Carina Shift Versus Plaque Shift for Aggravation of Side Branch Ostial Stenosis in Bifurcation Lesions
Volumetric Intravascular Ultrasound Analysis of Both Branches

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Background—Although carina shift and plaque shift are suggested as mechanisms of side branch ostial (SBo) compromise after main vessel (MV) stenting in bifurcation lesions, there are few direct evidence. Our purpose was to confirm the mechanism of SBo compromise after MV stent implantation.

Methods and Results—Intravascular ultrasound images of both MV and SB before procedure and immediately after MV stenting were evaluated in 44 bifurcation lesions. Three 5 mm segments of interest were volumetrically analyzed: the proximal MV, distal MV, and SBo. SBo compromise was defined as a lumen volume decrease, carina shift as a vessel volume decrease, and plaque shift as a plaque volume increase in the SBo segment after MV stenting. The vessel volume increased, and the plaque volume decreased significantly in the proximal MV and distal MV. In contrast, in the SBo, the vessel volume decreased (53.0±17.5 mm³ versus 50.4±16.2 mm³; P<0.001), with the accompanying small increase in plaque volume (23.0±9.8 mm³ versus 23.4±9.8 mm³; P<0.001). SBo compromise was significantly correlated with the carina shift (r=0.941; P<0.001), but not with the plaque shift (r=−0.019, P=0.90). Distal MV lumen volume increase was significantly correlated with SBo compromise (r=0.555; P<0.001) and carina shift (r=0.557; P<0.001), but not plaque shift (r=−0.228; P=0.14).

Conclusions—Our study indicates that carina shift, not plaque shift, is the major mechanism of SBo compromise after MV stent implantation, and the carina shift is primarily influenced by distal MV lumen expansion. (Circ Cardiovasc Interv. 2012;5:657-662.)

Key Words: angioplasty • bifurcation lesions • intravascular ultrasound • carina shift • plaque shift

Coronary bifurcation lesions account for 15% to 20% of coronary angioplasty procedures1,2 and remain a challenge for interventional cardiologists. Currently, the provisional approach of main vessel (MV) stenting followed by optional treatment of the side branch (SB) has become the default approach for most bifurcation lesions.3 During provisional approach, MV stent implantation often aggravates an SB ostial (SBo) stenosis, inducing SBo compromise, which is the most important procedural complication during bifurcation lesion percutaneous coronary intervention. Previously, the major mechanism of this complication was believed to be plaque shift from the MV to the SBo.4 However, intravascular ultrasound (IVUS) and pathological studies have demonstrated that the carina between the MV and SB is usually free of atherosclerotic plaques.5–7 Therefore, recent studies have suggested that carina shift may be a more important mechanism of SBo compromise.8–11 Unfortunately, there have been few systematic studies on relative contribution of carina shift and plaque shift using direct evaluation of SB. The aim of our study was to investigate the mechanism of aggravation of SB ostial stenosis after MV stent implantation in coronary bifurcation lesions by volumetric IVUS analysis of both MV and SBo.

Methods

Patient Population
Patients having a de novo coronary bifurcation lesion treated with provisional SB intervention were enrolled. The inclusion criteria were as follows: (1) a de novo lesion with >50% diameter stenosis in MV; (2) an MV reference diameter (RD) of ≥2.5 mm and an SB RD of ≥2.0 mm by visual estimation; and (3) available IVUS images for both the MV and SB before and immediately after MV stenting but before any SB angioplasty. The exclusion criteria were as follows: (1) no aggravation of SB stenosis after MV stenting by IVUS; (2) a totally occluded lesion; (3) an angiographically visible thrombus; (4) images unsuitable for the analysis, due to severe calcification or severe dissections; (5) the segments not long enough for volumetric analysis; or (6) predilatation of the SB before...
MV stent implantation. SBs without significant stenosis were not excluded from the study.

