Combined use of imaging and computational techniques to investigate fluid dynamics in stented coronary bifurcations

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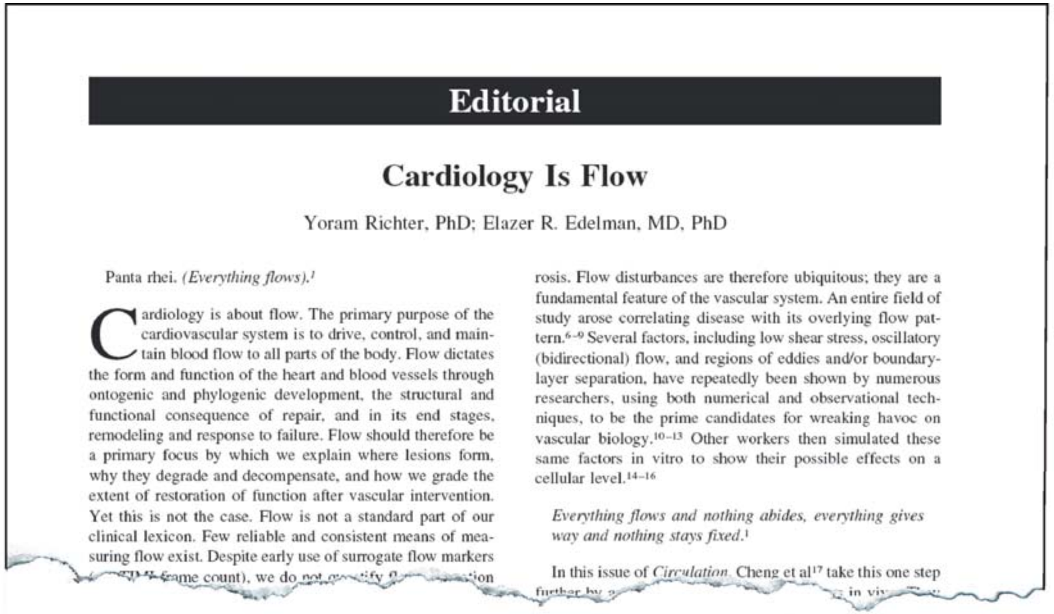
Plaque tends to form locally at:

- Bends
- Branches
- Bifurcations

We must remember that …



Editorial

Cardiology Is Flow

Yoram Richter, PhD; Elazer R. Edelman, MD, PhD

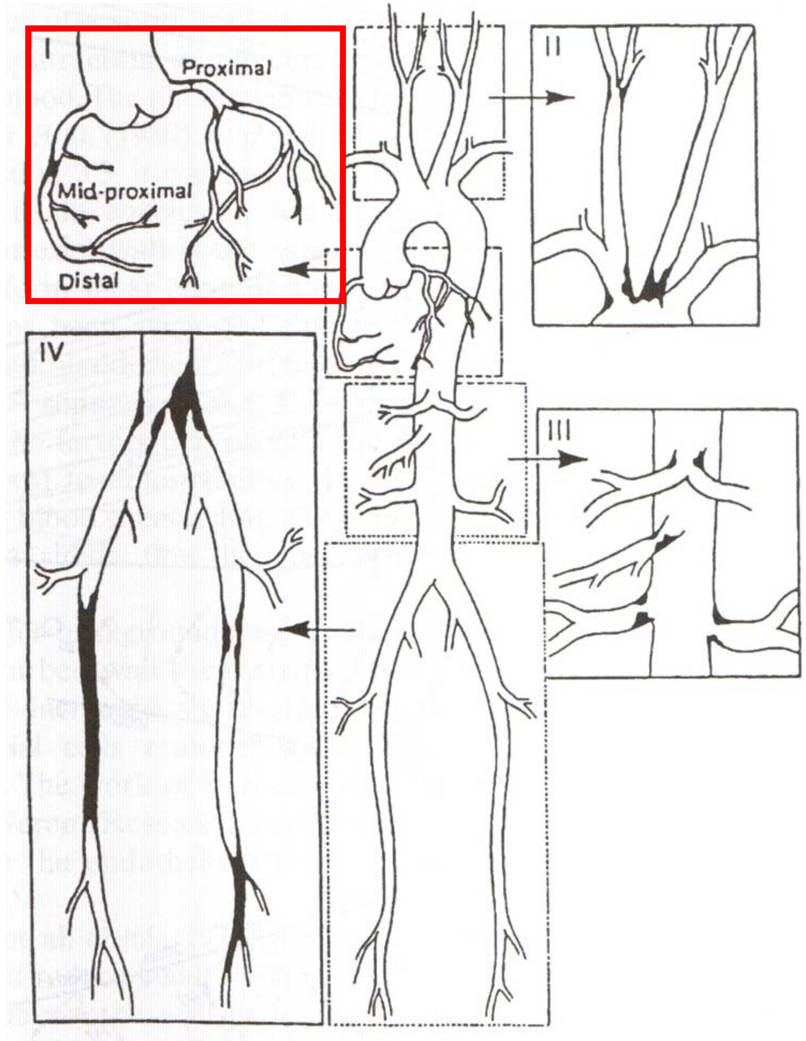
Panta rhei. *(Everything flows).*[1]

Cardiology is about flow. The primary purpose of the cardiovascular system is to drive, control, and maintain blood flow to all parts of the body. Flow dictates the form and function of the heart and blood vessels through ontogenic and phylogenic development, the structural and functional consequence of repair, and in its end stages, remodeling and response to failure. Flow should therefore be a primary focus by which we explain where lesions form, why they degrade and decompensate, and how we grade the extent of restoration of function after vascular intervention. Yet this is not the case. Flow is not a standard part of our clinical lexicon. Few reliable and consistent means of measuring flow exist. Despite early use of surrogate flow markers

rosis. Flow disturbances are therefore ubiquitous; they are a fundamental feature of the vascular system. An entire field of study arose correlating disease with its overlying flow pattern.[6–9] Several factors, including low shear stress, oscillatory (bidirectional) flow, and regions of eddies and/or boundary-layer separation, have repeatedly been shown by numerous researchers, using both numerical and observational techniques, to be the prime candidates for wreaking havoc on vascular biology.[10–13] Other workers then simulated these same factors in vitro to show their possible effects on a cellular level.[14–16]

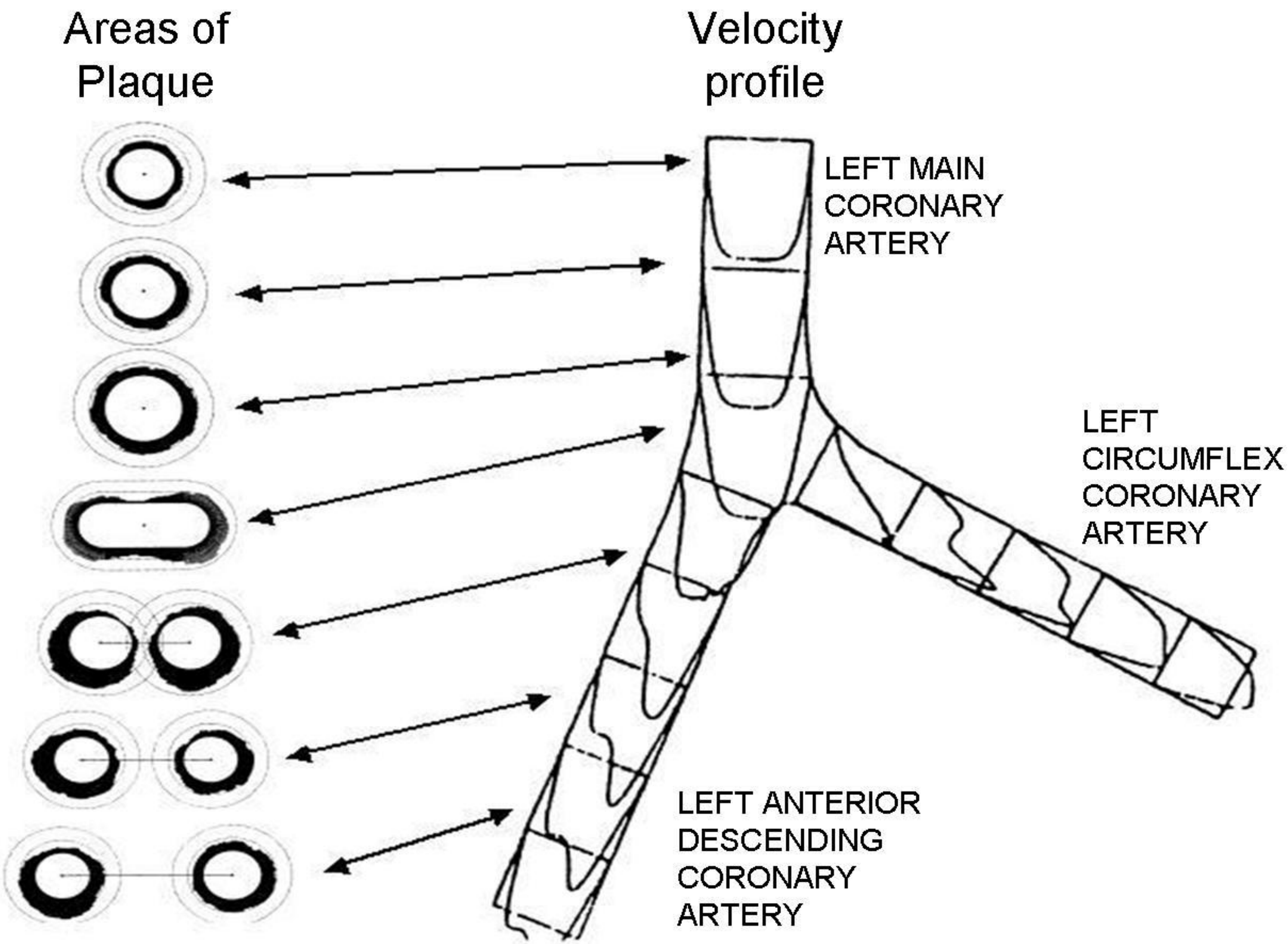
Everything flows and nothing abides, everything gives way and nothing stays fixed.[1]

