Sequential Intravascular Ultrasound Characterization of the Mechanisms of Rotational Atherectomy and Adjunct Balloon Angioplasty

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Objectives. The purpose of this study was to use sequential intravascular ultrasound imaging before intervention, after rotational atherectomy and after adjunct balloon angioplasty to characterize the mechanisms of lumen enlargement after each.

Background. Rotational atherectomy uses a high speed, rotating, diamond-tipped elliptic burr to abrade atherosclerotic plaque to increase lumen size. In vitro studies have shown that high speed rotational atherectomy selectively abrades hard, especially calcified, plaque elements. However, rotational atherectomy procedures usually require adjunct balloon angioplasty.

Methods. Forty-eight lesions in 46 patients were treated with rotational atherectomy followed by adjunct balloon angioplasty in 44. Quantitative coronary arteriographic and intravascular ultrasound measurements of the target lesion were made before intervention, after rotational atherectomy and after balloon angioplasty.

Results. Before intervention, target lesion external elastic membrane area measured 17.3 ± 5.9 mm², lumen area measured 1.8 ± 0.9 mm² and plaque plus media area measured 15.7 ± 4.1 mm². After rotational atherectomy, lumen area increased, plaque plus media area decreased, arc of target lesion calcium decreased and 26% of the target lesions had dissection planes. After adjunct balloon angioplasty, external elastic membrane area increased, lumen area increased, plaque plus media area did not change and 77% of the target lesions had dissection planes. Arterial expansion was seen in 80% of lesions. The pattern of dissection plane location, which was predominantly within calcified plaque after rotational atherectomy, became predominantly adjacent to calcified plaque after adjunct balloon angioplasty (p = 0.008).

Conclusions. Sequential intravascular ultrasound imaging shows that high speed rotational atherectomy causes lumen enlargement by selective ablation of hard, especially calcific, atherosclerotic plaque with little tissue disruption and rare arterial expansion. Adjunct balloon angioplasty further increased lumen area by a combination of arterial dissection and arterial expansion, especially of compliant, noncalcified plaque elements.

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images were grouped as follows: before rotational atherectomy, after rotational atherectomy and after adjunct balloon angioplasty. There were 32 men and 14 women with a mean age of 65 ± 10 years. Nineteen patients had an unstable anginal syndrome, whereas 27 had stable angina or silent ischemia on exercise testing.

Arteries treated and imaged were the left main coronary artery in 1, left anterior descending coronary artery in 23, left circumflex coronary artery in 12 and right coronary artery in 12. Target lesion location was ostial in 8, proximal in 21, midvessel in 18 and distal in 1. The rotational atherectomy procedure and intravascular ultrasound imaging protocols have the ongoing approval of the Washington Hospital Center Investigational Review Board.

Rotational atherectomy procedure. Details of the rotational atherectomy procedure have been described elsewhere (5,6,13). The largest final burr used was chosen to be 70% to 80% of the diameter of the reference vessel and most commonly was a 2.15-mm burr; a 2.50-mm burr (the largest coronary artery burr) was used in eight patients. Adjunct balloon angioplasty was performed if, angiographically, there was haziness, irregularity or residual stenosis >30%. The balloon diameter most commonly used was 3 mm. The largest balloon used was 4 mm.

Angiographic analysis. Quantitative coronary angiography was performed by an independent core angiographic laboratory using an automated edge detection algorithm (ARTREK, Quantitative Cardiac Systems). Lesion length, minimal lumen diameter and percent diameter stenosis before intervention, after rotational atherectomy and after adjunct balloon angioplasty were measured from orthogonal projections, and the results from the "worst" view were recorded. The presence or absence of calcium by fluoroscopy was noted. The presence or absence of dissections was noted.

Intravascular ultrasound imaging system. Intravascular ultrasound studies were performed using a commercially available system (InterTherapy Inc.). This imaging system incorporated a single-element 25-MHz transducer and an angled mirror mounted on the tip of a flexible shaft that was rotated at 1,800 rpm within a 3.9F (1.3 mm) short monorail polyethylene imaging sheath to form planar cross-sectional images in real time and that was withdrawn automatically at 0.5 mm/s. By using a reproducible landmark (usually the aortoostial junction or a large proximal side branch) and a known pullback speed, identical cross-sectional slices could be obtained and compared on serial studies.

There were no immediate complications of ultrasound imaging (including imaging before intervention), rotational atherectomy or adjunct balloon angioplasty. Transient chest pain was relieved by intracoronary nitroglycerin.

