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.1 The efficacy of stenting in preventing restenosis has been proven by randomized studies in different situations.2–7 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 stents8 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%9 to 67%.10 Lesions in vessels with small reference diameters represent a distinct group with respect to clinical and morphological characteristics.9 This translates into lower primary angioplasty success and more frequent major adverse cardiac events.9 An inverse relationship has been found between vessel size and angiographic restenosis rate.10 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.11–17 Very recently, 2 randomized studies comparing stent and angioplasty alone were published.18,19 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.

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From the Department of Medicine, Montreal Heart Institute (SD, L.B., J.L., F.H.), Montreal, Quebec, Canada; Department of Cardiology, Leiden University Medical Centre (M.J.S.), Leiden, The Netherlands; Ziekenhuis Oost Limburg (M.V.), Genk, Belgium; Royal Jubilee Hospital (D.H.), Victoria, Canada; Catholic University of Louvain-UCL-Mont-Godinne Hospital (P.C.), Mont-Godinne, Belgium; Cardiovascular Centre (B.d.B.), Aalst, Belgium; Chulalongkorn University (W.U.), Bangkok, Thailand; Escorts Heart Institute and Research Centre (A.S.), New Delhi, India; and Heart Core BV (J.H.C.R.), Leiden, The Netherlands.

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

Correspondence to Serge Doucet, 5000 Belanger St East, Montreal, Quebec, H1T 1C8, Canada. E-mail serge.doucet@sympatico.ca

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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 randomized 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 beStent Artist, available in 2.5-mm and 3-mm diameters (Medtronic Vascular). Unmounted beStents 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 $\text{atm}$. Protocols

Quantitative Coronary Angiography
Angiography was performed before intervention, 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 (MEDIS medical imaging system) was used in both laboratories after standardizing and validating inter–core laboratory variability. 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.

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.

Statistical Analysis
On the basis of the only preliminary data available on small arteries at the time of study design (published subsequently), 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$\pm SD$. 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,
TABLE 2. Procedural Outcomes and In-Hospital Clinical Events

<table>
<thead>
<tr>
<th>Variable</th>
<th>Angioplasty (n=182)</th>
<th>Stent (n=169)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procedural success</td>
<td>179 (98.3)</td>
<td>166 (98.2)</td>
<td>NS</td>
</tr>
<tr>
<td>Angiographic success</td>
<td>171 (93.9)</td>
<td>168 (96.2)</td>
<td>0.0065</td>
</tr>
<tr>
<td>Clinical success</td>
<td>160 (87.9)</td>
<td>161 (95.3)</td>
<td>0.0066</td>
</tr>
<tr>
<td>Crossover</td>
<td>37 (20.3)</td>
<td>4 (2.4)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>In-hospital events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>0</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Q-wave myocardial infarction</td>
<td>0</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Non-Q-wave myocardial infarction</td>
<td>9 (4.9)</td>
<td>3 (1.8)</td>
<td>0.142</td>
</tr>
<tr>
<td>Coronary bypass surgery</td>
<td>1 (0.5)</td>
<td>1 (0.6)</td>
<td>NS</td>
</tr>
<tr>
<td>Repeated angioplasty</td>
<td>5 (2.7)</td>
<td>1 (0.6)</td>
<td>0.217</td>
</tr>
<tr>
<td>Any event</td>
<td>13 (7.1)</td>
<td>5 (3.0)</td>
<td>0.076</td>
</tr>
</tbody>
</table>

Values are expressed as No. of patients (%).

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 (0.89±0.52 versus 0.48±0.60 mm, P<0.0001). The net gain at follow-up was thus not significantly different (0.51±0.55 versus 0.43±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

<table>
<thead>
<tr>
<th>Variable</th>
<th>Angioplasty</th>
<th>Stent</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>At baseline</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diameter of reference vessel, mm</td>
<td>2.45±0.34</td>
<td>2.50±0.37</td>
<td>0.22</td>
</tr>
<tr>
<td>Minimal lumen diameter, mm</td>
<td>0.93±0.29</td>
<td>0.93±0.30</td>
<td>0.90</td>
</tr>
<tr>
<td>Degree of stenosis, % of diameter</td>
<td>61.9±11.1</td>
<td>62.8±10.9</td>
<td>0.46</td>
</tr>
<tr>
<td>Lesion length, mm</td>
<td>10.2±4.1</td>
<td>10.8±4.2</td>
<td>0.14</td>
</tr>
<tr>
<td>Balloon-to-artery ratio, atm</td>
<td>1.03±0.14</td>
<td>1.08±0.12</td>
<td>0.01</td>
</tr>
<tr>
<td>Maximal balloon pressure, atm</td>
<td>10.5±3.1</td>
<td>11.7±2.4</td>
<td>0.0001</td>
</tr>
<tr>
<td>At 6 months</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diameter of reference vessel, mm</td>
<td>2.48±0.32</td>
<td>2.63±0.37</td>
<td>0.001</td>
</tr>
<tr>
<td>Minimal lumen diameter, mm</td>
<td>1.84±0.41</td>
<td>2.30±0.35</td>
<td>0.001</td>
</tr>
<tr>
<td>Degree of stenosis, % of diameter</td>
<td>25.6±15.1</td>
<td>12.4±9.0</td>
<td>0.001</td>
</tr>
<tr>
<td>Acute gain, mm</td>
<td>0.91±0.46</td>
<td>1.37±0.42</td>
<td>0.0001</td>
</tr>
<tr>
<td>Net gain, mm</td>
<td>0.43±0.54</td>
<td>0.51±0.55</td>
<td>0.28</td>
</tr>
<tr>
<td>Loss index, mm</td>
<td>0.46±0.94</td>
<td>0.67±0.40</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Values are mean±SD.
TABLE 4. Cumulative Major Cardiac Events at 6 Months After Procedure

