Vessel Size and Long-Term Outcome After Coronary Stent Placement

Shpend Elezi, MD; Adnan Kastrati, MD; Franz-Josef Neumann, MD; Martin Hadamitzky, MD; Josef Dirschinger, MD; Albert Schömig, MD

Background—The role of coronary stenting in the treatment of patients with small vessels is not well defined. The purpose of this study was to investigate the influence of vessel size on long-term clinical and angiographic outcome after coronary stent placement.

Methods and Results—The study comprised 2602 patients with successful stent implantation for symptomatic coronary artery disease. Patients were subdivided into 3 equally sized groups (tertiles) according to vessel size, with respective ranges of <2.8, 2.8 to 3.2, and >3.2 mm. Event-free survival at 1 year was 69.5% in the group with smaller vessels, 77.5% in the second group, and 81% in the group with larger vessels (P<0.001). Late lumen loss was similar between the 3 groups (1.12±0.73, 1.12±0.79, and 1.09±0.88 mm, respectively). Angiographic restenosis rate was significantly higher in the small-vessel group (38.6%, 28.4%, and 20.4% in groups 1, 2, and 3, respectively; P<0.001). The analysis identified subgroups with different risk for restenosis even among patients with small vessels. Within this group, the restenosis rate may be as low as 29.6% in patients without additional risk factors and as high as 53.5% in patients with diabetes and complex lesions.

Conclusions—Patients with small vessels present a higher risk for an adverse outcome after coronary stent placement because of a higher incidence of restenosis. However, the unusually high risk for restenosis is confined to those patients with small vessels who have concomitant risk factors such as diabetes and complex lesions. (Circulation. 1998;98:1875-1880.)

Key Words: angiography ■ coronary disease ■ restenosis ■ stents ■ vessels

Coronary stent placement is an established treatment for patients with symptomatic coronary artery disease. However, uncertainty about the results of stenting in small vessels has been the reason that only patients with target vessels ≥3 mm have been included in randomized trials. Therefore, stenting is generally recommended as a treatment option only for larger vessels. Indeed, this constitutes a major limitation for broadening the indications for coronary stent implantation. Previous studies with percutaneous coronary interventions have focused on the influence of vessel size on angiographic restenosis. Analyses after PTCA have generally shown an inverse relationship between vessel size and severity of angiographic restenosis at follow-up. Several studies have assessed the influence of vessel size on angiographic restenosis after coronary stent placement. If the proposed generalized model of restenosis based on a uniform late loss/acute gain ratio is also applicable to small vessels, stent placement is expected to offer advantages over the entire range of vessel size because of its better immediate results. Furthermore, angiographic assessment alone may not reliably reflect the clinical outcome of these patients. Concerns have been expressed about the possible dissociation between the angiogram and clinical outcome. It would be of interest to know whether all patients with smaller vessel size are affected by a similar risk or whether subgroups with different risks can be identified among them. Thus, much more information is needed before the role of coronary stenting in the treatment of patients with small vessels can be established.

The purpose of this study was to investigate the influence of vessel size on clinical and angiographic outcome after successful stent implantation in a large patient population.

Methods

Patient Population

The study population comprised all 2602 patients from the Deutsches Herzzentrum and 1. Medizinische Klinik der Technische Universität, Munich, with successful stent placement (stent at the desired position, residual stenosis <30%) during the period from May 1992 through February 1997. Excluded from the study were patients with cardiogenic shock or mechanical ventilation before PTCA. All patients who survived the first month after the procedure with neither stent vessel occlusion nor any other cardiac event (myocardial infarction, coronary bypass surgery, or PTCA) were considered eligible for follow-up angiography at 6 months. Follow-up angiography at a median of 188 days (interquartile range, 173 to 203) was performed in 2017 (80.5%) of the 2506 eligible patients.

