Stent Placement to Prevent Restenosis After Angioplasty in Small Coronary Arteries

Serge Doucet, MD; Martin J. Schalij, MD; Mathy C.M. Vrolix, MD; David Hilton, MD; Patrick Chenu, MD; Bernard de Bruyne, MD; Wasan Udayachalerm, MD; Ashok Seth, MD; Luc Bilodeau, MD; Johan H.C. Reiber, PhD; François Harel, MSc; Jacques Lespérance, MD; for the Stent In Small Arteries (SISA) Trial Investigators*

Background—Lesions in small-diameter vessels (<3 mm) define a group with distinct clinical and morphological characteristics. There is an inverse relationship between vessel size and angiographic restenosis rate. This study assessed whether stents reduce angiographic restenosis in small coronary arteries compared with standard balloon angioplasty.

Methods and Results—We randomly assigned 351 symptomatic patients needing dilatation of 1 native coronary vessel between 2.3 and 2.9 mm in size to angioplasty alone (n=182) or stent implantation (n=169). The primary end point was angiographic restenosis at 6 months. Secondary end points included death, myocardial infarction, bypass surgery, and target vessel revascularization in hospital and at 6 months. There were no significant differences between groups in terms of major in-hospital complications. There was a trend toward fewer in-hospital events in the stent group (3% versus 7.1% in angioplasty group, P=0.076). Crossovers to stent occurred in 37 patients (20.3%). Repeat angiography at 6-month follow-up was performed in 85.3% of patients. Angiographic restenosis occurred in 28% of the stent group and 32.9% of the angioplasty group (P=0.36). Target vessel revascularization was required in 17.8% versus 20.3% of patients (P=0.54), respectively.

Conclusions—Stenting and standard coronary angioplasty are associated with equal restenosis rate in small coronary arteries. With a lower in-hospital complication rate, stenting may be a superior strategy in small vessels. (Circulation. 2001;104:2029-2033.)

Key Words: angioplasty ■ stents ■ restenosis

Restenosis remains a major limitation to the long-term success of coronary angioplasty. The efficacy of stenting in preventing restenosis has been proven by randomized studies in different situations. However, increased risk of subacute thrombosis and uncertainty about the long-term results of stenting in small arteries have made target vessel <3 mm an exclusion criteria in randomized trials. Therefore, little prospective information on stent placement in small coronary arteries is available. The recently published American College of Cardiology consensus on coronary artery stents does not recommend stent implantation in small vessels to improve long-term results.

Small coronary arteries represent a fair amount of the day-to-day angioplasty practice. Depending on definition, prevalence ranges from 35% to 67%. Lesions in vessels with small reference diameters represent a distinct group with respect to clinical and morphological characteristics. This translates into lower primary angioplasty success and more frequent major adverse cardiac events. An inverse relationship has been found between vessel size and angiographic restenosis rate. Stenting could therefore be of great help in patients with lesions in small vessels. Retrospective and observational studies have been reported on stenting and small coronary arteries. Very recently, 2 randomized studies comparing stent and angioplasty alone were published. Safety and early efficacy were documented, but because of conflicting results, the antirestenotic potential remains to be confirmed. We therefore designed a multicenter randomized trial to test the hypothesis that routine implantation of a stent, compared with balloon angioplasty alone, would be associated with reduced angiographic restenosis at 6 months in small coronary arteries.

*Additional investigators participating in the Stent In Small Arteries (SISA) trial are listed in the appendix.

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Methods

Patients

Patients had stable angina, stabilized unstable angina (Braunwald class IIb), or documented silent ischemia and required coronary angioplasty of one de novo lesion with reference-vessel diameter $\geq 2.3$ mm and $\leq 2.9$ mm by online quantitative coronary angiography (QCA). The lesion length had to be $\leq 12$ mm without thrombus. Patients with left ventricular ejection fraction $<40\%$ or unable to take aspirin or ticlopidine were excluded. After giving written informed consent, patients were randomly stratified according to center with a block design and assigned to either angioplasty or stent placement.

Protocols

Angioplasty was performed in the conventional manner by femoral or radial approach, according to standard techniques at each center. Aspirin was prescribed and 10 000 IU of heparin was given before the procedure. A goal of $<30\%$ residual stenosis was set. Crossover to stent implantation was allowed in case of abrupt or threatened closure, defined as a dissection type C to F (NHLBI classification), TIMI flow $<3$, or $\geq 50\%$ residual stenosis with myocardial ischemia. The stents were 15 mm premounted Stent Artist, available in 2.5-mm and 3-mm diameters (Medtronic Vascular). Unmounted stents 8 mm and 25 mm were also available for very short lesions or to cover a long dissection postangioplasty. Stent implantation was performed after angioplasty according to routine clinical practice. Stents were selected with online QCA to achieve a balloon-to-artery ratio of 1.1. Final inflation pressure recommendation was 12 to 14 atmospheres. All patients received aspirin, 100 to 325 mg daily, after the procedure and ticlopidine, 250 to 500 mg daily for 1 month, if a stent was implanted. Platelet glycoprotein IIb/IIIa receptor inhibitors were discouraged but allowed if necessary.