<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>
<td>3 (1.6)</td>
<td>5 (3.0)</td>
<td>0.49</td>
</tr>
<tr>
<td>Repeated angioplasty</td>
<td>34 (18.7)</td>
<td>25 (14.8)</td>
<td>0.33</td>
</tr>
<tr>
<td>Target vessel revascularization</td>
<td>37 (20.3)</td>
<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 (%).

Table 4 shows the cumulative major cardiac events at 6 months after procedure. The incidence of death, myocardial infarction, or target-vessel revascularization at 6 months was not significantly different (22% angioplasty versus 18.3% stent, P = 0.40). The target-vessel revascularization rate was 20.3% in the angioplasty group and 17.8% in the stent group (P = 0.54). The angina-free rate at 6 months was 72.2% in the angioplasty group and 76.2% in the stent group (P = NS).

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 significantly different between the 2 treatment strategies.

Patients with lesions in vessels with small reference diameters constitute a distinct population. Their clinical characteristics are different, including more women, fewer white patients, and more patients with diabetes mellitus, heart failure, and peripheral vascular disease. Lesions in small vessels tend to be more complex and more commonly associated with multivessel disease. As a result, percutaneous revascularizations in small vessels show lower rates of procedural success and higher rates of in-hospital major events. Small vessels, particularly those with long or diffuse lesions, are more prone to restenosis than larger vessels after both standard angioplasty and stent placement. 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. There is a typical inverse relationship between arterial diameter and lesion length, which results in a higher plaque burden in small-vessel lesions.

Retrospective and observational studies have been reported on stenting in small coronary arteries. A subgroup analysis from the STRESS trial comparing stand-alone balloon angioplasty to stent implantation examined the benefit of stenting in small arteries, defined as a reference diameter <3 mm identified post hoc by quantitative coronary angiography. The mean reference diameter was 2.65 mm, and angiographic restenosis rates were 34% for stented lesions and 55% in the angioplasty group. Smaller trials in <3 mm or <2.8 mm arteries showed restenosis rates between 32% and 39% after stenting. Recently, 2 randomized trials comparing stent with balloon angioplasty in small vessels were published. In the ISAR-SMART study, 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 abciximab (none in our study), and the stent used 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 trial included only 120 patients, and the restenosis 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 difference in the primary end point of restenosis. Several explanations 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 remained 32.8%. One reason for the lack of significant difference 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 differences in populations. Compared with all other studies in which the efficacy of stenting to prevent restenosis had been proven, 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 balloon and the stent group. A higher balloon-to-artery ratio is associated with greater wall damage and consequently more potent intimal hyperplasia stimulus. The limitation of available 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 population. 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. Ultrasound may be the only way to obtain an accurate measurement of the true normal reference. 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. Ngarmukos, Chulalongkorn University Hospital, Bangkok, Thailand; H. D. Grafog, P. Yang, M. Paveone-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. Pieper, Herz-Zentrum Badense, Kreuzlingen, Switzerland; M. Curtis, M. Knudston, M. Traboulsi, T. Anderson, F. Spence, J. Hansen, Foothills Hospital, Calgary, Canada; D. Tresukosol, S. Chaithiraphan, Siriraj Hospital, Mahidol University, Bangkok, Thailand; B. J. O’Neill, L. M. Title, R. J. Teskey, C. J. Foster, C. M. Kells, Queen Elizabeth II Health Sciences Center, Halifax, Canada; G. Barbeau, L. Roy, O. Gleeton, S. Plante, G. Proulx, C. Juneau, Laval Hospital, Quebec, Canada; E. Schampaert, D. Palaisaîtis, P. Tessier, P. Terriault, D. Hamel, Hôpital du Sacré-Cœur, Montreal, Canada; G. Heyer, Invasive Kardiologie, Salzburg, Austria; L. Missault, L. Muylermans, St-Hôpital Brugge, Belgium; E. Von Hodenberg, U. Berninger, Heart Center, Lahr/Baden, Germany; H. Mudra, A. König, Medizinische Klinik, Klinikum Innenstadt, University of Munich, Munich, Germany; L. Giommi, E. Franceschini, G. Ristica, Ospedale Regionale Dell’Azienda, Trevisio, 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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