Stent Placement

All patients received 15 000 U of heparin and 500 mg of aspirin intravenously before PTCA. Short, 7-mm or standard articulated...
15-mm Palmaz-Schatz stents (Johnson & Johnson) were delivered under fluoroscopic guidance after they were hand-crimped on slightly oversized conventional angioplasty balloons. Adequacy of the final result was based solely on visual assessment of the stent site in the angiogram; intravascular ultrasound was used only in a minority of cases. Poststenting antithrombotic therapy consisted of either oral anticoagulant (initial phase) or combined antiplatelet therapy, as previously described.16

Quantitative Angiographic Analysis
Standardized image acquisition was used, consisting of multiple projections for each lesion, accurately reproduced in each angiographic session. Before each sequence, intracoronary injections of 0.1 to 0.3 mg of nitroglycerin were used to control for vasomotor tone. Quantitative analysis was performed on the baseline angiogram, on that containing the maximally inflated balloon, and on final poststenting and follow-up angiograms. An automated edge-detection algorithm (CMS 3.0, Medis Medical Imaging Systems) was used to obtain actual balloon diameter, interpolated reference diameter (RD), minimal lumen diameter (MLD), and diameter stenosis.

Definitions and Study End Points
Acute gain was calculated as the difference between the final and the original MLD; late loss was calculated as the difference between

### TABLE 1. Patient Clinical Characteristics

<table>
<thead>
<tr>
<th>Group 1: &lt;2.8 mm</th>
<th>Group 2: 2.8–3.2 mm</th>
<th>Group 3: &gt;3.2 mm</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n=870)</td>
<td>(n=866)</td>
<td>(n=866)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>25.7</td>
<td>24.6</td>
<td>18.9</td>
</tr>
<tr>
<td>Age, y</td>
<td>63.5±10.8</td>
<td>62.4±10.8</td>
<td>61.8±10.9</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>29.4</td>
<td>33.5</td>
<td>26.6</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>30.2</td>
<td>36.5</td>
<td>40.1</td>
</tr>
<tr>
<td>Arterial hypertension</td>
<td>64.3</td>
<td>59.2</td>
<td>61.3</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>18.0</td>
<td>13.4</td>
<td>16.9</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>38.2</td>
<td>38.6</td>
<td>39.1</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>9.9</td>
<td>8.4</td>
<td>15.0</td>
</tr>
<tr>
<td>Previous PTCA</td>
<td>10.7</td>
<td>11.5</td>
<td>11.2</td>
</tr>
<tr>
<td>Multivessel disease</td>
<td>72.1</td>
<td>67.9</td>
<td>74.3</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>39.4</td>
<td>42.9</td>
<td>45.6</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>14.3</td>
<td>16.5</td>
<td>21.0</td>
</tr>
<tr>
<td>Multilesion intervention</td>
<td>26.0</td>
<td>24.2</td>
<td>22.6</td>
</tr>
</tbody>
</table>

Data are mean±SD or percentages.