Quantitative Coronary Angiography

Angiography was performed preintervention, postintervention, and at 6-month follow-up or earlier if needed. QCA was performed at the QCA laboratory of the Montreal Heart Institute for Canadian centers and at Heart Core BV for other centers. The QCA-CMS system $^{20}$ (MEDIS medical imaging system) was used in both laboratories after standardizing and validating inter–core laboratory variability. $^{21}$ Offline QCA measurements were made in a single projection, showing the most severe stenosis. Whenever possible the same projection was used in all 3 angiograms. Each angiographic sequence was preceded by intracoronary injection of nitroglycerin. Minimal lumen diameter was measured, and percentage diameter-stenosis was calculated using the interpolated reference diameter approach. Standard morphological criteria were used to characterize the complexity of baseline lesions. $^{22}$

End Points

The primary end point was angiographic restenosis, defined as a percentage diameter stenosis of $\geq 50\%$ at follow-up. Secondary end points included the following: (1) angiographic success (reduction in stenosis to $<50\%$, QCA); (2) procedural success ($<50\%$ diameter stenosis with assigned devices alone or with adjunctive devices) by visual assessment; (3) clinical success (angiographic success without clinical events, including death, myocardial infarction, bypass surgery, and revascularization of the target vessel during the hospital stay); (4) target vessel revascularization at 6 months; (5) absolute minimal lumen diameter after procedure and at follow-up; and (6) Canadian Cardiovascular Society functional class, medication, and repeat revascularization at 1 year.

All deaths were considered cardiac unless an unequivocal noncardiac cause could be established. Myocardial infarction was defined by new Q-waves $>0.04$ second or elevation of the serum creatine kinase to greater than twice the upper limit of normal with an elevated MB fraction (measured at 12 and 24 hours). Target vessel revascularization required recurrent angina or signs of ischemia.

| TABLE 1. Baseline Clinical and Anatomic Characteristics of Patients |
|------------------------|------------------------|
| Variable               | Angioplasty (n=182)    |
|                        | Stent (n=169)          |
| Age, y                 | 59.9+10.5              |
|                       | 60.6+10.3              |
| Male sex, %            | 67.0                   |
|                       | 66.3                   |
| Diabetes, %            | 20.9                   |
|                       | 17.8                   |
| Hypertension, %        | 50.5                   |
|                       | 47.3                   |
| Hyperlipidemia, %      | 54.4                   |
|                       | 51.5                   |
| Present or past smoker, % | 57.7                     |
|                       | 63.9                   |
| Previous infarction, % | 35.1                   |
|                       | 31.9                   |
| Previous bypass surgery, % | 3.0                      |
|                       | 3.0                    |
| Previous angioplasty, % | 19.2                   |
|                       | 12.4                   |
| Unstable angina, %     | 29.1                   |
|                       | 34.3                   |
| Target vessel, %       |                        |
| Left anterior descending | 46.7                   |
|                       | 43.2                   |
| Left circumflex        | 33.5                   |
|                       | 33.7                   |
| Right coronary artery  | 19.8                   |
|                       | 23.1                   |
| Lesion type*           |                        |
| A                      | 17.5                   |
|                       | 13.3                   |
| B$_1$                  | 40.6                   |
|                       | 36.7                   |
| B$_2$                  | 41.3                   |
|                       | 47.6                   |
| C                      | 0.6                    |
|                       | 2.4                    |
| Ejection fraction, %   | 63.9+14.2              |
|                       | 63.3+12.7              |

*Type of lesion was determined according to the American Heart Association/American College of Cardiology classification, Core Laboratory Assessment.