In this issue of *Circulation*, Cheng et al[17] take this one step



Fung Y. Biomechanics: Mech. Prop. Living Tissues. 1993

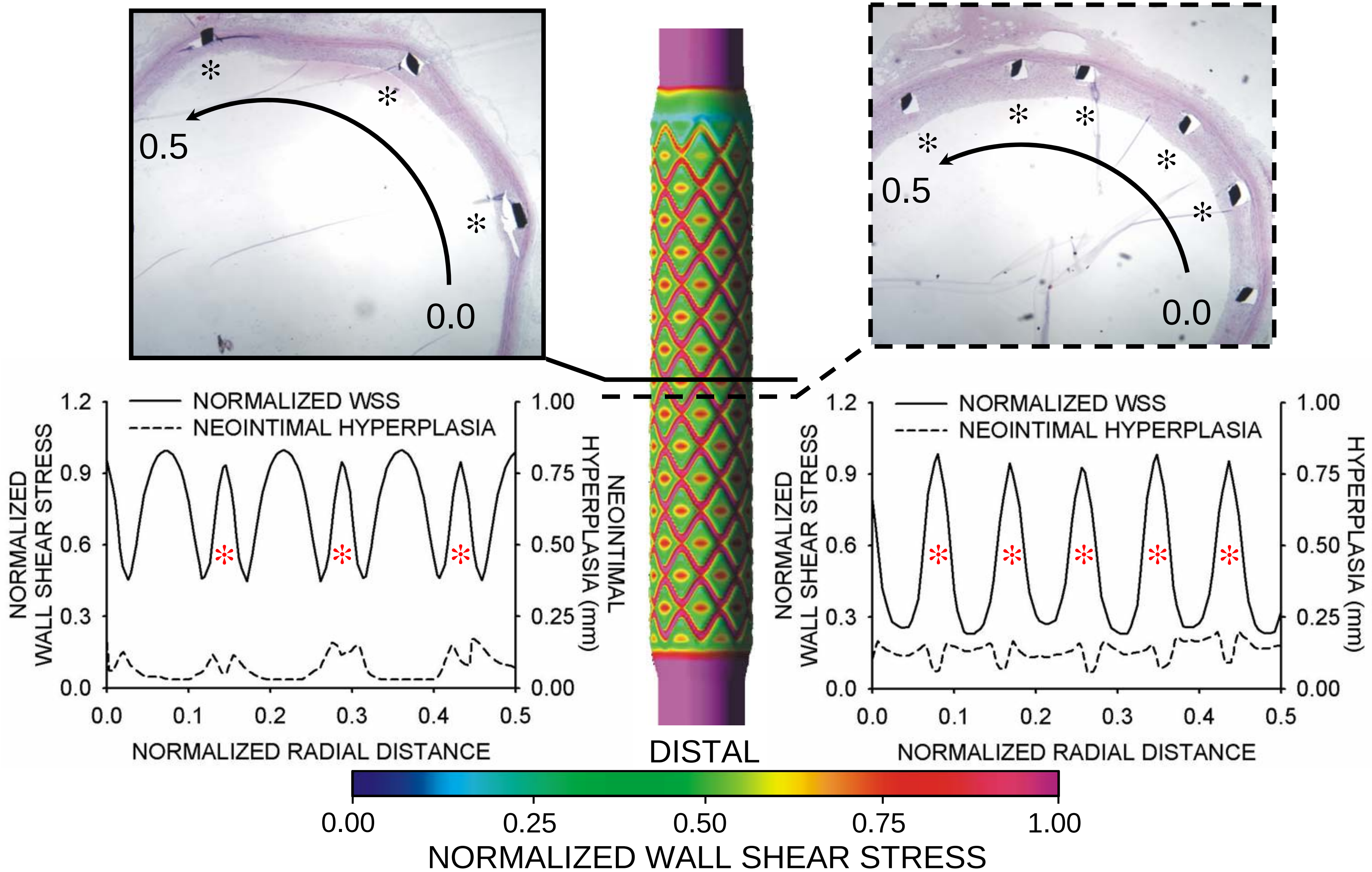
Hemodynamics in coronary artery bifurcations



Areas of low time-averaged WSS (< 4 dyn/cm^2) and high oscillatory WSS are prone to plaque

He & Ku. J Biomech Eng. 118: 74-82, 1996
Grøttum et al. Atherosclerosis. 47: 55-62, 1983

Distributions of WSS and NH after stenting



From LaDisa et al. AJP - Heart Circ Physiol 288: H2465-75, 2005.

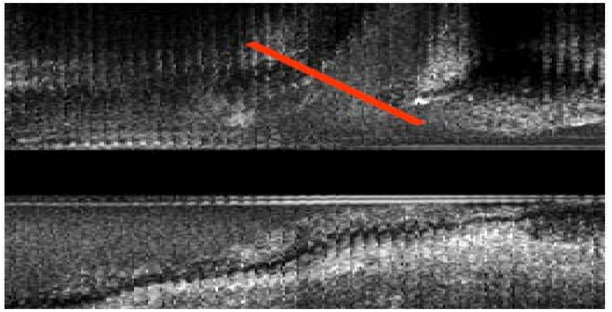
Background

- Restenosis rates vary with bare metal stent (BMS) type
- Stent type (geometry) influences flow patterns that *may impact thrombosis formation and dislodgement*
- 15-20% of all PCI involve bifurcation regions
- Restenosis after main branch bifurcation stenting is improved vs PTCA, but greater than in single vessels
- Restenosis is greater in bifurcation lesions treated with multiple BMS
- Drug-eluting (DES) and specialty bifurcation stents are associated with less than ideal restenosis rates
- Knowledge of local hemodynamic changes is necessary

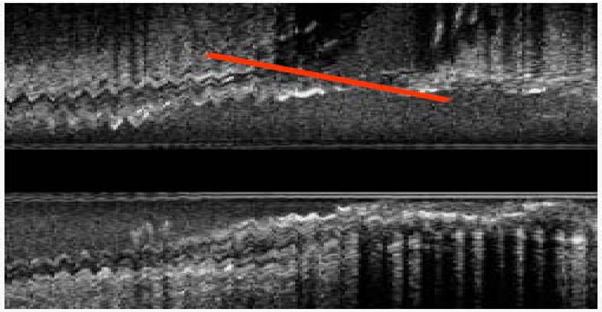
Investigation Objectives

1. Develop a process and methods to study altered hemodynamics in stented coronary bifurcations
2. Quantify altered hemodynamics in the LAD/D1 coronary bifurcation due to local geometry changes (carina shift) caused by stenting

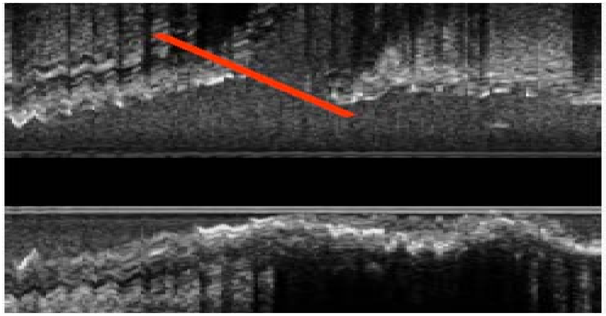
<u>IVUS data of carina shift from MB stenting and after SB angioplasty</u>



