# The Relationship and Threshold of Stent Length With Regard to Risk of Stent Thrombosis After Drug-Eluting Stent Implantation

Jon Suh, MD,\* Duk-Woo Park, MD, PhD,† Jong-Young Lee, MD,† In Hyun Jung, MD,† Seung-Whan Lee, MD, PhD,† Young-Hak Kim, MD, PhD,† Cheol Whan Lee, MD, PhD,† Sang-Sig Cheong, MD, PhD,‡ Jae-Joong Kim, MD, PhD,† Seong-Wook Park, MD, PhD,† Seung-Jung Park, MD, PhD,†

Bucheon, Seoul, and Gangneung, Korea

**Objectives** The aim of this study was to evaluate the association between the length of the stented segment and the risk of stent thrombosis (ST) after drug-eluting stent (DES) implantation and to determine the cutoff value of stent length in higher risk of ST in routine clinical practice.

**Background** Despite the recommendations of full lesion coverage to prevent angiographic restenosis, the length of the stented segment has been a risk factor for DES-related ST.

**Methods** A total of 3,145 consecutive patients (4,667 lesions) who underwent DES implantation were analyzed. The independent association of stent length with ST and its predictive value were evaluated for a median 29.6 months (interquartile range 21.6 to 37.5 months).

**Results** Stent thrombosis occurred in 68 patients (2.2%) at 3 years. The stent length/lesion was an independent predictor of ST (hazard ratio: 1.11, 95% confidence interval: 1.06 to 1.15, p < 0.001). The threshold of stent length for predicting ST was 31.5 mm (area under the receiver-operating characteristic curve: 0.746, 95% confidence interval: 0.699 to 0.793, p < 0.001), which had a sensitivity and specificity of 88.4% and 52.1%, respectively. Stent lengths ≥31.5 mm were associated with higher rates of ST (4.0% vs. 0.7%, p < 0.001), death (5.2% vs. 3.0%, p = 0.005), and myocardial infarction (2.4% vs. 0.7%, p = 0.001) at 3 years, as compared with stent lengths <31.5 mm.

**Conclusions** Length of the stented segment was independently associated with the incidence of ST and death or myocardial infarction after DES implantation. The value of stent length ≥31.5 mm is a threshold for the prediction of ST. (J Am Coll Cardiol Intv 2010;3:383–9) © 2010 by the American College of Cardiology Foundation

Use of drug-eluting stents (DES) has significantly reduced angiographic restenosis rates and the need for subsequent revascularization as compared with use of bare-metal stents (BMS) (1–3). Results from pivotal clinical trials show that DES are widely used in routine clinical practice including more complex patients and lesion characteristics, and full lesion coverage by using a longer stent has been regarded as the preferred strategy of percutaneous coronary intervention (PCI) to reduce the rate of restenosis. However, the widespread use of DES has raised concerns regarding the long-term safety of late stent thrombosis (ST).

Several clinical, anatomic, and procedural factors have been suggested to be important risk factors for development of ST after DES implantation (4–9). Previous studies have shown that the risk of ST is significantly associated with the total stent length (10,11). However, there have been limited

# Abbreviations and Acronyms

BMS = bare-metal stent(s)

CI = confidence interval

DES = drug-eluting stent(s)

HR = hazard ratio

IQR = interquartile range

MI = myocardial infarction

PES = paclitaxel-eluting stent(s)

PCI = percutaneous coronary intervention

ROC = receiver-operating

SES = sirolimus-eluting stent(s)

ST = stent thrombosis

TLR = target lesion revascularization

data regarding the prognostic impact of DES length on ST and clinical outcomes in "realworld" use.

Therefore we evaluated the independent relationship between stent length and the risk of ST after DES implantation and identified the threshold of stent length for predicting increased risk of ST and long-term cardiovascular events.

# **Methods**

Study population. The study included 3,157 consecutive patients who underwent coronary stent implantation with DES at 2 academic hospitals in Korea between February 2003 and February 2006. Drug-eluting

stents have been adopted as the default treatment for PCI treatment since February, 2003, at the Asan Medical Center, Seoul, and since May, 2003, at the Asan Medical Center, Gangneung.

