Comparison of Simple and Complex Stenting Techniques in the Treatment of Unprotected Left Main Coronary Artery Bifurcation Stenosis

Young-Hak Kim, MD, PhD, Seong-Wook Park, MD, PhD, Myeong-Ki Hong, MD, PhD, Duk-Woo Park, MD, Kyoung-Min Park, MD, Bong-Ki Lee, MD, Jong-Min Song, MD, PhD, Ki-Hoon Han, MD, PhD, Cheol Whan Lee, MD, PhD, Duk-Hyun Kang, MD, PhD, Jae-Kwan Song, MD, PhD, Jae-Joong Kim, MD, PhD, and Seung-Jung Park, MD, PhD*

We assessed the safety and feasibility of various stenting techniques using the sirolimus-eluting stent (SES) in the treatment of unprotected left main coronary artery (LMCA) bifurcation stenoses. One hundred sixteen patients with unprotected LMCA bifurcation stenoses underwent SES implantation. A simple stenting technique (simple group, n = 67) across the left circumflex artery (LCx) and a complex technique (complex group) comprising "kissing" stenting (n = 24) or a "crush" (n = 25) technique were used. Baseline clinical and angiographic characteristics were similar for the 2 groups, except for more multivessel involvement and narrower LCxs in the complex group. The procedural success rate was 100%. Angiographic restenosis rate at 6 months was lower in the simple group (5.3%) than in the complex group (24.4%, p = 0.024). In the complex group, restenosis rates were similar for the kissing (25.0%) and crush (23.8%) techniques (p = 1.0). There were no incidents of death or myocardial infarction during follow-up (median 18.6 months). Target lesion revascularization was performed in 6 patients only in the complex group (0% vs 12.2%, p = 0.005). At 18 months, survival rates without target lesion revascularization were 100 ± 0% in the simple group and 85.7 ± 5.6% in the complex group (p = 0.004). In conclusion, SES implantation for unprotected LMCA bifurcation stenoses appears to be safe and effective. Compared with the complex stenting technique, the simple technique was technically easier and appeared to be more effective in improving long-term outcomes in patients with normal LCxs. **(Am J Cardiol 2006; 97:1597–1601)**

The present study evaluated clinical, angiographic, and intravascular ultrasound outcomes of various stenting techniques using a sirolimus-eluting stent (SES) in the treatment of unprotected left main coronary artery (LMCA) bifurcation stenosis.

• • •

The study included 116 consecutive patients with de novo unprotected LMCA bifurcation stenoses who underwent elective SES implantation from March 2003 to November 2004. Inclusion criteria were symptomatic unprotected LMCA disease or documented myocardial ischemia and angiographic evidence of ≥50% diameter stenosis at the unprotected LMCA bifurcation that was considered suitable for stent placement. The LMCA was considered unprotected if there were no patent coronary artery bypass grafts to the left anterior descending artery or left circumflex artery (LCx). Patients with a contraindication for antiplatelet or anticoagulation therapy or severe left ventricular dysfunction (ejection fraction ≤40%) were excluded. Informed written consent was obtained from all patients, in accordance with the Declaration of Helsinki.

Unprotected LMCA bifurcation stenting was performed as previously described.[1,2] Lesions were treated with 1 of 2 stenting strategies according to quantitative angiographic and intravascular ultrasound measurements. The presence of ostial LCx disease (≥50% diameter stenosis) and the unprotected LMCA bifurcation reference size according to angiographic and intravascular ultrasound examinations were important considerations in selecting the stenting strategy. In 67 patients (57.8%) with a normal (<50% diameter stenosis) or small (≤2.0 mm) LCx, a simple stenting technique across the ostial LCx was used (simple group). Final kissing balloon inflation was performed in lesions with significant compromise (≥50% diameter stenosis) of the LCx after simple stenting. Alternatively, a complex technique such as a kissing stenting or a crush technique was preferred in 49 patients (42.2%) with a diseased LCx (complex group). In the complex group, 24 patients who had a relatively large unprotected LMCA that could accommodate 2 stents proximal to the bifurcation received the kissing stenting technique in an attempt to ensure optimal stent

The Department of Medicine, University of Ulsan College of Medicine, Asan Medical Center, Seoul, Korea. Manuscript received August 9, 2005; revised manuscript received and accepted December 20, 2005.

