Impact of final coronary flow velocity reserve on late outcome following stent implantation

T. Nishida1, C. Di Mario1, M. J. Kern2, T. J. Anderson3, I. Moussa4, R. Bonan5, T. Muramatsu6, A. C. Jain7, J. Suarez de Lezo8, S. Y. Cho9, I. T. Meredith10, J. W. Moses4 and A. Colombo1

1 Centro Cuore Columbus, Milan, Italy; 2 Saint Louis University Health Sciences Center, St. Louis, MO, U.S.A.; 3 Foothills Hospital, University of Calgary, Calgary, Alberta, Canada; 4 Lenox Hill Hospital, New York, NY, U.S.A.; 5 Institut de Cardiologie de Montreal, Montreal, Canada; 6 Social Insurance Kawasaki Hospital, Kawasaki, Kanagawa, Japan; 7 West Virginia University School of Medicine, Robert C. Byrd Health Sciences Center, Morgantown, WV, U.S.A.; 8 Hospital Reina Sofia, University of Cordoba, Spain; 9 Yonsei University College of Medicine, Seoul, South Korea; 10 Monash Medical Centre and Monash University, Department of Medicine, Clayton, Vic., Australia

Aims To assess whether coronary flow velocity reserve following stent implantation is predictive of the subsequent need of target lesion revascularization.

Methods and Results The outcome was examined of 417 patients enrolled in a multicentre prospective randomized study (DESTINI), who received a successful single vessel stent implantation in native coronary arteries and in whom coronary flow velocity reserve was measured. Logistic regression analysis and the receiver operator characteristic curve were used. When compared with 358 patients not requiring target lesion revascularization, 59 patients (14%) who underwent target lesion revascularization had a lower final coronary flow velocity reserve (2.33 ± 0.87 vs 2.48 ± 0.80, P = 0.20) and smaller final minimal lumen diameter (2.62 ± 0.66 mm vs 2.73 ± 0.60, P = 0.19); however, those differences were not statistically significant. Patients with a coronary flow velocity reserve of < 2.0 (n = 109, 26%) exhibited a significantly higher target lesion revascularization rate than patients with a coronary flow velocity reserve of ≥ 2.0 (22% vs 11%, P = 0.010). This difference remained significant (odds ratio = 2.01, 95% CI = 1.11 to 3.66) after adjustment for other variables that were also correlated with the incidence of target lesion revascularization.

Conclusion The presence of a final coronary flow velocity reserve of < 2.0 is an independent predictor of the need for target lesion revascularization after stent implantation in native coronary artery lesions.

Key Words: Intracoronary Doppler, stent, coronary flow velocity reserve.

See page 274, doi: 10.1053/euhj.2001.2829 for the Editorial comment on this article

Introduction

The coronary flow velocity reserve, distal to a coronary lesion measured with a Doppler flow wire, is a significant predictor of late outcome after balloon angioplasty[1]. Accordingly, coronary flow velocity reserve has been proposed as a physiological end-point following angioplasty in order to determine whether stent implantation is necessary[2]. The clinical usefulness and practical applicability of this strategy has been evaluated in the multicentre prospective randomized study DESTINI[3] (Doppler Endpoint STenting INTernational Investigation). The DESTINI study showed that achievement of a coronary flow velocity reserve of > 2.0 and a residual diameter stenosis of < 35% following balloon angioplasty alone was succeeded by a reduced need of target lesion revascularization and a low incidence of major adverse cardiac events. These results were similar to those obtained in the groups treated with stent implantation.

In the DESTINI study, an impaired final coronary flow velocity reserve (< 2.0) was observed in 26% of
lesions. This confirmed the results from preliminary reports showing that an impaired final coronary flow velocity reserve was common despite optimal angiographic results and stent expansion assessed by intravascular ultrasound\(^4\,^5\). However, these initial studies were small and did not allow the influence of the final coronary flow velocity reserve to be tested on the late outcome of coronary stenting.

Therefore, we studied 448 patients who were prospectively enrolled in the DESTINI study and had received stent implantation in order to assess the impact of the final coronary flow velocity reserve following stenting on the incidence of target lesion revascularization. The aim of this study was to test the hypothesis that an impaired final coronary flow velocity reserve after stent implantation is a determinant of the need for target lesion revascularization during follow-up.