Pre-stent



Post main branch stenting

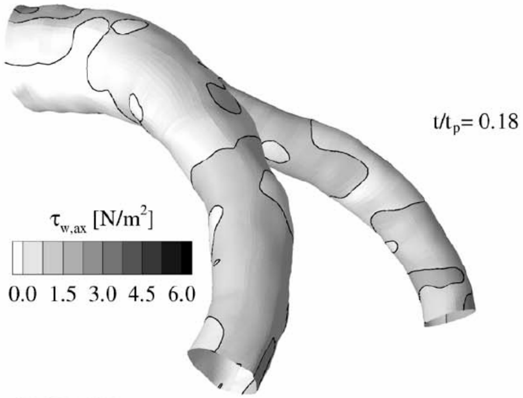


Post side branch angioplasty

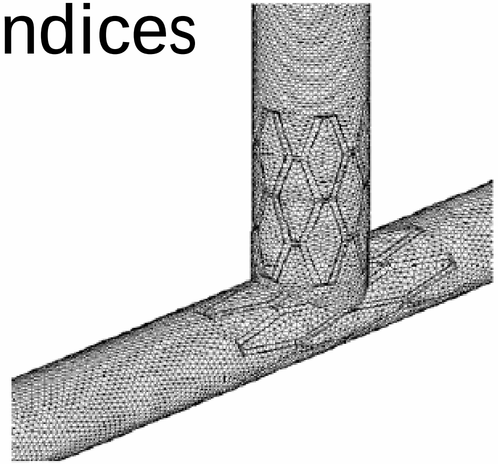
Images courtesy of Bon-Kwon Koo MD, PhD

Previous CFD studies of coronary bifurcations

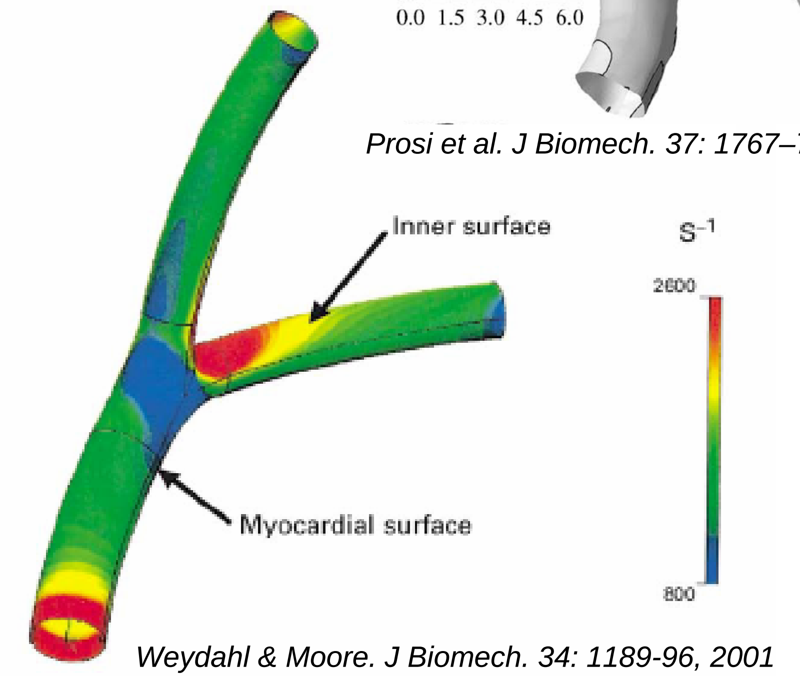
Computational fluid dynamics (CFD) is a tool to create vasculature models from medical imaging data and study hemodynamic indices



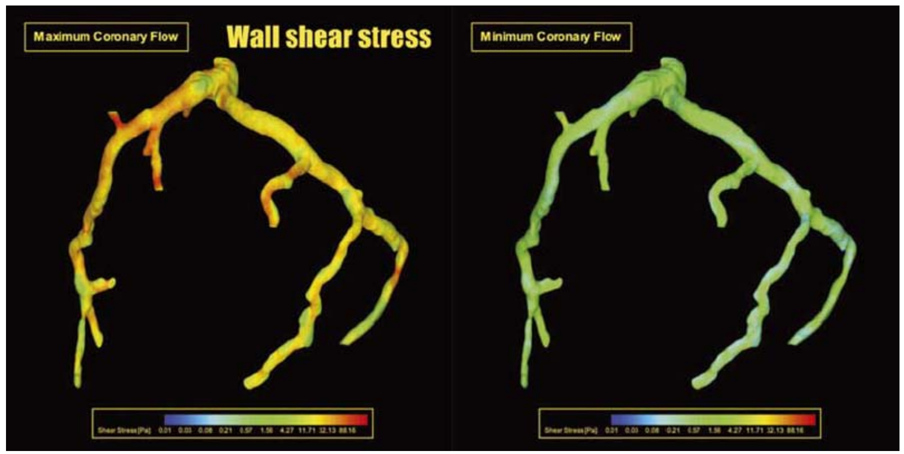
Prosi et al. J Biomech. 37: 1767–75, 2004.



Deplano et al. Med Biol Eng. Comput. 42: 650-9, 2004.



Weydahl & Moore. J Biomech. 34: 1189-96, 2001



Boutsianis et al. Eur J Cardiothorac Surg. 26: 248–56, 2004.

Previous CFD studies of coronary bifurcations

For CFD to be clinically applicable it should:

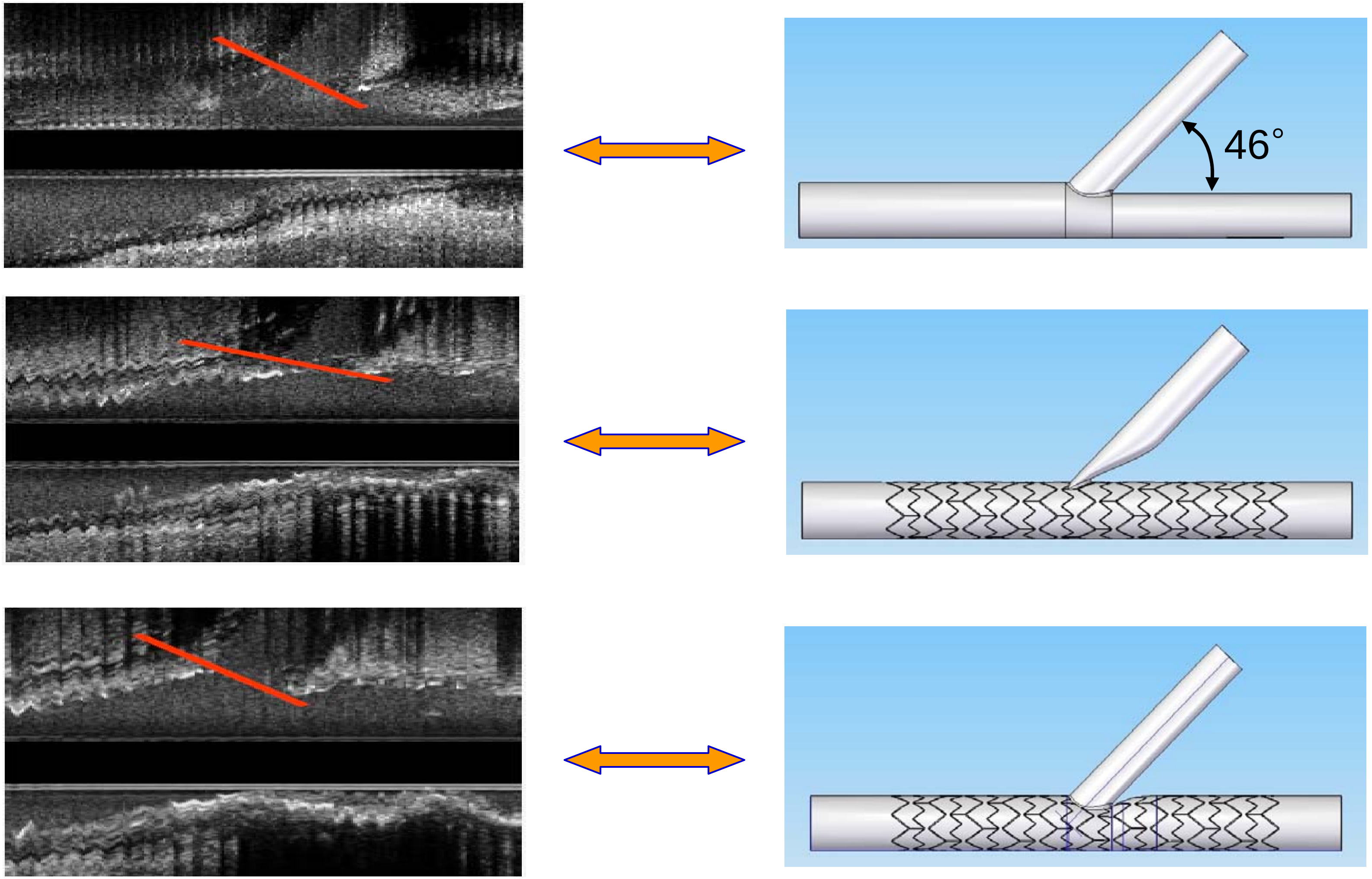
- Replicate blood flow, pressure, geometry and lumen motion measurements obtained clinically
- Apply inlet and outlet boundary conditions that replicate physiology
- Include the impact of pharmacological treatments (such as adenosine) by including vasoactive properties
- Incorporate the impact of devices including single and multiple stents, filters, etc.
- *No studies to date satisfy these criteria, or have been conducted in stented coronary bifurcations*

Representative bifurcations and Express stent

Pre-stent

Post main branch stenting

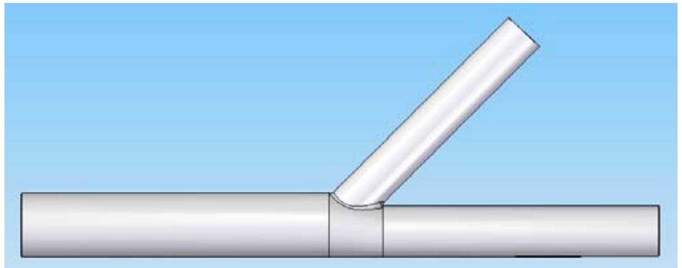
Post side branch angioplasty



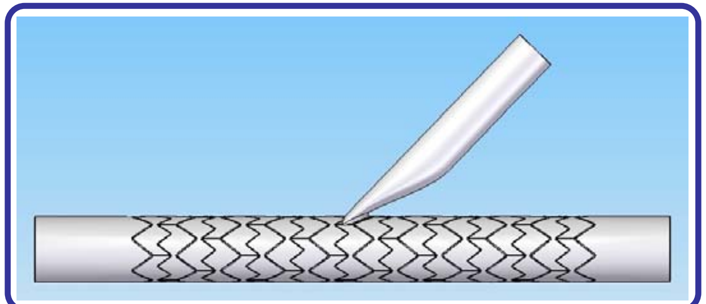
Representative bifurcations and Express stent