This study was supported in part by the CardioVascular Research Foundation, Seoul, Korea, by Grant 0412-CR02-0704-0001 from the Korea Health 21 R&D Project, Ministry of Health and Welfare, Seoul, Korea, and by Grants 2004-361 and 2005-361 from the Asan Institute for Life Sciences, Seoul, Korea.

*Corresponding author: Tel: 82-2-3010-4812; fax: 82-2-475-6898.

E-mail address: sjpark@amc.seoul.kr (S.-J. Park).

0002-9149/06/$ – see front matter

doi:10.1016/j.amjcard.2005.12.051

expansion and sufficient drug release when using SESs ≤3.5 mm in diameter. During kissing stenting, sequential high-pressure deployment of each stent was performed before simultaneous kissing balloon dilatation for optimal stent expansion.[3] The crush technique was used in 25 patients with moderately sized, unprotected LMCAs. After the crush technique, final kissing balloon dilatation was routinely attempted and was successful in 20 patients (80%) to ensure optimal stent apposition of the 2 stents at the bifurcation.[4] Intravascular ultrasound was used for guidance before and after the procedure.

Debulking atherectomy was used in 7 patients (6.0%) to facilitate stent delivery to the target lesions. An intra-aortic balloon pump was used in 7 patients (6.0%) for hemodynamic support. The use of glycoprotein IIb/IIIa inhibitors was left to the operators' discretion. All patients received aspirin (200 mg/day) indefinitely and a loading dose of 300 mg of clopidogrel followed by a single 75 mg/day dose for 6 months. In addition, 200 mg of cilostazol was administered as a loading dose, followed by 100 mg 2 times daily for 1 month.[5] All patients were evaluated clinically by office visits or telephone interviews at 1, 3, and 6 months after stenting and then every 4 months. For early detection of restenosis, repeat coronary angiography was routinely performed 6 months after stenting or sooner if clinically indicated. Target lesion revascularization was performed in patients with restenosis who had symptoms or objective evidence of myocardial ischemia.

Coronary angiography was performed after administering 0.2 mg of intracoronary nitroglycerin. Coronary angiographic results were analyzed by 2 experienced angiographers who were blinded to the stenting procedures. Using the guiding catheter for magnification calibration and an online quantitative coronary angiographic system (ANCOR 2.0, Siemens, Solna, Sweden), minimum lumen diameter, percent diameter stenosis, and reference vessel diameter were measured before and after intervention and at follow-up. Acute gain was calculated as the difference between minimum lumen diameters before and after the procedure, and late loss was defined as the difference between minimum lumen diameters after the procedure and at follow-up. Angiographic restenosis was defined as ≥50% diameter stenosis at the target site. An untreated, small LCx with ≥50% diameter stenosis after the procedure and at follow-up was not considered restenosed. Pre- and postintervention intravascular ultrasound images were obtained after administration of 0.2 mg of intracoronary nitroglycerin using a commercial intravascular ultrasound system (SciMed/Boston Scientific, Natick, Massachusetts) and motorized pullback of the ultrasound catheter at 0.5 mm/s. The external elastic membrane and lumen cross-sectional areas were measured with computerized planimetry.[6] Lesion sites of the unprotected LMCA bifurcation and ostial left anterior descending artery were defined as image slices with the smallest lumen cross-sectional area in the unprotected LMCA bifurcation and the site within 5 mm of the ostial left anterior descending artery, respectively. Plaque burden was measured as a percentage as 100 × (EEM CSA − lumen CSA)/EEM CSA, where CSA represents the cross-sectional area and EEM represents the external elastic membrane.

