Title

Incomplete Expansion of Palmaz-Schatz Stents despite High-Pressure Implantation Technique: Impact on Target Lesion Revascularization

Journal

Cardiology, 91(2)

ISSN

1422-9528

Authors

Blessing, Erwin
Hausmann, Dirk
Sturm, Matthias
et al.

Publication Date

1999

DOI

10.1159/000006888

Cardiac Catheterization

CARDIOLOGY

Cardiology 1999;91:102–108

Received: February 1, 1999
Accepted: February 8, 1999

Incomplete Expansion of Palmaz-Schatz Stents despite High-Pressure Implantation Technique: Impact on Target Lesion Revascularization

Erwin Blessing Dirk Hausmann Matthias Sturm Hans-Georg Wolpers
Ivo Amende Andreas Mügge

Division of Cardiology, Department of Internal Medicine, Hannover Medical School, Germany

Key Words
High-pressure implantation · Stent expansion · Intravascular ultrasound · Restenosis

Abstract
Improved expansion of stents using high-pressure implantation technique with subsequent antiplatelet therapy has improved patient outcome regarding the incidence of subacute stent thrombosis, bleeding complications and restenosis. Whether high-pressure implantation per se guarantees adequate stent expansion remains unclear. The aim of the study was to determine (1) stent expansion after high-pressure implantation technique and (2) whether stent expansion influences rate of target lesion revascularization within 6 months of follow-up. One hundred Palmaz-Schatz stents were implanted in 98 lesions (91 native vessels, 7 graft vessels) of 94 patients using high-pressure implantation technique (balloon pressure 12–20 atm). Stent expansion was investigated using intravascular ultrasound imaging (IVUS). Clinical follow-up of the patients was performed for 6 months. After implantation, stent/mean reference ratio was 0.81 ± 0.16. Noncompliant balloons used for implantation were chosen by angiographic criteria. Mean balloon/reference ratio was 1.08 ± 0.22; therefore balloons were not undersized. Additional balloon dilatation using higher pressures and/or larger balloons based on IVUS criteria and subsequent IVUS measurements was performed in 52 patients (55%); in these patients, stent expansion improved from 79 ± 16 to 91 ± 15% (mean ± SD) of average reference areas ($p < 0.002$). Within the 6 months' clinical follow-up, target lesion revascularization was performed in 19 patients (20%). The only prognostic factors for the development of in-stent restenosis requiring target lesion revascularization were the vessel size ($p < 0.05$) and the extent of plaque distal to the stents ($p < 0.05$). Implantation of Palmaz-Schatz stents using high-pressure technique does not guarantee adequate stent expansion. Additional dilatation with higher pressures and/or larger balloons improves stent expansion. The size of the stented vessel and the extent of plaque at the distal stent end (residual outflow stenosis) but not the degree of stent expansion were predictors for target lesion revascularization within 6 months' follow-up.

Introduction

Prior studies using intravascular ultrasound imaging (IVUS) demonstrated the absence of adequate stent expansion despite an acceptable angiographic result in

KARGER

Fax + 41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

0008–6312/99/0912–0102$17.50/0

Accessible online at:
http://BioMedNet.com/karger

Erwin Blessing, MD
University of Washington, Department of Pathobiology
Box 357238, Seattle, WA 98195-7238 (USA)
Tel. +1 206 543 5256, Fax +1 206 543 3873
E-Mail ebless@u.washington.edu

>80% of patients before the era of high-pressure implantation [1, 2]. In these days, stent thrombosis occurred in 3–4% of patients undergoing elective stent placement [3–5]. Consequent anticoagulation with heparin, coumadin and platelet inhibitors was therefore necessary. Anticoagulation, on the other hand, can lead to severe bleeding complications and also significantly prolongs the hospital stay. This has prompted investigators to develop a more aggressive strategy based on high-pressure balloon dilatation. Colombo and coworkers [6] showed that anticoagulation therapy was not obligatory as long as adequate stent expansion was achieved using high-pressure implantation technique, supporting the concept that stent thrombosis might be due to incomplete stent dilatation rather than to the thrombogenicity of the metallic struts.

Whether the incidence of acute complications as well as the restenosis rate after implantation of intracoronary stents can be further decreased using IVUS guidance is investigated in ongoing trials [7, 8]. The goal of this study was to determine (1) whether high-pressure implantation technique per se guarantees adequate stent expansion, and (2) whether stent expansion influences rate of target lesion revascularization at a 6-month follow-up.

Methods

Patients

Between October 1994 and July 1996 94 consecutive patients (80 male, 14 female; mean age 61 ± 10 years) underwent IVUS-guided implantation of 100 Palmaz-Schatz stents (Johnson & Johnson) in 98 coronary lesions (79 de novo lesions, 19 restenotic lesions). Indication for catheterization were stable angina pectoris (n = 59), unstable angina pectoris (n = 15) or prior myocardial infarction (n = 20). All patients were treated and imaged only after having given written, informed consent for participation in this study.