**Methods**

**Study population**

The DESTINI study was a multicentre randomized trial which compared the long-term outcome of 370 patients treated with elective stenting and 365 patients treated with balloon angioplasty alone. In this latter group, stenting was only allowed on a provisional basis when optimal morphological results (residual diameter stenosis \(<35\%\) measured with on-line quantitative coronary angiography, absence of threatening dissections) and optimal functional results (coronary flow velocity reserve \(>2\cdot0\) measured with a Doppler guidewire) could not be achieved with balloon angioplasty. Lesions which exhibited the following characteristics were excluded from the study: chronic total occlusions, graft and ostial stenoses, second restenosis after PTCA, in-stent restenosis, elective planned rotational or directional atherectomy, recent \((<24\ h)\) myocardial infarction or previous Q-wave myocardial infarction in the territory of the target vessel.

Among the 735 patients enrolled in the DESTINI study between August 1996 and April 1998, we selected 448 patients who received elective or provisional stenting of a single vessel lesion and achieved a final residual diameter stenosis of \(<35\%\) without suffering from death or target lesion revascularization (emergency coronary artery bypass surgery or repeat percutaneous intervention) during their hospital stay.

**Interventional procedures**

All patients were pre-treated with aspirin (100–325 mg.day\(^{-1}\)) and ticlopidine (250 mg twice daily); aspirin treatment was maintained indefinitely and ticlopidine for 1 month. The protocol recommended the use of tubular stents aiming for a final residual diameter stenosis of \(<30\%\). Intravascular ultrasound was used at the discretion of investigators in order to evaluate stent expansion and coronary dissection.

**Coronary flow velocity reserve and quantitative coronary angiography**

A 0·014-inch Doppler-tipped guidewire (FloWire, Endosonics, Rancho Cordova, CA, U.S.A.) was used to record the average peak velocity at baseline and maximal hyperaemia after a bolus injection of 12 or 24 \(\mu\)g of adenosine via the guiding catheter into the right or left coronary artery, respectively. The coronary flow velocity reserve was calculated as the ratio of the maximal hyperaemic to basal average peak velocity. The relative coronary flow velocity reserve was defined as the ratio of coronary flow velocity reserve in the target vessel to the coronary flow velocity reserve in an angiographically normal reference vessel (diameter stenosis \(<30\%\)). All measurements were made in duplicate and the average of the two measurements was reported.

All procedural angiograms were independently analysed in a core laboratory using a semi-automatic edge detection system (CMS MEDIS version 3-0).

**Major adverse cardiac events and follow-up**

Death was considered cardiac and related to the treated lesions unless an alternative cause could be demonstrated. Myocardial infarction was defined (1) as the development of new pathological Q waves in the distribution area of the treated artery or (2) an increase in the total creatine phosphokinase levels to twice the normal limit for each participating hospital, accompanied by a concomitant increase in the creatine phosphokinase myocardial band component. Target lesion revascularization was defined as either repeat percutaneous treatment or a coronary artery bypass graft operation caused by ischaemia due to occlusion or restenosis in the initially treated lesions.

Patients were contacted at 1, 6 and 12 months after treatment in order to document the occurrence of major adverse cardiac events (death, myocardial infarction and target lesion revascularization). All events were reviewed by an independent Critical Events Committee and the final event adjudication was made based on specific queries to the investigators and the review of source documents.

**Statistical analysis**

Continuous variables were expressed as means ± SD and were compared by the unpaired Student’s t-test. Discrete variables were expressed as counts and percentages and the Fisher exact test was used to compare proportions. A measure of the linear association between two variables was evaluated using the Pearson correlation coefficient and all statistical tests were two-tailed.

In order to determine independent predictors of the incidence of target lesion revascularization, all variables
that were accompanied by a \( P \) value of <0·10 in these basic univariate analyses were reevaluated using univariate and multivariate logistic regression analyses. When a statistically significant correlation was observed between the variables, i.e. between the number of deployed stents and the total length of the stented segment or the final basal average peak velocity and final coronary flow velocity reserve <2·0, only the variable with the higher predictive value, i.e. the total length of stented segment or a final coronary flow velocity reserve of <2·0, was used for logistic regression analysis [6].