Table 1
Clinical and procedural characteristics

Variable	Simple Group (n = 67)	Complex Group (n = 49)	p Value
Age (yrs)	59.6 ± 12.0	60.6 ± 8.5	0.604
Men	48 (71.6%)	38 (77.6%)	0.473
Cardiac risk factors			
Hypertension	34 (50.7%)	17 (34.7%)	0.085
Diabetes mellitus	24 (35.8%)	11 (22.4%)	0.121
Hypercholesterolemia (total cholesterol ≥200 mg/dl)	17 (25.4%)	8 (16.3%)	0.242
Current smoker	13 (19.4%)	15 (30.6%)	0.163
Previous percutaneous coronary intervention	8 (11.9%)	9 (18.4%)	0.334
Acute coronary syndrome	34 (50.7%)	29 (52.2%)	0.368
Multivessel involvement (≥2 vessels plus left main lesions)	46 (68.7%)	42 (85.7%)	0.047
Left ventricular ejection fraction (%)	59.2 ± 8.0	61.5 ± 7.3	0.110
Multiple lesion intervention	25 (37.3%)	18 (36.7%)	0.949
Debulking atherectomy	4 (6.0%)	3 (6.1%)	0.973
Use of glycoprotein IIb/IIIa inhibitor	3 (4.5%)	9 (18.4%)	0.027
Intravascular ultrasound guidance	60 (89.6%)	43 (87.8%)	0.762
Total stent length in main vessel (mm)	31.8 ± 19.3	35.4 ± 18.3	0.314
Stents used per lesion	1.4 ± 0.7	2.6 ± 0.8	<0.001
Use of intra-aortic balloon pump	5 (7.5%)	2 (4.1%)	0.697

Data are expressed as mean ± 1 SD for continuous variables and as frequencies for categorical variables. Differences between groups were assessed with chi-square or Fisher's exact test for categorical variables and Student's *t* test for continuous variables. Survival distributions without target lesion revascularization were estimated according to the Kaplan-Meier method. Log-rank test was used to compare survival without target lesion revascularization between the simple and complex groups. A p value <0.05 indicated a statistically significant difference. Statistical analysis was performed with SPSS 11 for Windows (SPSS, Inc., Chicago, Illinois).

Baseline clinical and procedural characteristics for the 2 groups are listed in Table 1. Baseline clinical characteristics did not differ between groups except for more multivessel involvement in the complex group. During the procedure, the complex group was administered more glycoprotein IIb/IIIa inhibitors and was implanted with more stents compared with the simple group. Stent lengths were similar for the 2 groups. Final kissing balloon inflation after stenting was performed in 28 patients (41.8%) in the simple group and 43 patients (87.8%) in the complex group (p <0.001). No patient in the simple group received provisional stenting at the LCx during or after the procedure. All procedures were successfully performed without any incidents of death,

Table 2
Quantitative angiographic analysis results

Variable	Simple Group (n = 69)	Complex Group (n = 49)	p Value
Patients with follow-up angiogram	57 (85.1%)	41 (83.7%)	0.837
LMCA			
Proximal reference diameter (mm)	3.61 ± 0.72	3.77 ± 0.74	0.240
Distal reference diameter (mm)	2.81 ± 0.60	2.75 ± 0.45	0.557
Minimal lumen diameter (mm)			
Before procedure	1.11 ± 0.47	1.01 ± 0.47	0.269
After procedure	2.97 ± 0.52	2.98 ± 0.36	0.931
At follow-up	2.91 ± 0.53	2.56 ± 0.67	0.006
Diameter stenosis (%)			
Before procedure	65.1 ± 13.9	68.2 ± 15.1	0.254
After procedure	9.6 ± 13.8	12.8 ± 14.0	0.219
At follow-up	11.3 ± 13.6	23.8 ± 18.4	<0.001
Lesion length (mm)	25.8 ± 17.1	26.2 ± 14.5	0.918
Acute gain (mm)	1.86 ± 0.58	1.96 ± 0.45	0.295
Late loss (mm)	0.13 ± 0.40	0.42 ± 0.63	0.009
Restenosis	0	4 (9.8%)	0.028
LCx			
Distal reference diameter (mm)	2.78 ± 0.66	2.64 ± 0.49	0.209
Minimal lumen diameter (mm)			
Before procedure	2.25 ± 0.76	1.39 ± 0.64	<0.001
After procedure	2.21 ± 0.77	2.65 ± 0.40	<0.001
At follow-up	1.98 ± 0.80	1.97 ± 0.81	0.958
Diameter stenosis (%)			
Before procedure	19.0 ± 21.8	47.8 ± 21.3	<0.001
After procedure	20.1 ± 22.7	−1.9 ± 13.7	<0.001
At follow-up	26.5 ± 20.6	26.3 ± 24.6	0.959
Acute gain (mm)	−0.04 ± 0.66	1.26 ± 0.60	<0.001
Late loss (mm)	0.20 ± 0.59	0.69 ± 0.72	<0.001
Restenosis	3 (5.3%)	7 (17.7%)	0.089
Overall restenosis	3 (5.3%)	10 (24.4%)	0.024