Stent Implantation

Preprocedural angiographic stenosis was ≥75% by visual estimate in all patients. The 98 lesions were located in the left anterior descending artery (n = 54), right coronary artery (n = 23), circumflex artery (n = 14) and saphenous vein grafts (n = 7). The types of Palmaz-Schatz stent implanted were: PS 104 (n = 23), PS 153 (n = 35), PS 154A (n = 30), ½ PS 153 (n = 7) and PS 204C (n = 5). Noncompliant balloon catheters used for stent implantation were chosen by angiographic criteria with a mean diameter of 3.3 ± 0.4 mm (2.5–5.0 mm). Inflation pressures for the initial stent deployment ranged between 12 and 20 atm (15.3 ± 3.9 atm). Patients were treated with a bolus dose of 10,000 U of heparin at the time of the sheath insertion and a repeat bolus dose of 5,000 U of heparin before stent implantation. After implantation, a combination of heparin and oral anticoagulant (n = 27) or antiplatelet (ticlopidine and/or aspirin) therapy (n = 68) was administered. In case of coumadin therapy, target range of international normalized ratio was set between 2.0 and 3.0 for 3 months. Ticlopidine therapy was discontinued after 4 weeks.

Ultrasound Imaging

After the angiographic completion of the interventional procedure, ultrasound imaging was performed. Over a 0.014-inch guidewire the 30-MHz single piezoelectric crystal catheter (Boston Scientific 3.5 french, CVIS, 2.9/3.2 french) was placed distal of the stents. Images were obtained during a manual or automated pullback with 1 mm/s. Two-dimensional images were displayed on a video monitor of 300 × 300 pixel matrix (RasterOps ST 24 V). Images were recorded on 0.5-inch S-VHS videotape for subsequent quantitative and qualitative analysis. Quantitative analysis was performed off-line using TapeMeasure® software (Indec Systems, Santa Cruz, Calif.). Reference sections were defined as most normal looking segments 1–5 mm proximal and distal to the proximal and distal edge of the stents. In the presence of side branches within that reference section, vessel borders were interpolated. Calcium was defined as a dense, echogenic mass located within the area of the plaque with corresponding shadowing of peripheral structures. Since calcium deposits obscure peripheral structures, the outer border of the plaque was measured only in the presence of a calcium arc smaller than 60° relative to the center of the lumen. ICUS images with image distortion were not included.

Quantitative measurements were performed at the proximal and distal reference areas as well as at the area of minimal stent lumen of the following parameters: minimal and maximal lumen diameter and lumen area. Stent expansion was defined as: (minimal stent lumen area/mean reference area) × 100%. Also, IVUS measurements of the angiographically silent (undetected) plaques proximal and distal to the stents (inflow/outflow) were performed. Residual plaque stenosis was calculated as: plaque area/total vessel area. Measurements were performed twice by an experienced investigator, the reported data is calculated as the mean of both measurements.

Statistical Analysis

Statistical analysis was performed using standard software. Results are presented as mean value ± SD. Comparisons between equivalent groups were performed by unpaired Student's t test. Subgroup comparisons of discrete variables were made by χ^2 analysis. A p value < 0.05 was considered statistically significant. Intra- and interobserver variability were reported previously from our laboratory [9]. Correlation coefficient for intra- and interobserver measurements were 0.96 and 0.93 for proximal and 0.94 and 0.92 for distal reference segments.

Results

Stent Implantation

IVUS imaging could be performed in all patients without complications. In 8 patients with ostial lesions, the proximal reference lumen area could not be determined. In these cases, only the distal reference was used to determine stent expansion. Mean lumen areas were 8.94 ± 2.92 mm^2 for the proximal and 8.26 ± 2.81 mm^2 for the distal reference section (mean ± SD). Area of minimal lumen within the stents was 6.90 ± 2.32 mm^2. Therefore average of all stent expansions was calculated as 81 ± 15%. Figure 1 shows the relation between minimal lumen area within the stents and the mean reference area; 89% of