Finet's Law: $D_m = 0.678(D_{d1} + D_{d2})$

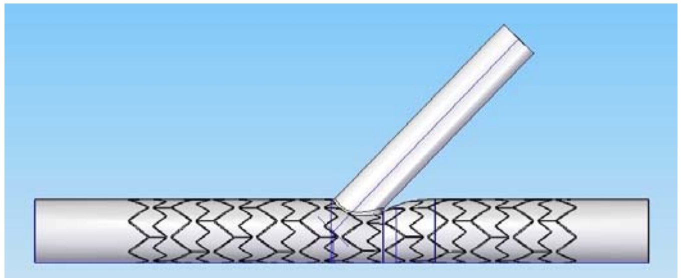
Pre-stent

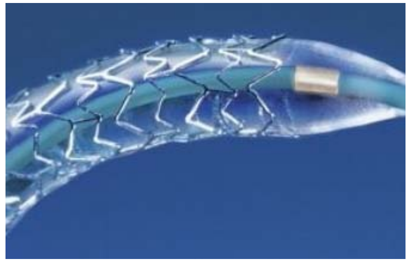


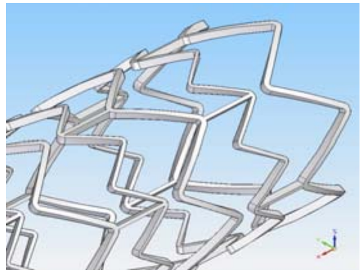
Post main branch stenting



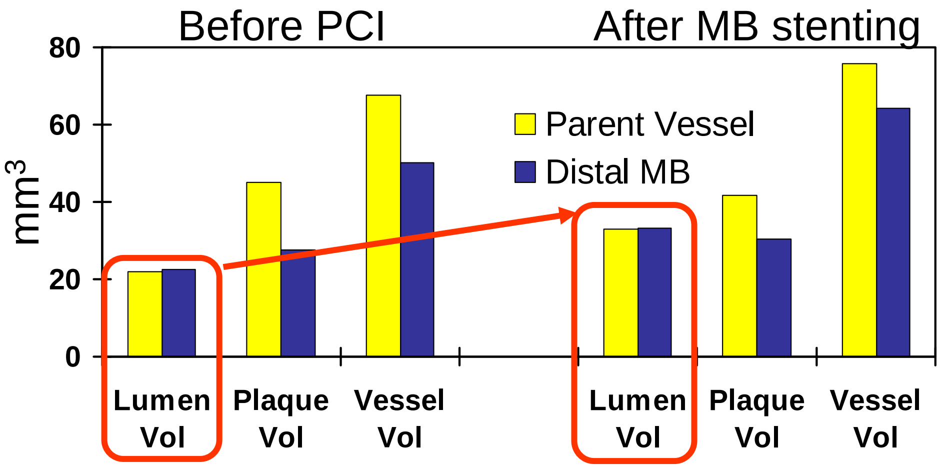
Post side branch angioplasty







Geometric changes after main branch stenting by IVUS (n =12)



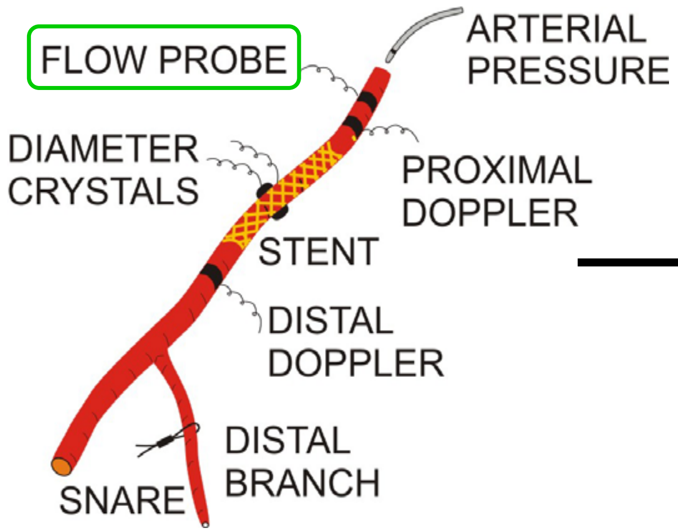
Distal lumen enlargement

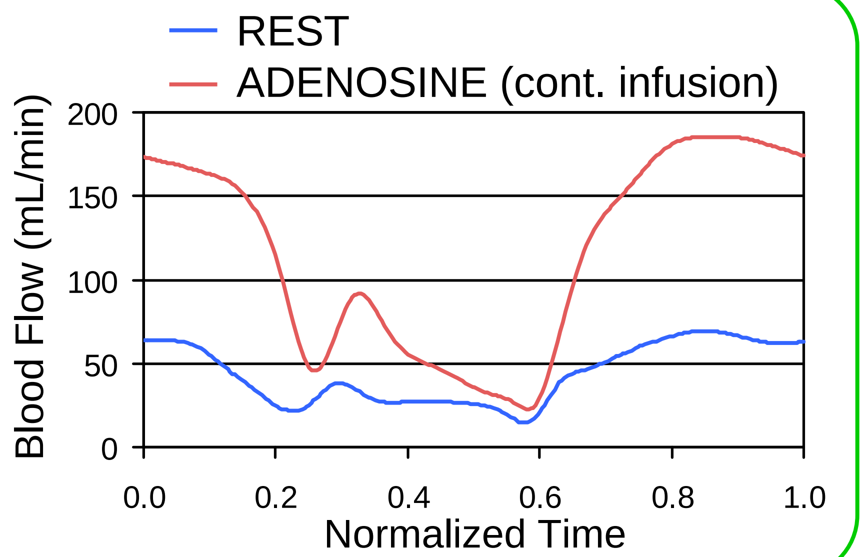
- 92% by vessel expansion
- 8% by plaque volume change

Boundary Conditions

Inflow waveforms and estimates of downstream vascular resistance obtained and implemented from previous studies