Study Procedure
Coronary stenting was performed with standard interventional techniques. Preprocedural IVUS examination was attempted before predilatation of any branches. If the stenosis of the MV was too tight to advance the IVUS catheter, predilatation of MV was performed with an undersized balloon prior to IVUS examination. After MV stenting, postdilatation of the MV stent was performed at the discretion of the operator. Thereafter, a second IVUS examination was performed before any SB intervention. All IVUS images were acquired with guidewires in both branches, to avoid the changes of bifurcation angles and the IVUS images. If necessary, SB dilatation and SB stenting were allowed after the poststenting IVUS examination. The Samsung Medical Center’s Institutional Review Board approved this study, and all subjects provided informed consent prior to participation.

Quantitative Coronary Angiography
Baseline and postintervention coronary angiographies were analyzed offline using an automated edge-detection system quantitative coronary angiography system (Intегris H3000, Philips, Hamburg, Germany). The minimum luminal diameter and RD of the MV and SB pre- and poststenting were measured in matched views. In this study, the proximal MV (MVp), distal MV (MVd), and SB were analyzed separately (Figure 1). For the percent diameter stenosis, the proximal RD was used for MVp lesions and the distal RD was used for MVd and SB lesions. The diameter stenosis was calculated by: 100% × (RD—minimum luminal diameter)/RD. Lesion length was measured as the length of contiguous coronary narrowing (defined as percent diameter stenosis >20%).

We measured 2 angles at the bifurcation site: the bifurcation angle and the carina angle. The bifurcation angle (angle A) was measured between the MVd and SB in the angiographic view with the widest angle. Measurement of the carina angle (angle α) is shown in Figure 1, and was performed by first identifying the view displaying the widest opening and without overlap between branches. A line was drawn parallel to the MV axis and through the apex of the carina. Next, a line parallel to the internal contour of the SB and intersecting the first line was drawn; the resulting angle was the carina angle α.

WHAT IS KNOWN
• Side branch compromise is a complication during coronary bifurcation stenting.
• Carina shift and the plaque shift have been considered to be the major mechanisms.

WHAT THE STUDY ADDS
• Systematic intravascular examination demonstrates that carina shift, not plaque shift, is the major mechanism of the side branch compromise in this setting.
• Furthermore, side branch compromise and the carina shift are significantly influenced by stent expansion in the distal main vessel.
• This observation suggests that the risk of side branch compromise may be decreased by implanting the main vessel stent sized to the distal vessel, followed by separate proximal main vessel stent dilation with a larger balloon.

IVUS Imaging and Analysis
The IVUS examinations were performed on the MV and the SB in a standard fashion. All IVUS images were obtained using a commercially available system (Boston Scientific Corporation/Cardiovascular Imaging System, San Jose, CA) consisting of a rotating 40-MHz transducer within a 3.2-F imaging sheath. Before each IVUS run, 200 µg of nitroglycerin was injected into the coronary artery. The catheter was advanced ≈10 mm beyond the lesion, and imaging was performed from there to ≈10 mm proximal to the lesion whenever possible. The transducer was pulled back automatically at a speed of 0.5 mm/s. Ultrasound studies were recorded digitally for offline analysis.

Quantitative IVUS analysis was performed using computerized planimetry (EchoPlaque, Indec System, Mountain View, CA) following the criteria of the American College of Cardiology Clinical Expert Consensus Document on IVUS. The cross-sectional areas of the lumen and the external elastic membrane were measured. The external elastic membrane area was considered as the vessel area. After stenting, the stent area was measured as the lumen area. Plaque area was calculated as the difference between the external elastic membrane area and the lumen area or the difference between the external elastic membrane area and stent area in the postintervention cross-sections. Minimal lumen area was measured at the narrowest luminal cross-section. The reference area was measured at the most normal-appearing cross-section within 10 mm of the lesion without intervening branches.

For volumetric analysis, the IVUS examinations were performed in three 5 mm long segments of interest: MVp (distal 5 mm segment of MVp), MVd (proximal 5 mm segment of MVd), and SBo (proximal 5 mm segment of SB, Figure 1). The carina cross-section was the frame immediately distal to the take-off of the SB in which both ostia of the MVd and SB could be visualized as a figure-8 shape. The starting point of MVp was the frame immediately proximal to the carina obtained from the prestenting IVUS analysis. For each segment of interest, the volumes of the vessel, lumen, and plaque were calculated using these measurements and Simpson’s rule. SBo compromise was defined as a lumen volume decrease, carina shift as a vessel volume decrease, and plaque shift as a plaque volume increase in the SBo segment after MV stenting.