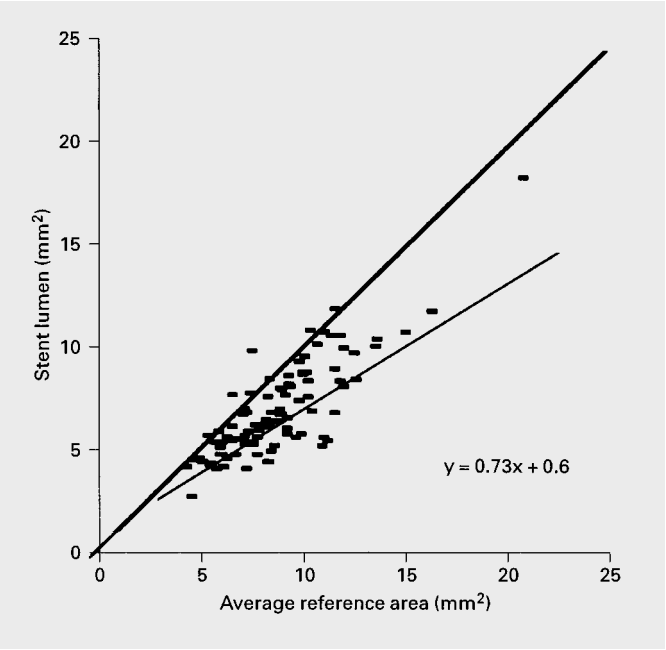

Fig. 1. Ratio of minimal stent lumen area within the stents and the mean reference areas. Almost all stents were smaller than the mean reference areas. Thick line shows line of equality. Thin line shows line of linear regression. Stent/reference: 81 ± 16% (mean ± SD).

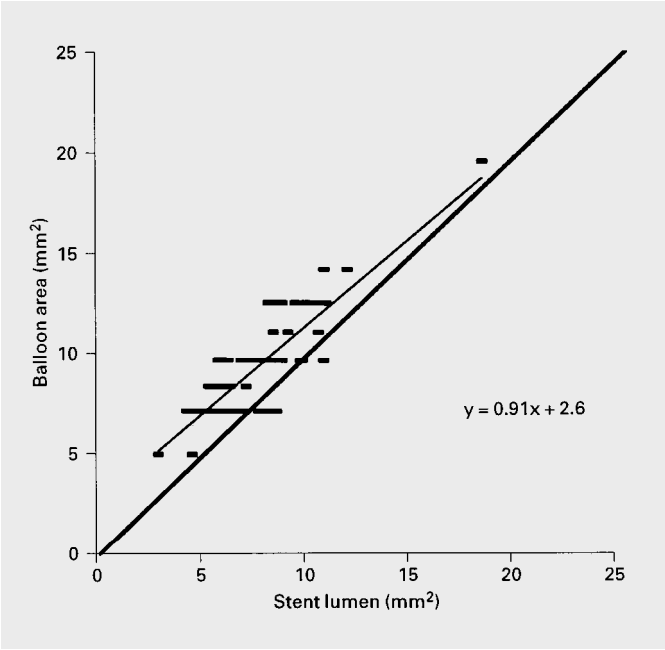

Fig. 3. Relation of nominal balloon area and minimal stent lumen. In almost all patients, balloon catheters were larger than the minimal lumen within the stents. Ballon/stent: 133 ± 21% (mean ± SD).

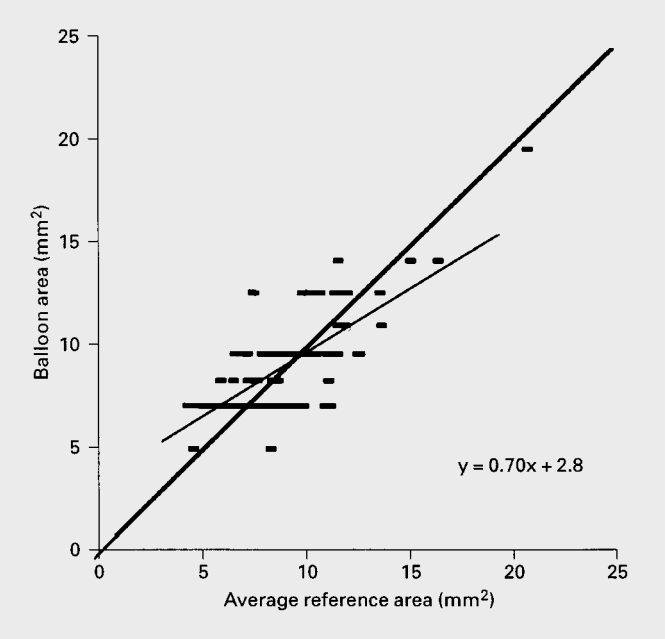

Fig. 2. Ratio of nominal areas of the catheters used for stent deployment and mean reference areas. It is shown that balloon catheters chosen by angiographic criteria were not undersized compared with the reference segments. Balloon/reference: 107 ± 23% (mean ± SD).

the stents were smaller than the mean reference lumen areas. Balloon catheters used for stent implantation had a nominal area of 8.88 ± 2.40 mm^2. The quotient of the nominal areas of the balloons used for stent deployment and the mean reference area was 1.07 on average, standard deviation was ±0.23 (y = 0.70x + 2.8). Therefore, balloon catheters chosen by angiographic criteria were not undersized compared with the reference segments (fig. 2). Comparison between nominal balloon areas and minimal stent lumen areas revealed a mean ratio of 1.33 ± 0.21 (y = 0.91x + 2.6). In almost all patients, balloons used for the interventions were larger than the minimal lumen within the stents (fig. 3).