Q-wave myocardial infarction, stent thrombosis, or emergency bypass surgery during hospitalization. Procedure-related creatine kinase-MB increases ≥3 times normal occurred in 4 patients (6.0%) in the simple group and in 3 patients (6.1%) in the complex group (p = 1.0).

Results of quantitative angiographic analyses are presented in Table 2. Angiographic data from the main vessel did not differ between the 2 groups before and after the procedure. However, the complex group had more diseased LCxs before the procedure and achieved more expanded LCxs after the procedure. Angiographic follow-up at 6 months was performed in 57 patients (85.1%) in the simple group and 41 patients (83.7%) in the complex group (p = 0.837). At the main vessel, late loss (0.13 ± 0.40 vs 0.42 ± 0.63 mm, p = 0.009) and angiographic restenosis rate (0% vs 9.8%, p = 0.028) were lower in the simple group than in the complex group. Likewise, late loss at the LCx was lower in the simple group than in the complex group (0.20 ± 0.59 vs 0.69 ± 0.72 mm, p <0.001). Lower late loss in the simple group, despite worse postprocedural angiographic outcomes, contributed to the suggestion of lower LCx restenosis than in the complex group (5.3% vs 17.7%, p = 0.089), but that difference did not reach statistical significance. Therefore, the simple group showed a lower overall angiographic restenosis rate than did the complex group (5.3% vs 24.4%, p = 0.024). Angiographic outcomes for the 2 complex stenting techniques are presented in Table 3. Except for the large reference diameter of the unprotected LMCA in the kissing technique group, the 2 complex stenting techniques had similar postprocedural and follow-up angiographic results at the main vessel and the side branch.

Table 3
Comparison of complex stenting techniques

Variable	Kissing Technique (n = 24)	Crush Technique (n = 25)	p Value
Patients with follow-up angiogram	20 (83%)	21 (84%)	
LMCA			
Proximal reference diameter (mm)	4.09 ± 0.69	3.46 ± 0.65	0.002
Distal reference diameter (mm)	2.92 ± 0.42	2.59 ± 0.42	0.009
Minimal lumen diameter (mm)			
Before procedure	0.91 ± 0.52	1.12 ± 0.40	0.111
After procedure	2.97 ± 0.35	2.99 ± 0.37	0.837
At follow-up	2.58 ± 0.70	2.54 ± 0.66	0.865
Lesion length (mm)	23.7 ± 13.3	28.6 ± 15.4	0.253
Acute gain (mm)	2.06 ± 0.40	1.87 ± 0.49	0.138
Late loss (mm)	0.39 ± 0.67	0.44 ± 0.61	0.790
Restenosis	3 (15.0%)	1 (4.8%)	0.343
LCx			
Distal reference diameter (mm)	2.73 ± 0.56	2.56 ± 0.40	0.229
Minimal lumen diameter (mm)			
Before procedure	1.48 ± 0.78	1.30 ± 0.47	0.332
After procedure	2.70 ± 0.36	2.60 ± 0.44	0.387
At follow-up	2.03 ± 0.78	1.91 ± 0.85	0.646
Acute gain (mm)	1.22 ± 0.72	1.30 ± 0.46	0.645
Late loss (mm)	0.72 ± 0.56	0.67 ± 0.85	0.824
Restenosis	3 (15.0%)	4 (19.0%)	1.000
Overall restenosis	5 (25.0%)	5 (23.8%)	1.000

Results of quantitative intravascular ultrasound measurement at the unprotected LMCA bifurcation and the ostial left anterior descending artery before and after the procedure are listed in Table 4. Before the procedure, lumen cross-sectional area at the unprotected LMCA bifurcation was larger in the simple group than in the complex group. However, postprocedural lumen cross-sectional area at the unprotected LMCA bifurcation was similar for the 2 groups. In contrast, after the procedure, lumen cross-sectional area at the ostial left anterior descending artery was larger in the simple group than in the complex group, despite a similar baseline character.