Excluded from the analysis were patients with contraindications to antiplatelet agents (i.e., active or major bleeding), those requiring antiplatelet therapy interruption within 1 month after the procedure, and those who underwent coronary brachytherapy. Thus, a total of 3,145 patients (4,667 lesions) were included in the analysis. Sirolimuseluting stents (SES) and paclitaxel-eluting stents (PES) were used in 2,478 patients (79%) and 667 patients (21%), respectively. The study was approved by the institutional ethics committee at each center, and written informed consent was obtained.

Stenting procedures and antiplatelet therapy. Stent implantation was performed according to current standard techniques. Primary PCI for the infarct-related culprit lesion for patients with ST-segment elevation acute myocardial infarction (MI) was performed within 12 h of symptom onset. During the procedure, patients received intravenous weightadjusted heparin treatment to achieve an activated clotting time of  $\geq$ 300 s. The use of glycoprotein IIb/IIIa inhibitors was at the physician's discretion. All patients received a loading dose of 100 to 200 mg aspirin and 300 or 600 mg clopidogrel before or during PCI. After the procedure, 100 or 200 mg aspirin daily was continued indefinitely in all patients. The prescription of at least 6 months of clopidogrel 75 mg daily was recommended for patients receiving DES irrespective of stent type. The duration of extended clopidogrel use was determined at the physician's discretion. However, the duration of prescribed clopidogrel was gradually extended to at least 12 months after the concerns of late thrombosis had been raised (12).

Clinical follow-up and definitions. Clinical observation was conducted for a median 29.6 months (interquartile range [IQR] 21.6 to 37.5). Information regarding baseline clinical characteristics, procedural data, and clinical events was prospectively collected in an electronic database system by experienced research personnel who were unaware of the objective of the study. Post-discharge clinical data were obtained via regular outpatient visits with an interval of 3 or 6 months or telephone interview. To ensure detailed information regarding clinical events during follow-up, survival data for all patients were obtained from the national registration system of the Ministry of Government Administration and Home Affairs in Korea with personal identification numbers. Also, data on repeat hospital stay for follow-up MI were obtained via the hospital disease code registration system, which was merged for reimbursement with the Health Insurance Review Agency in Korea.

The definition of ST corresponded to the levels of "definite," "probable," or "possible" proposed by Academic Research Consortium, and ST was classified on the basis of the timing of the event as early (0 to 30 days), late (31 to 360 days), or very late (>360 days) according to standardized definitions (13,14). Death was defined as all-cause mortality during the follow-up period. The diagnosis of acute MI was established in the presence of ischemic symptoms and cardiac enzyme elevation (creatine kinase-myocardial band elevation >3 times or creatine kinase elevation >2 times the upper limit of normal value) (15). Target lesion revascularization (TLR) was defined as target lesion reintervention inside the implanted stent or within 5 mm proximally or distally by either PCI or bypass surgery. Major adverse cardiac events were defined as a composite of death, MI, or TLR.

**Statistical analysis.** Continuous variables are presented as mean  $\pm$  SD or median (IQR) and were compared with

Student t test or Mann-Whitney U test. Categorical variables are presented as frequencies or percentages and were compared with chi-square or Fisher exact tests, as appropriate. Correlations between the incidence of ST and covariates were initially examined by use of univariate Cox proportional hazards model. Variables with a p value ≤0.2 at univariate analysis and clinically relevant factors were entered in a multivariate Cox regression model with a stepping algorithm to identify independent predictors of ST. A receiver-operating characteristic (ROC) curve analysis with Youden index measure was performed to determine the best cutoff value of optimal stent length for predicting the ST. Cumulative event curves were generated with the Kaplan-Meier method and compared with the log-rank test of significance. All statistical tests were 2-sided, and differences were considered statistically significant at p < 0.05. Statistical analysis was performed with SPSS version 12.0 for windows (SPSS, Inc., Chicago, Illinois).

#### Results

Incidence of ST and clinical events. The study population consisted of 3,145 consecutive patients (treated for 4,667 lesions) who received DES implantation (SES for 3,603 lesions [77%] and PES for 1,064 lesions [23%]) between

ACC/AHA = American College of Cardiology/American Heart Association; CI = confidence interval; ST = stent thrombosis

February 2003 and February 2006. The median follow-up duration was 29.6 months (IQR 21.6 to 37.5).