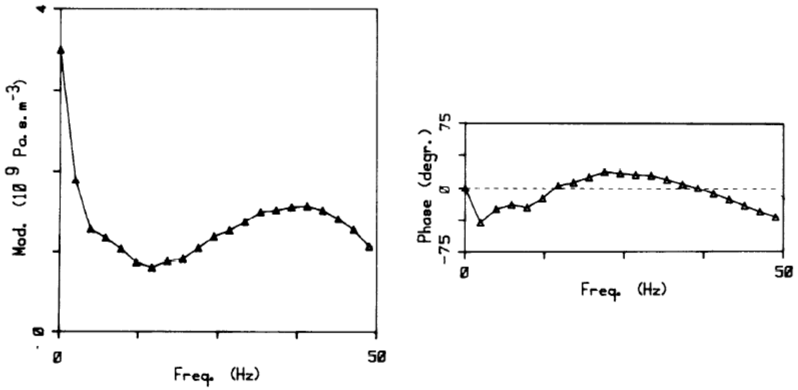
Inlet

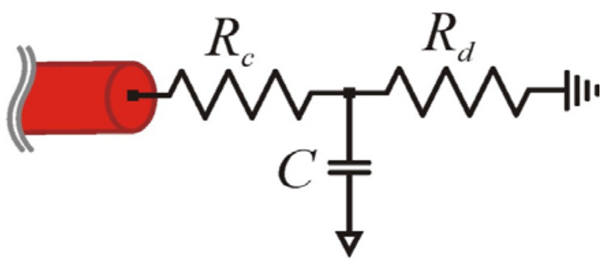




Systolic coronary impedance spectra

Outlets



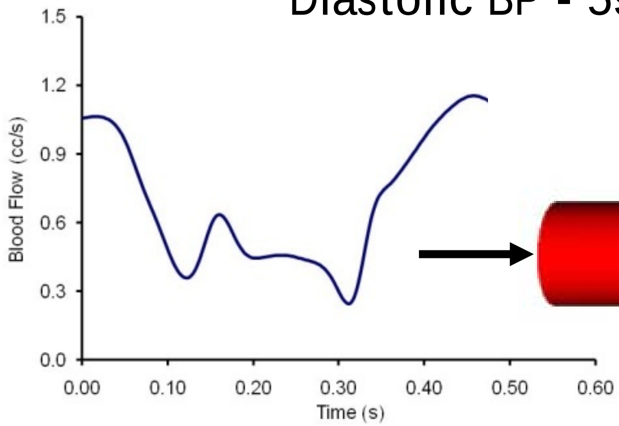


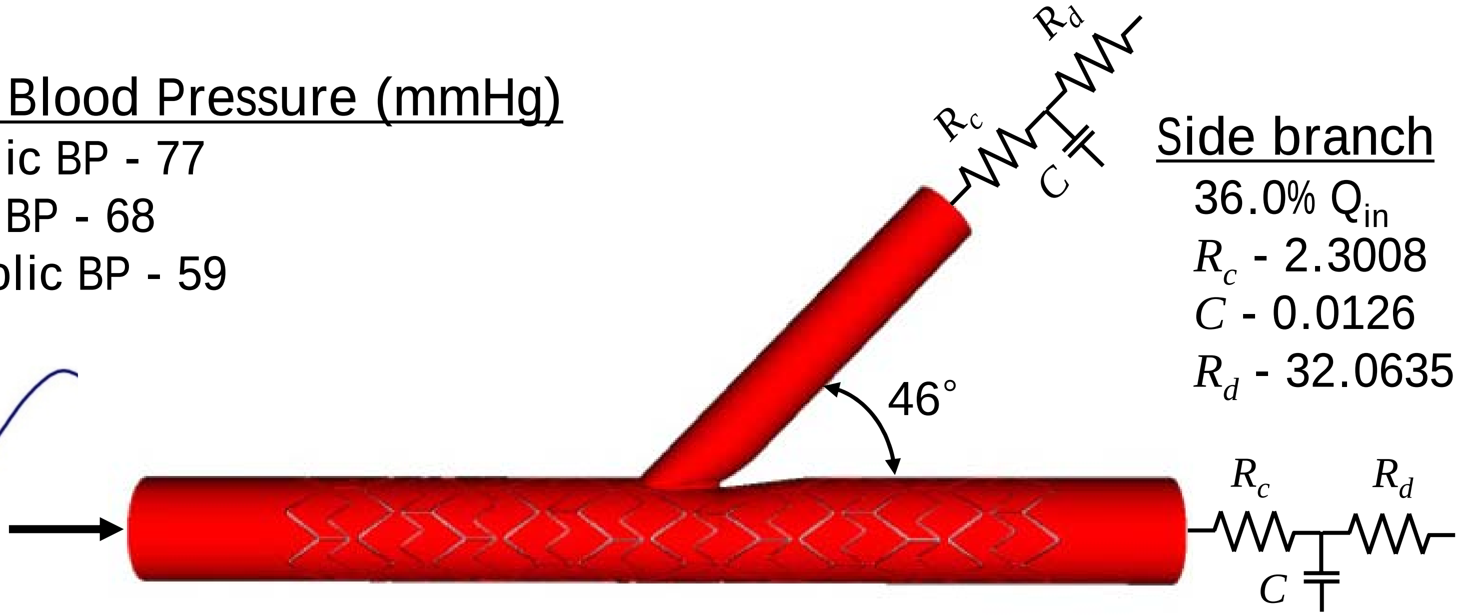
LaDisa et al. J Appl Physiol 93: 1939-46, 2002
Van Huis et al. AJP - Heart. 253 (22): H317-H324, 1987

Simulation Details

Aimed Blood Pressure (mmHg)

Systolic BP - 77
Mean BP - 68
Diastolic BP - 59





Side branch

36.0% Q_{in}
R_c - 2.3008
C - 0.0126
R_d - 32.0635

Distal LAD

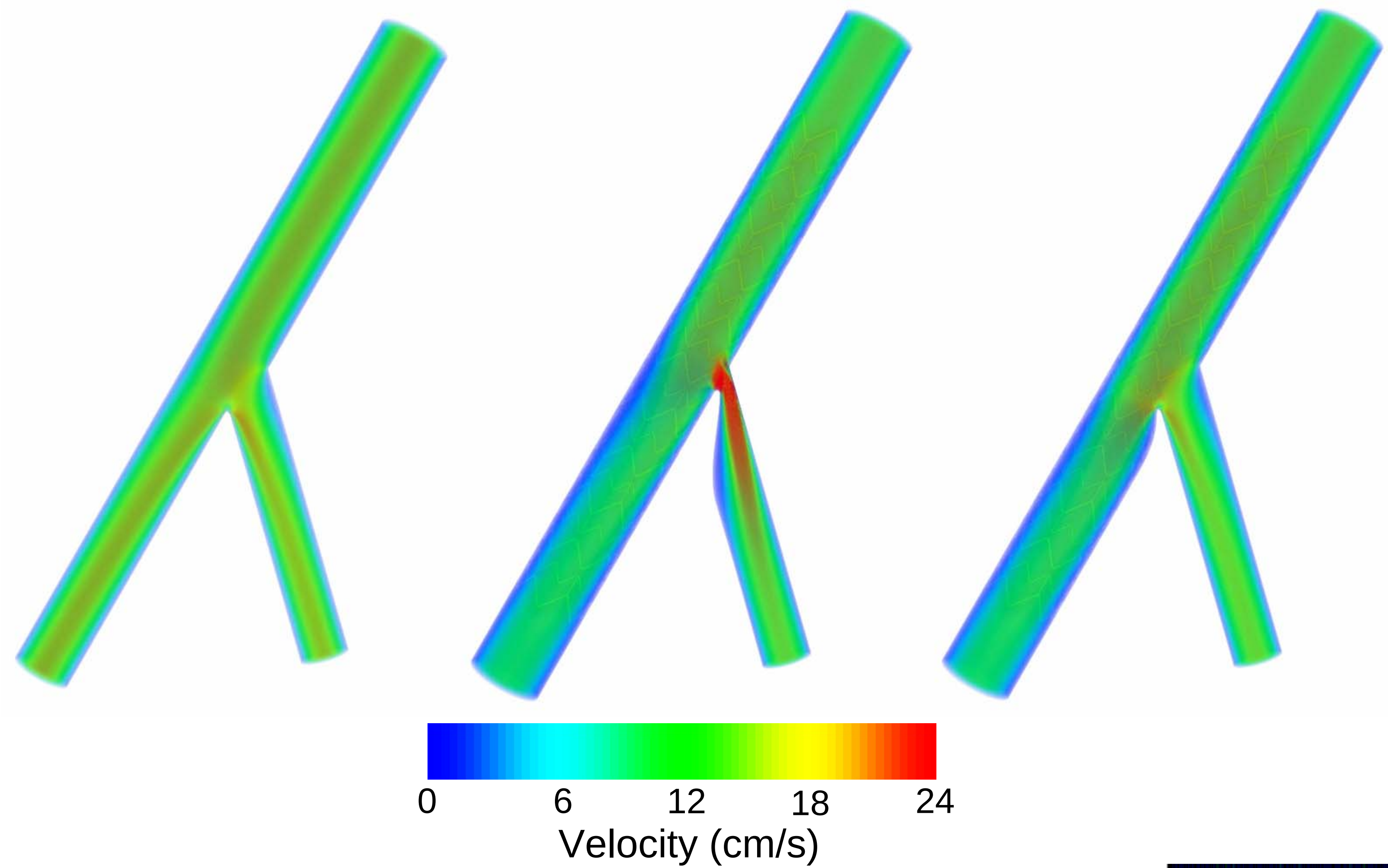
64.0% Q_{in}
R_c – 1.5686
C – 0.0224
R_d – 17.7806

Additional simulation details:

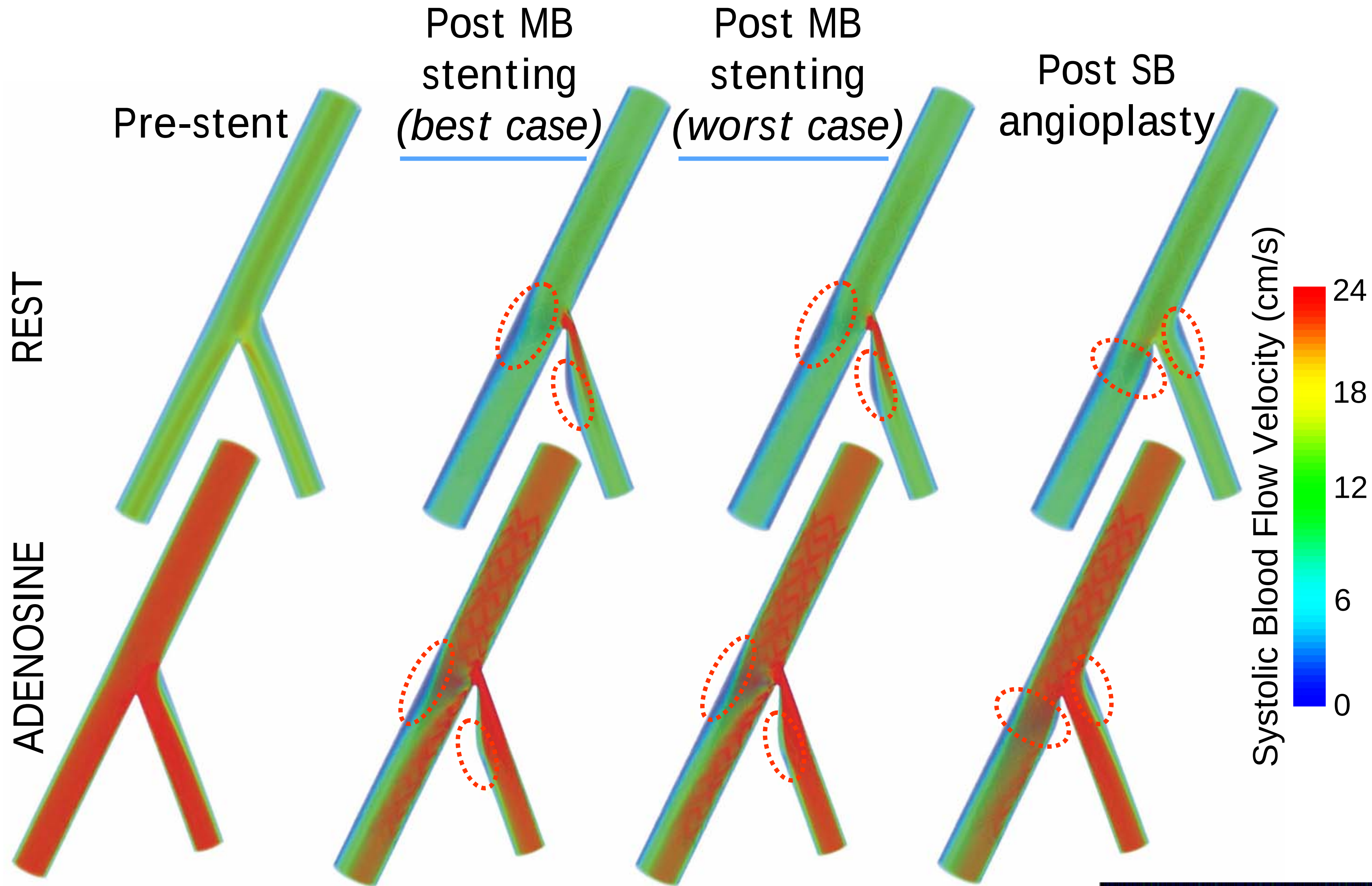
- Newtonian fluid, μ = 4.0 cP, ρ = 1.06 g/cm^3
- Vessel walls initially assumed to be rigid
- Equations describing pressure and velocity were solved using high performance computers