Qualitative and quantitative intravascular ultrasound criteria. Qualitative evaluation of plaque morphology and quantitative measurements of external elastic membrane cross-sectional area, residual lumen cross-sectional area, plaque plus media cross-sectional area and minor and major axis lumen diameters have been validated previously in vitro and in vivo (14–22). Plaque morphology was assessed for the presence and extent of calcification. Calcification produced echoes that were brighter than the reference external elastic membrane and were associated with acoustic shadowing. Because of acoustic shadowing, the thickness (and, therefore, cross-sectional area) of a calcified deposit could not be measured; therefore, calcium was quantified by measuring its total circumference (expressed as the arc of calcium in degrees) using a protractor centered on the imaging sheath (23); the reproducibility of the arc of calcium measurement is ±5º.

The use of intravascular ultrasound to detect dissections has also been validated (24). A dissection was an abrupt, focal interruption in the continuity of the plaque or intima that extends axially, radially or circumferentially, spanning normal tissue planes. Detection of dissections was facilitated by axial movement of the catheter by the motorized pullback device. Dissections were categorized as originating within the calcified deposit, if the most proximal or distal point of the dissection was surrounded on either side by calcium, or adjacent to the calcified deposit, if the most proximal or distal point of the dissection occurred at the junction of calcium and softer plaque elements.

Using computerized planimetry, the external elastic membrane cross-sectional area, residual lumen cross-sectional area and major and minor lumen diameters were measured at the lesion site. Plaque plus media cross-sectional area was calculated by subtracting the residual lumen area from the external elastic membrane cross-sectional area. Percent cross-sectional area narrowing was calculated by dividing the plaque plus media cross-sectional area by the external elastic membrane cross-sectional area and multiplying by 100. For long lesions, the smallest lumen area associated with the largest circumferential arc of calcium before intervention was chosen as the lesion site to be measured. The exact same cross-sectional slice was analyzed before intervention and after each subsequent intervention, and the results were compared. Arterial expansion was defined as an increase in external elastic membrane cross-sectional area >0.5 mm² after rotational atherectomy (vs. that before intervention) or after adjunct balloon angioplasty (vs. that after rotational atherectomy).

Although acoustic shadowing caused by lesion calcification tended to make measurements of external elastic membrane cross-sectional area difficult, the use of the motorized pullback device helped (13). Briefly, because the cross section of the coronary artery was more or less circular, extrapolation of the circumference of the external elastic membrane was possible provided that each calcific deposit did not shadow >60º of the adventitial circumference. Also, real-time axial movement of the transducer just distal and proximal to a calcific deposit (or to find the smallest circumferential arc of calcium within a large calcific deposit), unmasked and filled in contiguous parts of the adventitia that were otherwise shadowed by that deposit (Fig. 1). This
permitted extrapolation of the external elastic membrane cross-sectional area in all but five lesions.

Statistics. Statistical analysis was performed using StatView 4.01. Data are presented as mean value ± 1 SD. Two by two comparisons were performed using chi-square analysis. Comparisons between groups were performed using a paired Student t test. Comparison among groups was performed using analysis of variance for repeated measures, with post hoc analysis using the Fisher protected least significant difference (StatView SE).

Results

Lesion characteristics before rotational atherectomy. Angiographic findings. The minimal lumen diameter before intervention was 0.73 ± 0.39 mm, and the percent diameter stenosis was 70.7 ± 15.1%. Lesion length was 9.2 ± 4.9 mm (range 1.5 to 27.6). Fluoroscopic calcification was present in 33 lesions (69%). One lesion had a dissection by both angiography and intravascular ultrasound that was present before any intervention. The patient with this lesion had unstable angina, and the dissection was thought to be spontaneous. No further dissections occurred in this patient. No patient had an intracoronary thrombus.

Intravascular ultrasound findings. Before intervention, the lesion site external elastic membrane cross-sectional area was 17.3 ± 5.9 mm², the lumen cross-sectional area was 1.8 ± 0.9 mm², and the plaque plus media cross-sectional area was 15.7 ± 5.9 mm² (Table 1). The minor axis lumen diameter was 1.2 ± 0.2 mm, and the major axis diameter was 1.5 ± 0.3 mm. The percent cross-sectional area narrowing was 89.3 ± 5.5%.

By intravascular ultrasound, 44 (92%) of 48 sites were calcified (p = 0.0048 vs. angiography); the arc of calcium measured 227 ± 10°. Despite the frequent history of unstable angina, no patient had a suggestion of an intracoronary thrombus.

