

Plaque Formation and Rupture by Flow Hemodynamics: Clinical Implications

**The First International Geriatric
Cardiology Meeting in Vietnam**

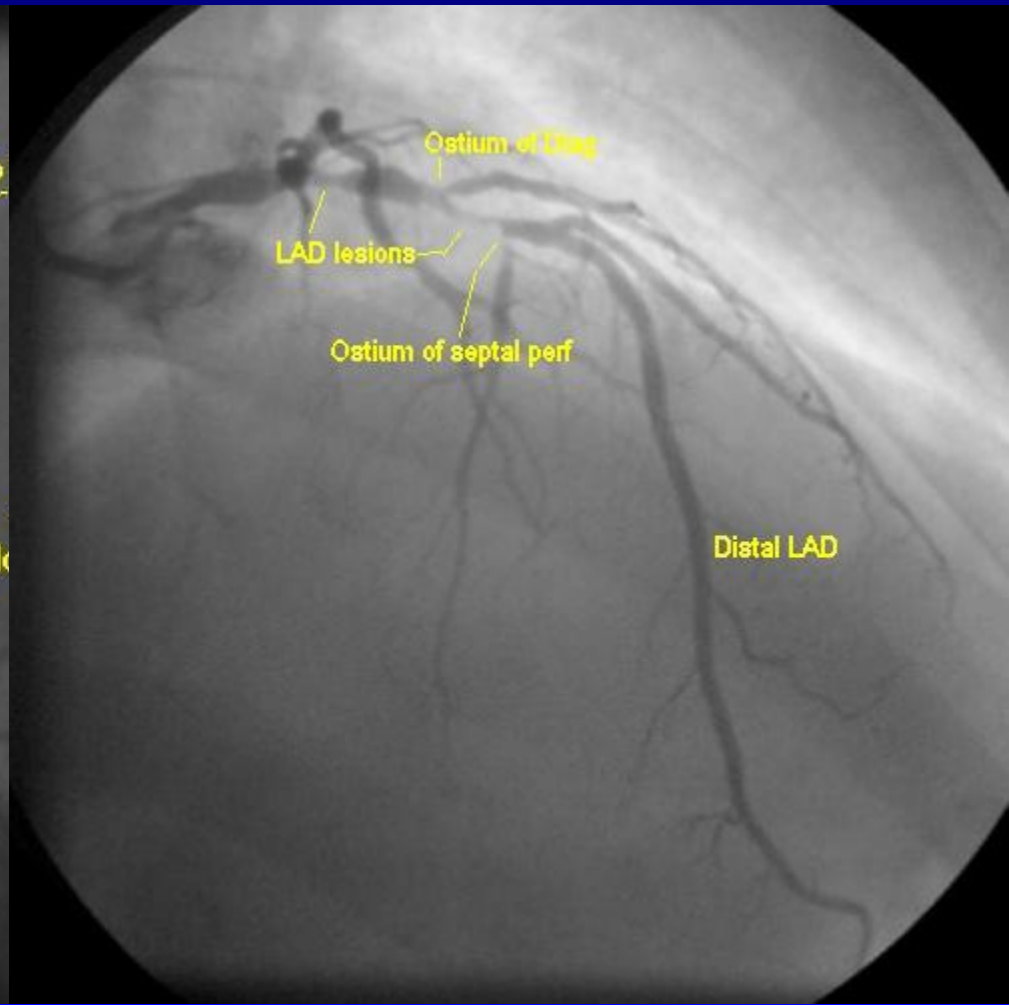
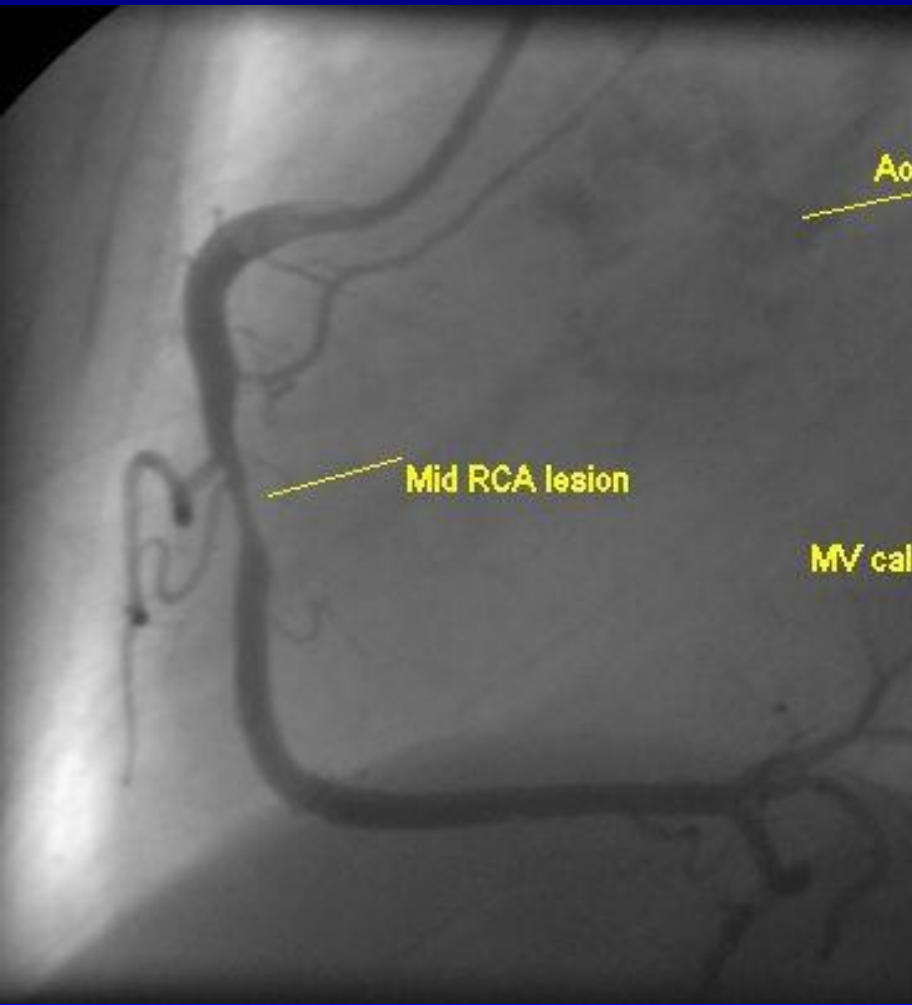
December 4th 2012