Volume-rendered Blood Flow Velocity - REST

Pre-stent | Post main branch (MB) stenting | Post side branch (SB) angioplasty



Systolic Blood Flow Velocity



Side branch jailing - impact on FFR

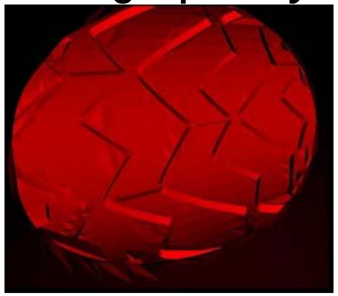
Post MB stenting
(best case)

Ostium area = 1.94 mm^2
Diameter stenosis = 54%
Area stenosis = 51%

Post MB stenting
(worst case)

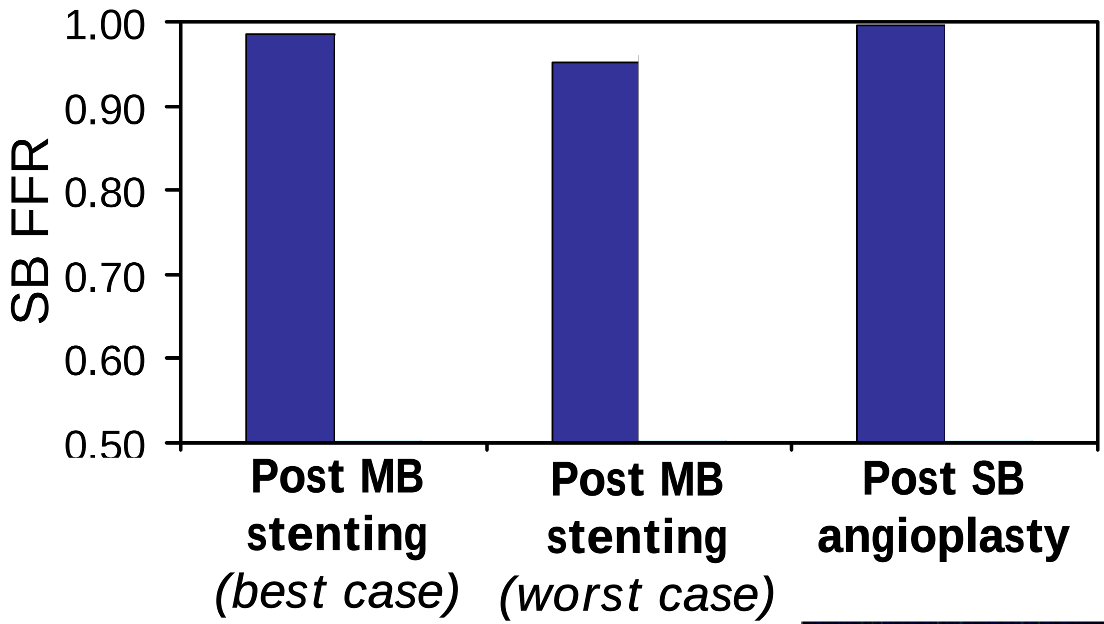
Ostium area = 1.88 mm^2
Diameter stenosis = 54%
Area stenosis = 52%