As shown in table 1, the degree of stent expansion did not differ significantly between the left anterior descending artery, the circumflex artery, right coronary artery and bypass grafts. Stent expansion was also comparable regarding the etiology of the stenosis (de novo stenosis, restenosis) and the different indications for stenting (primary, bail-out). Finally, there was no statistical difference for stent expansion regarding the different stent sizes.

In 71 of the 94 patients (76%), stent expansion by IVUS was considered as not optimal. In these patients, additional balloon dilatation was performed using larger

Table 1. Lesion characteristics and stent expansion

		n	%	
Vessel	LAD	54	79 ± 15	n.s.
	RCA	23	80 ± 11	
	LCx	14	82 ± 21	
	CABG	7	87 ± 28	
Stenosis	de novo	79	79 ± 15	n.s.
	restenosis	19	86 ± 16	
Stent	primary	50	79 ± 15	n.s.
	bail-out	50	82 ± 16	
	<3.5 mm	57	78 ± 15	n.s.
	>3.5 mm	43	85 ± 15	

Average stent expansion in the listed subgroups of the different treated vessels, lesion characteristics, indications for stenting and size of stented vessels are shown. No statistical significant difference in stent expansion was found between the vessels, the etiology of the lesions, the indications for stenting and the size of stented vessel. LAD = Left anterior descending artery; RCA = right coronary artery; LCx = Circumflex artery; CABG = coronary artery bypass graft; n.s. = nonsignificant.

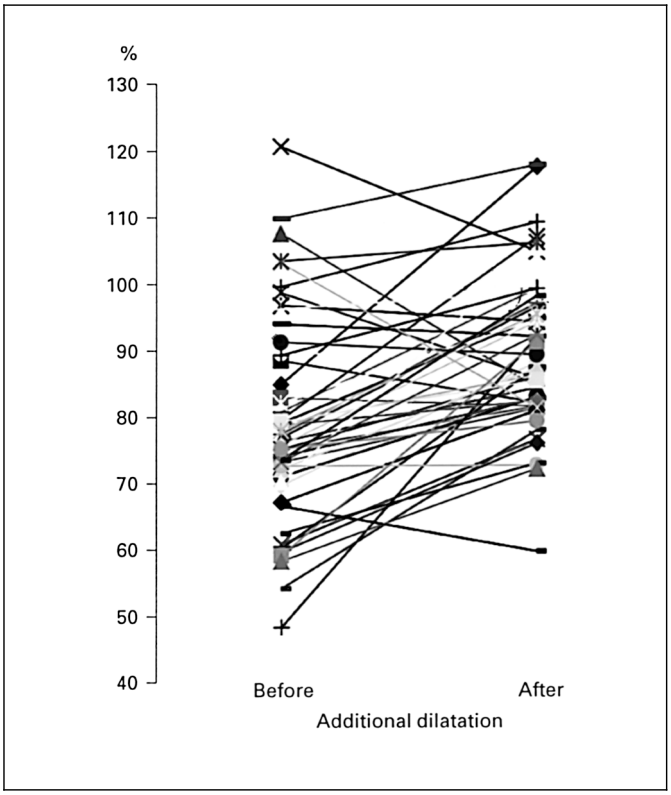

Fig. 4. Stent expansion before (79 ± 16%), and after (91 ± 15%; mean ± SD) additional balloon dilatation. Improvement of stent expansion was highly significant (n = 52; $p < 0.002$). However, due to a larger increase of reference area, relative stent expansion even decreased in some patients.

balloons (3.48 ± 0.48 mm, range 2.5–5.0 mm) and/or higher dilatation pressure (17.7 ± 2.6 atm, range 14–20 atm). In 52 of these 71 patients (73%), the final result was assessed with IVUS imaging. In this cohort of patients, stent expansion improved from 79 ± 16 to 91 ± 15% ($p < 0.002$; fig. 4). Residual stenosis of the angiographic silent plaque was calculated as 43.1 ± 12.5% for the inflow stenosis and 37.4 ± 14.7% for the outflow stenosis.

Follow-Up

27 patients have been anticoagulated with coumadin. 66 patients were treated with ticlopidine and aspirin, 2 received only aspirin. 2 patients had severe complications during their hospital stay: one patient had a subacute stent thrombosis resulting in an acute myocardial infarction, another patient underwent bypass surgery after a subacute stent thrombosis. Both patients were anticoagulated with coumadin. International normalized ratio was within the therapeutic range (2.0–3.0). Minimal stent lumen diameter was 4.85 mm in one and 3.89 mm in the other patient. Stent expansion was 92 and 98%, respectively. Thrombus formation, however, was already visible by IVUS at the time of the intervention and diminished after additional dilatation was performed. 8 patients underwent groin surgery, and 2 required blood transfusions after severe bleeding complications.