The logistic regression analyses were based on the maximum likelihood method. The forward stepwise method was used for multivariate analyses and the \( P \) values for inclusion and elimination were \( P=0·05 \) and \( P=0·10 \), respectively. The regression coefficient and standard error were used in the calculation of the odds ratio.

Receiver operator characteristic curves were used to assess the usefulness of an achieved logistic regression model for the prediction of the occurrence of target lesion revascularization, and the area under the curve, which indicates the predictive accuracy ranging from 50% to 100% (the higher is the better), was reported. All of the statistical analyses were performed using SPSS 10·0 (SPSS Inc.).

### Results

#### Baseline and procedural characteristics

The baseline characteristics of the patients in this study are reported in Table 1. Hypertension was observed in 45% of patients and diabetes mellitus in 19%. The reference vessel diameter was 3·09 \( \pm \) 0·43 mm and the lesion length 12·6 \( \pm \) 5·17 mm. A majority of lesions (57%) were angiographically complex lesions (type B2 or C according to the modified American College of Cardiology/American Heart Association angiographic classification[7]).

The procedural characteristics are listed in Table 2. Provisional stenting accounted for 29% of all lesions. Stents were expanded with an inflation pressure of 15 \( \pm \) 3·3 atm and 86% of lesions received a slotted tube stent. The total length of the stented segment was 18·6 \( \pm \) 9·1 mm and the minimal lumen diameter increased from 0·96 \( \pm \) 0·33 mm to 2·69 \( \pm \) 0·61 mm.
following the procedure. Platelet IIb/IIIa inhibitor was found to have been used in one patient who suffered target lesion revascularization (2%) and in 17 patients who did not suffer target lesion revascularization (5%) (P=0·49).

**Procedural outcome**

Of the 417 patients (93%) who completed the 12 month clinical follow-up period, 69 (17%) suffered at least one major adverse cardiac event. Death was observed in five patients (1%), four of which were considered cardiac and one due to intracranial haemorrhage. Myocardial infarction occurred in seven patients (2%). Coronary artery bypass surgery was performed in 12 patients (3%), all involving the target vessel, and repeat percutaneous intervention of the target lesion was performed in 47 patients (11%), yielding a cumulative target lesion revascularization occurrence of 14% (59 patients).

**Post-procedural coronary flow velocity reserve**

For the entire group, the final coronary flow velocity reserve was found to be 2·42 ± 0·80 and no significant correlation was found between the final coronary flow velocity reserve and the residual diameter stenosis (r = −0·078, P=0·13). There were no significant statistical differences in the mean values of the final coronary flow velocity reserve between patients who required target lesion revascularization and those who did not undergo target lesion revascularization (2·33 ± 0·87 vs 2·48 ± 0·80, respectively, P=0·20). However, patients who had a final coronary flow velocity reserve <2·0 had a significantly higher incidence of target lesion revascularization than the remaining patients (Fig. 1).

When the end-point is defined as death, myocardial infarction or target lesion revascularization, the final coronary flow velocity reserve of <2·0 still has a statistically significant correlation with the incidence of the end-points (P=0·010, sensitivity=0·41, specificity=0·76).

In all the patients, the 126 who were found to have a final coronary flow velocity reserve of <2·0 had a significantly higher final basal average peak velocity (28 ± 13 cm.s⁻¹ vs 19 ± 8·7 cm.s⁻¹, P<0·001) and a significantly lower hyperaemic average peak velocity (42 ± 18 cm.s⁻¹ vs 47 ± 19 cm.s⁻¹, P=0·016) than the remaining 322 patients.

**Post-procedural relative coronary flow velocity reserve**

The final relative coronary flow velocity reserve was measured in 168 patients (38%) and was found to be 0·93 ± 0·27. Among these patients, target lesion revascularization was needed in 25 of the 153 patients who required target lesion revascularization (16%) and in 17 patients who did not undergo target lesion revascularization (11%) (P=0·091).
who completed the 12-month follow-up period. The correlation between the final coronary flow velocity reserve and the relative coronary flow velocity reserve in these 153 patients is presented in Fig. 2, for which the previously reported cut-off point of normal relative coronary flow velocity reserve [4] is applied. A final relative coronary flow velocity reserve of \(<0.8\) was found in 48 patients (31\%) and a subset of 25 of these 48 patients had a final coronary flow velocity reserve \(<2.0\) (Fig. 2, subgroup II).