Lesion characteristics after rotational atherectomy. Angiographic findings. The percent diameter stenosis after atherectomy decreased to 36.8 ± 14.5%, and the minimal lumen diameter increased to 1.52 ± 0.37 mm (p < 0.0001 for each vs. before intervention). There were three dissections without ischemic consequences.

Intravascular ultrasound findings. Lesion site external elastic membrane cross-sectional area (16.6 ± 5.0 mm²) was unchanged after rotational atherectomy compared with that before intervention (p = NS) (Table 1). Residual lumen cross-sectional area increased to 3.8 ± 1.0 mm², and plaque

Table 1. Quantitative Intravascular Ultrasound Results

<table>
<thead>
<tr>
<th></th>
<th>Before (n = 39)</th>
<th>After (n = 43)</th>
<th>After PTCA (n = 39)</th>
<th>p Value (ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEM CS area (mm²)</td>
<td>17.3 ± 5.9</td>
<td>16.7 ± 4.8</td>
<td>18.8 ± 5.6</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Lumen CS area (mm²)</td>
<td>1.8 ± 0.9</td>
<td>3.9 ± 1.1</td>
<td>5.2 ± 1.2</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Plaque + media CS area (mm²)</td>
<td>15.7 ± 4.1</td>
<td>13.0 ± 4.7</td>
<td>13.7 ± 5.0</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Percent CS narrowing</td>
<td>89.3 ± 5.5</td>
<td>74.4 ± 15.0</td>
<td>71.3 ± 7.4</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Minor diameter (mm)</td>
<td>1.4 ± 0.2</td>
<td>2.0 ± 0.3</td>
<td>2.2 ± 0.3</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Major diameter (mm)</td>
<td>1.5 ± 0.3</td>
<td>2.3 ± 0.4</td>
<td>2.7 ± 0.4</td>
<td>&lt; 0.0001</td>
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</table>

Values presented are mean values ± SD. ANOVA = analysis of variance; CS = cross-sectional; EEM = external elastic membrane; PTCA = coronary balloon angioplasty; RA = rotational atherectomy.
plus media cross-sectional area decreased to 12.8 ± 4.9 mm² (p < 0.0001 for both vs. before intervention). The minor axis diameter increased to 2.0 ± 0.2 mm (p < 0.0001 vs. before intervention), and the major axis diameter increased to 2.2 ± 0.3 mm (p < 0.0001 vs. before intervention). Especially in lesions with circumferential calcification before atherectomy, the lumen was circular, and the intima/lumen interface was smooth after rotational atherectomy.

Expansion was detected by intravascular ultrasound in 6 (26%) of 23 lesion sites in which a change in external elastic membrane cross-sectional area could be measured. Dissections were detected by intravascular ultrasound in 17 of 43 sites and averaged 0.6 ± 0.9 dissections per vessel. The average depth of dissection was 0.7 ± 0.2 mm, the circumference was 77 ± 47° and the length was 2.7 ± 2.9 mm. Dissections originated within a calcific deposit in 12 lesions (Fig. 2), adjacent to a calcific deposit in 1 lesion and both within and adjacent to a calcific deposit in 4 lesions.

The arc of calcium decreased significantly after atherectomy to 209 ± 107° (p = 0.048 vs. before intervention). In some there was full thickness calcium removal (Fig. 3). However, significant calcium ablation also occurred in the absence of a measurable decrease in the arc of calcium. First, an increase in lumen cross-sectional area in a target lesion with circumferential superficial calcification that remained circumferential could only have occurred because of calcium removal; this was seen in four lesions (Fig. 4). Second, removal of superficial calcific elements uncovered deeper calcium deposits, with no change in the overall arc of calcium in 11 lesions. Third, images obtained after rotational atherectomy in seven lesions showed deeper adventitial structures in much more detail than was present on images before intervention (Fig. 3), suggesting that removal of some finite thickness of calcium permitted deeper penetration of the ultrasound beam.

Lesion characteristics after adjunct balloon angioplasty. Angiographic findings. Final postprocedural minimal lumen diameter increased to 2.01 ± 0.44 mm, and percent diameter stenosis decreased to 19.5 ± 11.9% (p < 0.0001 for each vs. before intervention). Four additional dissections were noted angiographically (p = NS), also without ischemic consequences.