Patients were followed up for 6 months with exercise tests and nuclear imaging if indicated; 8 patients (9%) were lost for follow-up as they did not appear in our outpatient clinic and could not be reached by phone or mail. 19 of the remaining 86 patients (22%) required target lesion revascularization within the 6-month period. In 6 patients in-stent conventional balloon angioplasty was performed, 7 patients underwent placement of another stent in the target vessel. In 4 patients conventional coronary bypass grafting was performed, in 2 patients revascularization was achieved using the minimal invasive approach. 1 patient presented with positive thallium stress testing but refused to undergo control angiography. All the other patients were clinically stable, showed no positive stress test and no significant restenosis of the stented vessel on control angiography, if performed. Table 2 shows the univariable comparison of patients with and without target lesion revascularization. The size of the stented vessels was significantly different in patients with

Table 2. Target lesion revascularization

	Group A	Group B	p
Stable/unstable/post-inf. angina	38/10/19	16/3/0	n.s.
LAD/LCx/RCA/CABG	36/10/18/3	13/3/2/1	n.s.
Balloon size, mm	3.6 ± 0.5	3.0 ± 0.2	$p < 0.001$
Maximal pressure, atm	15.2 ± 3.9	15.9 ± 3.9	n.s.
Stent expansion, %	90 ± 15	82 ± 13	n.s.
Minimal stent lumen, mm^2	7.5 ± 2.5	6.0 ± 1.8	$p < 0.05$
Inflow plaque stenosis, %	42 ± 12	46 ± 14	n.s.
Outflow plaque stenosis, %	35 ± 16	42 ± 11	$p < 0.05$

Statistical significant difference between patient *with* (group B) and *without* (group A) target lesion revascularization was found for minimal stent lumen, balloon size and degree of outflow plaque stenosis. Post-inf. angina = Postmyocardial infarction angina; LAD = left anterior descending artery; LCx = circumflex artery; RCA = right coronary artery; CABG = coronary artery bypass graft; n.s. = nonsignificant.

and without revascularization ($p < 0.05$). The degree of stent expansion at the time of primary implantation was not different (p = nonsignificant). The amount of plaque located at the distal end of the stents was significantly different in patients with and without target lesion revascularization ($p < 0.05$).

Discussion

There are established criteria for optimal stent expansion assessed by intravascular ultrasound [6]. A first criterion is a qualitative evaluation of the stent site involving the achievement of good stent apposition to the vessel wall. Second, minimal stent lumen area should be equal or greater than the reference lumen area (stent expansion ≥100%). Third, no significant residual stenosis (>60%) should be proximal or distal to the stent [6]. Previous studies showed that an incomplete apposition of the stent struts, a residual lumen narrowing or an irregular eccentric lumen in the stented segment was still present in up to 88% of the cases when the angiographic result was considered optimal by an experienced operator [2, 6]. This has prompted investigators to develop a more aggressive strategy based on high-pressure balloon dilatation [1, 2, 6, 10].

The question of whether high-pressure implantation technique per se guarantees adequate expansion of Palmaz-Schatz stents was addressed previously. Goerge et al. [11] compared two groups of patients undergoing either conventional implantation of Palmaz-Schatz stents (patients treated between 1991 and 1992) or using the high-pressure approach (between 1993 and 1994). Average minimal lumen diameter measured by IVUS increased from 2.55 ± 0.41 to 3.14 ± 0.37 mm in the group of patients undergoing high-pressure implantation. However, despite the use of high-pressure technique, not all patients had homogenous stent geometry or optimal stent expansion. In 9 out of the 24 patients, stent apposition was considered not optimal. Stone and coworkers [12] performed serial IVUS investigations after sequential balloon inflation of Palmaz-Schatz stents at 12, 15 and 18 atm using noncompliant balloons with a mean balloon to artery ratio of 1.13 ± 0.16. Minimal lumen area within the stents increased progressively from 7.7 ± 2.1 to 9.2 ± 2.4 mm^2 due to an increase of implantation pressure. Commonly used criteria for optimal stent expansion, however, were only met by 81% of the patients, even after applying 18 atm. In another study, Botas et al. [13] performed IVUS evaluations of stent expansion of different stent types. Stents were implanted with slightly oversized catheters (balloon/artery ratio of 1.15 ± 0.18) using 14.5 ± 2 atm. Mean stent expansion was calculated as 82 ± 20%, only 29% of the stents showed an expansion of over 90%. Interestingly, stent expansion did not vary among different stent types.

In our study, 89% of the stents were smaller than the mean reference lumen areas; mean stent expansion was 81 ± 15%. Results of stent implantation was considered as not optimal by IVUS criteria in 76% of the patients. Additional balloon dilatations improved stent expansion significantly. The ratio of the nominal balloon areas used for stent implantation and the mean reference area was 1.07 ± 0.23, indicating that the balloon catheters chosen by angiographic criteria were not undersized compared with the reference segments. Comparison between nominal balloon areas and minimal stent lumen areas revealed a ratio of 1.33 ± 0.21. Therefore, almost all balloons used

for the interventions were larger than the minimal lumen within the stents.