Statistical Analysis
Data are expressed as mean±SD for continuous variables and frequencies for categorical variables. Continuous variables were analyzed using the paired t test or the Wilcoxon signed-rank test. Correlations between volumetric parameters of the 3 segments
were evaluated by Spearman rank correlation analysis. $P$ values were 2-tailed, and $P<0.05$ was considered significant. All analyses were performed using SPSS software, version 15.0 (SPSS Inc, Chicago, IL).

**Results**

**Patient and Lesion Characteristics**

From July 2007 to October 2010, there was a total of 234 de novo coronary bifurcation lesions successfully treated with provisional SB intervention. IVUS images for both the MV and SB before and after MV stenting, but before SB balloononing was available in 84 lesions. Among these, the following lesions were excluded: 25 lesions with no aggravation in SBo; 10 lesions with the IVUS run not long enough for the analysis in MVp segment; 4 lesions with severe SB calcification; and 1 lesion with postintervention SB dissection. Finally, 44 lesions in 44 patients were included in our analysis. In all lesions, all images were available for the analysis of MVp, MVd, and SBo segments both before and after MV stenting. In 12 cases, baseline IVUS for the MV was performed after predilatation (balloon diameter, 2.1±0.3 mm; balloon-to-artery diameter ratio, 0.80±0.13). Clinical, angiographic, and procedural characteristics are shown in Table 1.

**Angiographic Analysis**

Stenosis severity and lesion length were greater in the MVd compared with the MVp (Table 2). The minimum luminal diameter of the MVp and MVd significantly increased after MV stenting. In SBo segment, the minimum luminal diameter decreased significantly after the MV stenting, without a significant change of RD.

**Volumetric IVUS Analysis Before and After MV Stenting**

The vessel and the lumen volume increased, whereas the plaque volumes decreased significantly in both MVp and MVd segments (Table 3). In SBo segment, both the lumen and the vessel volume decreased, whereas the plaque volume slightly increased. Representative images are presented in Figure 2. The increase of the vessel and the lumen volume were greater in the MVd compared with the MVp (16.2±7.1 mm$^3$ versus 11.0±9.6 mm$^3$; $P=0.004$ and 19.5±8.9 mm$^3$ versus 13.8±11.8 mm$^3$; $P=0.020$, respectively), although there was no significant difference in plaque volume change between MVp and MVd (3.3±3.3 mm$^3$ versus 2.8±5.7 mm$^3$; $P=0.61$) (Figure 3). Plaque shift explained 85±23% of SBo compromise.

**Correlations Between Volumetric IVUS Parameters**

The SBo compromise was significantly correlated with the carina shift ($r=0.941$; $P<0.001$) (Figure 4A), but not with the plaque shift ($r=0.019$; $P=0.90$) (Figure 4B). The carina shift was significantly correlated with MVd vessel volume increase ($r=0.463$; $P=0.002$) (Figure 4C), but not with MVp vessel volume increase ($r=0.061$; $P=0.695$). Meanwhile, the plaque shift was significantly correlated with MVp plaque

### Table 1. Clinical and Procedural Characteristics

<table>
<thead>
<tr>
<th>Patient characteristics (n=44)</th>
<th></th>
</tr>
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<tbody>
<tr>
<td>Male</td>
<td>38 (86)</td>
</tr>
<tr>
<td>Age, y</td>
<td>60±9</td>
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<tr>
<td>Diabetes mellitus</td>
<td>11 (25)</td>
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<tr>
<td>Hypertension</td>
<td>23 (52)</td>
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<tr>
<td>Hyperlipidemia</td>
<td>5 (11)</td>
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<tr>
<td>Smoking</td>
<td>13 (30)</td>
</tr>
<tr>
<td>FHX of CAD</td>
<td>7 (16)</td>
</tr>
<tr>
<td>Acute coronary syndrome</td>
<td>10 (23)</td>
</tr>
</tbody>
</table>

### Table 2. Quantitative Coronary Angiographic Analysis of 3 Segments Before and After MV Stenting

$\text{MVp}$ indicates proximal main vessel; $\text{MVd}$, distal main vessel; $\text{SBo}$, SB ostial; $\text{RD}$, reference diameter; and $\text{MLD}$, minimal luminal diameter.

