Journal of the American College of Cardiology
© 2002 by the American College of Cardiology Foundation
Published by Elsevier Science Inc.
Vol. 40, No. 3, 2002
ISSN 0735-1097/02/$22.00
PII S0735-1097(02)01989-7

In-Stent Restenosis in Small Coronary Arteries

Impact of Strut Thickness

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OBJECTIVES We sought to evaluate whether strut thickness may impact the restenosis rate after stent implantation in small coronary arteries.

BACKGROUND Small vessel size (<3.0 mm) is an independent risk factor for the occurrence of in-stent restenosis. It has been reported that vessel damage induced during stent deployment is an important factor in restenosis.

METHODS From our database, we selected all patients who had successful stenting in small native vessels, with angiographic follow-up available, between March 1996 and April 2001. The strut was defined as thin when <0.10 mm and thick when ≥0.10 mm. According to these criteria, we identified two subgroups: a thin group and a thick group.

RESULTS A total of 821 (57%) of the 1,447 patients had angiographic follow-up available and were included in the analysis. The thin group included 400 patients with 505 lesions. The thick group included 421 patients with 436 lesions. The restenosis rate was 28.5% in the thin group and 36.6% in the thick group (p = 0.009; odds ratio [OR] 1.44, 95% confidence interval [CI] 1.09 to 1.90). The study group was classified into three subgroups according to the reference vessel diameter: ≤2.50 mm, 2.51 to 2.75 mm and 2.76 to 2.99 mm. Strut thickness influenced the restenosis rate only in the subgroup with a reference vessel diameter between 2.76 and 2.99 mm, with rates of 23.5% in the thin group and 37% in the thick group (p = 0.006). By logistic regression analysis, predictors of restenosis were stent length (OR 1.03, 95% CI 1.01 to 1.04; p = 0.001), strut thickness (OR 1.68, 95% CI 1.23 to 2.29; p = 0.001) and diabetes mellitus (OR 2.10, 95% CI 1.21 to 3.68; p = 0.007).

CONCLUSIONS This study supports that strut thickness is an independent predictor of restenosis in coronary arteries with a reference diameter of 2.75 to 2.99 mm. (J Am Coll Cardiol 2002;40:403–9)
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The restenosis rate after stent implantation is higher in small (<3.0 mm reference diameter) versus large coronary arteries (1–3). Randomized, observational studies have found that stent-selected lesions located in small vessels lead to results equivalent to or better than those achieved with balloon angioplasty (4–8). Kastrati et al. (9) recently demonstrated that the use of a thinner strut device is associated with a significant reduction in angiographic and clinical restenosis after coronary artery stenting in vessels with a reference diameter >2.8 mm.

The purpose of the present study was to evaluate whether strut thickness may impact the restenosis rate after stent implantation in small coronary arteries.

METHODS

Patient group. From March 1996 to April 2001, 2,911 consecutive patients with 3,334 lesions underwent coronary stent implantation in small (<3.0 mm reference diameter) native coronary arteries in two institutions. All patients with self-expandable stents (n = 57), coil stents (n = 208), covered stents (n = 4) radioactive stents (n = 100), a combination of different stents (n = 973) and atherectomy (both rotational and directional; n = 81) were excluded from the analysis. Patients with successful stent placement and no major adverse cardiac events (MACE) during the first 30 days after the intervention were considered eligible for angiographic follow-up. After the exclusion of 65 patients in whom the procedure failed (success rate 96%) and 75 patients who developed at least one MACE during the first 30 days (30-day incidence of MACE 4.9%), all other patients were asked to undergo coronary angiography at six months or earlier if they had recurrence of symptoms. Angiographic follow-up was available in 821 (57%) of the 1,447 patients at an average of 8 ± 2 months after the procedure. Therefore, the final study group included consisted of 821 consecutive patients with 941 lesions successfully treated with slotted-tube or multicellular stent implantation, or both, in small coronary arteries. All patients gave written, informed consent for both the intervention and follow-up angiography.