Intravascular ultrasound findings. Lesion site external elastic membrane cross-sectional area after adjunct balloon angioplasty increased to 18.8 ± 5.6 mm², and residual lumen cross-sectional area also increased to 5.2 ± 1.2 mm² (p < 0.001 and p < 0.0001 for each, respectively, vs. after atherectomy); plaque plus media cross-sectional area did not change (13.7 ± 5.0 mm², p = NS) (Table 1). Minor axis diameter increased to 2.2 ± 0.3 mm (p < 0.0001 vs. after atherectomy). The portion of the vessel that was normal or that was occupied by softer plaque elements expanded preferentially (Fig. 5). Arterial expansion was noted in 24
(80%) of 30 lesion sites in which a change in external elastic membrane cross-sectional area could be measured (p < 0.0001 vs. after rotational atherectomy). In vessels with circumferential calcification and a circular lumen after rotational atherectomy, the lumen remained circular after adjunct balloon angioplasty with displacement of calcified elements (Fig. 4 and 6).

Dissections occurred in 30 (77%) of 39 sites after adjunct balloon angioplasty (p = 0.0006 vs. after rotational atherectomy), and there were significantly more dissections per site, 1.8 ± 1.5 (p < 0.0001 vs. after atherectomy). Twenty-one of these 30 sites had new dissection planes; 9 others had an increase in the number of dissection planes. The depth (0.7 ± 0.2 mm), length (3.3 ± 0.4 mm) and circumferential extent (74 ± 41°) were not significantly different after adjunct balloon angioplasty than after rotational atherectomy. However, dissections originated within a calcified deposit in 9 target lesions, adjacent to a calcific deposit in 14 target lesions (Fig. 7) and both within and adjacent to calcium in 7 target lesions (Table 2) (p = 0.008 vs. after atherectomy).

Dissection and arterial expansion commonly occurred together after adjunct balloon angioplasty. Of the 30 sites imaged after rotational atherectomy and after adjunct balloon angioplasty in which both the presence of dissections and arterial expansion could be assessed, 21 (70%) had both.

Discussion

High frequency intravascular ultrasound provides high quality tomographic images of the coronary artery lumen, lumen-intima interface, atherosclerotic plaque and vessel wall in vivo. Several studies have documented its validity for assessments of plaque morphology (18,19,21,22) and for measurements of lumen cross-sectional area, external elastic membrane cross-sectional area and plaque plus media cross-sectional area (14–18,20–22). It has proved to be useful for evaluating results of various transcatheter therapies, including balloon angioplasty (7,9–12) and directional (8) and rotational atherectomy (13). Intravascular ultrasound images acquired before intervention are essential for the accurate assessment of changes in plaque and vessel wall morphology that occur in direct response to intervention. Using the 3.9F imaging catheter, we routinely obtain intravascular ultrasound images in our patients with angiographically severe stenoses before rotational atherectomy (or other interventions) without adverse clinical sequelae.

Similarly, sequential imaging is essential for separating primary device effect from adjunctive balloon effect. The motorized pullback device produces contiguous image slices that are 17 µm apart. By always pulling the ultrasound catheter back to an easily reproducible landmark (e.g., the...
Figure 6. Left anterior descending coronary artery anatomic cross section before rotational atherectomy (pre RA), after rotational atherectomy (post RA) and after adjunct balloon angioplasty (post PTCA). Both before and after rotational atherectomy with a 2.5-mm burr there is circumferential calcification. Significant lumen enlargement could have occurred only with partial thickness calcium ablation. After adjunct balloon angioplasty, the circular lumen was preserved, and there was uniform displacement of the calcified elements.

aortoostial junction), we can identify and compare the exact same tomographic slice before and after each intervention. This allows us to assess morphologic changes due to that intervention and make accurate comparative measurements.

Using intravascular ultrasound sequentially (before intervention, after rotational atherectomy and after adjunct balloon angioplasty), we showed that rotational atherectomy increases coronary artery lumen cross-sectional area in heavily calcified target lesions primarily by the removal of atherosclerotic plaque with selective ablation of calcific elements. Adjunct balloon angioplasty further increases lumen area by a combination of vessel wall expansion and plaque dissection without further reduction in plaque mass.

Baseline target lesion characteristics. The patients treated successfully with rotational atherectomy in our study had stenoses that were angiographically severe (minimal lumen diameter before intervention 0.73 ± 0.39 mm), moderately long (9.2 ± 4.9 mm) and complex. The severity, length and complexity of these target lesions before intervention were comparable to those reported in other studies using angiography to evaluate the effect of rotational atherectomy (4-6).