Clinical information was collected on all patients for the follow-up period (median 18.6 months). Mean follow-up durations were 18.0 ± 5.3 months in the simple group and 18.0 ± 5.6 months in the complex group (p = 0.83). No cases of death, stent thrombosis, or myocardial infarction occurred during follow-up. Target lesion revascularization was performed in no patient in the simple group and 6 patients (12.2%) in the complex group (p = 0.005). At 18

Table 4
Intravascular ultrasound analysis results

Variable	Simple Group (n = 21)	Complex Group (n = 18)	p Value
LMCA bifurcation			
Before procedure			
External elastic membrane area (mm^2)	21.7 ± 6.0	20.6 ± 4.0	0.391
Lumen area (mm^2)	6.2 ± 2.2	4.8 ± 1.7	0.003
Plaque burden (%)	70.8 ± 8.9	76.1 ± 9.1	0.012
After procedure			
External elastic membrane area (mm^2)	23.9 ± 5.7	24.0 ± 3.9	0.905
Lumen area (mm^2)	11.7 ± 2.7	12.5 ± 2.7	0.191
Plaque burden (%)	50.2 ± 8.4	47.7 ± 8.8	0.184
Ostial left anterior descending artery			
Before procedure			
External elastic membrane area (mm^2)	15.2 ± 4.4	14.4 ± 3.3	0.339
Lumen area (mm^2)	4.5 ± 2.0	4.2 ± 1.8	0.548
Plaque burden (%)	69.7 ± 11.8	70.6 ± 9.9	0.707
After procedure			
External elastic membrane area (mm^2)	18.2 ± 4.0	17.7 ± 2.6	0.523
Lumen area (mm^2)	9.7 ± 2.0	8.0 ± 1.7	<0.001
Plaque burden (%)	45.8 ± 10.2	54.8 ± 7.5	<0.001

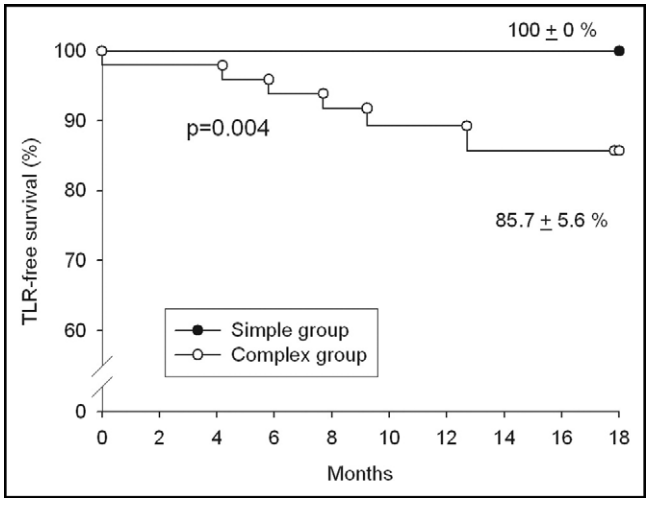

Figure 1. Kaplan-Meier curves for survival without target lesion revascularization (TLR) at 18 months in the simple and complex groups show a statistically significant difference between groups (p = 0.004).

months, survival rates without target lesion revascularization were 100 ± 0% in the simple group and 85.7 ± 5.6% in the complex group (p = 0.004; Figure 1).

• • •

The major finding of this study was that percutaneous intervention using the SES appears safe and effective in treating unprotected LMCA bifurcation stenoses. Such an approach was not associated with death, myocardial infarction, or stent thrombosis. In addition, the stenting strategy adopted on the basis of lesion characteristics affected clinical and angiographic outcomes, which were more favorably associated with the simple stenting technique than with the complex stenting techniques.

Some studies have reported that percutaneous intervention for unprotected LMCA bifurcation is safe in low-risk patients.[1,7–10] Nevertheless, because the unprotected LMCA bifurcation stenosis invariably involves the ostia of 2 major epicardial arteries (left anterior descending artery and LCx), in-stent restenosis at the target lesion may lead to serious adverse cardiac events and often requires bypass surgery for treatment.[8] Restenotic rates of unprotected LMCA bifurcation treatment with drug-eluting stents remain diverse.[2,11–13] The present study, which had a target lesion revascularization rate of 5.2% without any incidents of death or myocardial infarction, suggests that SES implantation may be an effective treatment for unprotected LMCA bifurcation stenosis.