Stent implantation procedure. Intracoronary stenting was performed using techniques previously described (10). All patients received 325 mg aspirin before stent deployment. All patients received 70 IU/kg of an intra-arterial bolus of

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Manuscript received February 5, 2002; revised manuscript received March 25, 2002, accepted April 17, 2002.

Abbreviations and Acronyms
CI = confidence interval
DS = diameter stenosis
OR = odds ratio
MACE = major acute cardiac events
MLD = minimal lumen diameter

unfractionated heparin. Glycoprotein IIb/IIIa inhibitors were administered according to the operator's preference.

Definitions. All stents with a strut thickness <0.10 mm were considered as *thin*, whereas all stents with a strut thickness ≥0.10 mm were considered as *thick* (11). Thin stents (thin group) included the Palmaz-Schatz (Cordis, Johnson & Johnson Company, Warren, New Jersey), ACS Multilink (Guidant Corp., Santa Clara, California), Biodyvisio (Biocompatibles Ltd., Surrey, UK), BeStent (Medtronic, Minneapolis, Minnesota), Jostent Flex (Jomed International AB, Helsingborg, Sweden), Diamond (Phytis Medical Devices GmbH, Berlin, Germany), V-Flex (Global Therapeutic, Inc., Bloomington, Indiana) and Carbostent (Sorin Biomedica Cardio, Saluggia, Italy). Thick stents (thick group) included the NIR (Medinol/Scimed Life Systems, Maple Grove, Minnesota), ACS Duet (Guidant Corp.), BX Velocity (Cordis, Johnson & Johnson Company), AVEII (Medtronic), Crossflex LC (Cordis, Johnson & Johnson Company) and Bard XT (CR Bard Inc., Billerica, Massachusetts).

Postprocedural management and follow-up. After successful stent implantation was achieved, no further heparin was administered, and the sheaths were removed in 4 to 6 h. Aspirin (325 mg/day) and ticlopidine (250 mg twice daily for 30 days) were prescribed to all patients. Follow-up was performed in all patients by means of an interview or telephone conversation with the patient or with the referring physician. Follow-up angiography was performed at six months, unless an early re-study was indicated by symptoms.

Angiographic measurements were performed, as previously described, with an automated computer-based system (QCA-CMS version 3.0, MEDIS, Leiden, The Netherlands) (12). The lesions were characterized according to the modified American College of Cardiology/American Heart Association classification (13). *Angiographic success* was defined as final angiographic residual stenosis of <20% by a visual estimate. Restenosis was defined in a dichotomous manner as diameter stenosis (DS) ≥50% at follow-up angiography. The analysis included assessment of the minimal lumen diameter (MLD) and percent DS immediately after stenting and at follow-up, as well as their cumulative distributions. According to a continuous geometric model of restenosis, we examined: 1) acute gain (MLD [after stenting] − MLD [before procedure]); 2) relative gain (acute gain/reference diameter before stenting); 3) late loss (MLD [after stenting] − MLD [at follow-up]); and 4) loss index (late loss/acute gain).

Intravascular ultrasound imaging was performed with a 3.9F monorail system equipped with a 25-MHz transducer-tipped catheter (Interpret Catheter, Inter-Therapy/CVIS) or a 2.9F or 3.2F monorail system equipped with a 30-MHz transducer-tipped catheter (Scimed-Boston Scientific, Maple Grove, Minnesota).

Statistical analysis. Continuous variables are expressed as the mean value ± SD. Paired and unpaired *t* tests were performed to determine the differences between mean values for baseline continuous variables. The Fisher exact test was used to analyze categorical variables at baseline. The study group was classified into three subgroups (tertiles) according to the reference vessel diameter: ≤2.50 mm for the first group; 2.51 to 2.75 mm for the second group; and 2.76 to 2.99 mm for the third group. Angiographic data were analyzed by a mixed linear model with the interaction between group (thin and thick), vessel diameter tertiles and group-by-vessel diameter tertiles as a fixed effect and patient indicator as a random term to take into account clustered data (more lesions within the same patient). A comparison

Table 1. Clinical Characteristics of the Two Groups According to Stent Strut Thickness