The majority of target lesion sites were heavily calcified both by angiography (69%) and ultrasound (92%). Heavily calcified target lesion sites often respond to balloon angioplasty with dissection or insufficient dilation; directional atherectomy has limited utility in heavily calcified target lesions owing to its inability to cut calcium. At our institution, patients with heavily calcified target lesions are treated preferentially with high speed rotational atherectomy.

Mechanism of rotational atherectomy. From histologic studies, it has been postulated that the rotational atherectomy burr is deflected by soft atheroma, whereas it selectively pulverizes "harder" plaque elements (especially calcium) to produce a smooth lumen-atheroma interface (1,2). In our study, plaque mass (plaque plus media cross-sectional area as determined by intravascular ultrasound) decreased significantly after high speed rotational atherectomy (from 15.7 ± 4.1 to 13.0 ± 4.7 mm², p < 0.0001). However, despite a lumen with a cross-sectional area that averaged 3.9 mm² after rotational atherectomy, there remained a significant residual plaque and a percent cross-sectional area narrowing averaging 74%. In part, this significant residual disease occurs because at our institution the final burr size is chosen to be 70% to 80% of the reference vessel angiographic lumen to prevent complications. Whether more aggressive plaque removal with a larger burr would be preferable is not known.

Intravascular ultrasound also provides evidence that rotational atherectomy selectively removes calcium. The mean arc of calcium decreased significantly after high speed rotational atherectomy (p = 0.048), and 32% of lesions had >30°
reduction in the arc of calcium. Because calcium shadows
structures deep to it, the thickness (and cross-sectional area)
of a specific calcific deposit cannot be measured; therefore,
only full thickness calcium removal results in a measurable
decrease in the arc of calcium. However, there can be partial
thickness decalcification without a reduction in the total arc
of calcium. This is suggested by intravascular ultrasound
images acquired after rotational atherectomy in 64% of the
lesions that showed more deep plaque or adventitial detail
than before intervention. In addition, 17% of the lesions had
circumferential superficial target lesion calcification that
remained circumferential after rotational atherectomy de-
spite lumen enlargement; this could only have occurred with
partial thickness calcium removal.

In our study, dissections due to rotational atherectomy
alone were infrequent, occurring in 6% of lesions by angiog-
raphy and 26% by ultrasound. This is similar to the 29% inci-
dence of dissection detected by ultrasound after rota-
tional atherectomy reported previously (15) and is a much
lower incidence than occurs after primary balloon angiop-
asty of calcified target lesions (12). When plaque disruption
does occur after rotational atherectomy, the dissection plane
most often originates within a calcified deposit, not adjacent
to it. This is distinctly different from dissections due to
angioplasty that originate at the junction of calcified and
noncalcified plaque (11).

After rotational atherectomy, only 6 of 23 lesions had
arterial expansion. Thus, arterial expansion, one of the
markers of the "Dotter" effect or of balloon angioplasty-
induced barotrauma, is unusual after rotational atherectomy
alone.

**Mechanism of adj ced balloon angioplasty.** Waller (25)
described three potential mechanisms of lumen enlargement
due to balloon angioplasty: 1) compression of soft plaque
elements, 2) fracture of the plaque and 3) stretching of
normal arterial wall or of the more elastic plaque elements.
We assessed the contributions of each of these three poten-
tial mechanisms to successful adjunct balloon angioplasty
after rotational atherectomy.

**After adjunct balloon angioplasty, the residual plaque
mass did not differ from that after atherectomy. Therefore,
plaque compression does not play a role in the lumen
enlargement resulting from adjunct balloon angioplasty.**

Dissection detected by intravascular ultrasound occurs in
significantly more sites (77% vs. 26%, p = 0.0004) after
adjunct balloon angioplasty than after rotational atherect-
omy. The 77% of calcified sites that dissected is similar to
the 66% incidence of dissection after primary balloon angiop-
asty of calcified target lesions reported by Honve et al. (10)
and the 73% incidence reported by Potkin et al. (12). How-
ever, the distribution of dissection location after ad-
junct balloon angioplasty is distinctly different from the
distribution of dissections after either rotational atherectomy
or angioplasty alone (11). Although dissections due to rota-
tional atherectomy originate within a calcific deposit, addi-
tional dissections after adjunct balloon angioplasty occur
both within and adjacent to calcium (p = 0.008). Rotational
atherectomy produces microscopic fissures within calcium
deposits (1). This "fissuring" may "weaken" the deposit
and promote intraplaque fracture after balloon angioplasty
barotrauma. Additionally, like primary angioplasty, adjunct
balloon angioplasty also results in dissections adjacent to
calcific deposits, presumably as a result of shear forces
(26,27). Using intravascular ultrasound to evaluate the role
of calcium deposits in dissection after primary balloon angiop-
asty, Fitzgerald et al. (11) found that dissections
commonly occur immediately adjacent to focal calcification
and originate infrequently at other locations. High tensile
stress is present at the junction of atherosclerotic plaque
elements having different elastic properties (26,27).