No difference was observed in the target lesion revascularization rate between the 48 patients who had a final relative coronary flow velocity reserve of \(<0.8\) and the remaining 105 patients who had a relative coronary flow velocity reserve of \(\geq 0.8\) (13\% vs 18\%, respectively, \(P=0.48\)). The final relative coronary flow velocity reserve was similar for the 25 patients who underwent target lesion revascularization and the 128 patients who did not (1.0 \(\pm 0.3\) vs 0.9 \(\pm 0.3\), respectively, \(P=0.61\)).

**Determinants of target lesion revascularization**

In order to establish the determinants for the incidence of target lesion revascularization, all the variables presented in Tables 1 and 2 were compared between patients who underwent target lesion revascularization and patients not requiring target lesion revascularization. Intravascular ultrasound was more frequently used in lesions with a final coronary flow velocity reserve of \(<2.0\) compared with the remaining lesions (28\% vs 12\%, \(P=0.001\)). For this reason, the use of intravascular ultrasound was not entered into the multivariate analysis. Table 3 reports the result of the logistic regression analysis and indicates that four independent predictors of target lesion revascularization (hypertension, reference vessel diameter, total length of stented segment and presence of a final coronary flow velocity reserve \(<2.0\)) were found.

A final coronary flow velocity reserve \(<2.0\) alone was accompanied by an area under the receiver operator characteristic curve of 59\% (95\% confidence interval=50\% to 67\%, \(P=0.037\)) for the prediction of the occurrence of target lesion revascularization (sensitivity=0.41, specificity=0.76, positive predictive value=0.22, negative predictive value=0.89). When the predictive power of all four obtained predictors was adjusted in the multivariate analysis, the area under the curve for this logistic regression model increased to 69\% (95\% confidence interval=62\% to 77\%, \(P<0.001\)).

**Discussion**

**Effects of percutaneous coronary interventions on coronary flow velocity reserve**

A decreased maximum blood flow and impaired coronary flow velocity reserve correlates with the angiographic severity of coronary stenoses [8,9] and more closely with the presence of abnormal myocardial perfusion assessed by scintigraphy [10,11], even in stenoses of intermediate angiographic severity. Despite a successful lumen enlargement of lesions with stents, the presence of an impaired final coronary flow velocity reserve is commonly observed for 21\% to 31\% of lesions [4,12]. It has been reported that by using intracoronary
An impaired coronary flow velocity reserve immediately after optimal stent implantation has been reported to be caused by an elevated basal average peak velocity rather than an attenuated augmentation of the average peak velocity during hyperaemia. A significant elevation of the basal average peak velocity after stenting was also confirmed in the present study. A high basal velocity after angioplasty is considered to be a confounding factor leading to a falsely low coronary flow velocity reserve. In reality, an increase in the basal velocity after treatment may be due to widespread microembolization, a phenomenon more likely to be induced by long lesions or large plaque burdens, which are considered to be risk factors of restenosis. This may explain why the basal velocity was found to have a statistically significant, but weak, correlation with the occurrence of target lesion revascularization in the present study. Unlike other reports, in the current study the hyperaemic average peak velocity was found to be significantly lower in patients with a final coronary flow velocity reserve of <2·0 compared with the remaining patients, suggesting a true reduction of maximal flow in this group. However, the coronary flow velocity reserve was similar in patients who underwent target lesion revascularization and in those who did not. The large individual variability of the hyperaemic average peak velocity in the current study population and the role of high basal velocity may explain this phenomenon.

