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Coronary Revascularization

Intracoronary Stenting and Angiographic Results: Strut Thickness Effect on Restenosis Outcome (ISAR-STEREO-2) Trial

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OBJECTIVES We tested the hypothesis that thinner-strut stents are associated with a reduced rate of restenosis when comparing two stents with different design.

BACKGROUND We have previously shown that, for two stents with similar design, the risk for restenosis is dependent on the strut thickness. It is unknown whether strut thickness preserves its relevance as a determinant of restenosis even in the presence of different stent designs.

METHODS A total of 611 patients with symptomatic coronary artery disease were randomly assigned to receive either the thin-strut ACS RX Multilink stent (Guidant, Advanced Cardiovascular Systems, Santa Clara, California) (strut thickness 50 μm, interconnected ring design; n = 309) or the thick-strut BX Velocity stent (Cordis Corp., Miami, Florida) (strut thickness 140 μm, closed cell design; n = 302). The primary end point was angiographic restenosis (≥50% diameter stenosis at follow-up angiography). Secondary end points were the incidence of target-vessel revascularization (TVR) and the combined rate of death and myocardial infarction (MI) at one year.

RESULTS The incidence of angiographic restenosis was 17.9% in the thin-strut group and 31.4% in the thick-strut group, relative risk, 0.57 (95% confidence interval, 0.39 to 0.84), $p < 0.001$. A TVR due to restenosis was required in 12.3% of the thin-strut group and 21.9% of the thick-strut group, relative risk, 0.56 (95% confidence interval, 0.38 to 0.84), $p = 0.002$. No significant difference was observed in the combined incidence of death and MI at one year.

CONCLUSIONS When two stents with different design are compared, the stent with thinner struts elicits less angiographic and clinical restenosis than the thicker-strut stent. (J Am Coll Cardiol 2003; 41:1283–8) © 2003 by the American College of Cardiology Foundation

Stenting has become the dominant percutaneous coronary intervention (1). An increasing number of randomized clinical trials have demonstrated that stent characteristics are an important determinant of restenosis (2–7). Although the exact underlying mechanisms of this influence are not clearly understood, stent architecture (2,3,8), material composition of stent surface (4–6), and strut thickness (7,9) have been indicated as factors that may affect the process of restenosis after stent implantation. In the Intracoronary Stenting and Angiographic Results: Strut Thickness Effect on Restenosis Outcome (ISAR-STEREO) trial (7), patients were randomly assigned to receive one of two stents with similar interconnected ring design but a different strut thickness. One year after stenting, patients who received stents with thin struts had a considerably lower restenosis rate than those receiving thick-strut stents (7). The magnitude of difference in restenosis in the ISAR-STEREO (42% risk reduction with the thin-strut stent) suggests that strut thickness plays a major role in this process, with relevant implications for stent technology (7). However, the ISAR-STEREO trial could not answer the question of whether the role of strut thickness is evident only when comparing two stents with similar interconnected ring design and is not applicable for stents with different design. This was the rationale of the present trial, ISAR-STEREO-2, in which the objective was to assess the influence of strut thickness on restenosis when two stents with different design are compared.

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METHODS

This randomized, multicenter study included 611 patients with symptomatic coronary artery disease and coronary

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Abbreviations and Acronyms

CK	= creatine kinase
ISAR-STEREO	= Intracoronary Stenting and Angiographic Results: Strut Thickness Effect on Restenosis Outcome trial
MI	= myocardial infarction
RAVEL	= Randomized Study With the Sirolimus Coated Velocity Balloon Expandable Stent in the Treatment of Patients With De Novo Native Coronary Lesions trial
RR	= relative risk
TVR	= target-vessel revascularization

lesions situated in native vessels and was conducted according to the principles of the Declaration of Helsinki and approved by the local ethics committees. All patients had given their informed consent for participation in this trial.