<table>
<thead>
<tr>
<th></th>
<th>MVp (n=44)</th>
<th></th>
<th>MVd (n=44)</th>
<th></th>
<th>SBo (n=44)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>$P$ Value</td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>RD, mm</td>
<td>3.7±0.6</td>
<td>3.9±0.8</td>
<td>0.012</td>
<td>2.7±0.5</td>
<td>2.9±0.6</td>
</tr>
<tr>
<td>MLD, mm</td>
<td>2.0±1.2</td>
<td>3.4±0.7</td>
<td>&lt;0.001</td>
<td>1.0±0.6</td>
<td>2.5±0.8</td>
</tr>
<tr>
<td>Diameter stenosis, %</td>
<td>45±28</td>
<td>12±12</td>
<td>&lt;0.001</td>
<td>64±22</td>
<td>11±20</td>
</tr>
<tr>
<td>Lesion length, mm</td>
<td>5.8±6.3</td>
<td>13.2±9.3</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
volume decrease (r=0.495; P=0.001) (Figure 4D), but not with MVd plaque volume decrease (r=0.095; P=0.54). The MVd lumen volume increase was also significantly correlated with the carina shift (r=0.555; P<0.001) and the SBo compromise (r=0.557; P<0.001), but not with the plaque shift (r=0.228; P=0.14).

**Correlates of the Angles**

Angle A was not correlated with the SBo compromise (r=0.117; P=0.45), the carina shift (r=0.114; P=0.46), or the plaque shift (r=0.168; P=0.28). There was an insignificant trend of correlations between the angle α and the SBo compromise (r=0.263; P=0.084), the angle α and the carina shift (r=0.296; P=0.051), but not between the angle α and the plaque shift (r=-0.039; P=0.80). There was also a trend of correlation between the angle α and the MVd lumen expansion (r=0.286; P=0.059).

**Discussion**

We performed volumetric IVUS analysis of bifurcation lesions, including not only the MV but also the SB, to investigate the mechanism of aggravation of SB ostial stenosis after MV stent implantation. The principal findings of this study were as follows: (1) the carina shift, not the plaque shift, is the major mechanism of SBo compromise after MV stent implantation; (2) the carina shift is primarily influenced by MVd lumen expansion.

**Mechanism of Aggravation of SB Ostial Stenosis**

MV stent implantation often results in the aggravation of SB ostial stenosis. The mechanism of SBo compromise was previously thought to be plaque shift from the MV, the presence of stent struts in the ostium, and SB vasospasm. However, some angiographic studies have demonstrated that a stent-induced increase in lumen diameter of the MVd shifted the carina into the SBo, and this carina shift was the main mechanism of SB ostial narrowing after MV stent implantation. A recent IVUS study indicated that the vessel and lumen volumes increased in both the MVp and MVd, whereas only the plaque volume of the MVp decreased and the plaque volume of the MVd remained unchanged after MV stenting. The authors speculated that the degree of luminal narrowing of an SB after stenting of the MV was a result of carina shift. However, this study was limited as it utilized only indirect evidence of the MV, without analysis of SB volume changes.

In our study, SB vessel volume decrease accounted for 85% of SB lumen volume decrease, much more than the contribution of plaque volume increase. When considering the correlations, the SBo compromise (SB lumen volume decrease) was only correlated with the carina shift (SB vessel volume decrease), but not with the plaque shift (SB plaque volume increase). These findings indicate that carina shift, not the plaque shift, is the major mechanism of SBo compromise after MV stent implantation.

Notably, the MVd lumen volume increase was significantly correlated with the SBo compromise and the carina shift.