Thach Nguyen, M.D. FACC FSCAI

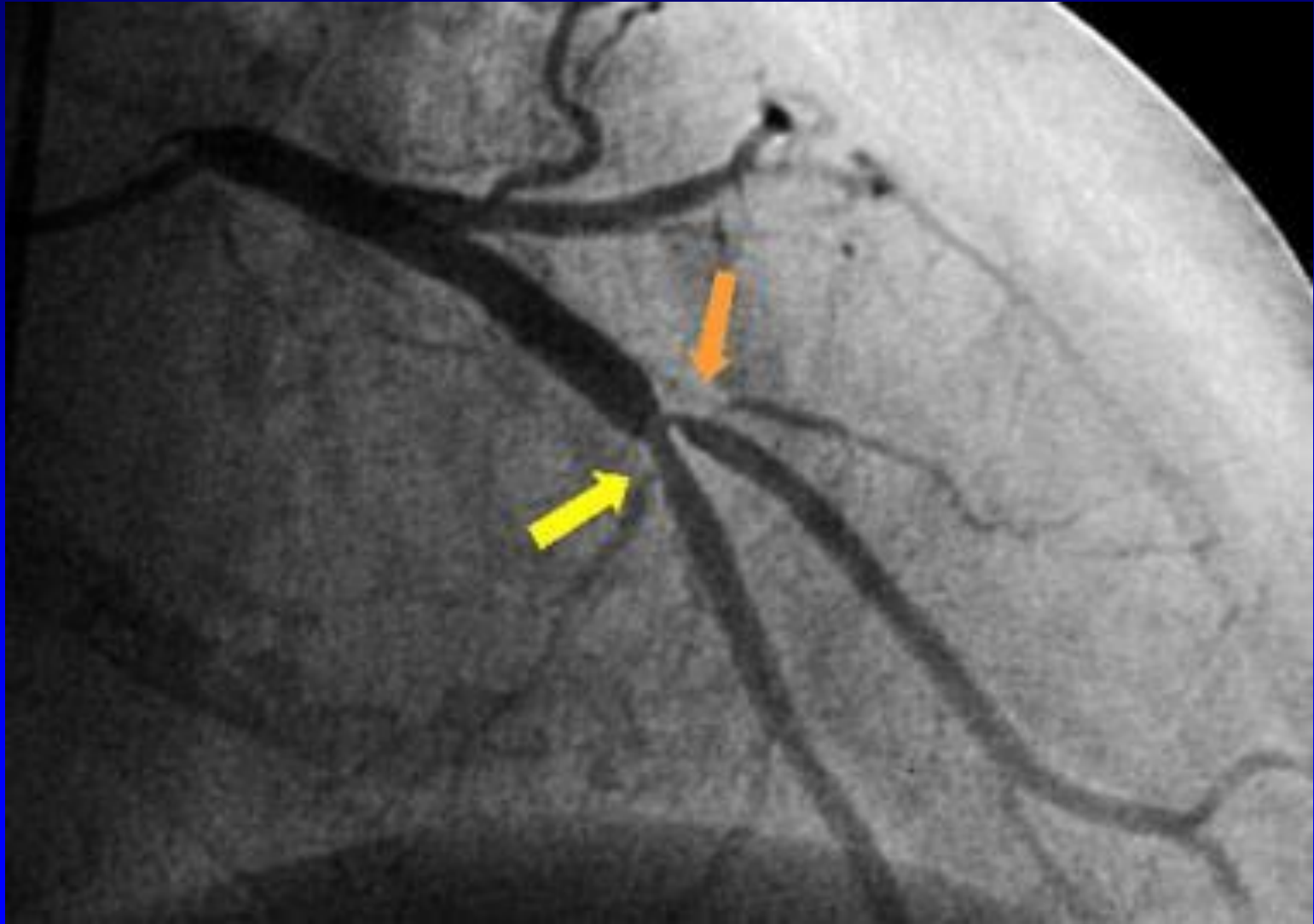
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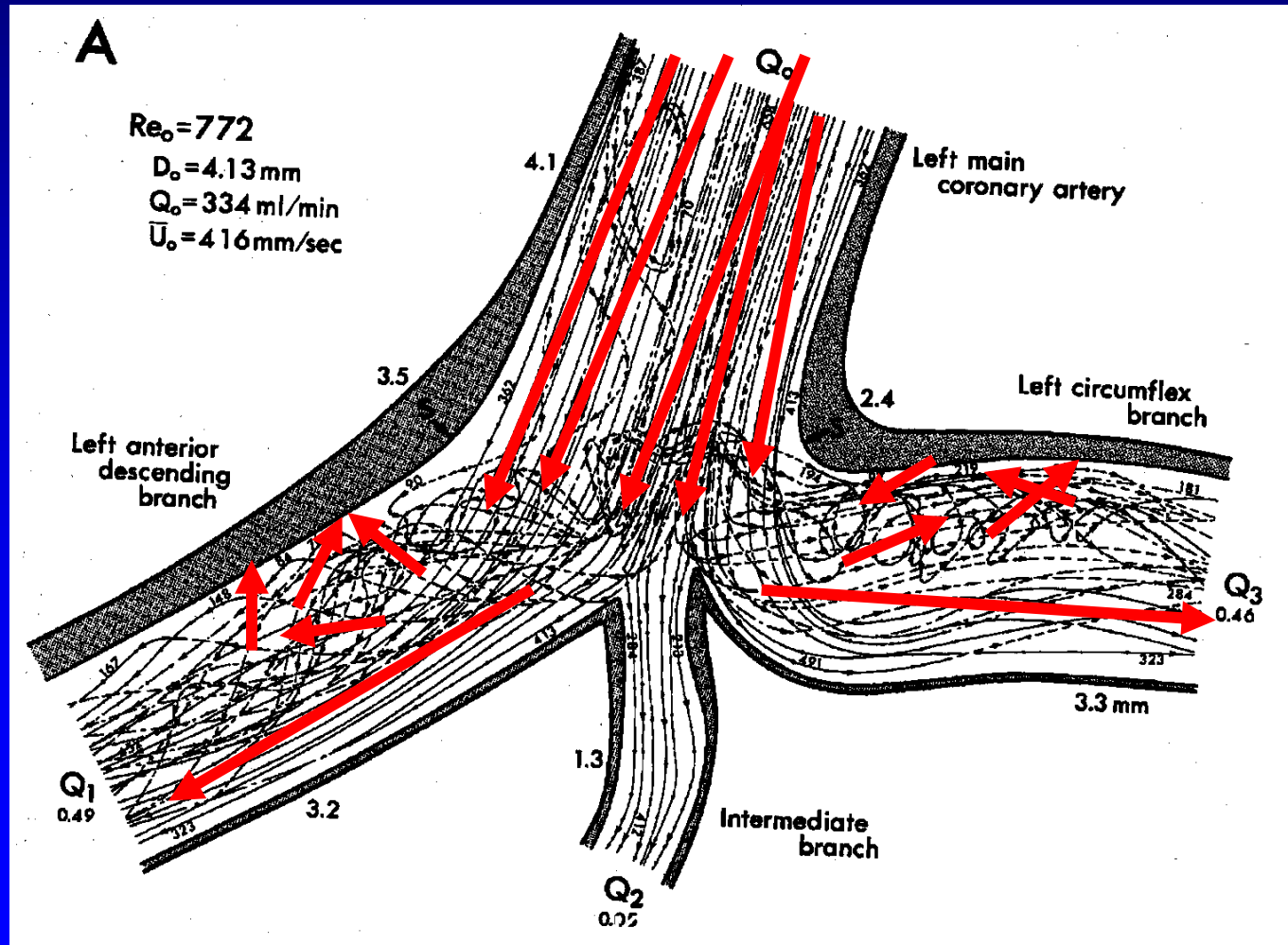
OBSERVATION 1: Coronary artery lesions