### TABLE 2. Lesion- and Procedure-Related Characteristics

<table>
<thead>
<tr>
<th>Group 1: &lt;2.8 mm</th>
<th>Group 2: 2.8–3.2 mm</th>
<th>Group 3: &gt;3.2 mm</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n=870)</td>
<td>(n=866)</td>
<td>(n=866)</td>
<td></td>
</tr>
<tr>
<td>Lesion location</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left main artery</td>
<td>0.2</td>
<td>1.2</td>
<td>3.5</td>
</tr>
<tr>
<td>LAD</td>
<td>52.7</td>
<td>45.3</td>
<td>29.2</td>
</tr>
<tr>
<td>LCx</td>
<td>25.6</td>
<td>16.7</td>
<td>11.5</td>
</tr>
<tr>
<td>RCA</td>
<td>19.4</td>
<td>33.9</td>
<td>46.9</td>
</tr>
<tr>
<td>Saphenous vein graft</td>
<td>2.1</td>
<td>3.0</td>
<td>8.8</td>
</tr>
<tr>
<td>Restenotic lesions</td>
<td>11.7</td>
<td>13.2</td>
<td>11.8</td>
</tr>
<tr>
<td>Chronic occlusion</td>
<td>6.0</td>
<td>7.5</td>
<td>6.5</td>
</tr>
<tr>
<td>Dissection before stenting</td>
<td>45.6</td>
<td>46.3</td>
<td>44.0</td>
</tr>
<tr>
<td>RD before intervention, mm</td>
<td>2.51±0.23</td>
<td>3.05±0.12</td>
<td>3.67±0.36</td>
</tr>
<tr>
<td>RD after intervention, mm</td>
<td>2.63±0.27</td>
<td>3.13±0.20</td>
<td>3.69±0.38</td>
</tr>
<tr>
<td>MLD before intervention, mm</td>
<td>0.66±0.38</td>
<td>0.77±0.47</td>
<td>0.88±0.61</td>
</tr>
<tr>
<td>MLD after intervention, mm</td>
<td>2.55±0.35</td>
<td>2.93±0.31</td>
<td>3.36±0.43</td>
</tr>
<tr>
<td>Balloon pressure, atm</td>
<td>13.8±3.2</td>
<td>13.9±3.1</td>
<td>14.1±3.1</td>
</tr>
<tr>
<td>Balloon-to-vessel ratio</td>
<td>1.12±0.13</td>
<td>1.05±0.11</td>
<td>0.99±0.09</td>
</tr>
<tr>
<td>No. of stents/lesion</td>
<td>1.12±0.65</td>
<td>1.18±0.67</td>
<td>1.24±0.68</td>
</tr>
<tr>
<td>DS before intervention, %</td>
<td>73.6±14.8</td>
<td>74.7±15.3</td>
<td>76.2±16.0</td>
</tr>
<tr>
<td>DS after intervention, %</td>
<td>3.1±10.3</td>
<td>6.1±9.1</td>
<td>8.5±8.9</td>
</tr>
<tr>
<td>Acute gain, mm</td>
<td>1.88±0.49</td>
<td>2.16±0.55</td>
<td>2.48±0.66</td>
</tr>
</tbody>
</table>

LAD indicates left anterior descending coronary artery; LCx, left circumflex; RCA, right coronary artery; and DS, diameter stenosis. Data are mean±SD or percentages.
final poststenting MLD and MLD measured at follow-up angiography. Loss index was the calculated ratio of late loss and acute gain. Acute gain and late loss were also adjusted for vessel size, and relative gain and relative loss parameters were obtained. Binary restenosis was defined as a diameter stenosis $\geq 50\%$ at control angiography. Lesions were also qualitatively classified by use of the modified American College of Cardiology/American Heart Association grading system, and type B2 and C lesions were considered complex lesions.

Major adverse cardiovascular events were defined as death of cardiac or procedure-related origin, nonfatal myocardial infarction, and target-lesion revascularization (CABG or repeat PTCA of the stented vessel). All deaths were considered of cardiac origin unless a noncardiac cause was established by autopsy. The diagnosis of acute myocardial infarction was made in the presence of a clinical episode of prolonged chest pain, a rise in serum cardiac enzyme levels to at least twice the upper normal limit, or the appearance of $\geq 1$ new pathological Q wave. Cardiac events were monitored throughout the follow-up period. The diagnosis of stent vessel occlusion was always based on symptom-driven or routinely scheduled coronary angiography in the presence of Thrombolysis In Myocardial Infarction (TIMI) flow grade 0 or 1.

The primary clinical end point of the study was the probability of event-free survival at 1 year. The primary angiographic end point was restenosis at follow-up as assessed by binary restenosis and late lumen loss. Secondary end points were the occurrence of any major adverse cardiovascular event or stent vessel occlusion during the first 30 days after the procedure.

### Statistical Analysis

Statistical analyses were performed with S-Plus (Mathsoft, Inc) or SPSS statistical software packages on a per-patient basis. For patients with multileesion interventions, only 1 lesion was randomly selected for analysis. However, for primary end points (both clinical and angiographic), this approach was validated through a repeated analysis confined to patients with single-lesion intervention. The study population was subdivided into 3 groups (tertiles) according to RD; the ranges were $\leq 2.8$ mm for the first group, 2.8 to $3.2$ mm for the second, and $>3.2$ mm for the third. Group differences were assessed by ANOVA or Kruskal-Wallis H test for continuous variables and $\chi^2$ analysis for categorical variables.