After successful passage of the guidewire through the target vessel, the patients were randomly assigned to receive one of the following two premounted stent types: the thin-strut stent, ACS RX Multi-Link (Guidant, Advanced Cardiovascular Systems, Santa Clara, California), has a strut thickness of 50 μm, a strut width of 100 μm, and an interconnected ring design; it was available in lengths of 15, 25, and 35 mm. The thin-strut stent model is no longer commercially available. The thick-strut stent, BX Velocity (Cordis Corporation, Miami, Florida), has a strut thickness of 140 μm, a strut width of 130 μm, and a closed cell design; it was available in lengths of 8, 13, 18, 23, 28, and 33 mm. The operator decided which balloon pressure to use for final stent expansion on the basis of the evidence provided by previous randomized trials (10). The procedure was considered successful when stent placement was associated with a residual stenosis of $<$30% and Thrombolysis in Myocardial Infarction flow grade $\geq$2. During the intervention, patients received heparin and aspirin, intravenously. The patients considered at a higher risk for thrombotic events (e.g., patients with acute myocardial infarction (MI), thrombus-containing lesions, and suboptimal procedural results) received abciximab as a bolus, followed by a 12-h intravenous infusion and heparin dosage reduced by 50%. Post-procedural therapy comprised aspirin 100 mg twice daily indefinitely and clopidogrel 75 mg/day for at least four weeks. A follow-up angiography at six months was scheduled for all patients.

Quantitative coronary angiographic evaluation. Operators of the core angiographic laboratory who performed the quantitative assessment were blinded to the randomly assigned treatment. Angiograms recorded before and immediately after the procedure as well as at six-month follow-up were assessed with the aid of the automated edge-detection system CMS (Medis Medical Imaging System, Nuenen, the Netherlands). All measurements were performed on cineangiograms recorded after intracoronary nitroglycerin administration. The same projections were used at all time points. The contrast-filled non-tapered catheter tip was used for calibration. Early lumen gain was calculated as the difference between minimal lumen diameter at the end of the intervention and before the balloon dilation. Late lumen loss was the difference in the minimal lumen diameter between that immediately after the procedure and that at follow-up, and loss index was the ratio between late lumen loss and early lumen gain. Angiographic restenosis was defined as a diameter stenosis $\geq$50% at follow-up angiography measured at any point within the stented segment or in the 5-mm proximal or distal segments adjacent to the stent.

Clinical evaluation. Adverse events were monitored throughout the follow-up period: by telephone interview at 30 days, a clinical visit at six months, and an additional telephone interview at one year after the intervention. If patients reported cardiac symptoms during the telephone interview, at least a clinical and electrocardiographic follow-up visit was performed at the outpatient clinic or by the referring physician. All information available from hospital re-admission records, the referring physician, or the outpatient clinic was entered into a computer database. Death, MI, and target-vessel revascularization (TVR) (percutaneous transluminal coronary angioplasty or bypass surgery) were considered as adverse events. The diagnosis of acute MI required the presence of at least two of the following criteria: prolonged ($>$20-min duration) and typical chest pain, new pathological Q waves on the electrocardiogram, and an increase of creatine kinase (CK) or its MB isoenzyme $>$2 times the upper limit; CK was determined before and immediately after the procedure, every 8 h for the first 24 h after stenting, and daily afterward until discharge. The criteria for TVR included the presence of restenosis accompanied by symptoms and/or positive exercise test.

End points of the study and sample size calculation. The primary end point of the study was the incidence of angiographic restenosis (see the definition in the preceding text). The sample size of the trial was calculated on the basis of the findings of a previous randomized trial (7): a 15.0% restenosis rate with the thin-strut stent and 25.8% with the thick-strut stent. We intended to be able to show a similar difference for the two stents in this study as well, with an 80% power and a two-sided α-level of 0.05. To achieve this objective, 440 patients with follow-up angiography were required. We included 611 patients to accommodate for expected missing angiographic examinations at six months.

Secondary end points focused on clinical events occurring during one year after the procedure: the incidence of TVR and the combined incidence of death and MI.

Statistical analysis. The main analyses were performed on an intention-to-treat basis. Data are presented as mean $\pm$ SD or as proportions (%). The differences between groups were assessed by chi-square test or Fisher exact test for categorical data and t test for continuous data. Survival

Table 1. Baseline Clinical Characteristics

	Thin-Strut (n = 309)	Thick-Strut (n = 302)	p Value
Age, yrs	66 ± 11	66 ± 12	0.94
Women, n (%)	79 (26)	75 (25)	0.84
Diabetes, n (%)	68 (22)	67 (22)	0.96
Current smoker, n (%)	66 (21)	65 (22)	0.96
Cholesterol level, mg/dl	209 ± 54	211 ± 47	0.53
Acute myocardial infarction, n (%)	39 (13)	36 (12)	0.79
Unstable angina, n (%)	98 (32)	93 (31)	0.81
Previous myocardial infarction, n (%)	54 (18)	69 (23)	0.10
Previous bypass surgery, n (%)	22 (7)	24 (8)	0.70

Data are mean ± SD or number of patients (%).

parameters were compared using the log-rank test. The relative risk (RR) and its 95% confidence interval (CI) were calculated for each study end point. A p value < 0.05 was considered statistically significant.