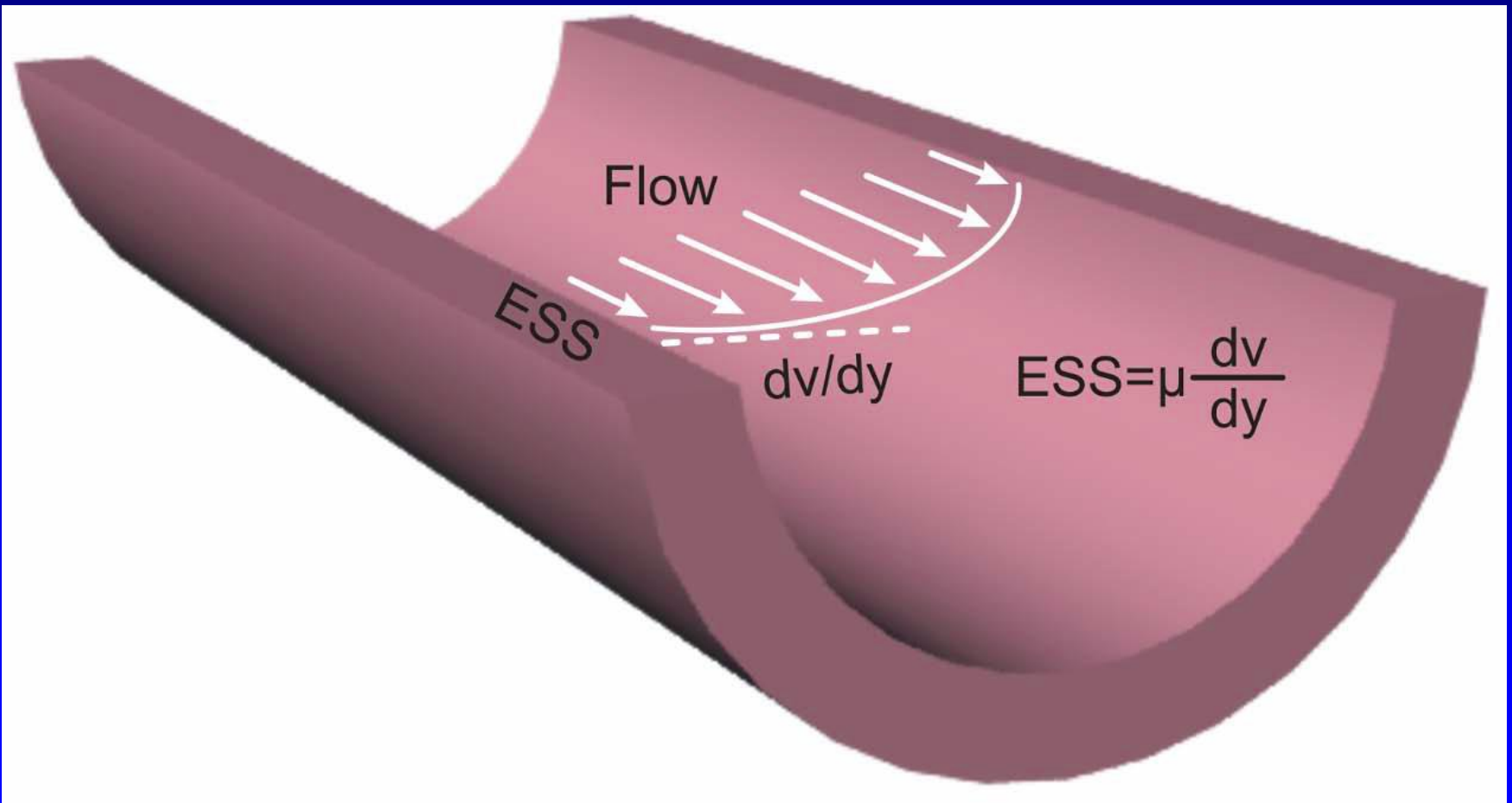
OBSERVATION 1: Coronary artery lesions



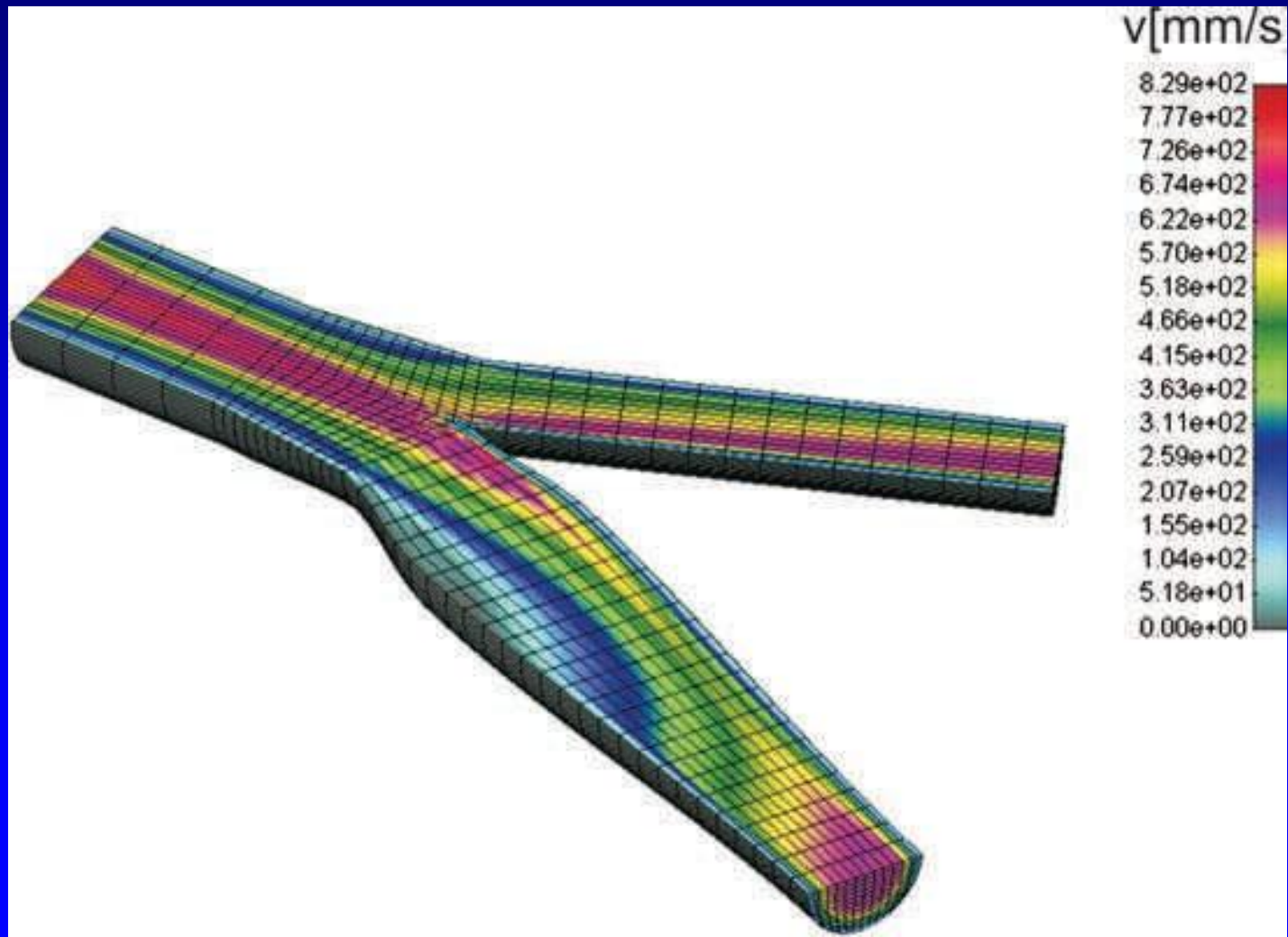
CURRENT EXPLANATION: Recirculation area