Multivariate logistic regression was used to assess the independent role of vessel size in restenosis after adjustment for other covariates. Event-free survival curves for all cardiac events and specifically for myocardial infarction were constructed by means of the Kaplan-Meier method. Survival probabilities of the 3 groups were compared with log-rank test. In addition, assessment of the independent role of vessel size in event-free survival was made by use of Cox proportional hazards regression model and adjustment for other covariates. A $\chi^2$ automatic interaction detection (CHAID) algorithm (SPSS Inc) was used to identify subgroups of patients with different risk for restenosis within the group with small vessels.

Data are expressed as mean $\pm$ SD for continuous variables and as percentages for discrete variables. Differences were considered to be statistically significant when the respective $P$ values were $<0.05$.

### Results

Baseline characteristics of patients are shown in Tables 1 and 2.

#### Clinical Outcome

Table 3 shows clinical outcome during the first 30 days. The incidence of any major adverse cardiovascular event during this period was $4.5\%$ in the first group, $3.3\%$ in the second, and $2.9\%$ in the third ($P=0.65$). There was a significant difference for repeat PTCA between the 3 groups, with the highest incidence occurring in the group with small vessels.

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Plot of Kaplan-Meier event-free survival curves.

| Group 1:  
| $\leq 2.8$ mm  
| (n=870)  
| Group 2:  
| 2.8–3.2 mm  
| (n=866)  
| Group 3:  
| $>3.2$ mm  
| (n=866)  
| Major adverse cardiovascular event  
| 39 (4.5)  
| 29 (3.3)  
| 25 (2.9)  
| 0.183  
| Death  
| 9 (1.0)  
| 6 (0.7)  
| 10 (1.2)  
| 0.593  
| Nonfatal myocardial infarction  
| 18 (2.1)  
| 13 (1.5)  
| 10 (1.2)  
| 0.303  
| CABG  
| 1 (0.1)  
| 3 (0.3)  
| 1 (0.1)  
| 0.447  
| PTCA  
| 31 (3.6)  
| 19 (2.2)  
| 14 (1.6)  
| 0.027  
| Subacute stent occlusion  
| 21 (2.4)  
| 22 (2.5)  
| 17 (2.0)  
| 0.702  

Data are number (%).
In 32% of the patients in the group with small vessels, the reason for reintervention was a large residual dissection. After 1 year, the probability of survival free of myocardial infarction was not significantly different (94.7%, 96.2%, and 95.4% in groups 1, 2, and 3, respectively; \( P = 0.34 \)). There was also no significant difference between the 3 groups in the probability of survival free of myocardial infarction when the analysis was restricted to patients with single-lesion interventions (95.0%, 97.0%, and 95.5% in groups 1, 2, and 3, respectively; \( P = 0.38 \)). However, the probability of event-free survival was significantly different (69.5% in the first group, 77.5% in the second, and 81% in the third; Figure 1). This was due to a higher rate of repeat PTCA in the first group (26.3%) compared with the second and third groups (18.8% and 14.3%, respectively; \( P < 0.001 \)), whereas the rate of CABG was similar in all 3 groups (2.5% versus 2.2% versus 1.6%; \( P = 0.41 \)). Significant differences between the 3 groups (\( P < 0.001 \)) were also recorded for event-free survival when the analysis was confined to patients with single-lesion interventions. The results of the Cox proportional hazards regression model that included 21 explanatory variables demonstrated that small vessel size is an independent risk factor for the occurrence of major adverse cardiovascular events during 1 year of follow-up. Vessel size was entered into the model as a continuous variable, and a comparison between vessel sizes of 2.7 mm (1st quartile) and 3.4 mm (3rd quartile) yielded a hazard ratio of 1.56 (95% CI, 1.37 to 1.75). Additional independent risk factors were age, diabetes, complex lesions, multiple stent placement, and lower balloon-to-vessel ratio. Figure 2 is a graphic demonstration of the relation between vessel size and adjusted risk for an adverse outcome as derived from the Cox model. The risk shows a gradual increase as vessel size decreases.