RESULTS

Table 1 shows the baseline clinical characteristics of the patients in the two groups defined by the randomly assigned device, and no statistical difference is evident. Table 2 displays the baseline angiographic and hemodynamic characteristics. Although the vast majority of the parameters show no statistical difference, the lesions in the thin-strut stent group were significantly more complex ($p < 0.001$). The group of the thick-strut stent showed, in turn, a trend toward a higher proportion of patients with chronic occlusions ($p = 0.06$).

Procedural data. Table 3 shows the procedural data. A significantly higher number of stents was implanted in the patients of the thin-strut stent group ($p = 0.02$). We performed post-stent dilations with semi-compliant balloons in the large majority of the patients. Actual maximal balloon diameter measured in the quantitative coronary angiographic core laboratory was not significantly different between the two groups (Table 3) ($p = 0.62$). Of note, there were no differences in early lumen gain and final diameter stenosis between the two groups. There was a high procedural success rate in both groups according to the intention-to-treat principle. However, although this success was always achieved with the randomly assigned device in the thick-strut stent group, the placement of a stent other than that randomly assigned, was necessary in 38 patients of the thin-strut group, which translated into a significantly lower device success rate in the latter group ($p < 0.001$). Of the 38 patients who received a stent other than the thin-strut

Table 3. Procedural Data

	Thin-Strut (n = 309)	Thick-Strut (n = 302)	p Value
Administration of abciximab, n (%)	154 (50)	149 (49)	0.90
Multilesion intervention, n (%)	122 (39)	114 (38)	0.66
Maximal balloon pressure, atm	12.1 ± 2.0	12.3 ± 2.1	0.18
Maximal balloon diameter, mm	3.39 ± 0.51	3.37 ± 0.51	0.62
Balloon-to-vessel ratio	1.17 ± 0.15	1.17 ± 0.15	0.90
Number of implanted stents	1.3 ± 0.6	1.2 ± 0.4	0.02
Length of stented segment, mm	22.0 ± 10.3	20.7 ± 9.6	0.12
Early lumen gain, mm	1.97 ± 0.61	2.00 ± 0.66	0.54
Final minimal lumen diameter, mm	2.89 ± 0.48	2.85 ± 0.52	0.32
Final diameter stenosis, %	4.3 ± 9.7	4.2 ± 11.6	0.78
Procedural success, n (%)	307 (99)	299 (99)	0.64
Device success, n (%)	269 (87)	299 (99)	< 0.001

Data are mean ± SD or number of patients (%).

Table 2. Baseline Angiographic and Hemodynamic Characteristics

	Thin-Strut (n = 309)	Thick-Strut (n = 302)	p Value
Heart rate, min^{-1}	72.9 ± 12.0	71.7 ± 11.9	0.21
Mean arterial blood pressure, mm Hg	102.5 ± 20.7	103.9 ± 19.4	0.39
Left ventricular ejection fraction, %	57.7 ± 15.5	58.1 ± 15.0	0.73
Number of diseased vessels			0.41
One vessel, n (%)	96 (31)	80 (27)	
Two vessels, n (%)	92 (30)	101 (33)	
Three vessels, n (%)	121 (39)	121 (40)	
Target vessel			0.79
Left main, n (%)	3 (1)	3 (1)	
LAD, n (%)	138 (45)	123 (41)	
LCx, n (%)	76 (24)	77 (25)	
RCA, n (%)	92 (30)	99 (33)	
Complex lesions (B2/C lesions)*	255 (82)	213 (70)	< 0.001
Chronic occlusions, n (%)	15 (5)	26 (9)	0.06
Restenotic lesions, n (%)	10 (3)	13 (4)	0.49
Lesion length, mm	13.9 ± 7.8	14.1 ± 7.8	0.77
Vessel size, mm	2.93 ± 0.50	2.91 ± 0.51	0.68
Diameter stenosis, %	68.2 ± 18.9	70.8 ± 20.4	0.11

*Lesion complexity was assessed on the basis of the modified classification of the American College of Cardiology/American Heart Association. Data are mean ± SD or number of patients (%).

LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; RCA = right coronary artery.

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Table 4. Angiographic Data at Follow-up

	Thin-Strut (n = 229)	Thick-Strut (n = 236)	p Value
Minimal lumen diameter, mm	1.96 ± 0.76	1.70 ± 0.83	< 0.001
Diameter stenosis, %	33.4 ± 21.5	42.4 ± 24.1	< 0.001
Late lumen loss, mm	0.93 ± 0.61	1.19 ± 0.69	< 0.001
Loss index	0.51 ± 0.37	0.65 ± 0.44	< 0.001
Incidence of restenosis, n (%)	41 (17.9)	74 (31.4)	< 0.001

Data are mean ± SD or number of patients (%).

model, 31 received the BX Velocity stent, four the AVE stent (Medtronic, Minneapolis, Minnesota), and three the Multi-Link Tetra stent (Guidant, Advanced Cardiovascular Systems). Postprocedurally, an increase in CK or CK-MB 3× above the normal limit, as defined previously (11), was observed in 17 patients (6.4%) of the thin-strut stent group and 12 patients (4.5%) of the thick-strut stent group (p = 0.34).

Angiographic data at six months. Procedural failure, death, or TVR during the first 30 days were considered ineligibility criteria for the six-month angiography. Accordingly, 10 patients (3.2%) of the thin-strut stent group and six patients (2.0%) of the thick-strut stent group were ineligible for follow-up angiography (p = 0.33). Follow-up angiography was carried out in 229 of the 299 eligible patients (77%) of the thin-strut group and 236 of the 296 eligible patients (80%) of the thick-strut group (p = 0.35). Angiographic findings at follow-up are displayed in Table 4. All parameters are concordant in showing a significantly greater risk for lumen re-narrowing with the thick-strut stent. Figure 1 shows that, although early lumen gain was practically similar in both groups, late lumen loss was significantly more considerable in the thick-strut stent group.

The primary end point of the study, the incidence of angiographic restenosis, was encountered in 17.9% of the patients in the thin-strut group and 31.4% of the patients in the thick-strut group (Fig. 2, left panel) (p < 0.001). Thus, the use of the thin-strut stent was associated with a 43% reduction of the risk for restenosis, compared with the thick-strut stent, RR, 0.57 (95% CI, 0.39 to 0.84).

We performed two additional analyses after excluding specific subgroups of patients. When we excluded patients who did not receive the randomly assigned stent, the restenosis rates were 17.6% in the thin-strut group and 31.4% in the thick-strut group; when we excluded patients with chronic occlusions, the restenosis rates were 16.6% in the thin-strut group and 29.3% in the thick-strut group.

Clinical data at one-year follow-up. During one year after the procedure, a TVR due to restenosis was required in 38 patients of the thin-strut group (12.3%) and 66 patients of the thick-strut group (21.9%) (Fig. 2, right panel) (p = 0.002). This indicates a 44% risk reduction with the use of the thin-strut stent (RR, 0.56 [95% CI, 0.38 to 0.84]). During the same follow-up period, 15 patients of the thin-strut group (4.9%) and 19 patients of the thick-strut group (6.3%) died or incurred an MI (RR, 0.77 [95 CI, 0.39 to 1.52], p = 0.46). No significant differences were observed in one-year mortality rates, with 3.9% in the thin-strut group and 4.6% in the thick-strut group (p = 0.66).

DISCUSSION

We used two stainless steel stents with different architecture and strut thickness. The thin-strut stent has been the first model of interconnected ring design in the Multi-Link series. Although several other stent models share a similar design, the most peculiar characteristic of the thin-strut stent used in the present study is its strut thickness of only 50 μm. The thick-strut stent, BX-Velocity, has a strut thickness of 140 μm, similar to several other stents that are currently used by interventional cardiologists. Its thickness is identical to that of the thick-strut stent used in the ISAR-STEREO trial (7). The thick-strut stent used in the present trial has a closed-cell design markedly different from that of the thick-strut stent implanted in the ISAR-STEREO trial (7). The design of the BX-Velocity stent enabled a high device success rate that was much better than that achieved with the system carrying the ACS RX Multi-Link thin-strut stent. The patients were followed up for one year, and an angiography scheduled at six months was performed in a high proportion of patients even in the absence of symptoms. The stents assessed in our trial were associated with a comparable one-year incidence of death and MI. The main finding of the study indicates, however, that when two stents with different design are compared, the stent with thinner struts elicits less angiographic and clinical restenosis.