During the follow-up period, ST occurred in 68 patients (2.2%), and these were categorized as 27 definite, 7 probable, and 34 possible. Early ST occurred in 9 patients (0.3%), late ST occurred in 28 patients (0.9%), and very late ST occurred in 31 patients (1.0%). The median time after the procedure was 4 days (IQR 2.0 to 9.3 days) for early ST occurrence, 7.0 months (IQR 4.6 to 8.5 months) for late ST, and 20.3 months (IQR 14.3 to 29.4 months) for very late ST. In ST patients, the mean duration of clopidogrel use was 11.5  $\pm$  8.8 months. After discontinuation of clopidogrel use, 21 (75.0%) patients developed late ST and 24 (77.4%) patients developed very late ST.

During follow-up, the cumulative incidence of major adverse cardiac events at 3 years was 5.9%. Death occurred in 4.2%, MI occurred in 1.6%, and TLR occurred in 2.7% of patients. Of the 68 ST patients, 62% (42 of 68) died, 49% (33 of 68) had MI, and 38% (26 of 68) had TLR.

Baseline characteristics. The baseline clinical and lesion characteristics comparing ST and non-ST patients as well as corresponding results of univariate Cox proportional hazard analysis for ST development are presented in Table 1. The ST and non-ST patients were similar in terms of age, sex, and coronary risk factors, but ST patients had higher

Variable	ST (n = 68)	No ST (n = 3,077)	Univariate Hazard Ratio (95% CI)	p Value
Age, yrs	59.4 ± 13.2	60.6 ± 10.2	0.99 (0.97–1.01)	0.40
Male sex	51 (75.0)	2,166 (70.5)	1.23 (0.71–2.13)	0.46
Hypertension	36 (52.9)	1,534 (49.9)	1.13 (0.70–1.82)	0.61
Diabetes mellitus	22 (32.4)	834 (27.1)	1.29 (0.78–2.14)	0.33
Hypercholesterolemia	15 (22.1)	679 (22.2)	0.98 (0.55-1.74)	0.94
Current smoking	20 (30.3)	871 (28.8)	1.04 (0.62–1.78)	0.88
Previous myocardial infarction	8 (11.7)	307 (10.0)	2.51 (0.71–8.90)	0.28
Previous coronary angioplasty	10 (14.7)	521 (16.9)	0.81 (0.41–1.59)	0.54
Previous bypass surgery	3 (4.4)	82 (2.7)	1.74 (0.55–5.54)	0.35
Acute myocardial infarction	21 (30.9)	336 (10.9)	3.79 (2.26–6.33)	< 0.001
Renal failure	7 (10.3)	36 (1.2)	8.06 (3.68–17.65)	< 0.001
Left ventricular ejection fraction, %	52.9 ± 11.4	$58.7 \pm 8.7$	0.95 (0.93-0.97)	< 0.001
Discontinuation of antiplatelet ≤6 months	16 (23.5)	294 (9.6)	2.71 (1.55–4.75)	< 0.001
Duration of antiplatelet therapy, months	$11.5 \pm 8.8$	$13.0 \pm 9.4$	0.98 (0.96–1.01)	0.14
Treated vessel and lesion characteristics				
Left anterior descending	32 (46.4)	2,265 (49.3)	0.81 (0.28–2.31)	0.69
Left main	8 (11.6)	317 (6.9)	1.43 (0.68–2.97)	0.34
Chronic total occlusion	4 (5.8)	252 (5.5)	0.85 (0.31–2.33)	0.75
Restenotic lesion	7 (10.1)	327 (7.1)	1.12 (0.51–2.46)	0.77
Ostial lesion	7 (10.1)	375 (8.2)	1.20 (0.55–2.63)	0.64
Bifurcation lesion	14 (20.3)	743 (16.2)	0.99 (0.55–1.78)	0.96
ACC/AHA type B2 or C	53 (76.8)	3,861 (73.1)	1.51 (0.34–6.78)	0.59