Regarding the mechanisms of residual lumen stenosis after high-pressure stent implantation, quantitative coronary angiography and IVUS studies were performed. They support the hypothesis that balloon underexpansion as well as elastic recoil are responsible for residual lumen stenosis, suggesting that plaque characteristics and stent radial strength deserve further investigations [14].

Optimal ultrasound-guided stent expansion was initially targeted to the prevention of subacute thrombosis, but this strategy can also have an impact on the reduction of restenosis. Preliminary studies have shown that IVUS-guided stent placement may result in a lower restenosis rate as compared to the angiographic-guided approach [7, 8, 15–17]. Data from the CRUISE trial, the IVUS substudy of the STARS trial suggest that ultrasound guidance results not only in a significantly larger minimal stent lumen, but also in a 39% relative reduction in target vessel revascularization (8.9 vs. 14.8%) [18].

As shown in previous studies, development of in-stent restenosis depends on several factors, including the size of the stented vessel [3, 4, 19–21]. In our study, vessel size also proved to be a prognostic factor regarding the incidence of target lesion revascularization. Minimal stent lumen in the group of patients requiring target lesion revascularization was 6.0 ± 1.8 compared with 7.5 ± 2.5 mm^2 in those without a revascularization procedure ($p < 0.05$). However, expansion of the stents did not differ significantly in both groups of patients. Residual plaque stenosis proximal to the stents (inflow) was 45 ± 14% in patients *with* and 42 ± 12% in patients *without* target lesion revascularization (nonsignificant). Most interestingly, data for the residual plaque stenosis distal to the stents (outflow) were 43 ± 10 and 35 ± 15%, respectively ($p < 0.05$). These data suggest that focusing on the minimal stent lumen and avoidance of residual outflow stenosis seems to be a more appropriate strategy than focusing on the mere stent expansion. Of course, the aggressive use of larger balloons and higher pressure must be tempered with the risk of vessel rupture and increased intimal response due to vessel injury [22–27].

Limitations of the Study

Considering the issue of stent thrombosis, it should be mentioned that the quoted studies employed antiplatelet therapy in addition to high-pressure technique. Several recent studies showed significant reductions in stent thrombosis solely by employment of antiplatelet therapy and elimination of anticoagulation with coumadin [28–30]. However, adequacy of stent deployment remains an important issue regarding the rate of target lesion revascularization. One limitation of the present study is that nominal balloon diameters (diameter of the balloon catheters provided by the manufacturers depending on inflation pressure) were used to assess balloon/reference and balloon/stent ratio. No QCA measurements were performed to determine actual balloon diameters. Due to radial forces provided by the plaque, the vessel wall and the stents themselves, balloon diameters might be overestimated. In the 8 patients with ostial lesions, proximal reference could not be determined. Therefore, only the distal reference area was used to calculate stent expansion which could lead to overestimation of stent expansion in those patients. Furthermore, final IVUS was performed only in 53 out of the 72 patients, who underwent additional dilatation after initial IVUS imaging. Angiographic follow-up data at 6 months was available in only 73 patients (78%); again, QCA was not performed for quantitative analysis. Target lesion revascularization depends on several factors and cannot be used to exactly assess the rate of in-stent restenosis. 8 patients were lost for clinical follow-up, possibly due to a better clinical outcome than those who underwent regular follow-up visits or control angiography which might increase the relative rate of target lesion revascularization in the followed-up patients. In 28 of the 86 patients, final IVUS was not performed after additional balloon dilatation; therefore actual stent expansion by IVUS after completion of the procedure was only known in 58 patients (67.4%). Furthermore, new stent designs, providing flexibility without an articulation site may already help to overcome the problem of plaque prolapse into the lumen contributing to in-stent restenosis, as observed in the first generation of Palmaz-Schatz stents.

Conclusions

Implantation of Palmaz-Schatz stents using high-pressure implantation technique and angiographic criteria does not guarantee 'adequate' stent expansion, as defined by IVUS criteria. Additional dilatation with higher pressure and/or larger balloons improves stent expansion significantly. The size of the stented vessel and the outflow plaque stenosis, but not the degree of stent expansion had an influence on the rate of target lesion revascularization within 6 months' follow-up. These data suggest that minimal stent lumen and residual plaque stenosis seem to be more predictive parameters than mere stent expansion regarding the rate of target lesion revascularization.

References

1 Goldberg SL, Colombo A, Nakamura S, Almagor M, Maiello L, Tobis JM: Benefit of intracoronary ultrasound in the deployment of Palmaz-Schatz stents. J Am Coll Cardiol 1994;24: 996–1003.