	Thin Group (n = 400)	Thick Group (n = 451)	p Value
Age (yrs)	59 ± 10	60 ± 10	0.20
Male	354 (88.5%)	381 (84.5%)	0.10
Unstable angina	137 (34.3%)	157 (34.8%)	0.53
Previous MI	204 (51%)	230 (51%)	0.92
LV ejection fraction	56 ± 11%	60 ± 12%	0.28
Previous bypass surgery	40 (10%)	52 (11.5%)	0.48
Systemic hypertension	316 (79%)	360 (80%)	0.96
Diabetes mellitus	44 (11%)	22 (5%)	0.001
Hypercholesterolemia	208 (52%)	225 (50%)	0.83
Smokers (current and former)	160 (60%)	275 (61%)	0.25
Coronary artery disease			0.001
Single-vessel	170 (42.6%)	124 (27.5%)	
Double-vessel	148 (37%)	182 (40.4%)	
Triple-vessel	82 (20.4%)	145 (32.1%)	

Data are presented as the mean value ± SD or number (%) of patients.
LV = left ventricular; MI = myocardial infarction.

Table 2. Angiographic Characteristics of the Two Groups According to Stent Strut Thickness

	Thin Group (n = 505)	Thick Group (n = 436)	p Value
Vessel treated			0.002
LAD	260 (51.5%)	162 (38.4%)	
LCx	79 (15.6%)	90 (20.6%)	
RCA	89 (17.6%)	100 (22%)	
Diagonal branch	26 (5.1%)	30 (6.9%)	
Intermedial branch	26 (5.1%)	36 (8.3%)	
Obtuse marginal branch	16 (3.2%)	7 (1.6%)	
Others branches	9 (1.8%)	11 (2.5%)	
Lesion site			0.012
Ostial	40 (7.9%)	46 (10.6%)	
Proximal	193 (38.2%)	140 (32.1%)	
Mid vessel	221 (43.8%)	181 (41.5%)	
Distal	51 (10.1%)	69 (15.8%)	
Lesion types			0.004
A	22 (4.4%)	21 (4.8%)	
B1	168 (33.2%)	103 (23.6%)	
B2	209 (41.4%)	196 (44.9%)	
C	106 (21%)	116 (26.7%)	
Diameter stenosis (%)			
Before stenting	68 ± 16	66 ± 17	0.022
After stenting	1 ± 10	1 ± 7	0.80
Follow-up	36 ± 25	40 ± 24	0.005
Bifurcation lesions	105 (21%)	102 (23.5%)	0.33
Chronic total occlusion	30 (6%)	31 (7.2%)	0.45
Lesion length (mm)	11 ± 7	13 ± 9	< 0.001
Reference diameter (mm)			
Before stenting	2.60 ± 0.33	2.58 ± 0.32	0.38
After stenting	2.62 ± 0.36	2.64 ± 0.36	0.38
Follow-up	2.67 ± 0.45	2.61 ± 0.43	0.25
MLD (mm)			
Before stenting	0.83 ± 0.41	0.89 ± 0.44	0.018
After stenting	2.52 ± 0.50	2.53 ± 0.50	0.80
Follow-up	1.77 ± 0.80	1.60 ± 0.75	0.005
Acute gain (mm)	2.00 ± 0.60	1.84 ± 0.58	< 0.001
Relative gain (mm)	0.77 ± 0.22	0.72 ± 0.24	0.002
Late loss (mm)	1.04 ± 0.79	1.16 ± 0.76	0.031
Loss index	0.54 ± 0.43	0.66 ± 0.47	< 0.001

Data are presented as the mean value ± SD or number (%) of patients.
LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; MLD = minimal lumen diameter; RCA = right coronary artery.

of angiographic measurements in the three subgroups was performed by using proper, mixed linear model contrasts and by adjusting the p value using the Tukey approach for multiple comparisons. The SAS Proc Mixed (Cary, North Carolina) was used to obtain maximal likelihood solutions of the mixed linear models. The influence of clinical, angiographic and procedural variables on restenosis was evaluated by univariate and stepwise logistic regression analyses. All variables with a p value <0.10 in the univariate analysis were entered into the multivariate model of restenosis to test for independent effects. The analysis was made per lesion. A p value of <0.05 was considered statistically significant. Data were analyzed with SPSS version 10.0 (SPSS Inc., Chicago, Illinois) for Windows.