Variable	ST (n = 68)	No ST (n = 3,077)	Univariate Hazard Ratio (95% CI)	p Value
Multivessel PCI	16 (23.5)	1,034 (33.6)	0.59 (0.34–1.04)	0.07
Primary PCI	11 (16.1)	239 (7.7)	2.54 (1.33–4.87)	0.005
Bifurcation stenting	6 (8.7)	224 (4.9)	1.42 (0.61–3.28)	0.41
Direct stenting	8 (11.6)	759 (17.0)	0.81 (0.39–1.69)	0.57
Overlapping stenting	37 (54.4)	1,234 (26.8)	2.18 (1.36–3.50)	0.01
PCI with paclitaxel-eluting stent	18 (26.5)	649 (21.1)	0.77 (0.45-1.31)	0.33
Number of stents/lesion	$1.7 \pm 0.9$	$1.4 \pm 0.6$	1.71 (1.32–2.20)	< 0.001
Total stent length/lesion, mm	45.2 ± 27.3	34.2 ± 18.8	1.02 (1.01–1.03)	< 0.001
Number of stents/patient	2.2 ± 1.5	1.9 ± 1.1	1.18 (0.97–1.41)	0.06
Total stent length/patient, mm	57.3 ± 42.1	56.9 ± 35.6	1.01 (1.00–1.02)	0.001
Maximal balloon size, mm	3.7 ± 0.4	$3.6\pm0.6$	1.07 (0.81–1.43)	0.63
Maximal balloon inflation, atm	15.8 ± 4.3	$15.9 \pm 3.9$	0.98 (0.93-1.04)	0.56
Lesion length, mm	32.6 ± 19.3	26.2 ± 14.6	1.02 (1.00–1.03)	0.03
Reference vessel diameter, mm	$3.0\pm0.4$	$2.9\pm0.5$	1.18 (0.68–2.06)	0.55
Minimal luminal diameter, mm				
Before intervention	$0.9\pm0.6$	$0.9\pm0.5$	1.04 (0.67–1.60)	0.87
After intervention	$2.8 \pm 0.4$	$2.8\pm0.5$	0.90 (0.52–1.54)	0.69
Diameter stenosis, %				
Before intervention	68.1 ± 19.3	67.6 ± 17.6	1.00 (0.98–1.02)	0.75
After intervention	4.8 ± 11.2	1.9 ± 13.4	1.02 (0.99–1.04)	0.17
Acute gain, mm	1.9 ± 0.5	1.9 ± 0.6	0.77 (0.44–1.33)	0.35

incidence of MI on presentation and renal failure and lower left ventricular ejection fraction than non-ST patients. Apart from the duration of antiplatelet therapy, discontinuation of antiplatelet therapy  ${\leq}6$  months was more frequent in ST patients than in non-ST patients. The angiographic and procedural findings according to ST are presented in Table 2. The ST patients underwent more overlapping stents and more primary PCI and had more stents, longer stents, and longer lesions than non-ST patients.

Data represent mean  $\pm$  SD for continuous variables and n (%) for dichotomous variables. PCI = percutaneous coronary intervention; other abbreviations as in Table 1.

Independent predictors of stent thrombosis. Stepwise multivariate Cox regression analysis showed that renal failure,

Table 3. Independent Predictors of Stent Thrombosis					
Variables	Hazard Ratio (95% CI)	p Value*			
Overall stent thrombosis					
Renal failure	7.97 (2.24–28.36)	0.001			
Discontinuation of antiplatelet ≤6 months	2.77 (1.12-6.88)	0.028			
Total stent length/lesion	1.11 (1.06–1.15)	< 0.001			
Very late stent thrombosis					
Left ventricular ejection fraction	0.95 (0.91-1.00)	0.035			
Total stent length/lesion	1.15 (1.05–1.25)	0.001			
Diameter stenosis, post	3.23 (1.42–7.42)	0.006			
*Predictors with a p value $\leq$ 0.2 were entered into multivariate Cox-regression analysis with a					

stepping algorithm.