A number of mechanisms of impairment of the coronary flow velocity reserve after stenting have been reported to include the pre-existent impairment of the vasodilatory response in microcirculation, the residual
Table 3  Determinants for the incidence of target lesion revascularization

<table>
<thead>
<tr>
<th></th>
<th>Univariate analysis</th>
<th></th>
<th>Multivariate analysis</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>P</td>
<td>B</td>
<td>OR</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.76</td>
<td>0.008</td>
<td>0.65</td>
<td>1.92</td>
</tr>
<tr>
<td>Reference diameter, mm</td>
<td>-0.93</td>
<td>0.010</td>
<td>-0.96</td>
<td>0.38</td>
</tr>
<tr>
<td>Coil stent*</td>
<td>0.92</td>
<td>0.029</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Total length of stented segment, mm</td>
<td>0.04</td>
<td>&lt;0.001</td>
<td>0.04</td>
<td>1.04</td>
</tr>
<tr>
<td>Final coronary flow velocity reserve &lt;2.0</td>
<td>0.79</td>
<td>0.007</td>
<td>0.70</td>
<td>2.01</td>
</tr>
</tbody>
</table>

B=coefficient; OR=odds ratio; CI=confidence interval.
*Use of coil stent was not selected as a determinant in multivariate analysis.

Conduit obstruction and acute attenuation of the microcirculatory vasomotor responses by distal embolization and neurohumoral stimuli[4]. In order to elucidate and distinguish these mechanisms, measurement of relative coronary flow velocity reserve[4,5], an evaluation of the residual minimal lumen cross-sectional area with intravascular ultrasound[16–18] and an assessment of the fractional flow reserve with pressure guidewires in the stented vessel[19,20] are useful.

In particular, the presence of a final coronary flow velocity reserve <2.0 and relative coronary flow velocity reserve <0.8, which accounted for nearly half of the lesions with a final coronary flow velocity reserve of <2.0 in the present study, suggests an unappreciated residual conduit obstruction (Fig. 2, subgroup II). If this mechanism can be excluded using intravascular ultrasound or by the measurement of the fractional flow reserve, an alternative explanation is a transient or persistent impairment of the vasodilatory response that is limited to the distribution area of the target vessel, possibly caused by the microembolization or previous myocardial infarction of the target vessel area. However, a final coronary flow velocity reserve <2.0 and a normal (≥0.8) relative coronary flow velocity reserve suggests diffuse impairment of the vasodilatory response of the coronary microcirculation either chronically or acutely (Fig. 2, subgroup I).

As the coronary flow velocity reserve is influenced by the basal average peak velocity, a method of normalization of the coronary flow velocity reserve for the basal average peak velocity has recently been proposed[21]. This concept seems to be useful for discriminating between conditions directly affecting the vasodilator reserve, such as unappreciated residual conduit obstruction and conditions primarily affecting the basal average peak velocity.

**Prognostic implication of impaired final coronary flow velocity reserve**

In the presence of a coronary flow velocity reserve ≥2.0, percutaneous intervention can safely be deferred in patients having coronary artery stenoses of intermediate angiographic severity[22]. A final coronary flow velocity reserve of >2.0 has been reported to help define lesions in which stenting is not necessary[3]. In the absence of any physiological guidance, balloon angioplasty with provisional stenting has been associated with a worsened short- and long-term outcome compared to that for routine stent implantation[23], suggesting that the measurement of a post-procedural coronary flow velocity reserve adds valuable information to angiography.

The receiver operator characteristic curve revealed that a final coronary flow velocity reserve <2.0 in the current analysis exhibited an area under the curve similar to that of the final coronary flow velocity reserve in the DEBATE study, which was 58% (95% confidence interval =50% to 66%) for the prediction of target lesion revascularization at 6 months. In the present study, measurement of final coronary flow velocity reserve after stent implantation had a clinically relevant predictive value for the occurrence of target lesion revascularization at 12 months and thus prompts a strict follow-up.

It has been reported in animals that in the common carotid artery after balloon injury, a low blood flow and decreased shear stress facilitates the migration of smooth muscle cells and the subsequent development of intimal thickening[24]. In the present analysis, patients with a final coronary flow velocity reserve <2.0 had significantly lower hyperaemic average peak velocity compared with the remaining patients. Nevertheless, the mechanism linking a low coronary flow velocity reserve and neointimal proliferation after stenting is questionable and has yet to be established.