2 Nakamura S, Colombo A, Gaglione S, Almagor Y, Goldberg SL, Maiello L, Finci L, Tobis JM: Intracoronary ultrasound observations during stent implantation. Circulation 1994;89:2026–2034.

3 Serruys PW, de Jaegere P, Kiemeneij F, Macaya C, Rutsch W, Heyndrickx G, Emanuelsson H, Marco J, Legrand V, Materne P, Belardi J, Sigwart U, Colombo A, Goy J-J, van den Huevel P, Delcan J, Morel MA, for the Benestent Study Group: A comparison of balloon expandable stent implantation with balloon angioplasty in patients with coronary artery disease. N Engl J Med 1994;331:489–495.

4 Fischman DL, Leon MB, Baim D, Schatz RA, Penn I, Detre K, Savage MP, Veltri L, Ricci D, Nobuyoshi M, Cleman M, Heuser R, Almond D, Teirstein P, Fisch D, Colombo A, Brinker J, Moses J, Hirshfeld J, Bailey S, Ellis S, Rake R, Goldberg S: A randomized comparison of coronary stent placement and balloon angioplasty in the treatment of coronary artery disease. N Engl J Med 1994;331:496–501.

5 Schatz RA, Baim DS, Leon M, Ellis SG, Goldberg S, Hirshfeld J, Cleman MW, Cabin HS, Walke C: Clinical experience with the Palmaz-Schatz coronary stent: Initial results of a multicenter study. Circulation 1991;83:148–161.

6 Colombo A, Hall P, Nakamura S, Almagor Y, Maiello L, Martini G, Gaglione A, Goldberg S, Tobis J: Intracoronary stenting without anticoagulation accomplished with intravascular ultrasound guidance. Circulation 1995;91: 1676–1688.

7 Mudra H, Sunamura M, Figulla H, Almagor Y, Maiello L, Martini G, Bilodeau L, Penn I, Colombo A, Hamm C, Bartorelli A, Rothman M, Nabuyoshi M, Yamaguchi T, Voudris V, Makovski S, Hausmann C, de Jaegere P: Six month clinical and angiographic outcome after IVUS guided stent implantation (abstract). J Am Coll Cardiol 1997;29:171A.

8 Mudra H, Macaya C, Zahn R, Grip L, Di Mario C, Rutsch W, Voudris V, Schachinger V, Henneke K-H: Interim analysis of the 'Optimization with ICUS to Reduce Stent Restenosis' OPTICUS) trial (abstract). Circulation 1998; 98(suppl II).

9 Blessing E, Hausmann D, Sturm M, Wolpers HG, Amende I, Mügge A: Intravascular ultrasound guidance of stent implantation: Intraobserver and interobserver variability. Am Heart J 1999;137:368–371.

10 Serruys PW, Emanuelsson H, van der Giessen W, Lunn A, Kiemeney F, Macaya C, Rutsch W, Heyndrickx G, Suryapranata H, Legrand V, Goy JJ, Materne P, Bonnier H, Morice MC, Fajadet J, Belardi J, Colombo A, Garcia E, Ruygrok P, de Jaegere P, Morel MA: Heparin-coated Palmaz-Schatz stents in human coronary arteries. Early outcome of the Benestent-II pilot study. Circulation 1996;93:412–422.

11 Goerge G, Haude M, Ge J, Voegele E, Gerber T, Rupprecht HJ, Meyer J, Erbel R: Intravascular ultrasound after low and high inflation pressure coronary artery stent implantation. J Am Coll Cardiol 1995;26:725–730.

12 Stone GW, Linnemeier T, St Goar F, Hodgson J, Coverdale J, Yock P, Fitzgerald P: Defining the relationship between stent implantation pressure and optimal expansion – Core lab analysis from the OSTI trial (abstract). Circulation 1996;94(suppl I):I-259.

13 Botas J, Elizaga J, Garcia E, Bermejo J, Soriano J, Abeytua M, Osende J, Fernandez A, Delcan JL: Intravascular ultrasound evaluation of stent implantation using high balloon pressures (abstract). Eur Heart J 1996;17:1020.

14 Bermejo J, Botas J, Garcia EJ, Elizaga J, Soriano J, Abeytua M, Sarnago F, Delcan JL: Mechanism of residual lumen stenosis after high pressure stent implantation: A QCA and IVUS study (abstract). Circulation 1996;94(suppl I): I-199.

15 Blasini R, Zitzmann E, Schalkhauser F, Paloncy R, Boekenhamp J, Walter H, Hadamitzki M, Neumann FJ, Richardt G, Schmitt C, Alt E, Schoemig A: Bedeutung einer IVUS-gesteuerten Optimierung koronarer Palmaz-Schatz Stent Implantation auf den klinischen und angiographischen Langzeitverlauf (abstract). Z Kardiol 1996;85(suppl II):476.