Abbreviations as in Table 1.

discontinuation of antiplatelet ≤6 months, and total stent length/lesion were independent risk factors of ST (Table 3). Left ventricular ejection fraction and total stent length/lesion were major predictors of very late ST. Of the procedural variables, total stent length/lesion was the only

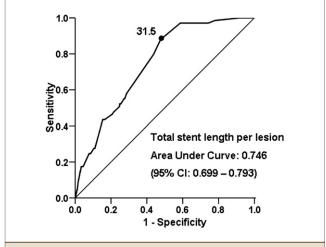


Figure 1. Receiver-Operating Characteristic Curve

A receiver-operating characteristic curve to identify optimal stent length for predicting stent thrombosis. The number on the curve represents the best predictive value of optimal stent length. CI = confidence interval.

predictor of ST (hazard ratio [HR]: 1.11, 95% confidence interval [CI]: 1.06 to 1.15, p < 0.001).

Stent length as a predictor of ST. An ROC curve to determine the cutoff value of stent length for predicting ST occurrence is shown in Figure 1. The area under the curve was 0.746 (95% CI: 0.699 to 0.793, p < 0.001), showing fair discrimination, and the stent length that best predicted ST was 31.5 mm. In the present study, 1,761 (56%) patients had stents longer than 31.5 mm. Patients with stent lengths ≥31.5 mm had a higher 3-year cumulative ST incidence rate compared with patients with stent lengths <31.5 mm (4.0% vs. 0.7%, [log rank] p < 0.001) (Fig. 2A). Adjusted Cox regression analysis showed that stent lengths ≥31.5 mm were accompanied by an increased risk of ST (HR: 7.87, 95% CI: 2.57 to 24.17, p < 0.001) (Fig. 3).

Stent lengths  $\geq$ 31.5 mm predicted ST with a sensitivity of 88.4% and a specificity of 52.1%.

Relationship between stent length and clinical outcomes.

The 3-year cumulative clinical outcomes according to the stent length prediction value are depicted in Figure 2. The 3-year mortality was higher in patients with stent lengths ≥31.5 mm as compared with those with stent lengths <31.5 mm (5.2% vs. 3.0%, [log rank] p = 0.005) (Fig. 2B). The 3-year follow-up MI rate was also higher in patients with stent lengths ≥31.5 mm compared with those with stent lengths <31.5 mm (2.4% vs. 0.7%, [log rank] p = 0.001) (Fig. 2C). Accordingly, patients with stent lengths ≥31.5 mm had a higher association with overall rate of death or MI, compared with those with stent lengths <31.5 mm (6.9% vs. 3.5%, [log rank] p < 0.001) (Fig. 2D).

## **Discussion**

The main finding of the present study was that longer stent lengths were significantly associated with a higher risk of ST and major coronary events (i.e., cardiac death and MI) in

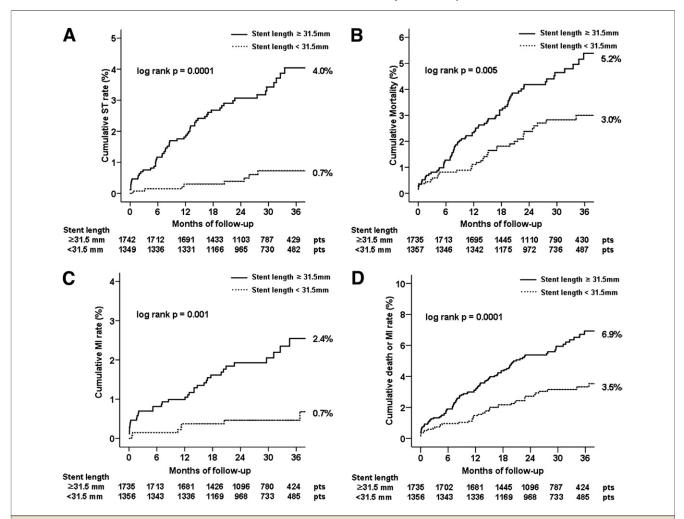
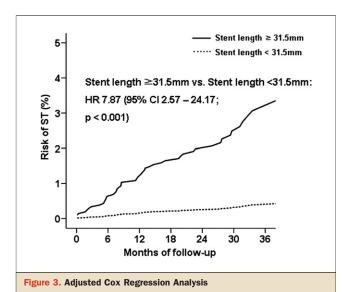


Figure 2. Kaplan-Meier Curves of 3-Year Cumulative Incidence of Clinical Outcomes

Comparison of 3-year cumulative adverse outcomes according to the optimal predictive stent length of 31.5 mm. (A) Stent thrombosis (ST), (B) death, (C) myocardial infarction (MI), (D) death/myocardial infarction.