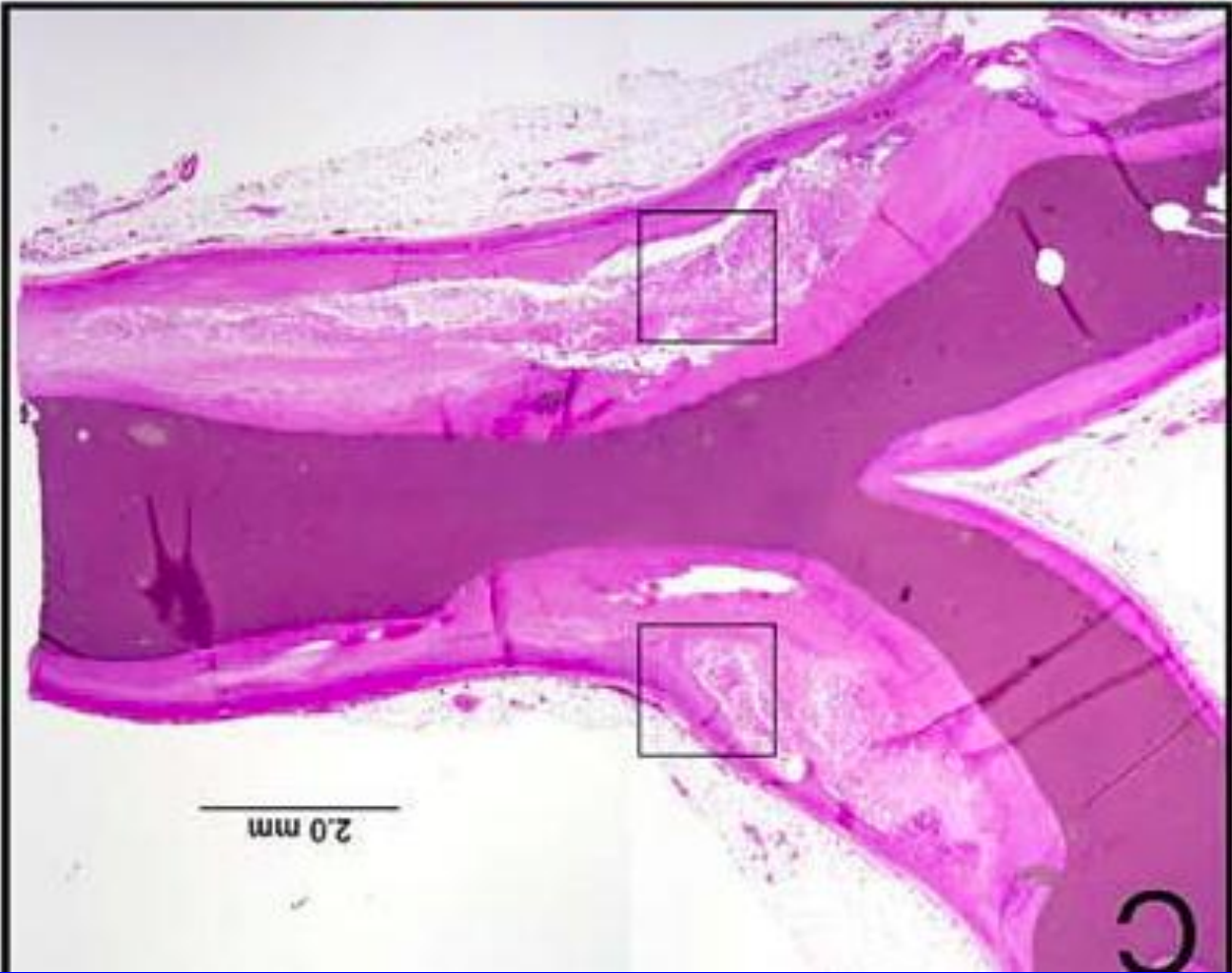
Shear stress is the tangential force derived from the friction of the flowing blood on the endothelium