Comparison with previous trials. The incidence of restenosis at follow-up angiography with the thin-strut stent was 17.9%. This appears to be slightly higher than the incidence

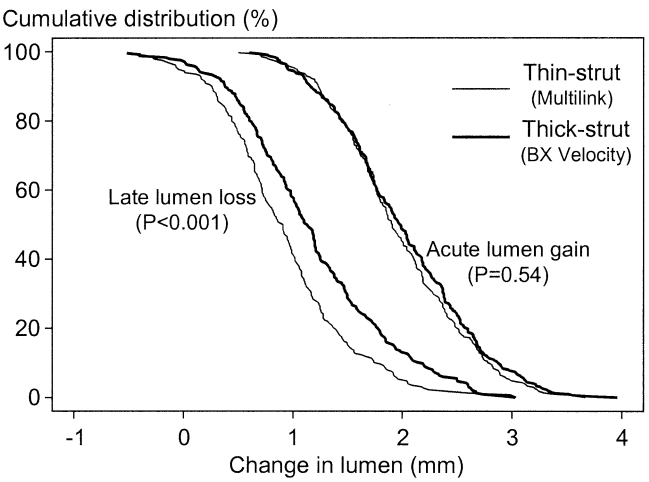

Figure 1. Cumulative distribution curves of early ("acute") lumen gain (enlargement of lumen achieved by stenting and measured immediately after the procedure) and late lumen loss (lumen narrowing occurred during the period between intervention and follow-up angiography at 6 months). Note that similar early results (as indicated by the **curves depicting early lumen gain**) do not necessarily translate into similar results at 6 months (as indicated by the **curves depicting late lumen loss**).

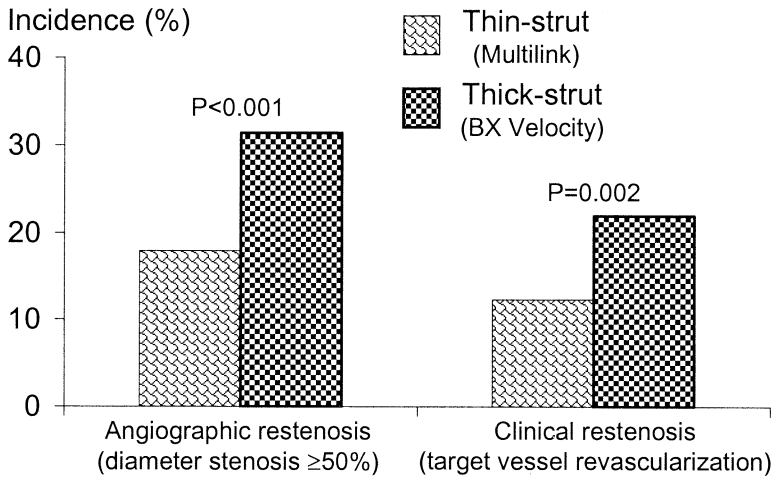

Figure 2. Incidence of angiographic **(left)** and clinical **(right)** restenosis in each group.

of restenosis of 15.0% reported from in the previous ISAR-STEREO trial with the same stent type (7). The reason may be found in the difference in vessel size between the populations included in the two ISAR-STEREO trials, with smaller coronary vessels treated in the present trial. Altogether, the findings of the ISAR-STEREO trials as well as those of another randomized trial (12) demonstrate that consistently low restenosis rates can be achieved with thin-strut stents.

