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Prominent Role of Tensile Stress in Propagation of a Dissection After Coronary Stenting
Computational Fluid Dynamic Analysis on True 3D-Reconstructed Segment

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A 61-year-old man underwent direct stenting in the mid right coronary artery with a 3.5×18-mm stent deployed at a pressure of 14 atm. Without further balloon inflation, the control angiogram showed a stepdown-like pattern at the distal edge of the stent (Figure 1A). At that site, intravascular ultrasound (IVUS) imaging (see Movie, available at http://www.circulationaha.org) showed a ruptured plaque and a minor dissection. Shortly after IVUS, the dissection extended distally and became subocclusive (Figure 1, B and C). Deployment of 2 additional stents arrested propagation of the dissection. The final angiogram showed a satisfactory result, with TIMI 3 flow. Despite the importance of edge dissection, the mechanism of propagation in coronary arteries has not been described. It seems reasonable that the pressure force induced by flow on a dissected flap would stimulate propagation. To study this flow-induced force, the coronary artery lumen and wall were reconstructed in true 3D (Figure 2A) by fusion of angiographic and IVUS data. Application of computational fluid dynamics to the reconstruction visualized the pressure development in the axial direction (Figure 2B) as well as that over the cross sections surrounding the protruding flap (Figure 2C). The pressure difference found between the intimal and luminal sides of the flap is ≈1 mm Hg. In contrast, with this low value, greater local changes in pressure and associated force can be deduced from the Laplace equation applied to the changing morphology of the dissected vessel wall. Normally, the tensile force exerted by the fibrous cap and thickened intima creates a significant pressure drop from the lumen to the subintimal space. This pressure drop is proportional to the ratio of intima thickness to wall thickness, eg, for respective thicknesses of 0.2 mm and 1 mm, this amounts to one fifth of luminal pressure. In case of an intimal rupture, blood enters the subintimal space. This eliminates the lumen-to-subintima pressure drop and results in retraction of the inner vessel wall layers. Moreover, the outer wall layers then become exposed to the full blood pressure and expand (Figure 2A). Both effects lead to propagation of the dissection. A similar mechanism has been described for aortic dissection. Indeed, this also explains retrograde expansion of a dissection, as is frequently observed after balloon dilatation. Nevertheless, a major flow-related difference between retrogradely or distally propagating dissections occurs when the dissection spreads circumferentially over >180°. Then, only for distally propagating dissections, the flow-induced pressure difference becomes important, because this forces the flap to flip over, thus causing a total vessel occlusion.
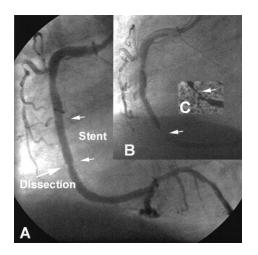
Figure 1. A, Angiographic left anterior oblique view of right coronary artery showing implanted 3.5×18-mm stent (arrows) and marked indentation ("stepdown") at distal edge with good distal flow. B, Suboclusive dissection with TIMI 2 flow shortly after IVUS pullback. C, Improved visualization of suboclusion. Animated version: ECG-gated motorized pullback IVUS recording (0.5 mm/step) (ClearView, CVIS, Boston Scientific Corp, with UltraCross 30-MHz imaging catheter) shows cross sections of artery from 3 mm downstream of distal edge of stent to 1 mm within stented segment. IVUS image shows a large fibrotic cap between 2 o'clock and 7 o'clock positions, dissected over a 1.5-mm length in longitudinal direction (arrow). Behind flap, motion of blood in cavity is visualized by moving speckles. Also, contrast remnants (black) can be recognized at 3 o'clock position.
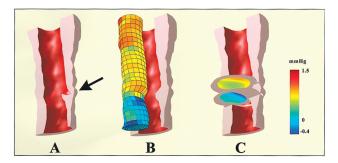

Figure 2. A, 3D reconstruction of lumen (red) and wall of stented and dissected part of right coronary artery. Stent (not visualized) is immediately proximal (top of reconstruction) to dissection. Outer surface represents external elastic membrane. Opened subintimal space was manually edited into reconstruction for didactic purposes. Some local bulging of outer vessel wall after dissection can be recognized (arrow). B, Luminal mesh for application of computational fluid dynamics to solve Navier-Stokes equations and to derive from these spatial luminal pressure values. Pressure is calculated relative to exit cross section (bottom) and shown color coded at luminal wall. Total pressure drop over segment was ≈1.5 mm Hg. For entrance flow condition, a high mean velocity of 40 cm/s was selected to avoid underestimation of flow-induced pressure effects. Blood was modeled as a Newtonian fluid with a viscosity of 3.5×10^{-3} Pa · s. C, Two cross-sectional planes are indicated at 1-mm distance, 1 at distal stent edge and 1 at minimal lumen cross-sectional area caused by flap protruding into lumen. Color-coded planes show pressure distribution over lumen cross sections. A small local maximum in pressure (0.8 mm Hg) at medial entrance of dissection can be observed. With current mesh, pressure in subintimal space behind flap could not be calculated, but it may be assumed that this pressure nearly equals that at dissection entrance regarding subintimal low-flow-velocity conditions. Interestingly, a negative pressure (−0.4 mm Hg) has developed at luminal side of flap because of flow acceleration (Venturi effect). Resulting maximal difference in pressure between both sides of flap is only ≈1 mm Hg.