Adjusted cumulative hazard ratio (HR) of stent thrombosis (ST) up to 3 years according to the optimal predictive stent length of 31.5 mm. Abbreviations as in Figure 1.

patients who received DES implantation. In addition, the value of 31.5 mm is a threshold above which there is a higher likelihood of ST with a sensitivity and specificity of 88% and 52%, respectively. Hence, patients with stent lengths ≥31.5 mm had a higher cumulative incidence rate of ST, death, and MI as compared with those with stent lengths <31.5 mm.

Stent length as a predictor of ST and clinical events. The increased off-label use of DES has raised issues regarding how to manage complex lesions in terms of complete revascularization. Use of multiple or longer stents for complex intervention increases the risk of ST, regardless of the presence or absence of conventional ST risk. However, the long-term clinical impact of stent length on ST development in the DES era remains to be determined. Previous studies showed that ST incidence after BMS implantation was related to stent length (16). Similarly, recent reports also showed an association between stent length and increased clinical risk of ST after DES implantation (11,17,18).

A major finding of the present study was that stent length/lesion was the most important procedure-related risk factor for development of ST during long-term follow-up. This result is consistent with the ARTS II (Arterial Revascularization Therapies Study Part II) analysis, showing that total stent length was an independent predictor of ST at 3 years (HR: 1.14, 95% CI: 1.04 to 1.25, p=0.0037) (17,19). Although the present study had a lower-risk procedural profile than for the ARTS II analysis, total stented length was also found to be an independent predictor of ST in the current study. Moreover, in the current study, the threshold of predictive stent length was found to be 31.5 mm, with stents longer than 31.5 mm associated with a remarkably

higher risk of ST. Indeed, of the 68 ST patients, 60 (88%) had stent lengths/lesion longer than 31.5 mm.

Another major finding of this study was that the incidence of death or MI was significantly proportional to stent length. The incidence of adverse clinical events associated with long DES in the present study was lower than that reported to be associated with BMS (20,21). These were reassuring results compared with previous BMS studies showing a positive association between stent length and adverse cardiac events (22).

Although the long DES seem to be safer and more effective than BMS, to date, data regarding DES length and adverse events are lacking. It seems that the present findings showing an association between stent length and coronary events after implantation should be considered when assessing safety relating to DES use.

Study limitations. Although all variables were included by backward elimination step-wise manner in multivariable analysis, there are too many candidate variables for the multivariable Cox models, given the number of end points. Including death, there are competing events that might interfere with the ST event risk. The present report describes an observational study from 2 centers. The association between ST risk and stent length in terms of DES type was not evaluated. Also, mostly SES were used in this study. Therefore, it was a nonrandomized study design, and the procedures used were those recommended by each institution. Furthermore, the lack of procedure-supported intravascular ultrasound data evaluation diminished the power of the study to show the effectiveness of DES procedures. However, we performed PCI with intravascular ultrasound guidance in more than approximately 70% of patients, because such a procedure is likely of clinical benefit.

## **Conclusions**

The present study demonstrated that stent length is an independent predictor of ST after DES implantation. The risk of ST as well as death or MI was higher if the stented segment was ≥31.5 mm long. Therefore, longer stent length was related to greater risk of ST, death, and MI after DES implantation.

Reprint requests and correspondence: Dr. Seung-Jung Park, Department of Cardiology, Asan Medical Center, University of Ulsan College of Medicine, Cardiac Center, 388-1 Poongnap-dong, Songpa-gu, Seoul 138-736, Korea. E-mail: sjpark@amc.seoul.kr.

#### REFERENCES

 Morice MC, Serruys PW, Sousa JE, et al. A randomized comparison of a sirolimus-eluting stent with a standard stent for coronary revascularization. N Engl J Med 2002;346:1773–80.