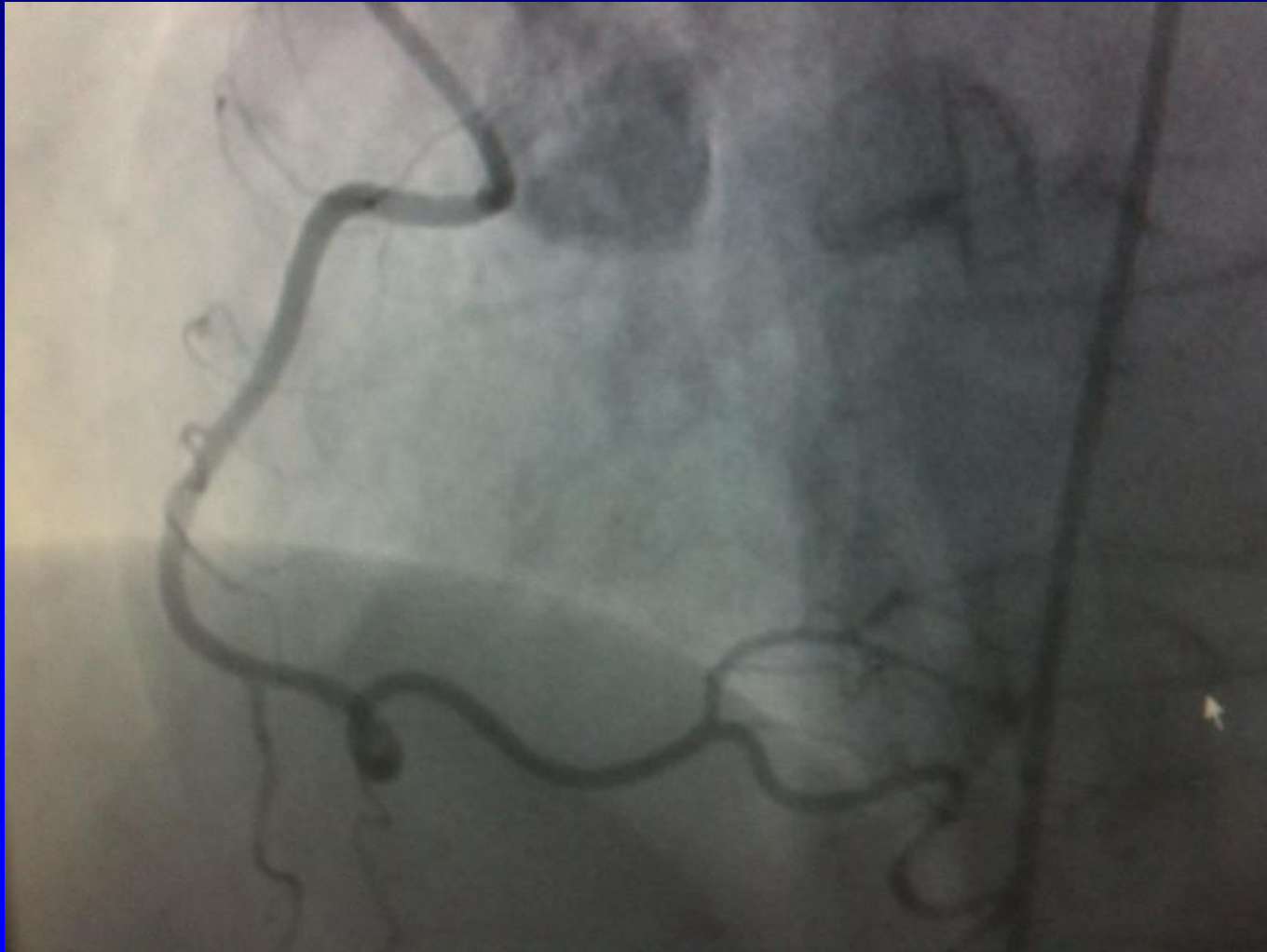
CURRENT EXPLANATION: Low shear stress area is the place for plaque formation







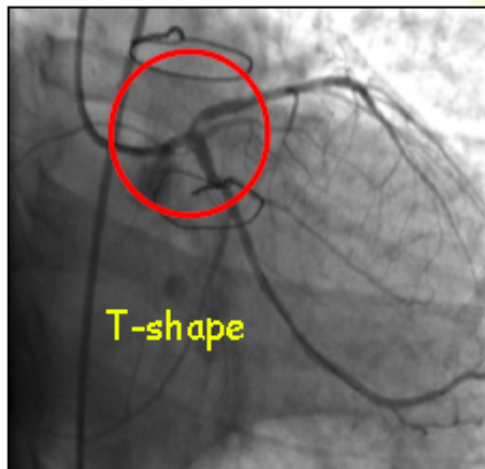
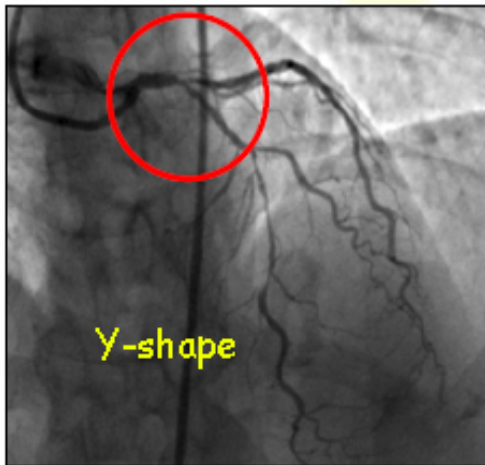
QUESTION 1: Why Does not every patient with curved arteries and low shear stress have plaque?



QUESTION 2: What is the effect of Bifurcation Angle on Shear Stress and Plaque formation (90 degree versus Y angle)?