Distension or expansion of angiographic "plaque-free"
wall segments in eccentric stenoses is thought to be an
infrequent mechanism of lumen enlargement after balloon
angioplasty (25). By comparing the target lesion to a prox-
imal reference site, previous studies using intravascular
ultrasound to analyze the effects of balloon angioplasty alone
have demonstrated global arterial expansion. However, pre-
vious studies could not demonstrate eccentric (or localized)
expansion because imaging before intervention was not
routinely performed (13,14). In this study the external elastic
membrane cross-sectional area at the lesion site is larger
after adjunct balloon angioplasty than after rotational
atherectomy in 80% of lesions (p < 0.001); this represents
actual stretching of the vessel wall at the lesion site. Examin-
ation of individual images reveals eccentric distension of
more compliant plaque elements in lesions with no circum-
fugal calcification but more concentric expansion in
lesion sites with circumferential calcification. Diffuse micro-
fissuring of the calcium by the burr may permit more even
circumferential distribution of radial shear stresses and
result in uniform displacement of individual calcific elements
(Fig. 4 and 6).

Commonly, distension of compliant plaque elements and
arterial dissection occur together as the arterial response to
adjunct balloon angioplasty; 70% of lesion sites have con-
comitant dissection and arterial expansion. After rotational
atherectomy alone, only 11% exhibited both plaque dissec-
tion and vessel wall expansion.
Intravascular ultrasound versus quantitative angiography:
The minimal lumen diameter measured by quantitative angiography was smaller than that measured by intravascular ultrasound both before intervention and after rotational atherectomy (both p < 0.0001) and after adjunct balloon angioplasty (p < 0.0003). Because the diameter of the ultrasound catheter is 3.9F, all lumen diameters equal to or smaller than this are reported as 1.3 mm to produce misleadingly large mean ultrasound lumen diameters. This is only important in imaging before intervention, when lumens are smaller than the imaging catheter. Alternatively, quantitative coronary angiography may underestimate lumen diameters compared with intravascular ultrasound; however, this is probably not a significant factor because after adjunct balloon angioplasty, minimal lumen diameters determined by the two techniques were not significantly different.

Study limitations. There are several limitations to this study. 1) We evaluated the effect of rotational atherectomy only on heavily calcified arteries. Noncalcified lesions may respond differently to the burr. 2) Not all patients were imaged before intervention, after rotational atherectomy and after adjunct balloon angioplasty. However, the results of subgroup analysis of only patients studied serially before and after each of the interventions did not differ significantly from larger group analysis. 3) Ultrasound images before intervention were obtained at sites with angiographic diameters that were smaller than that of the ultrasound catheter; a “Dotter” effect by the transducer itself might have affected interpretation of these images. However, it is unlikely that these heavily calcified target lesions would be significantly altered by the small catheter. 4) Measurement of external elastic membrane cross-sectional areas is difficult in heavily calcified coronary arteries. Careful movement of the transducer in small increments (possible with the motorized pullback device) unmasks contiguous portions of the external elastic membrane otherwise shadowed by calcium. Nevertheless, we were unable to measure external elastic membrane area in five lesion sites.

Conclusions. Sequential intravascular ultrasound imaging is the only technique that can directly assess the mechanism by which percutaneous transcatheter devices achieve increases in lumen cross-sectional area in vivo. This approach is also necessary for studying the interactions of multiple transcatheter therapies. Using new, smaller intravascular ultrasound transducers, we can obtain high quality tomographic images of the coronary arteries before intervention without any adverse clinical outcomes in the majority of patients. By comparing serial intravascular ultrasound images, we can separate the distinct arterial responses and mechanisms of lumen enlargement after rotational atherectomy from those produced by adjunct balloon angioplasty. Rotational atherectomy removes atherosclerotic plaque with selective ablation of fibrous and calcific elements and without extensive plaque disruption or arterial expansion. Further increases in lumen cross-sectional area produced by adjunct balloon angioplasty are a result of combined dissection and expansion of more compliant plaque elements without further atheroma ablation or plaque compression.

References