### Angiographic Outcome

Subacute stent occlusions occurred in 21 patients (2.4%) in the first group, 22 (2.5%) in the second group, and 17 (2.0%) in the third group (Table 3). Angiographic outcome at 6 months is presented in Table 4. Restenosis rate demonstrated a steady decrease as vessel size increased: 38.6% in the first group, 28.4% in the second, and only 20.4% in the third (\( P < 0.001 \)). Differences in restenosis rate between the 3 groups remained significant when the analysis was restricted to patients with single-lesion intervention (37.0%, 27.3%, and 19.9% in groups 1, 2, and 3, respectively; \( P < 0.001 \)). Late lumen loss was not statistically different despite the significant difference in acute gain observed between the 3 groups. In addition, linear regression analysis demonstrated that variations in relative gain could explain <0.6% of the variability in relative loss.

In the multivariate logistic regression model that included 21 explanatory variables, small vessel size remained a significant independent risk factor for restenosis, with an odds ratio of 1.75 (95% CI, 1.49 to 2.08) for a vessel size of 2.7 versus 3.4 mm; additional independent risk factors were diabetes, previous PTCA, complex lesions, diameter stenosis before intervention, a lower balloon-to-vessel ratio, and multiple stent placement. Figure 3 displays the relation between vessel size and probability of restenosis for 2 arbitrarily assumed cutoff points of balloon-to-vessel ratio (0.90 and 1.10), adjusted for the effect of all other covariates in the model. A lower risk for restenosis is expected for a balloon-to-vessel ratio of 1.1 over the entire range of vessel sizes. The results of CHAID analysis, illustrated in Figure 4, demonstrate that patients with small vessels can be further divided into subgroups with different risks for restenosis. A particularly high risk for restenosis is associated with complex lesions in diabetic patients (53.5%). On the other hand, almost 25% of patients with small vessels have a combination of favorable characteristics (simple lesions, no diabetes) that

### Table 4. Results of Follow-Up Angiography

<table>
<thead>
<tr>
<th>Group</th>
<th>Vessel Size</th>
<th>Restenosis Rate</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;2.8 mm</td>
<td>38.6%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.8–3.2 mm</td>
<td>28.4%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;3.2 mm</td>
<td>20.4%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(n=664)</td>
<td>(n=676)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(n=677)</td>
<td>(n=707)</td>
<td></td>
</tr>
</tbody>
</table>

DS indicates diameter stenosis. Data are mean±SD or percentages.

Figure 3. Relationship between vessel size and risk for restenosis for 2 arbitrary values of balloon-to-vessel ratio after adjustment for effect of other covariates included in multiple logistic regression model. A progressive increase in risk is noted with decrease in vessel size for both levels of balloon-to-vessel ratio. In addition, risk of restenosis is higher with lower balloon-to-vessel ratio for each level of vessel size.

Figure 4. CHAID diagram showing factors (diabetes mellitus, complex lesions) that define subgroups with unusually high risk for restenosis among patients with small vessels. Boxes represent different subgroups with respective restenosis rate (middle row) and number of patients (bottom row).
considerably reduces the risk of restenosis to <30%. The subgroups with higher risk for restenosis (patients with complex lesions or diabetes) did not differ significantly from those with lower risk with respect to either vessel size ($P=0.5$) or balloon-to-vessel ratio used during the procedure ($P=0.4$).