Statistical Analysis

On the basis of the only preliminary data available on small arteries at the time of study design (published subsequently), $^{11}$ the sample size required to demonstrate a restenosis-rate reduction from 52% to 35% or from 43% to 25% by a 2-sided test with an $\alpha$ error of 0.05 and a power of 0.90 was 275 and 234 patients, respectively. This 35% reduction of restenosis rate by stenting was considered clinically significant. To compensate for unsuccessful interventions, crossovers, and losses to follow-up, the sample size was increased by 25% to 350 patients. The results are expressed as mean+SD. Comparisons between treatment groups were performed on an intent-to-treat basis. Unpaired Student’s $t$ tests were used to assess differences in continuous variables. Categorical data, presented as rates, were compared by $\chi^2$ test or Fisher’s exact test. Two-tailed probability values were calculated, with $P<0.05$ considered to indicate statistical significance.

Results

From October 1997 to July 1999, 351 patients were assigned to angioplasty (182 patients) or stenting (169 patients). There were no significant differences in baseline characteristics between groups (Table 1).

Procedural and Early Outcome

Of the 169 patients assigned to stenting, 166 (98.2%) had procedural success. In 2 patients, the wire failed to cross the lesion, and 1 patient required emergency coronary artery bypass surgery for type D dissection after predilatation. Of the 182 angioplasty-only patients, 179 (98.3%) had procedural success. In 1 patient, the wire failed to cross the lesion, 1 patient required emergency coronary artery bypass surgery,
and 1 patient had a total occlusion and was treated medically. In the stent group, 4 patients (2.4%) were switched to angioplasty because of inability to cross the lesion with the stent. In the angioplasty group, 37 patients (20.3%) crossed over to stent implantation as a bailout procedure.

Procedural and in-hospital outcomes are shown in Table 2. Angiographic success was achieved in 98.2% of stent patients and 93.9% of angioplasty-only patients (P=0.0065). Clinical success was also greater in the stent group than the angioplasty group (95.3% versus 87.9%, respectively, P=0.0066). There were no significant differences in major in-hospital cardiac complications. There was a trend toward fewer non-Q-wave myocardial infarctions in the stent group (1.8% versus 4.9% in the angioplasty group, P=0.142) and fewer repeated angioplasties (0.6% versus 2.7%, respectively, P=0.217). There was a trend toward fewer occurrences of in-hospital events in the stent group (3% versus 7.1% in the angioplasty group, P=0.076).

**Angiographic Results**

Coronary angiography was repeated 6±2 (mean±SD) months after initial procedure in 289 of 339 (85.3%) patients eligible for angiographic follow-up. Angiography was not repeated or angiograms could not be analyzed in 23 patients in the stent group because of refusal (20 patients), technical problems in quantification (2), or death (1). In the angioplasty group, 30 patients refused follow-up angiography, 2 were unsuitable for quantification, and 1 died. Quantitative angiographic results are shown in Table 3. Immediately after intervention, a larger gain in luminal diameter was achieved in stented patients (1.37±0.42 versus 0.91±0.46 mm, P<0.0001), whereas the late loss in luminal diameter was higher after stenting (0.49±0.52 versus 0.89±0.60 mm, P<0.0001). The net gain at follow-up was thus not significantly different (0.51±0.55 versus 0.54±0.54 mm for stent versus balloon, P=0.28). The minimal lumen diameter at 6 months was 1.44±0.53 in the stent group and 1.37±0.57 mm in the angioplasty group (P=0.34). Cumulative frequency distributions of minimal lumen diameter are shown in Figure.

When results were analyzed according to intention-to-treat principles, restenosis occurred in 28% of stented patients and in 32.9% of patients in the angioplasty-only group (P=0.36). To assess results on the basis of actual treatment received, patients who crossed over from angioplasty to stent were put together with patients randomized to stenting, creating a population of 170 patients treated with stent and 119 patients treated by angioplasty alone. The restenosis rates according to this analysis showed no statistically significant differences (28.8% stent group versus 32.8% angioplasty group, P=NS).

### Late Clinical Events

The cumulative major cardiac event rate at follow-up is shown in Table 4. The rate of occurrence of any events...
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events.9 Small vessels, particularly those with long or diffuse
revascularizations in small vessels show lower rates of
vasculopathy. 11 The mean reference diameter was 2.65 mm, and
3 mm identified post hoc by quantitative coronary angiog-
thesis from the STRESS trial comparing stand-alone balloon
angioplasty in small arteries, defined as a reference diameter
are more prone to restenosis than larger vessels after
lesions, are more prone to restenosis than larger vessels after
angiographic restenosis rates were 34% for stented lesions
and 32% having unstable angina and a lesion length of
arteries and better than the predicted range of 35% to 52%.
Even excluding crossovers to stent, the restenosis rate re-
mained 32.8%. One reason for the lack of significant differ-
ce could be the excellent performance of the balloon. The
availability of stents for bailout may presently permit a more
aggressive dilatation strategy and provides better results with
balloon angioplasty only. Another explanation may be differ-
ences in populations. Compared with all other studies in
which the efficacy of stenting to prevent restenosis had been
proven,2–7 our trial included a higher-risk population. With
46% of the population having complex lesions (type B2 or C)
and 32% having unstable angina and a lesion length of
10.4 mm, our population was clearly different from that of
previously published trials. Therefore, the efficacy of stenting
to prevent restenosis may have been negated by lesion
characteristics. Another potential explanation for lack of
significant differences is that the balloon-to-artery ratio and
final pressure were significantly different between the bal-
loon and the stent group. A higher balloon-to-artery ratio is
associated with greater wall damage and consequently more
potent intimal hyperplasia stimulus.24 The limitation of avail-
able space for the hyperplastic response in small vessels may
highlight the need for a more gentle approach in this
particular population.