Post SB
angioplasty

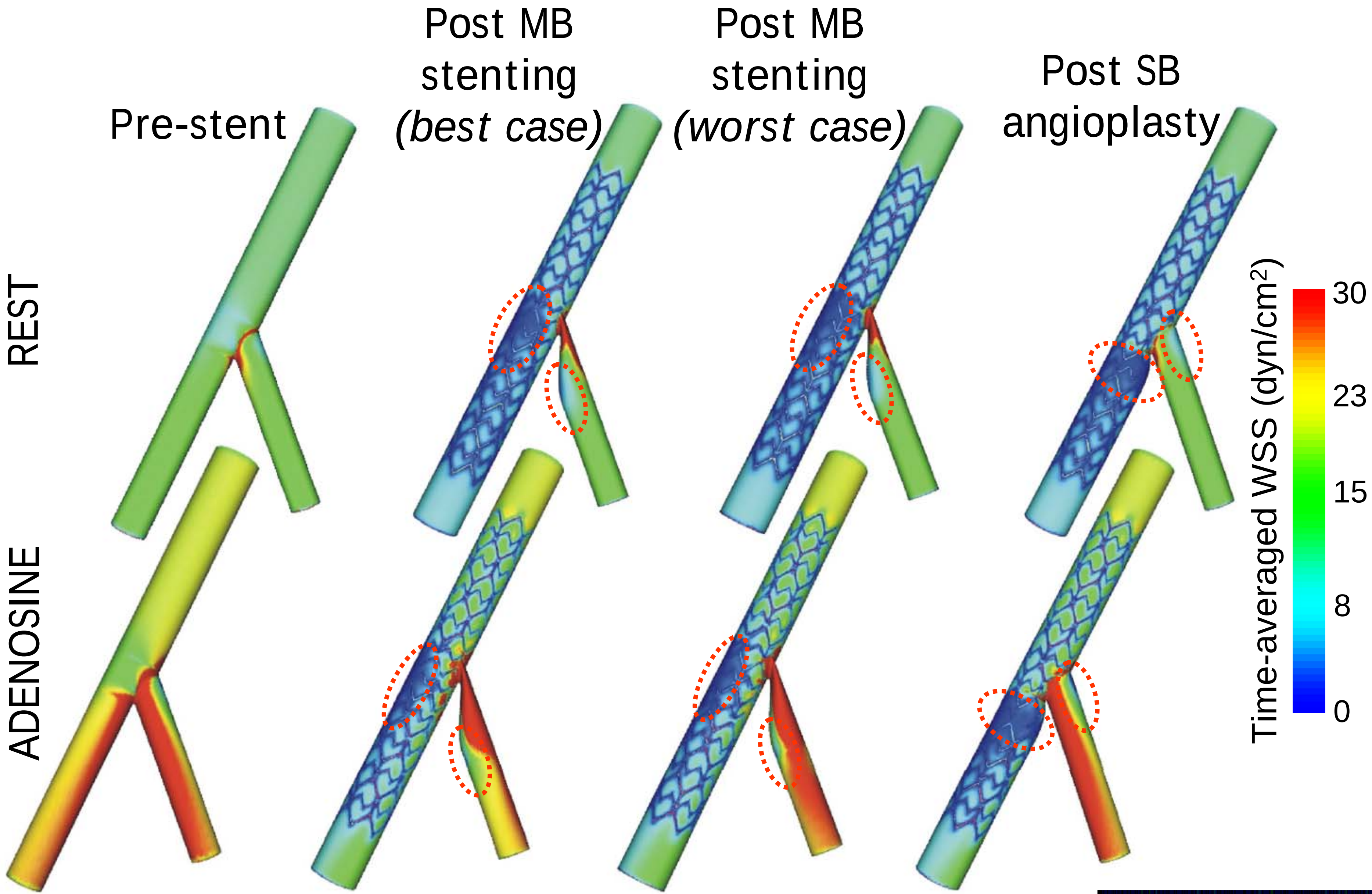


Ostium area = 3.89 mm^2
Diameter stenosis = 0%
Area stenosis = 0%

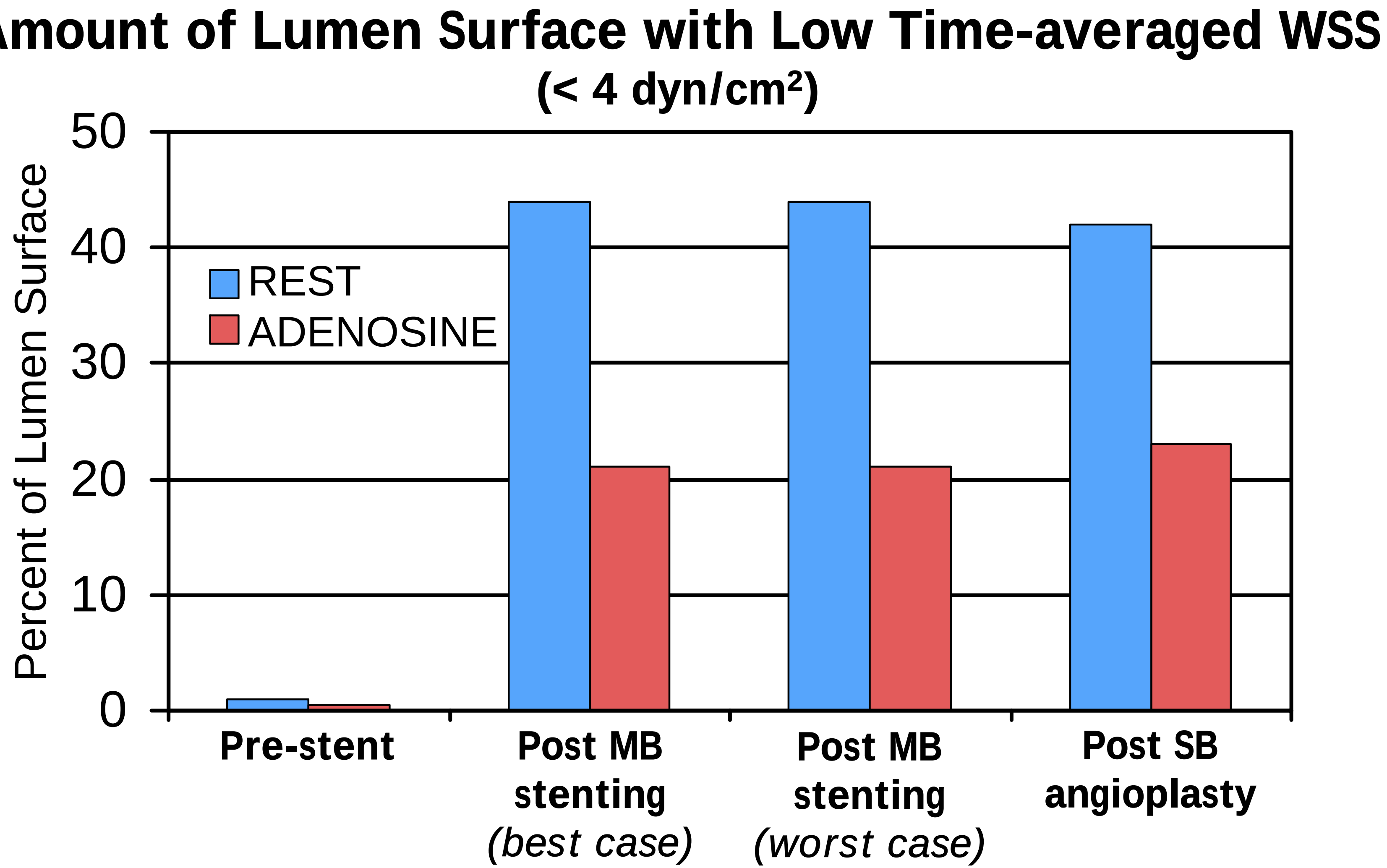
$$FFR = \frac{Q^S_{max}}{Q^N_{max}} \approx \frac{P_d}{P_a}$$



Time-averaged wall shear stress

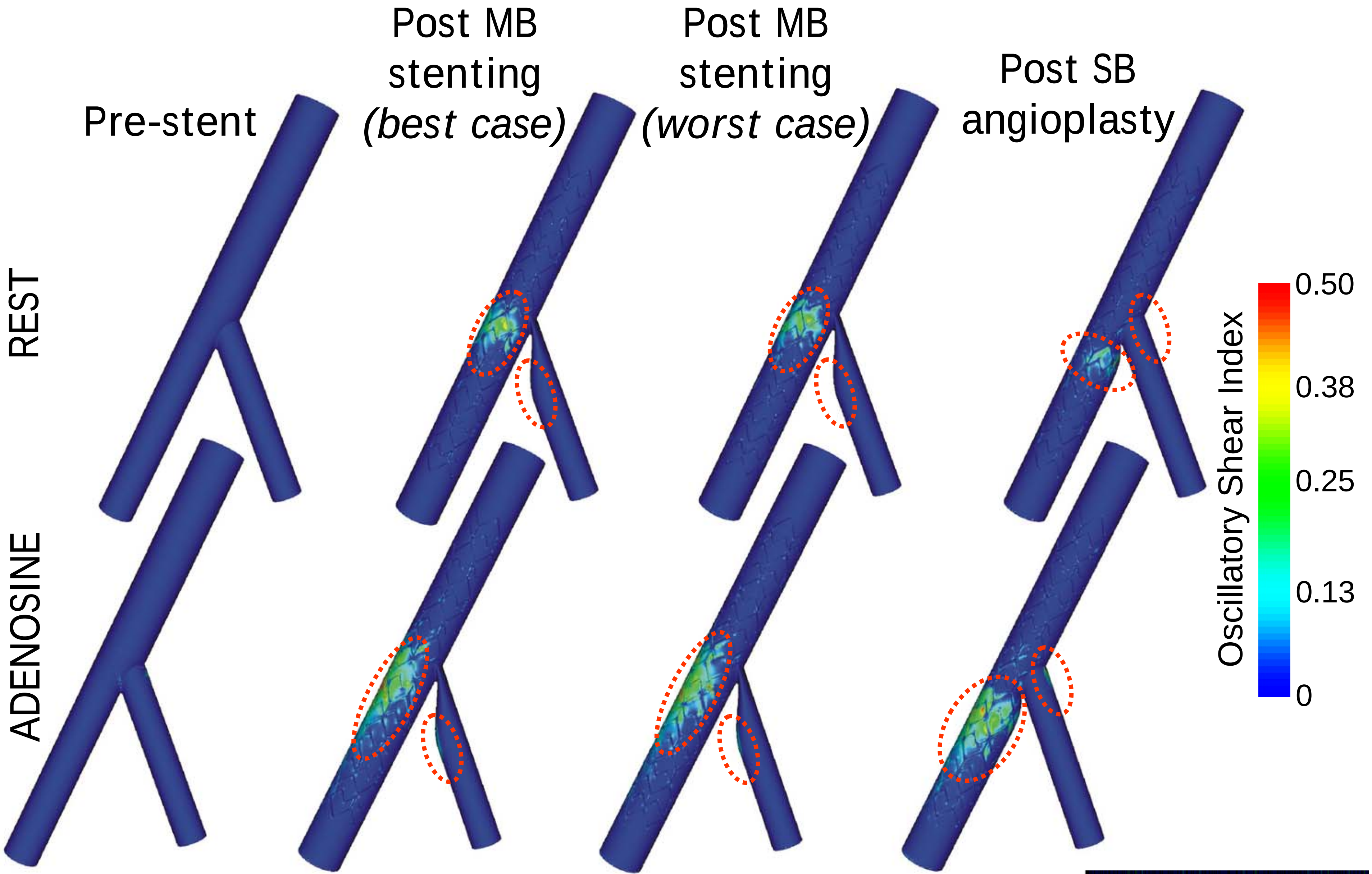


Quantification of Low WSS areas

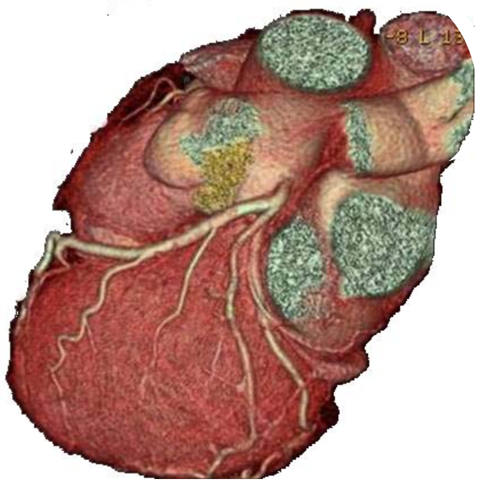


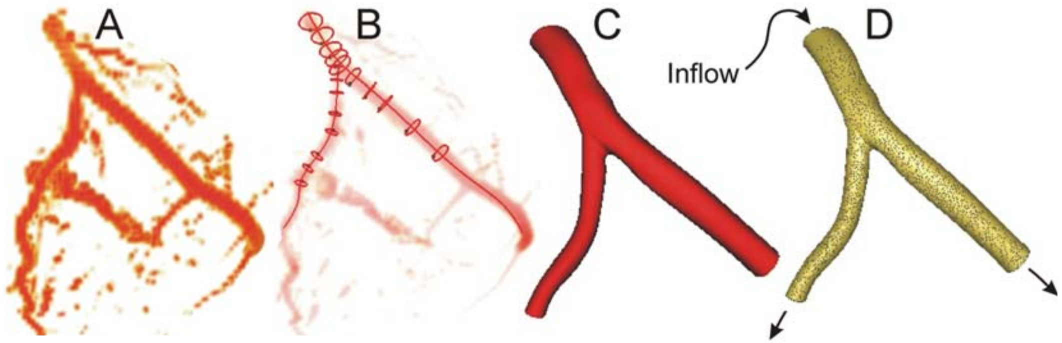
There are no differences in the total area of low TAWSS so the potential for neointimal hyperplasia or thrombus are the same from a fluid dynamics perspective

Oscillatory shear index (OSI)