RESULTS

The thin group included 400 patients with 505 lesions. Thin stents included the Palmaz-Schatz (39.5%), ACS Multilink (9.2%), Biodyvisio (10.2%), BeStent (10%), Jostent Flex (4%), Diamond (7.6%), V-Flex (5.6%) and Carbostent (13.9%). The thick group included 421 patients with 436 lesions. Thick stents were the NIR (25.5%), ACS Duet (33.5%), BX Velocity (18%), AVEII (18%), Crossflex LC (2.8%) and Bard XT (2.2%).

Clinical characteristics. Patients in the thin group were more often diabetics and had a lower rate of multivessel disease (Table 1).

Angiographic and procedural characteristics. The reference artery size was similar between the two groups. In contrast, there were significant differences in MLD, percent DS, lesion length, target vessel distribution, lesion type and lesion site (Table 2). Indeed, the lesions were more often complex (e.g., B2/C), longer and located in the ostium in the thick group versus the thin group.

Intravascular ultrasonography was performed more often in the thin group than in the thick group (63% vs. 33%; p <

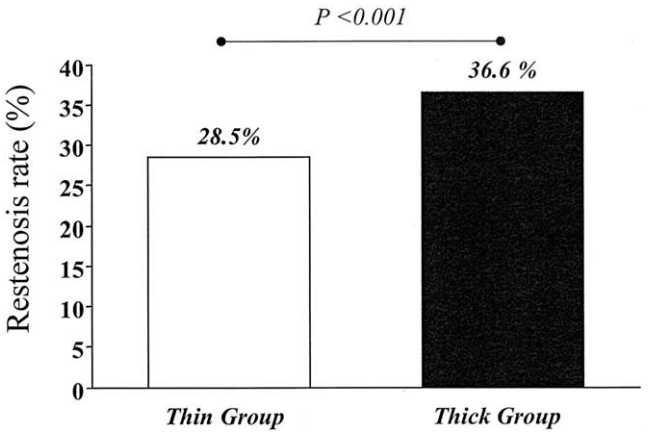

Figure 1. Restenosis rates in lesions treated with a stent with a strut thickness of <0.10 mm (thin group; **open bar**) and a stent with a strut thickness of ≥0.10 mm (thick group; **solid bar**).

0.001), whereas direct stenting was similar (10%) for both groups (p = 0.90). The balloon-to-artery ratio and inflation pressure were higher in the thin group than in the thick group (1.3 ± 0.2 atm vs. 1.2 ± 0.2 atm and 15 ± 3 atm vs. 14 ± 3 atm, respectively; p < 0.001 for both). Acute gain was higher in the thin group, whereas the final MLD was similar between the two groups (Table 2). The final stent length was longer in the thin group (20 ± 11 mm vs. 19 ± 11 mm, p = 0.037). Administration of glycoprotein IIb/IIIa inhibitors was similar between the thin group (17%) and thick group (16%; p = 0.85).

Long-term angiographic outcome. The restenosis rate was significantly lower in the thin group (28.5%) than in the thick group (36.6%; p = 0.009; odds ratio [OR] 1.44, 95% confidence interval [CI] 1.09 to 1.90) (Fig. 1). This finding was also confirmed when deleting from the analysis the carbon-coated (Carbostent and Diamond) stents (27.6% vs. 34.7%; p = 0.016; OR 1.40, 95% CI 1.06 to 1.83). Of note, the loss index was significantly lower in the thin group (Table 2). The estimated mean relative surface area coverage of the vessel (11) was slightly higher in the thin group than in the thick group (18 ± 2% vs. 17 ± 2%; p < 0.001) but was not different according to restenosis occurrence (17 ± 3% in case of restenosis vs. 17 ± 3%; p = 0.86). Because of important differences in some clinical and angiographic characteristics that may potentially impact the restenosis rate, we identified the independent predictors of restenosis in our study group. Predictors of restenosis by univariate and multivariable analyses are summarized in Table 3. By stepwise logistic regression analysis, predictors of restenosis in the entire study group were stent length (OR 1.03, 95% CI 1.01 to 1.04; p = 0.001), strut thickness (OR 1.68, 95% CI 1.23 to 2.29; p = 0.001) and diabetes mellitus (OR 2.1, 95% CI 1.21 to 3.68; p = 0.007).