- 2. Moses JW, Leon MB, Popma JJ, et al. Sirolimus-eluting stents versus standard stents in patients with stenosis in a native coronary artery. N Engl J Med 2003;349:1315–23.
- 3. Stone GW, Ellis SG, Cox DA, et al. A polymer-based, paclitaxel-eluting stent in patients with coronary artery disease. N Engl J Med 2004;350:221–31.
- Iakovou I, Schmidt T, Bonizzoni E, et al. Incidence, predictors, and outcome of thrombosis after successful implantation of drug-eluting stents. JAMA 2005;293:2126–30.
- Urban P, Gershlick AH, Guagliumi G, et al. Safety of coronary sirolimus-eluting stents in daily clinical practice: one-year follow-up of the e-Cypher registry. Circulation 2006;113:1434–41.
- Kuchulakanti PK, Chu WW, Torguson R, et al. Correlates and long-term outcomes of angiographically proven stent thrombosis with sirolimus- and paclitaxel-eluting stents. Circulation 2006;113:1108–13.
- 7. Biondi-Zoccai GGL, Agostoni P, Sangiorgi GM, et al. Incidence, predictors, and outcomes of coronary dissections left untreated after drug-eluting stent implantation. Eur Heart J 2006;27:540-6.
- 8. Fujii K, Carlier SG, Mintz GS, et al. Stent underexpansion and residual reference segment stenosis are related to stent thrombosis after sirolimus-eluting stent implantation: an intravascular ultrasound study. J Am Coll Cardiol 2005;45:995–8.
- Spertus JA, Kettelkamp R, Vance C, et al. Prevalence, predictors, and outcomes of premature discontinuation of thienopyridine therapy after drug-eluting stent placement: results from the PREMIER registry. Circulation 2006;113:2803–9.
- 10. Park DW, Park SW, Park KH, et al. Frequency of and risk factors for stent thrombosis after drug-eluting stent implantation during long-term follow-up. Am J Cardiol 2006;98:352–6.
- Moreno R. Drug-eluting stent thrombosis. J Am Coll Cardiol 2005; 45:954–9.
- 12. McFadden EP, Stabile E, Regar E, et al. Late thrombosis in drugeluting coronary stents after discontinuation of antiplatelet therapy. Lancet 2004;364:1519–21.
- 13. Laskey WK, Yancy CW, Maisel WH. Thrombosis in coronary drug-eluting stents: report from the meeting of the Circulatory System

- Medical Devices Advisory Panel of the Food and Drug Administration Center for Devices and Radiologic Health, December 7–8, 2006. Circulation 2007;115:2352–7.
- Cutlip DE, Windecker S, Mehran R, et al. Clinical end points in coronary stent trials: a case for standardized definitions. Circulation 2007;115:2344–51.
- Thygesen K, Alpert JS, White HD, Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. Universal definition of myocardial infarction. J Am Coll Cardiol 2007;50:2173–95.
- Cutlip DE, Baim DS, Ho KKL, et al. Stent thrombosis in the modern era: a pooled analysis of multicenter coronary stent clinical trials. Circulation 2001;103:1967–71.
- Serruys PW, Daemen J, Morice MC, et al. Three-year follow-up of the ARTS-II—sirolimus-eluting stents for the treatment of patients with multivessel coronary artery disease. Euro Intervention 2008;3:450-9.
- Chieffo A, Bonizzoni E, Orlic D, et al. Intraprocedural stent thrombosis during implantation of sirolimus-eluting stents. Circulation 2004;109:2732-6.
- Serruys PW, Ong AT, Morice MC, et al. Arterial Revascularization Therapies Study Part II—sirolimus-eluting stents for the treatment of patients with multivessel de novo coronary artery lesions. EuroIntervention 2005;1:147–56.
- Degertekin M, Arampatzis CA, Lemos PA, et al. Very long sirolimuseluting stent implantation for de novo coronary lesions. Am J Cardiol 2004;93:826–9.
- 21. Kim YH, Park SW, Lee CW, et al. Comparison of sirolimus-eluting stent, paclitaxel-eluting stent, and bare metal stent in the treatment of long coronary lesions. Catheter Cardiovasc Interv 2006;67:181–7.
- 22. Serruys PW, Foley DP, Suttorp MJ, et al. A randomized comparison of the value of additional stenting after optimal balloon angioplasty for long coronary lesions: final results of the additional value of NIR stents for treatment of long coronary lesions (ADVANCE) study. J Am Coll Cardiol 2002;39:393–9.

**Key Words:** drug-eluting stent ■ stent length ■ stent thrombosis.