There are some potential limitations to this trial. First,
small coronary arteries were defined as vessels 2.3 to 2.9 mm
according to online QCA interpolation of the proximal and
distal references. This is an arbitrary definition that can create
an inhomogeneous group in terms of restenosis risk, although
it is clearly distinct from the previously studied stent popu-
lation. A second limitation is related to the atherosclerosis
process, which may be diffuse in nature, so that the diseased
vessel may be reduced in caliber along its entire length,
giving the angiographic impression of a small vessel. Ultra-
sound may be the only way to obtain an accurate measure-
ment of the true normal reference.25 We then could have
included in our trial larger vessels with diffuse disease that

<table>
<thead>
<tr>
<th>Event</th>
<th>Angioplasty (n=182)</th>
<th>Stent (n=169)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>1 (0.5)</td>
<td>1 (0.6)</td>
<td>NS</td>
</tr>
<tr>
<td>Q-wave myocardial infarction</td>
<td>3 (1.6)</td>
<td>2 (1.2)</td>
<td>NS</td>
</tr>
<tr>
<td>Non-Q-wave myocardial infarction</td>
<td>12 (6.6)</td>
<td>5 (3.0)</td>
<td>0.11</td>
</tr>
<tr>
<td>Coronary bypass surgery</td>
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<td>0.49</td>
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<td>Repeated angioplasty</td>
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<td>25 (14.8)</td>
<td>0.33</td>
</tr>
<tr>
<td>Target vessel revascularization</td>
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<td>30 (17.8)</td>
<td>0.54</td>
</tr>
<tr>
<td>Any event</td>
<td>40 (22.0)</td>
<td>31 (18.3)</td>
<td>0.40</td>
</tr>
</tbody>
</table>

Values are No. of patients (%).

Discussion

In this study comparing stent to angioplasty alone in small
coronary arteries, despite a better initial gain in luminal
diameter, angiographic success and clinical success with
stent, restenosis, and revascularization rates were not signif-
ically different between the 2 treatment strategies.

Patients with lesions in vessels with small reference diam-
eters constitute a distinct population. Their clinical character-
istics are different, including more women, fewer white
patients, and more patients with diabetes mellitus, heart
failure, and peripheral vascular disease.9 Lesions in small
vessels tend to be more complex and more commonly
associated with multivessel disease.9 As a result, percutane-
ous revascularizations in small vessels show lower rates of
procedural success and higher rates of in-hospital major
events.8 Small vessels, particularly those with long or diffuse
lesions, are more prone to restenosis than larger vessels after
both standard angioplasty10 and stent placement.12 At least
part of this difference can be ascribed to the amount of late
loss that can be accommodated before clinically important
luminal narrowing recurs.13 There is a typical inverse rela-

Angioplasty (n=182) Stent (n=169) P

Death 1 (0.5) 1 (0.6) NS
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Target vessel revascularization 37 (20.3) 30 (17.8) 0.54
Any event 40 (22.0) 31 (18.3) 0.40

Angiographic

restenosis (≥50% diameter stenosis) was found in 35.7% of
stented patients and 37.4% of PTCA patients (P=0.74). The
protocol was very similar to this study with a 404-patient
population, an average vessel size of 2.4 mm, and a 16.5%
crossover rate to stent. All patients received adjunctive
therapy with abiciximab (none in our study), and the stent
was the MULTI-LINK stent. Also, in the ISAR-SMART
study, there was no difference in adverse events at 30 days. In
comparison, our trial showed better clinical success with stent
(95.3% versus 87.9%, P=0.0066) and a trend to fewer
complications (3% versus 7.1%, P=0.076). The second
randomized trial19 included only 120 patients, and the reste-
nosis rate was 30.9% in the angioplasty group and 35.7% in
the stent group (P=NS) using the 7-cell NIR 2.5-mm stent.