To better clarify the impact of strut thickness on restenosis as a function of vessel size, we further analyzed three subgroups (tertiles) of vessels, according to the reference diameter: 1) vessel diameter ≤2.50 mm (300 lesions); 2) vessel diameter 2.51 to 2.75 mm (291 lesions); and 3) vessel diameter 2.76 to 2.99 mm (350 lesions) (Tables 4 and 5). Strut thickness influenced the restenosis rate only in the subgroup with a reference vessel diameter between 2.76 and 2.99 mm (23.5% in the thin group and 37% in the thick group; p = 0.006; OR 1.91, 95% CI 1.19 to 3.0) (Fig. 2). As represented in Figure 3, the loss index was similar in vessels with a reference diameter ≤2.50 mm, although it was lower in the thin group, as the reference vessel diameter increased, and it was statistically lower only in vessels with a reference diameter between 2.76 and 2.99 mm.

DISCUSSION

The main result of the present study is that strut thickness appears to be an independent predictor of restenosis after stent implantation in vessels with a reference diameter <3.0 mm. The implantation of stents with a thin (<0.10 mm) strut may reduce the restenosis rate in small coronary arteries. In particular, implantation of thin-strutted stents implies an adjusted risk reduction of 56% for angiographic restenosis. The restenosis rate observed in the thin group was lower, even when there was a higher percentage of diabetic patients and a longer final stent length, both of which are well-known unfavorable factors of the long-term outcome, especially in small vessels (1,2). The lesions in the thick group were predisposed to restenosis by being more

Table 3. Predictors of Restenosis in the Entire Study Group, as Assessed by Univariate and Multivariable Analyses

	Restenosis Rate (%)		Univariate Analysis			Multivariable Analysis		
Variable	Factor Present	Factor Absent	Chi-Square Statistic	p Value	Odds Ratio (95% CI)	Chi-Square Statistics	p Value	Odds Ratio (95% CI)
Stent length ≥16 mm	38.5	27.6	9.14	< 0.001	1.64 (1.24–2.16)	19.61	0.001	1.03 (1.01–1.04)
Strut thickness ≥0.10 mm	27.4	34.6	5.33	0.009	1.44 (1.09–1.90)	4.65	0.001	1.68 (1.23–2.29)
Diabetes mellitus	45.5	31.2	5.68	0.017	1.84 (1.10–3.04)	8.25	0.007	2.10 (1.21–3.68)
Age ≥60 yrs	31.8	28.6	5.69	0.017	1.40 (1.06–1.84)		>0.1	
Ostial lesion	42.4	31.3	4.34	0.037	1.61 (1.03–2.54)		>0.1	
Lesion type B2/C	34.4	28	3.86	0.049	1.35 (1.00–1.82)		>0.1	
Inflation pressure ≥14 atm	29.9	37.9	5.47	0.019	0.67 (0.52–0.94)		>0.1	

For continuous variables (i.e., age, stent length and inflation pressure), the median value was used as a cut-off point to define the two subgroups with the characteristic (i.e., restenosis) present or absent. Stepwise regression analysis was performed by putting into the model all the variables significant in the univariate analysis. Variables not significant at the 0.1 level were deleted from the model.

CI = confidence interval.

