be related to greater diameter loss between 3 and 6 months after stent implantation; however, only about 15% of the total diameter loss by 6 months occurred between 3 and 6 months, suggesting that this process should have been completed by 6 months. Leon et al. (22) reported the result of longer term angiographic follow-up, showing no further reduction in lumen diameter after 6 months. Previously, we recommended follow-up angiography at 3 months after balloon angioplasty to detect restenosis earlier. Considering the much lower restenosis rate and minimal but nonnegligible progression until 6 months, the restenosis rate after stenting should be evaluated by follow-up angiography at 6 months.

Study limitations. This study has several important limitations. 1) Although serial angiographic follow-up was designed prospectively, comparison of the balloon and stent groups was retrospective. Selection criteria for the balloon group were arbitrary. 2) There were several important differences in baseline characteristics between the two groups. The restenosis rate is reported to be influenced by several variables, such as lesion length and location, vessel size and severity of stenosis before the procedure. To evaluate differences in restenosis rates, a comparison must be made between the two groups matched for these baseline characteristics. 3) Stenosis in the stent group was measured with calipers, whereas videodensitometry was utilized in the balloon group. After balloon angioplasty, the angiographic appearance of the dilated lesions is often shaggy and hazy, making caliper measurement less accurate. Caliper measurements, however, are reliable for measuring the round, smooth lumens such as those that occur after stent implantation. 4) The sample size is still too small to identify the actual restenosis rate of the Palmaz-Schatz stent. In addition, most lesions were carefully selected for stent implantation. The present results are not directly applicable to smaller arteries and longer lesions. Despite these study limitations, promising results in preventing restenosis warrant continued clinical research and randomized trials with other interventional devices.

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