### Table 3. The Volumes Measured With IVUS (mm³) Before and After MV Stent Implantation

<table>
<thead>
<tr>
<th></th>
<th>MVp (n=44)</th>
<th></th>
<th>MVd (n=44)</th>
<th></th>
<th>SBo (n=44)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>P Value</td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Vessel volume, mm³</td>
<td>93.9±26.8</td>
<td>104.9±27.1</td>
<td>&lt;0.001</td>
<td>59.2±16.5</td>
<td>75.4±18.8</td>
</tr>
<tr>
<td>Lumen volume, mm³</td>
<td>40.0±19.4</td>
<td>53.8±16.8</td>
<td>&lt;0.001</td>
<td>19.5±7.4</td>
<td>39.0±9.0</td>
</tr>
<tr>
<td>Plaque volume, mm³</td>
<td>53.9±18.5</td>
<td>51.1±17.0</td>
<td>0.002</td>
<td>39.7±15.1</td>
<td>36.4±13.1</td>
</tr>
</tbody>
</table>

**IVUS** indicates intravascular ultrasound; **MVp**, proximal main vessel; **MVd**, distal main vessel; and **SBo**, side branch ostium.

**Figure 2.** Angiographic and intravascular ultrasound (IVUS) images pulled back from side branch to proximal main vessel. This figure shows the angiogram of left anterior descending (LAD)-diagonal bifurcation (left), cross-sectional IVUS image at the level of the carina (middle), and longitudinal IVUS image (right) before (upper) and after (lower) main vessel stent implantation. After stent implantation, the carina (white arrow) was shifted to the side branch side, which resulted in the eccentric luminal narrowing of the side branch ostium. The carina site was plaque free before main vessel stenting, while there was plaque shift from the main vessel to the side branch ostium after stenting. Area change was lumen −1.07 mm², vessel −0.88 mm², and plaque 0.19 mm², separately.
These findings suggest that aggressive stent expansion in the MVd segment may be an important procedural determinant of SBo compromise mostly by the carina shift.

**Clinical Implications**

Carina shift is the major mechanism of SBo compromise and is significantly correlated by the MVd lumen volume increase in MVd segment, which suggests that aggressive stent expansion in the MVd may be the major mechanism of SBo compromise. If MV stent overexpansion in the MVd is avoided,

![Figure 3](image1.png)

**Figure 3.** Absolute volume changes of vessel, lumen, and plaque of each segment. *P* = 0.004: comparison between proximal main vessel (MVp) and distal main vessel (MVd) vessel volume change; †P = 0.02: comparison between MVp and MVd lumen volume change. SB indicates side branch.

![Figure 4](image2.png)

**Figure 4.** Correlations between side branch (SB) lumen volume decrease and vessel volume decrease (A), SB lumen volume decrease and plaque volume increase (B), SB vessel volume decrease and distal main vessel (MVd) vessel volume increase (C), SB plaque volume increase and proximal main vessel (MVp) plaque volume decrease (D), MVd lumen expansion and SB ostial (SBo) compromise (E), and also between MVd lumen expansion and carina shift (F).
the risk of SBo compromise can be decreased. It may be possible to decrease the risk of SBo compromise to implant the MV stent with an optimal size to MVd segment, followed by the proximal optimization of MVp with a larger balloon. For this purpose, the IVUS examination of vessel size in each segment may decrease the risk of SBo compromise.

Study Limitations

There are several limitations to our study. First, because the sample size was relatively small, it is difficult to perform further analysis of lesion characteristics, such as degree of calcification, plaque composition, plaque burden, and clinical characteristics. Second, there may have been a selection bias for mild SBo compromise, as we included only lesions with available IVUS examination for the SB after MV stenting. However, there is no feasible way to study severely compromised lesions after MV stenting. Third, physiological assessment of SB using fractional flow reserve was not performed. Therefore, we could not confirm the functional significance of SBo compromise. Fourth, because there is no clinical follow-up, the relationship between these IVUS findings and long-term clinical outcomes could not be investigated.

Conclusion

Our study suggests that the carina shift, not the plaque shift, is the major mechanism of SBo compromise after MV stent implantation, and the carina shift is primarily influenced by MVd lumen expansion. Careful selection of the stent diameter may decrease the risk of SBo compromise.

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Disclosures

None.

References

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