MACE after PCI of LM in the LM Taxus French Registry



2-years FU	Y-Shape (137)	T-Shape (84)	<i>P</i> value
Stent thrombosis* (%)	0	2.3	<0.05
TVR (%)	8.7	8.3	0.41
Cardiac death (%)	2.9	9.5	0.021
Death (%)	4.4	17.8	0.001

* Definite and probable stent thrombosis according to ARC definition

WHY?

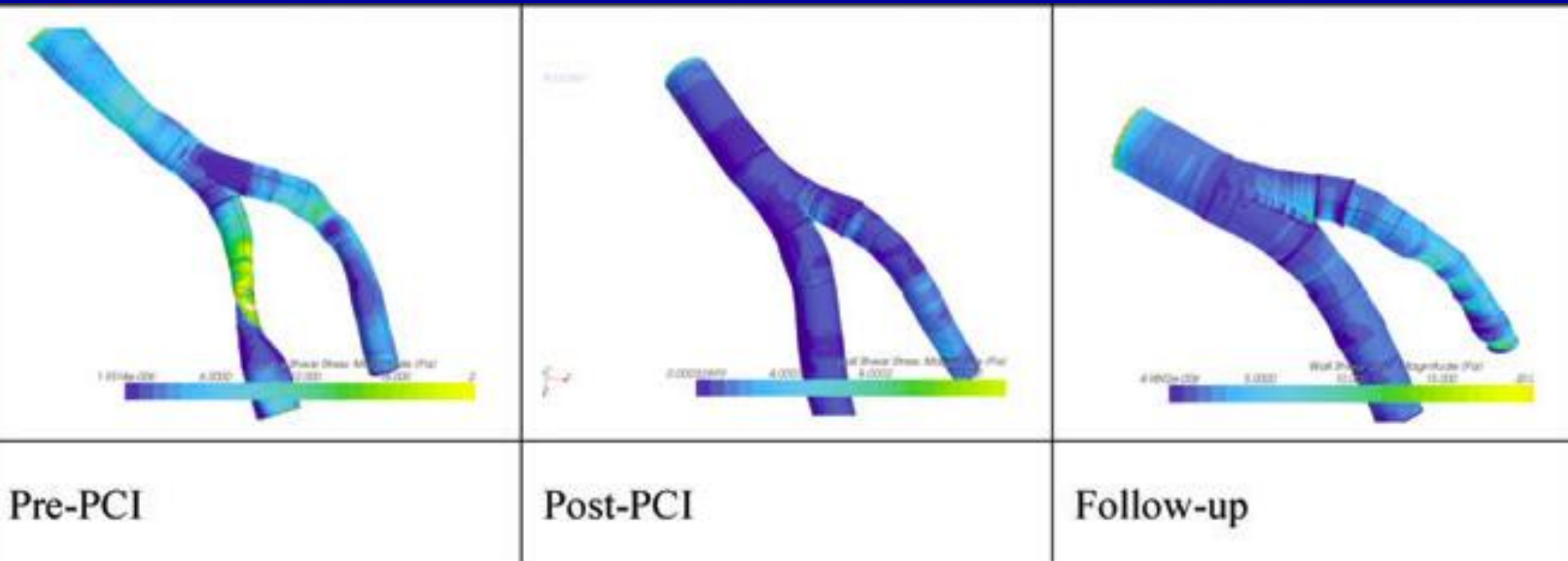
QUESTION 3: Does the large or small size of the side branch have effect on shear stress and plaque formation? **WHY?**



QUESTION 4: Does the length of the side branch have effect on shear stress and plaque formation? **WHY?**



Observation 5: After stenting, low shear stress area happens in all area so the stent. How it can be the cause of neo-intimal hyperplasia at the ostium of the SB after DES?