Table 4. Angiographic and Procedural Characteristics of the Three Subgroups (Tertiles) According to Vessel Reference Diameter in the Thin and Thick Groups

	<2.50 mm VD		2.50–2.75 mm VD		2.76–2.99 mm VD	
	Thin Group (n = 155)	Thick Group (n = 145)	Thin Group (n = 154)	Thick Group (n = 137)	Thin Group (n = 200)	Thick Group (n = 150)
Vessel size (mm)	2.23 ± 0.22	2.20 ± 0.24	2.65 ± 0.09	2.64 ± 0.06	2.88 ± 0.05	2.90 ± 0.07
MLD (mm)						
Before stenting	0.81 ± 0.34	0.83 ± 0.29	0.85 ± 0.43	0.97 ± 0.37	0.86 ± 0.36	0.90 ± 0.44*
After stenting	2.28 ± 0.52	2.26 ± 0.47*	2.64 ± 0.40	2.63 ± 0.39	2.90 ± 0.42	2.90 ± 0.48
Follow-up	1.51 ± 0.72	1.53 ± 0.68	1.69 ± 0.78	1.56 ± 0.75*	2.02 ± 0.80	1.67 ± 0.73*
DS (%)						
Before stenting	63 ± 15	62 ± 12	66 ± 16	63 ± 14	66 ± 12	65 ± 15
After stenting	0 ± 3.62	0 ± 3.08	1.00 ± 6.05	0.79 ± 5.02	0 ± 4.45	1.00 ± 8.63
Follow-up	39 ± 22	38 ± 24	38 ± 26	43 ± 26	31 ± 25	40 ± 24*
Restenosis rate (%)	31.8	34.9	32	37.9	23.5	37*
Acute gain	1.90 ± 0.51	1.94 ± 0.44	2.14 ± 0.59	1.99 ± 1.47	2.32 ± 0.49	2.11 ± 0.64*
Relative gain	0.85 ± 0.21	0.90 ± 0.22	0.81 ± 0.22	0.76 ± 0.18*	0.80 ± 0.17	0.76 ± 0.22
Late loss	1.05 ± 0.76	1.18 ± 1.72*	1.12 ± 0.83	1.25 ± 0.75	1.12 ± 0.88	1.50 ± 1.74*
Loss index	0.56 ± 0.40	0.62 ± 0.39	0.57 ± 0.47	0.64 ± 0.38	0.48 ± 0.37	0.78 ± 0.37*
Lesion length (mm)	10 ± 6	11 ± 6	10 ± 7	11 ± 6	10 ± 6	11 ± 9
Stent length (mm)	19 ± 13	18 ± 8	21 ± 12	19 ± 12	22 ± 13	18 ± 8
Pressure inflation (atm)	16 ± 3	14 ± 3*	16 ± 3	15 ± 3*	15 ± 3	15 ± 3
BA ratio	1.4 ± 0.2	1.4 ± 0.2	1.3 ± 0.1	1.2 ± 0.1	1.2 ± 0.1	1.1 ± 0.2

*$p < 0.05$ for comparisons between groups (thin and thick) within the same reference vessel diameter range. Data are presented as the mean value ± SD.
BA = balloon-to-artery; DS = diameter stenosis; MLD = minimal lumen diameter; VD = vessel diameter.

complex, longer and more commonly located in the ostium. The favorable effect of thin struts was clinically apparent only in vessels with a reference diameter between 2.76 and 2.99 mm. The loss index was significantly lower for stents implanted in vessels that were 2.76 to 2.99 mm (Fig. 3), despite no difference in relative gain (Table 4). The fact that no advantage was found in very small vessels supports the concept that the modest benefit obtained by stents with a thin strut cannot override the small residual lumen present in very small vessels. This may be due to the relatively high metal/vessel ratio present when a stent is implanted in a small vessel.