In the present study, to prevent LCx compromise during and after the procedure, complex stenting techniques were used in truly unprotected LMCA bifurcation lesions with diseased LCxs. Two complex stenting strategies were predetermined to ensure complete coverage of the 2 lesions, optimal stent expansion, and sufficient drug diffusion, i.e., kissing stenting after sequential high-pressure dilatation and a crush technique followed by final kissing balloon inflation.[3,4,14] We found that the long-term outcomes for the 2 complex strategies were acceptable, a finding consistent with previous results for bifurcation interventions using drug-eluting stents.[15–17] Compared with simple stenting, complex stenting was performed in lesions with narrower LCxs, which meant that the higher occurrence of LCx restenosis in the complex group could be partly explained by the greater complexity of the initial lesions. However, lesion character may not be the only explanation for the higher restenotic rate in the main vessel and the side branch. Main vessel angiographic findings did not differ between the 2 groups. Moreover, postprocedural angiographic outcomes for the LCx were better in the complex group. Intravascular ultrasound analysis indicated this could be partly attributed to stent underexpansion distal to the bifurcation. Postprocedural intravascular ultrasound measurements showed that the ostial left anterior descending artery was less dilated in the complex group than in the simple group. Stent underexpansion has been considered a significant cause of drug-eluting stent restenosis in bifurcation and nonbifurcation lesions.[14,18,19] These findings indicate that further studies are needed to define a dedicated bifurcation stenting strategy using drug-eluting stents, or a new drug-eluting stent platform, to improve outcomes in true bifurcation lesions at the unprotected LMCA.

The present study had several limitations. First, the study population was not large enough to compare the angiographic and intravascular ultrasound results of the stenting strategies with sufficient statistical power. Second, we could not perform complete postprocedural intravascular ultrasound evaluation of the LCx, which was the major restenosis loca-

tion. Third, because of a nonrandomized study design, selection bias that was introduced in choosing strategies might have influenced the outcomes of the 2 stenting strategies. However, the findings are particularly relevant because treatment strategies were selected and applied very stringently based on angiographic and intravascular ultrasound lesion assessments.