16 Albiero R, Rau T, Schlueter M, DiMario C, Reimers B, Mathey DG, Tobis JM, Schofer J, Colombo A: Comparison of immediate and intermediate-term results of intravascular ultrasound versus angiography-guided Palmaz-Schatz stent implantation in matched lesions. Circulation 1997;96:2997–3005.

17 Stone GW, Hodgson JM, St Goar FG, Frey A, Mudra H: Improved procedural result of coronary angioplasty with ultrasound guided balloon sizing: The CLOUT pilot trial. Circulation 1997;95:2044–2052.

18 Fitzgerald PJ, Hayase M, Mintz GS, Kuntz R, Moses JW, Diver DJ, Deutsch E, Simonton CA, Baim DS, Leon MB, Yock PG, for the CRUISE Investigators: Can routine intravascular ultrasound influence stent expansion? Analysis of outcomes (abstract). J Am Coll Cardiol 1998;31(suppl A):396A.

19 Hoffmann R, Mintz GS, Mehran R, Pichard AD, Kent KM, Satler LF, Popma JJ, Hongsheng W, Leon MB: Intravascular ultrasound predictors of angiographic restenosis in lesions treated with Palmaz-Schatz stent. J Am Coll Cardiol 1998;31:43–49.

20 Mintz GS, Hoffmann R, Mehran R, Pichard AD, Kent KM, Satler LF, Popma JJ, Leon MB: In-stent restenosis – The Washington Hospital Center experience. Am J Cardiol 1998;81/7A: E7–E13.

21 Abizaid A, Mehran R, Abizaid AS, Leiboff B, Pappas C, Walsh CL, Ezibe M, Kent KM, Mintz GS: Clinical, angiographic, and intravascular ultrasound predictors of target lesion revascularization in >1,000 consecutive patients with native vessel lesions treated with Palmaz-Schatz stents (abstract). Circulation 1998; 98(suppl I):I-229.

22 Alfonso F, Goicolea J, Hernandez R, Fernandez-Ortiz A, Segovia J, Banuelos C, Aragoncillo P, Phillips P, Macaya C: Arterial perforation during optimization of coronary stents during high-pressure balloon inflations. Am J Cardiol 1996;78:1169–1172.

23 Nobuyoshi M, Kimura T, Ohishi H, Horiuchi H, Nosaka H, Hamasaki N, Yokoi H, Kim K: Restenosis after percutaneous transluminal coronary angioplasty: Pathologic observations in 20 patients. J Am Coll Cardiol 1991;17:433–439.

24 Schwartz R, Murphy JG, Edwards WD, Camrud AR, Vliestra RE, Holmes DR: Restenosis after balloon angioplasty: A practical proliferation model in porcine coronary arteries. Circulation 1990;82:2190–2200.

25 de Jaegere P, Serruys PW, Bertrand M, Wiegand V, Marquis JF, Vrolicx M, Piessens J, Valeix B, Kober G, Bonnier H: Angiographic predictors of recurrence of restenosis after Wiktor stent implantation in native coronary arteries. Am J Cardiol 1993;72:165–170.

26 Savage MP, Fischman DL, Douglas JS, Pepine CJ, Werner JA, Bailey SR, Rake R, Goldberg S, for the SAVED Investigators: The dark side of high pressure stent deployment (abstract). J Am Coll Cardiol 1997;29(suppl A):368A.

27 Fernandez-Aviles F, Alonso JJH, Duran JM, Gimero F, Garcia-Moran E, Paniagua J, Garcimartin I, Munoz JC: High-pressure increases late loss after coronary stenting (abstract). J Am Coll Cardiol 1997;29(suppl A):369A.

28 Schomig A, Neumann JF, Kastrati A, Schuhlen H, Blasini R, Hadamitzky M, Walter H, Zitzmann-Roth EM, Richardt G, Alt E, Schmitt C, Ulm K: A randomized comparison of antiplatelet and anticoagulant therapy after the placement of coronary-artery stents. N Engl J Med 1996;334:1084–1089.

29 Morice MC, Zemour G, Benveniste E, Biron Y, Bourdonnec C, Faivre R, Fajadet J, Gaspard P, Glatt B, Joly P: Intracoronary stenting without coumadin: One month results of a French multicenter study. Cathet Cardiovasc Diagn 1995; 35:1–7.

30 Karrillon GJ, Morice MC, Benveniste E, Bunouf P, Aubry P, Cattan S, Chevalier B, Commeau P, Cribier A, Eiferman C, Grollier G, Guerin Y, Henry M, Lefevre T, Livarek B, Louvard Y, Marco J, Makowski S, Monassier JP, Pernes JM, Rioux P, Spaulding C, Zemour G: Intracoronary stent implantation without ultrasound guidance and with replacement of conventional anticoagulation by antiplatelet therapy. 30-day clinical outcome of the French Multicenter Registry. Circulation 1996;94: 1519–1527.