Small artery size, as assessed by angiography, is an independent risk factor for the occurrence of restenosis and MACE at follow-up after stent implantation (1–3). The proposed mechanisms of such an unfavorable outcome are: 1) a high degree of vessel stretch and injury; 2) a small postprocedural lumen; and 3) a high metal density. Vascular injury and foreign body reaction are important mechanisms by which stent implantation can provoke neointimal hyper-

Table 5. Main and Subgroup Analysis of Angiographic Data Obtained by a Mixed Linear Model With Group, Diameter and Group × Diameter Interaction and Fixed Effects

	Between Groups	Between Diameters	Interaction Group × Diameter	Thin Group vs. Thick × Diameter ≤2.50 mm	Thin Group vs. Thick × Diameter <2.51–2.75 mm	Thin Group vs. Thick × Diameter 2.76–2.99 mm
MLD						
Before stenting	0.005	<0.001	0.043	—	—	−0.16 ± 0.04 (0.001)
After stenting	0.73	0.001	0.006	—	—	—
Follow-up	0.03	<0.001	0.007	—	—	0.29 ± 0.08 (<0.001)
DS (%)						
Before stenting	0.030	0.015	0.069	—	—	5.12 ± 1.54 (0.003)
After stenting	<0.001	0.17	0.007	—	−2.83 ± 1.05 (0.020)	−3.73 ± 0.93 (<0.001)
Follow-up	0.019	0.018	0.055	—	—	−7.46 ± 2.53 (0.009)
Acute gain	0.004	<0.001	0.008	—	—	0.23 ± 0.06 (0.001)
Relative gain	0.001	<0.001	0.11	—	—	0.08 ± 0.02 (0.001)
Late loss	0.056	0.17	0.43	—	—	—
Loss index	0.007	0.027	0.28	—	—	−0.01 ± 0.05 (0.015)
Stent length	0.49	0.003	0.55	—	—	—
Pressure inflation	<0.001	0.094	0.018	1.10 ± 0.34 (0.004)	1.47 ± 0.35 (<0.001)	—
BA ratio	<0.001	<0.001	0.43	0.07 ± 0.01 (<0.001)	0.05 ± 0.01 (0.018)	0.04 ± 0.01 (0.035)

P values obtained from the mixed linear model analysis of angiographic data, together with the significant estimated group (thin and thick) differences ± standard error and Tukey adjusted p values (in parentheses) for each subgroup (according to the reference vessel diameter ≤2.50, 2.51–2.75 and 2.76–2.99 mm) analysis. For the subgroup analyses, only statistically significant results ($p \leq 0.05$) are reported.
Abbreviations as in Table 4.

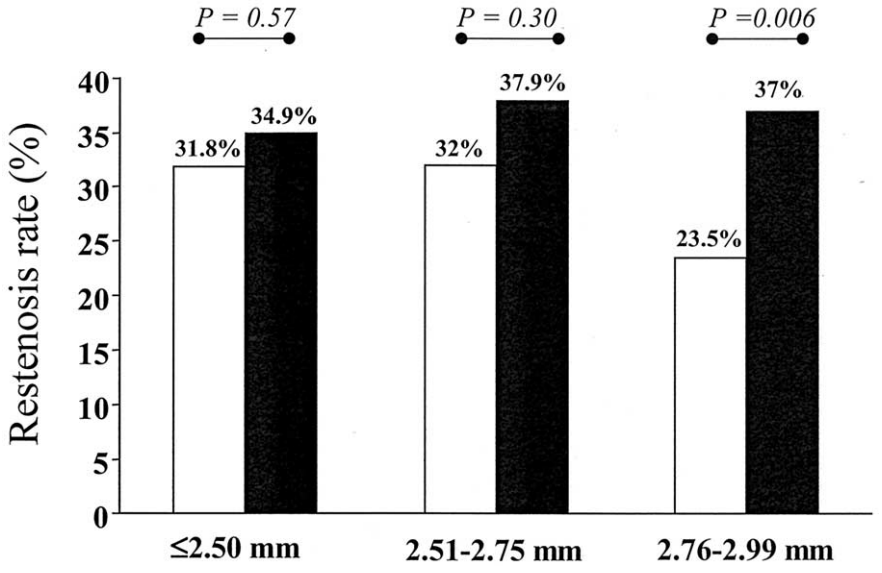

Figure 2. Restenosis rates in lesions treated with a stent with a strut thickness of <0.10 mm (thin group; **open bars**) and a stent with a strut thickness ≥0.10 mm (thick group; **solid bars**), according to the reference vessel diameter (≤2.50, 2.51 to 2.75 and 2.76 to 2.99 mm).

plasia (14–16). Understanding the factors involved in vascular injury imposed during stent deployment might allow optimization of the stent design (e.g., stent/strut geometry and stent material) to reduce restenosis.