MAIN JOB FOR US

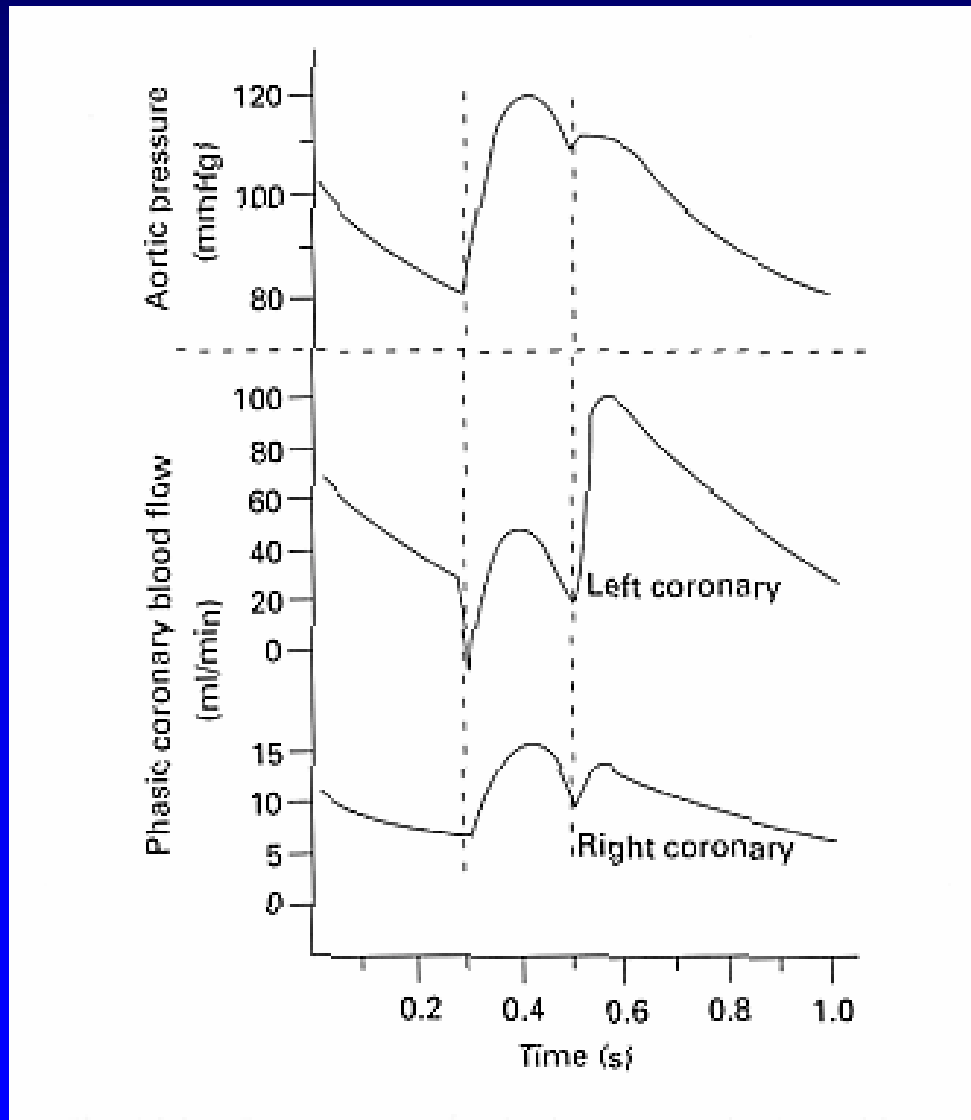
To Connect All the DOTS



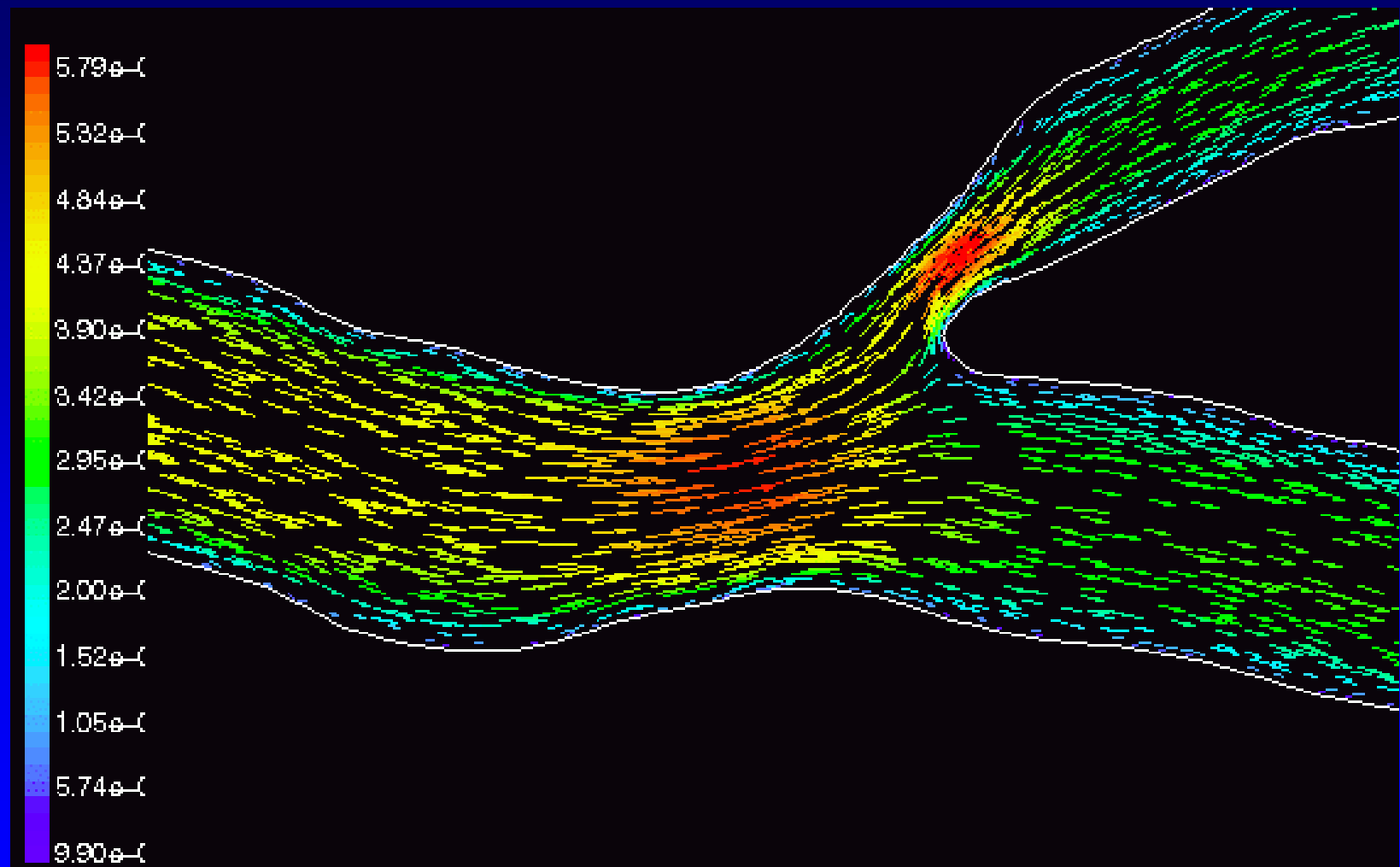
Observation 2

The Flow is Pulsatile (not laminar)