In the present trial, the incidence of restenosis at follow-up angiography with the thick-strut stent was 31.4%. The same type of stent has been used in the uncoated stent arm of the Randomized Study With the Sirolimus Coated Velocity Balloon Expandable Stent in the Treatment of Patients With de novo Native Coronary Lesions (RAVEL) (13). In the RAVEL trial, which included a control group of 118 patients treated with the uncoated BX-Velocity stent, the angiographic restenosis rate in this arm was 26% (13). Considering that the study lesions in the ISAR-STEREO 2 trial were more complex, there is essentially no difference between our trial and the RAVEL trial with respect to the incidence of angiographic restenosis observed in the uncoated, thick-strut arm. Consistent with these results are also the findings of clinical restenosis, with a revascularization rate of 21.9% in the present trial and 22% in the RAVEL trial (13).

Clinical implications. Although there was no difference in mortality and the incidence of MI, the group assigned to the thin-strut stent had a 43% decrease in the risk for restenosis at follow-up angiography and a 44% reduction in the risk for re-intervention, compared with the group assigned to the thick-strut stent. Similar reduction in the risk for restenosis was also observed in the group of the thin-strut stent in the first ISAR-STEREO trial (7). The consistent reduction of restenosis with the thin-strut stent compared with two thick-strut stents with different architecture in both ISAR-STEREO trials strongly supports a role for the strut thickness that is largely independent of other stent characteristics. We do not know the exact mechanisms underlying the reduction of restenosis after the use of thin-strut stents. A few possibilities have already been discussed in the previous publication on the same subject (7). Despite the paucity of the mechanistic data that may help to explain the influence of strut thickness on restenosis (9), the findings of the present trial have important implications because most of the stents currently in use have a strut thickness similar to that of the thick-strut stent used in this and in the previous ISAR-STEREO trial. Drug-eluting stent technology has surely opened new promising prospects for our efforts against restenosis (13), and hopefully, large studies in the near future will be able to prove the abolishment of restenosis through this technology.

Study limitations. The design of the present study aimed at complementing the previous ISAR-STEREO trial (7). Although both trials suggest a relevant role for strut thickness in in-stent restenosis, other stent-related factors might also have contributed to the differences in restenosis observed between the study groups. The impact of stent geometry on restenosis has not been evaluated in controlled clinical trials, and it is not known which geometrical characteristics enable reduction of restenosis. The differences in stent geometry between the thick- and the thin-strut models, which were subtle in the previous ISAR-STEREO trial (7) and much more conspicuous in the present trial, might also have contributed to the overall better result achieved with the thin-strut stent. Therefore, further work is needed to quantify the degree in which strut thickness per se influences in-stent restenosis and to identify the optimal strut thickness to use during stent manufacturing.

Two additional limitations should also be acknowledged. First, due to the failure of thin-strut device, the procedural success in 12% of the patients in the thin-strut group was achieved after crossover to the thick-strut model. We tried to adjust for this inconvenience by performing an additional analysis confined to patients without crossover, but both the

nature and magnitude of the possible bias introduced by the crossover phenomenon remain still unknown. Second, the difference in radiolucency between the thin- and the thick-strut models might have interfered with the quantitative angiographic assessment. A specifically designed study concluded that quantitative angiography can be used as an accurate method of lumen assessment after implantation of steel stents with different thickness and that only the highly opaque tantalum stent may reduce the accuracy to some degree (14). However, our angiographic data are also supported by the incidence of reinterventions, which favors the thin-strut model.

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APPENDIX

The following centers and investigators participated in the ISAR-STEREO-2 trial:

Steering Committee: A. Schömig (chairman), A. Kastrati, J. Dirschinger.

Data Coordinating Center: A. Kastrati, M. Hadamitzky, Deutsches Herzzentrum, Munich.

Angiographic Core Laboratory: J. Mehilli, A. Redl, D. Kiemoser, S. Pinieck, Deutsches Herzzentrum, Munich.

Clinical Follow-up Center: H. Holle, K. Hösl, F. Rodrigues, S. Koch, M. Müller; Deutsches Herzzentrum, Munich.

Patient-Enrolling Centers: Deutsches Herzzentrum, Munich: J. Dirschinger (principal investigator), C. Schmitt, R. Blasini, M. Gawaz, J. Pache; 1. Medizinische Klinik rechts der Isar, Munich: F.-J. Neumann (principal investigator), H. Schühlen, M. Seyfarth; Medizinische Klinik I, Garmisch-Partenkirchen: F. Dotzer (principal investigator), M. Fleckenstein. Medizinische Klinik I, Ingolstadt: C. Pfafferott (principal investigator), U. Sattelberger.