A number of stent configurations are now available. Differences have been reported in flexibility, tracking ability, expansion, radiovisibility, side-branch access and resistance to compression and recoil. Four different direct comparisons of first-generation Palmaz-Schatz slotted-tube stents and second-generation stents have been made ("stent vs. stent" equivalency trials). In three studies (17–19), there were no significant differences in restenosis at follow-up, including MLD, percent DS, late loss and the binary restenosis rate. Randomized trials often include selected patients who are generally not representative of "real-life" stenting. Selected subsets may lead to equalization of outcomes and mask differences among the devices being tested (20).

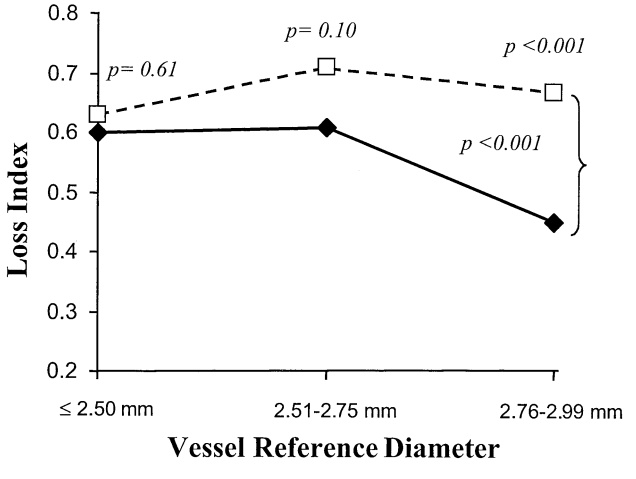

Figure 3. The loss index according to the vessel reference (2.50, 2.51 to 2.75 and 2.76 to 2.99 mm) in lesions treated with a stent with a strut thickness <0.10 mm **(solid diamonds on continuous line)** and a stent with a strut thickness ≥0.10 mm **(open squares on dotted line)**.

Strut thickness and vessel damage. Kastrati et al. (9) recently demonstrated that the use of a device with a thinner strut is associated with a significant reduction in angiographic and clinical restenosis after coronary artery stenting in vessels >2.8 mm in reference diameter. The adjusted risk of restenosis associated with the thin-strutted stent was 0.42 (95% CI 0.26 to 0.68). One mechanism of stent-induced damage is strut-imposed vascular injury, which corresponds the extent of intimal thickening in experimental animals (14,21–23). The struts of the expanding stent impose focal, deep vascular trauma in comparison to the less controlled stretching and fracturing of the vessel wall caused by balloon inflation alone (23). In addition to the deep injury associated with stent expansion, more superficial vascular injury occurs during stent expansion in areas removed from stent struts themselves. Furthermore, Rogers et al. (15) demonstrated that, using stents with a similar total surface area and strut thickness but a different geometric configuration, the stent design in which the struts created a more complex and closed area (corrugated-ring design) permitted 33% less injury in the spaces bounded by each strut, as compared with the stent design in which the inter-strut areas were more simple and open in shape (slotted-tube design).

Study limitations. This is a nonrandomized, retrospective study. Furthermore, the rate angiographic follow-up was low. However, it was similar in the two groups (56% in the thin group vs. 57% thick group; p = 0.80); therefore, any potential selection bias should be equally distributed in the two groups.

Conclusions. This study supports the concept that strut thickness is an independent predictor of angiographic restenosis in coronary arteries with a reference diameter of 2.76

to 2.99 mm. Even with the availability of drug-eluting stents, which seem to drastically reduce the restenosis rate, the result of the present study may contribute to a further reduction in the restenosis rate after stent implantation in small coronary arteries.

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