The relative coronary flow velocity reserve has been proposed as an alternative to overcome the limitations of the absolute coronary flow velocity reserve. In the studies supporting the usefulness of relative coronary flow velocity reserve, however, a highly selected patient population was examined and conditions that induce non-uniform microvasculatory impairment (i.e. myocardial infarction) were often excluded[4]. A high correlation between the absolute coronary flow velocity reserve and relative coronary flow velocity reserve after balloon angioplasty or stent implantation (r=0.93) has been previously reported by van Liebergen et al[5].
present study confirms the statistically significant correlation between absolute coronary flow velocity reserve and relative coronary flow velocity reserve but the correlation was relatively weak \( (r=0.52, P<0.001) \). Compared with those in van Liebergen’s report, the patients in the present study exhibit higher percentages of diabetes mellitus (19% vs 2%) and hypertension (45% vs 33%), conditions known to reduce absolute coronary flow velocity reserve\[^{25,26}\]. When the absolute coronary flow velocity reserve is abnormal (<2.0), patients having those conditions may have a normal relative coronary flow velocity reserve. Although patients undergoing a previous Q-wave myocardial infarction and akinaesia in the area of the target vessel were excluded from the present study, patients with a previous history of non-Q-wave myocardial infarction were included (38%). The low coronary flow velocity reserve in the infarct areas may have affected the weaker correlation between absolute coronary flow velocity reserve and relative coronary flow velocity reserve. Accordingly, the inclusion criteria for the study population, which reflect common patient groups in daily practice, may have led to the finding that the relative coronary flow velocity reserve did not have a significant impact on the incidence of target lesion revascularization in the present study.

### Study limitations

Several limitations of this study should be recognized. Although the data acquisition was performed prospectively, based on the DESTINI study protocol, analyses in the current study were conducted as a retrospective review. Parameters known to influence the coronary flow reserve such as left ventricular hypertrophy\[^{23}\] and serum cholesterol levels\[^{28}\] were not evaluated in the current analysis, although categorical variables related to those parameters such as hypertension and hypercholesterolaemia were used. Given that a final coronary flow velocity reserve in itself did not correlate with the incidence of target lesion revascularization, the best cut off point of impaired coronary flow velocity reserve could not be determined and a pre-defined cut-off point was used. A time-dependent recovery of coronary flow velocity reserve after percutaneous coronary intervention has been reported\[^{5,14}\], although the coronary flow velocity reserve measured immediately after the interventional procedure may not reflect the late recovery of microcirculatory impairment. Despite this limitation, it is a great practical advantage that a grossly abnormal coronary flow velocity reserve, immediately after stenting, still correlates with the occurrence of target lesion revascularization. A final relative coronary flow velocity reserve was measured in only a minority of cases. However, the present study comprises one of the largest study populations in which relative coronary flow velocity reserve and absolute coronary flow velocity reserve have been measured after stenting.

### Conclusion

A final coronary flow velocity reserve of <2.0 after stent implantation in native coronary artery lesions was found to be an independent predictor of the need for long-term target lesion revascularization.

The authors wish to thank all the investigators who participated in the DESTINI study.