1. Park SJ, Lee CW, Kim YH, Lee JH, Hong MK, Kim JJ, Park SW. Technical feasibility, safety, and clinical outcome of stenting of unprotected left main coronary artery bifurcation narrowing. *Am J Cardiol* 2002;90:374–378.
2. Park SJ, Kim YH, Lee BK, Lee SW, Lee CW, Hong MK, Kim JJ, Mintz GS, Park SW. Sirolimus-eluting stent implantation for unprotected left main coronary artery stenosis: comparison with bare metal stent mplantation. *J Am Coll Cardiol* 2005;45:351–355.
3. Sharma SK, Choudhury A, Lee J, Kim MC, Fisher E, Steinheimer AM, Kini AS. Simultaneous kissing stents (SKS) technique for treating bifurcation lesions in medium-to-large size coronary arteries. *Am J Cardiol* 2004;94:913–917.
4. Colombo A. Bifurcational lesions and the "crush" technique: understanding why it works and why it doesn't—a kiss is not just a kiss. *Catheter Cardiovasc Interv* 2004;63:337–338.
5. Lee SW, Park SW, Hong MK, Kim YH, Lee BK, Song JM, Han KH, Lee CW, Kang DH, Song JK, et al. Triple versus dual antiplatelet therapy after coronary stenting: impact on stent thrombosis. *J Am Coll Cardiol* 2005;46:1833–1837.
6. Hong MK, Park SW, Lee CW, Kang DH, Song JK, Kim JJ, Park SJ, Hong MK, Mintz GS, Leon MB. Intravascular ultrasound findings in stenting of unprotected left main coronary artery stenosis. *Am J Cardiol* 1998;82:670–673.
7. Tan WA, Tamai H, Park SJ, Plokker HW, Nobuyoshi M, Suzuki T, Colombo A, Macaya C, Holmes DR Jr, Cohen DJ, et al, for the ULTIMA Investigators. Long-term clinical outcomes after unprotected left main trunk percutaneous revascularization in 279 patients. *Circulation* 2001;104:1609–1614.
8. Takagi T, Stankovic G, Finci L, Toutouzas K, Chieffo A, Spanos V, Liistro F, Briguori C, Corvaja N, Albero R, et al. Results and long-term predictors of adverse clinical events after elective percutaneous interventions on unprotected left main coronary artery. *Circulation* 2002;106:698–702.
9. Silvestri M, Barragan P, Sainsous J, Bayet G, Simeoni JB, Roquebert PO, Macaluso G, Bouvier JL, Comet B. Unprotected left main coronary artery stenting: immediate and medium-term outcomes of 140 elective procedures. *J Am Coll Cardiol* 2000;35:1543–1550.
10. Hu FB, Tamai H, Kosuga K, Kyo E, Hata T, Okada M, Nakamura T, Fujita S, Tsuji T, Takeda S, et al. Intravascular ultrasound-guided directional coronary atherectomy for unprotected left main coronary stenoses with distal bifurcation involvement. *Am J Cardiol* 2003;92:936–940.
11. Arampatzis CA, Lemos PA, Tanabe K, Hoye A, Degertekin M, Saia F, Lee CH, Ruiter A, McFadden E, Sianos G, et al. Effectiveness of sirolimus-eluting stent for treatment of left main coronary artery disease. *Am J Cardiol* 2003;92:327–329.
12. Chieffo A, Stankovic G, Bonizzoni E, Tsagalou E, Iakovou I, Montorfano M, Airoldi F, Michev I, Sangiorgi MG, Carlino M, et al. Early and mid-term results of drug-eluting stent implantation in unprotected left main. *Circulation* 2005;111:791–795.
13. Valgimigli M, van Mieghem CA, Ong AT, Aoki J, Granillo GA, McFadden EP, Kappetein AP, de Feyter PJ, Smits PC, Regar E, et al. Short- and long-term clinical outcome after drug-eluting stent implantation for the percutaneous treatment of left main coronary artery disease: insights from the Rapamycin-Eluting and Taxus Stent Evaluated At Rotterdam Cardiology Hospital registries (RESEARCH and T-SEARCH). *Circulation* 2005;111:1383–1389.
14. Ormiston JA, Currie E, Webster MW, Kay P, Ruygrok PN, Stewart JT, Padgett RC, Panther MJ. Drug-eluting stents for coronary bifurcations: insights into the crushing technique. *Catheter Cardiovasc Interv* 2004;63:332–336.
15. Colombo A, Moses JW, Morice MC, Ludwig J, Holmes DR Jr, Spanos V, Louvard Y, Desmedt B, Di Mario C, Leon MB. Randomized study to evaluate sirolimus-eluting stents implanted at coronary bifurcation lesions. *Circulation* 2004;109:1244–1249.
16. Tanabe K, Hoye A, Lemos PA, Aoki J, Arampatzis CA, Saia F, Lee CH, Degertekin M, Hofma SH, Sianos G, et al. Restenosis rates following bifurcation stenting with sirolimus-eluting stents for de novo narrowings. *Am J Cardiol* 2004;94:115–118.
17. Pan M, de Lezo JS, Medina A, Romero M, Segura J, Pavlovic D, Delgado A, Ojeda S, Melian F, Herrador J, et al. Rapamycin-eluting stents for the treatment of bifurcated coronary lesions: a randomized comparison of a simple versus complex strategy. *Am Heart J* 2004;148:857–864.
18. Takebayashi H, Kobayashi Y, Dangas G, Fujii K, Mintz GS, Stone GW, Moses JW, Leon MB. Restenosis due to underexpansion of sirolimus-eluting stent in a bifurcation lesion. *Catheter Cardiovasc Interv* 2003;60:496–499.
19. Takebayashi H, Kobayashi Y, Mintz GS, Carlier SG, Fujii K, Yasuda T, Moussa I, Mehran R, Dangas GD, Collins MB, et al. Intravascular ultrasound assessment of lesions with target vessel failure after sirolimus-eluting stent implantation. *Am J Cardiol* 2005;95:498–502.