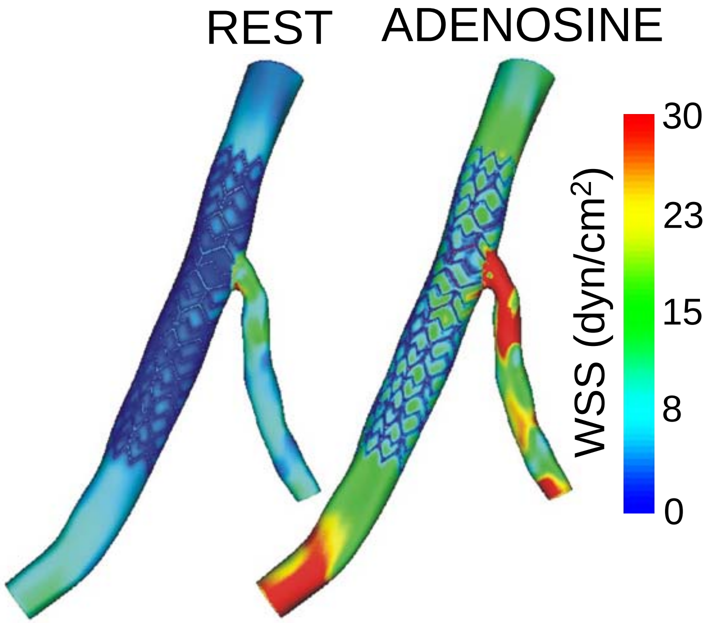
Next Steps – Patient specific modeling

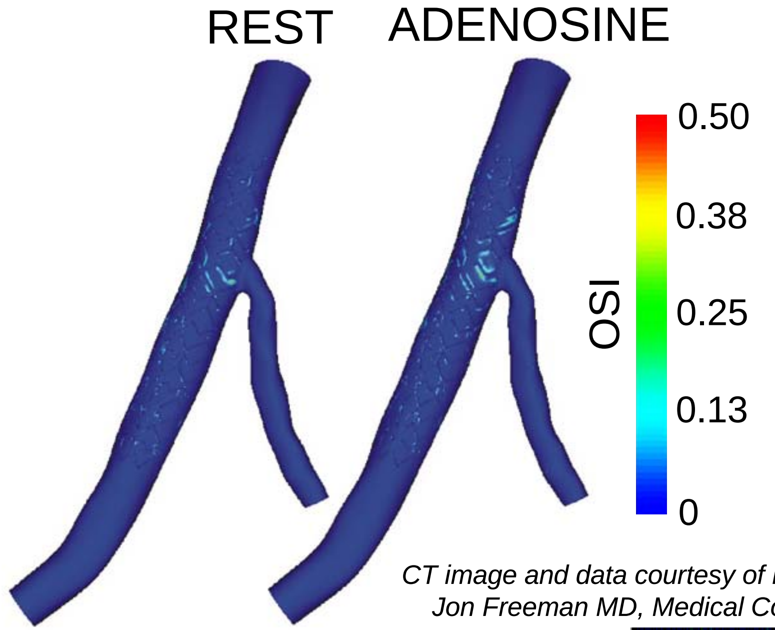




Methodology

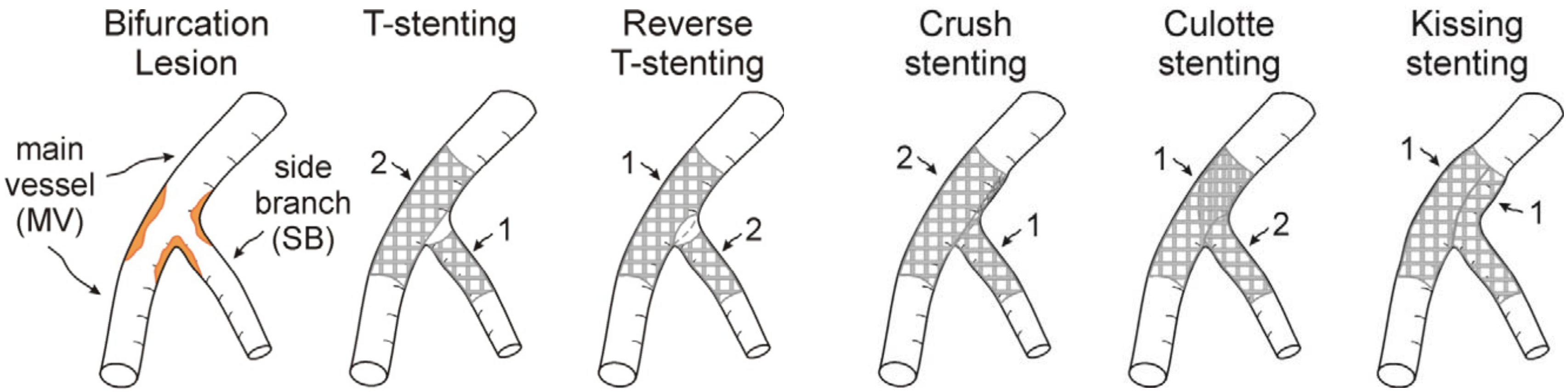
A. Identify artery
B. Create center-line paths and segments
C. Crate model and add stent
D. Create mesh and run simulation

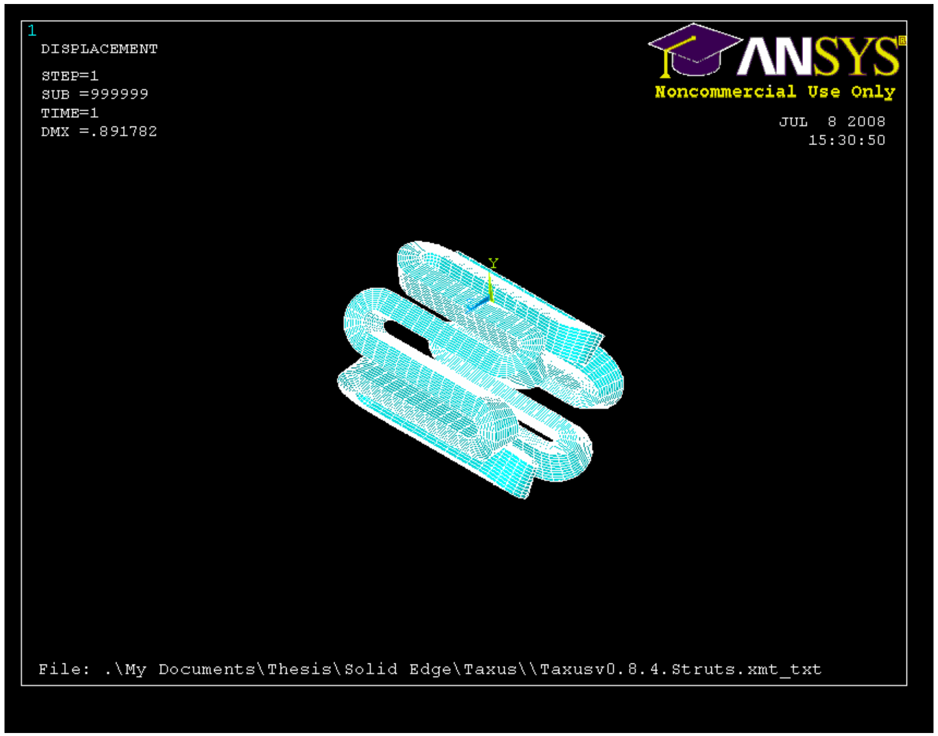


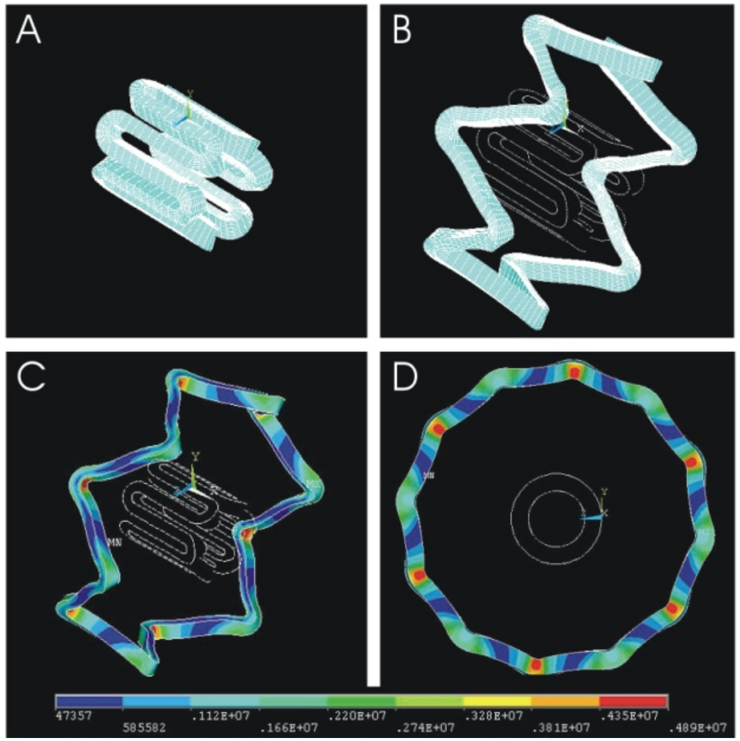


CT image and data courtesy of Ray Migrino MD and Jon Freeman MD, Medical College of Wisconsin

Next Steps – Modeling multi-stent deployment







Summary

- CFD can be used to quantify and increase our knowledge of altered hemodynamics in the stented LAD/D1 bifurcation
- Simple MB stenting caused flow disturbances
  1. Stenting caused low time-averaged WSS (TAWSS) near struts that diminished during adenosine infusion
  2. MB stenting caused eccentric areas of low time-averaged WSS and elevated OSI opposite the carina
- SB angioplasty did not alleviate these flow disturbances
  1. The total area of low TAWSS was the same for MB stenting and after SB angioplasty
  2. SB angioplasty restored carina position, but caused concentric low TAWSS and high OSI in the distal MB