### Appendix

**Responsible investigators, participating centres and numbers of patients enrolled in the DESTINI study**

T. Anderson, J. Knutson, Foothills Hospital, Calgary, Canada (50); J. Moses, I Moussa, Lenox Hill Hospital, New York, U.S.A. (50); R. Bonan, J. C. Tardif, Institut de Cardiologie de Montreal, Montreal, Canada (50); T. Muramatsu, Social Insurance Kawasaki Hospital, Kanagawa, Japan (50); A. Colombo, C. Di Mario, EMO Centro Cuore Columbus, Milan, Italy (43); A. Jain, West Virginia University, Robert C. Byrd Health Sciences Center, Morgantown, U.S.A. (36); J. Suarez de Lezo, M. Pan, Hospital Reina Sofia, Cordoba, Spain (33); S. Y. Cho, Y. S. Jang, Yonsei University Hospital, Seoul, South Korea (30); M. Kern, R. Bach, St. Louis University, St. Louis, U.S.A. (28); I. Meredith, Monash Medical Center, Clayton, Australia (28); S. Kazziha, St. John’s Hospital, Detroit, U.S.A. (25); B. Weiner, University of Massachusetts Medical Center, Worcester, U.S.A. (24); V. Aharonian, Kaiser Foundation Hospital, Los Angeles, U.S.A. (23); S. J. Park, S. W. Park, Asan Medical Center, Seoul, Korea (21); H. Mudra, Immenstadt Munchen, Munchen, Germany (21); A. Frey, Herzzentrum, Bad Krozingen, Germany (20); M. L. Simard, White Memorial Hospital, Los Angeles, U.S.A. (20); T. Fischell, Borgess Medical Center, Kalamazoo, U.S.A. (19); E. Verna, S. Repetto, Ospedale di Circolo, Varese, Italy (15); B. W. Choi, Ajou University School of Medicine, Suwon, Korea (13); E. Caracciolo, Veterans Hospital John Cochran, St. Louis, U.S.A. (11); M. Mosseri, Hadassah-Hebrew University Medical Center, Jerusalem, Israel (10); H. Madyoon, L. Chroushore, St. Joseph’s, Stockton, California, U.S.A. (9); H. Nonogi, National Cardiovascular Center, Osaka, Japan (9); M. Leon, A. Pichard, Washington Hospital Center, Washington, U.S.A. (7); M. Ayres, V. Rhule, Fort Sanders Regional Medical Center, Knoxville, U.S.A. (7); T. Akasaka, Kobe General Hospital, Chuo-ku Kobe, Japan (7); T. Kondo, Komaki Hospital, Komaki City, Japan (7); S. Brener, Cleveland Clinic Hospital, Cleveland, U.S.A. (6); A. H. Gershlick, Glenfield General Hospital, Leicester, U.K. (6); T. Suzuki, National Toyohashi Hijashi Hospital, Toyohashi, Japan (6); F. Crea, A. Maseri, Policlinico Universitario Gemelli, Roma, Italy (6); I. Penn, Vancouver Hospital, Vancouver, Canada (5);
Coronary flow velocity reserve in stent implantation

M. Nobuyoshi, Kokura Memorial Hospital, Kitakyushu City, Japan (3); C. Seiler, B. Meier, Inselspital Bern, Bern, Switzerland (4); W. Kussmaul, J. D. Joye, Allegheny University Hospital, Philadelphia, U.S.A. (4); D. Senior, N. Xenopoulous, Jewish Hospital, Louisville, U.S.A. (4); A. Anwar, Baylor University Medical Center, Dallas, U.S.A. (3); R. Stewart, University of Wisconsin, Madison, U.S.A. (3); S. Werns, University of Michigan, Ann Arbor, U.S.A. (3); R. White, Baptist Hospital, Oklahoma City, U.S.A. (2); R. Bowerman, University Community Hospital, Tampa, U.S.A. (2); P. Overlie, Methodist Hospital, Lubbock, U.S.A. (2); K. Parr, Methodist Hospital, Indianapolis, U.S.A. (2); M. Tobias, Advocate Lutheran General Hospital, Park Ridge, U.S.A. (2); N. Margolis, Miami Heart Institute, Miami Beach, U.S.A. (1); J. M. Ruggio, Baptist Hospital, Oklahoma City, U.S.A. (1); E. B. Johnson, Cardiovascular Associates of South Texas, Laredo, U.S.A. (1); J. M. Lablanche, CHU de Rennes, Rennes, France (1); J. M. Ruggio, Villa Bianca, Bari, Italy (1); E. Kapilowicz, Villa Bianca, Bari, Italy (1); N. Kapilowicz, R. Heyar, Rambam Hospital, Haifa, Israel (1); T. Yamaguchi, Ohashi Hospital, Toho University, Tokyo, Japan (2); S. Lieberman, East Texas Medical Center, Tyler, U.S.A. (2); M. Bertrand, J. M. Lablanche, Centre Hospitalier Regional et Universitaire, Lille, France (1); J. M. Ruggio, Pacific Cardiovascular Associates Medical Group, Fountain Valley, U.S.A. (1); J. Margolis, Miami Heart Institute, Miami Beach, U.S.A. (1); V. Sethi, Hackensack Medical Center, Hackensack, U.S.A. (1); J. Lawson, Stony Brook, U.S.A. (1).

References