**Discussion**

This study indicates that patients with smaller vessel size have a less-favorable clinical outcome at 1 year after coronary stent placement than patients with larger vessels. Most of the difference in the 1-year clinical outcome was generated after the first month. In fact, patients with smaller vessels had very similar rates for the most adverse clinical events, ie, death and myocardial infarction. The difference in the overall event-free survival arose from a major need for revascularization procedures in patients with smaller vessels. This must be attributed to a restenosis rate for small vessels that was $\geq 1.5 \times$ higher than that demonstrated for larger vessels. A worse clinical outcome was reported for subsets of patients with small vessel size from the BENESTENT$^9$ and STRESS$^{12}$ trials, especially when small vessel size was combined with greater lesion length.$^{19}$ Several studies failed to demonstrate a significant independent role for vessel size in the restenosis process.$^6$–$^9,19,20$ Although it was part of the analysis, the relation between vessel size and restenosis was not the principal focus of these studies, and the limited number of patients in these studies may have imparted insufficient power in this regard. Most of these studies entered postprocedural MLD into the multivariate models, which may have blunted the independent role of vessel size in outcome. Recently, Serruys et al$^{21}$ demonstrated that a multivariate model based on vessel size and residual stenosis (similar to our model) was more powerful than that based on postprocedural MLD. This model is even more appropriate for studies that are focused on the influence of vessel size. Our angiographic findings are in line with those previously reported by Foley et al$^5$ in a large study with PTCA.

Although it is evident from the present study that a higher incidence of restenosis is responsible for the more adverse clinical outcome of patients with smaller vessels, the reason smaller vessels carry a higher risk for restenosis is not clear. The explanation must be sought in our results relative to late lumen loss. We found similar values of late lumen loss in all 3 vessel-size groups despite the significantly different acute gain achieved. Similar findings have also emerged from studies with PTCA.$^{19,22}$ It is obvious that the same lumen loss entails many more consequences for small vessels than for larger vessels. A greater balloon-to-vessel ratio was used in the small-vessel group, which may have led to greater vessel wall injury and more considerable reactive neointimal hyperplasia.$^{23}$ However, it is difficult to accept this as a mechanism for more restenosis in the small-vessel group. Our multivariate model of restenosis demonstrated that the adjusted risk for restenosis decreases if a greater balloon-to-vessel ratio is used during the intervention (Figure 4). The influence of balloon-to-vessel ratio on the risk for early adverse events must be specifically assessed before the strategy of using oversized balloons is recommended as a remedy for the excessive restenosis verified in the group with small vessels.

Another mechanism of greater lumen loss in small vessels has recently been suggested by studies with intravascular ultrasound. Hoffmann et al$^{24}$ found that preinterventional plaque burden, measured with intravascular ultrasound as the ratio between plaque and total artery area, was a very strong predictor of restenosis. Plaque burden is expected to be greater in smaller vessels, thereby representing more stimulus for lumen renarrowing. This may be particularly true for patients with diabetes.$^{25}$ Other plausible explanations may come from differences in accompanying characteristics that may favor more restenosis in the small-vessel group. We found significant differences in age, sex distribution, frequency of diabetes, and proportion of lesions located in the left anterior descending coronary artery. In particular, a major presence of diabetes and the location of lesions in the left anterior descending coronary artery have frequently been associated with a higher risk for restenosis.$^{26–28}$

The stratification scheme produced by the specific analysis for the small-vessel group has clinical implications. We found that the unusually high risk for restenosis does not equally affect all patients with small vessels. Particularly unfavorable results were confined to $\approx 10\%$ of these patients who had both complex lesions and diabetes. For almost 25% of the patients, an acceptable restenosis rate is expected that is not different from the usual incidence of this complication after stenting.

A major limitation of the present study is that the analysis was restricted to stent implantation, and no other interventional devices were used. Thus, the results of this study enable us to state that small vessels are associated with higher risk after stenting but do not provide any information about the most appropriate treatment in this setting. The lack of intravascular ultrasound assessment must be considered an additional limitation of the present study. Such assessment might have provided more insights into potential specific restenosis mechanisms present in small vessels.$^{29}$

**Conclusions**

Patients with small vessels present a higher risk for an adverse outcome during the 12 months after coronary stent placement. This higher risk is generated by a major need for target-lesion revascularizations because of a higher incidence of restenosis in small vessels. However, the unusually high risk for restenosis is confined to a small percentage of patients who have concomitant risk factors such as diabetes and complex lesions. If such adverse characteristics are not present, stenting may be safely performed in patients with small coronary vessels, and favorable long-term results are to be expected.

**References**

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