The findings of the present trial are in agreement with these
2 recently-published randomized trials in showing no differ-
ence in the primary end point of restenosis. Several explana-
ations could account for this lack of significant differences. In
the power calculation, the predicted restenosis rate for the
stent arm was set between 43% and 25%. The observed rate
was 28%. On the other hand, for the balloon group, the actual
restenosis rate was 32.9%, which is relatively low for small
arteries and better than the predicted range of 35% to 52%.
Even excluding crossovers to stent, the restenosis rate re-
mained 32.8%. One reason for the lack of significant differ-
ce could be the excellent performance of the balloon. The
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ment of the true normal reference.25 We then could have
included in our trial larger vessels with diffuse disease that
represent a completely different population compared with small vessels with discrete lesions. A third limitation is related to stent design and strut thickness, which can influence outcomes in small vessels. Our findings may not be generalizable to all types of stents. Finally, technical variables, especially regarding stent deployment and maximum atmosphere, were left to operator’s choice and may have introduced bias in this large multicenter trial.

In conclusion, stenting and PTCA are associated with equal restenosis rate in small coronary arteries. With lower inhospital complication rates, stenting may be a superior strategy in this population.

Appendix

The following institutions and investigators, in addition to the authors, participated in the Stent In Small Arteries (SISA) trial: R. Bonan, G. Côté, J. Crépeau, P. de Guise, R. Gallo, G. Gosselin, J. Grégoire, M. Joyal, J.-F. Tanguay, Montreal Heart Institute, Montreal, Canada; J. Van Lierde, Ziekenhuis Oost Limburg, Genk, Belgium; W.P. Klinke, R.D. Kinloch, M.B. Williams, Royal Jubilee Hospital, Victoria, Canada; O. Gorne, E. Schroeder, Catholic University of Louvain-UCL-Mont-Godinne Hospital, Mont-Godinne, Belgium; W. Wijns, G.R. Heyndrickx, Cardiovascular Center, Aalst, Belgium; P. Chandra, A. Mathur, Escorts Heart Institute and Research Center, New Delhi, India; P. Gurnagulski, Chulalongkorn University Hospital, Bangkok, Thailand; H.D. Glogur, P. Yang, M. Pavone-Gyöngyösi, University of Vienna Medical Center, Vienna, Austria; F. Reeves, G. Leclerc, R.M. Gagnon, A. Rivard, CHUM, Notre-Dame Pavillon, Montreal, Canada; L. Schwartz, Toronto General Hospital, Toronto, Canada; M.J. Peper, Herz-Zentrum Bodensee, Kreuzlingen, Switzerland; M. Curtis, M. Knudston, M. Traboulsi, T. Anderson, F. Spence, J. Hansen, Foothills Hospital, Calgary, Canada; D. Tresukosol, S. Chaithiraphan, Siriraj Hospital, Bangkok, Thailand; H. Mudra, A. König, Medezinische Klinik, Klinikum Innenstad, University of Munich, Munich, Germany; L. Missault, L. Muyldermans, St-Jan Hospital, Brugge, Belgium; E. Von Hodenberg, U. Berninger, Heart Center, Laher/Baden, Germany; H. Mudra, A. König, Medezinische Klinik, Klinikum Innenstad, University of Munich, Munich, Germany; L. Giommi, E. Franceschini, G. Ristic, Ospedale Regionale Dell’Azienda, Treviso, Italy; A. Chauhan, Blackpool Victoria Hospital, Blackpool, United Kingdom; C. Costantini, Clinica Cardiologica C. Constantini, Curitiba, Brazil. Steering Committee: S. Doucet (Chairperson) and L. Bilodeau, Montreal, Canada; M. Schalji, Leiden, The Netherlands; F. van Leeuwen, Maastricht, The Netherlands. Adverse Event Adjudication Committee: V.L. Legrand, Liège, Belgium; P. van den Heuvel, Middelheim, Belgium. Data and Safety Monitoring Board: M.G. Bourassa, Montreal, Canada; M. Bertrand, Lille, France; A.V.G. Bruschke, Leiden, The Netherlands. Quantitative Angiographic Core Laboratories: A.W.M. van Weert, J.H.C. Reiber, E. Hekking, J. ter Horst and Y. Ishii, Leiden, The Netherlands; J. Lesperance, L. Bilodeau, F. Belanger, C. Desjardins and M.J. Dussault, Montreal, Canada. Data Coordinating Management: S. Jacobs, Maastricht, The Netherlands. Sponsor: Medtronic Vascular, Maastricht, The Netherlands.

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References

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