Coronary Flow in Diastole or Systole



T= 50msec

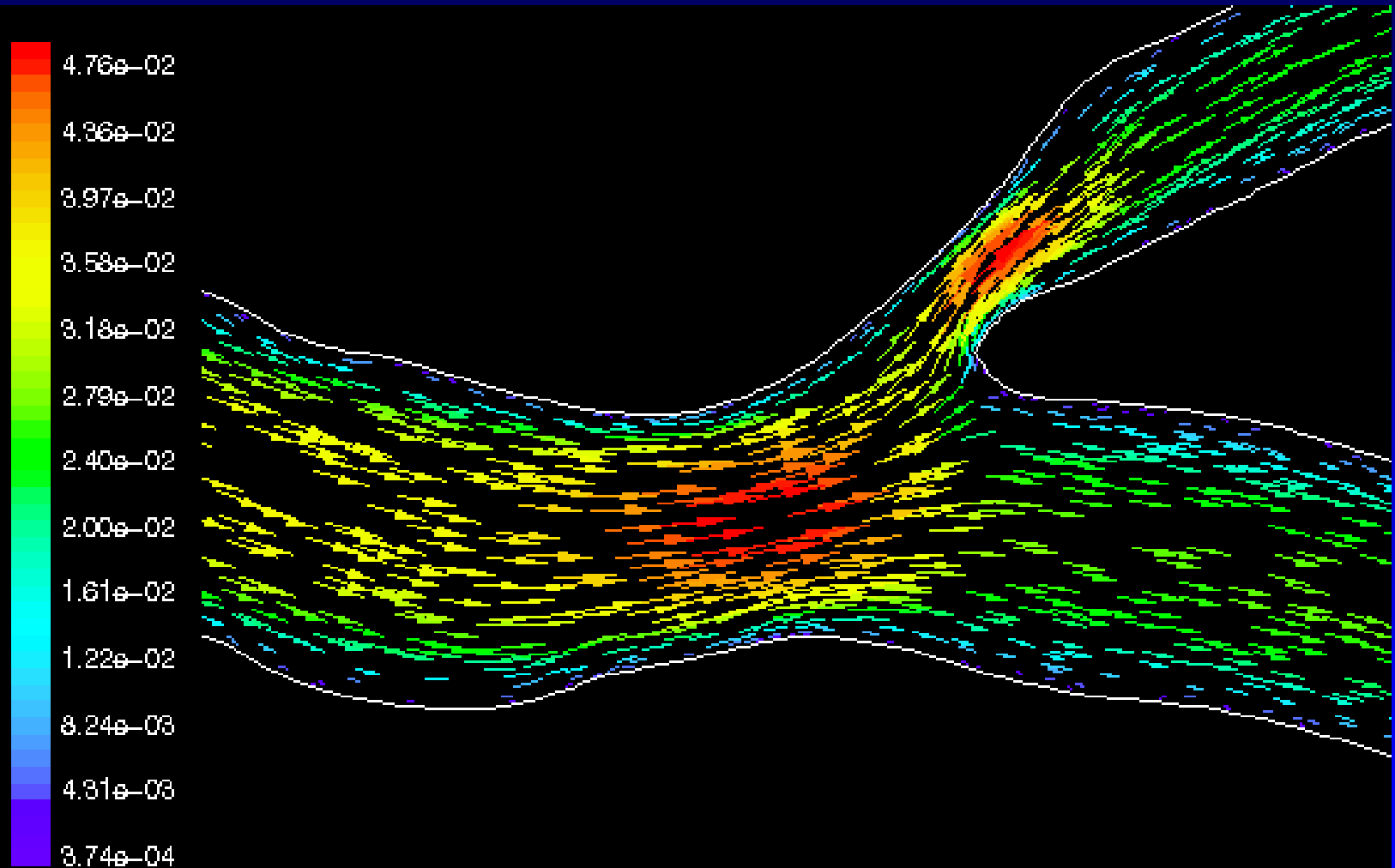


Velocity Vectors Colored By Velocity Magnitude (m/s) (Time=9.1743e-03)

Oct 04, 2000

ELIENIT 5.3 (2d de accelerated km unsteady)

T= 110msec

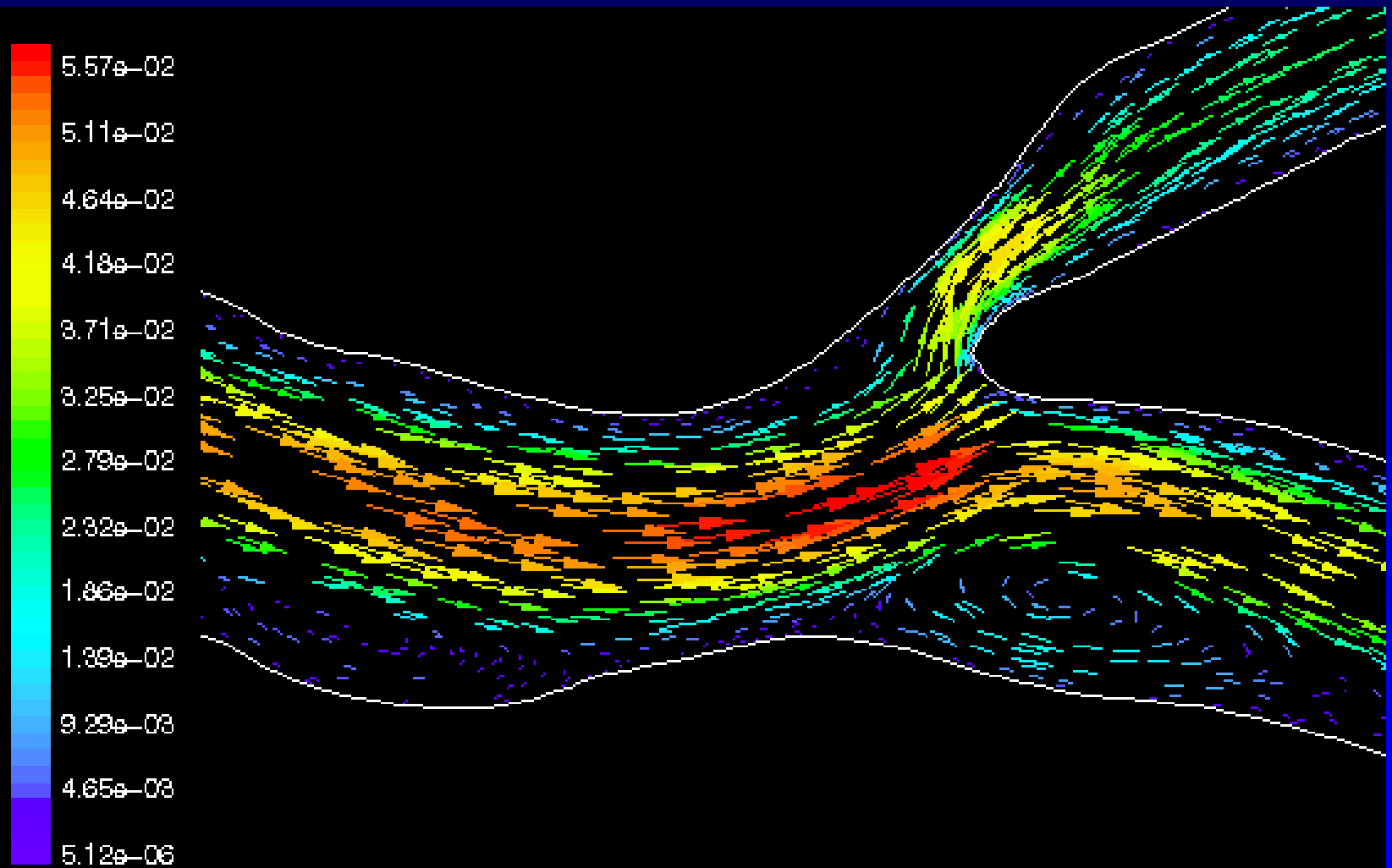


Velocity Vectors Colored By Velocity Magnitude (m/s) (Time= 1.1926×10^{-1})

Sep 26, 2000

FLUENT 5.3 (2d, dp, segregated, lam, unsteady)

T= 605msec

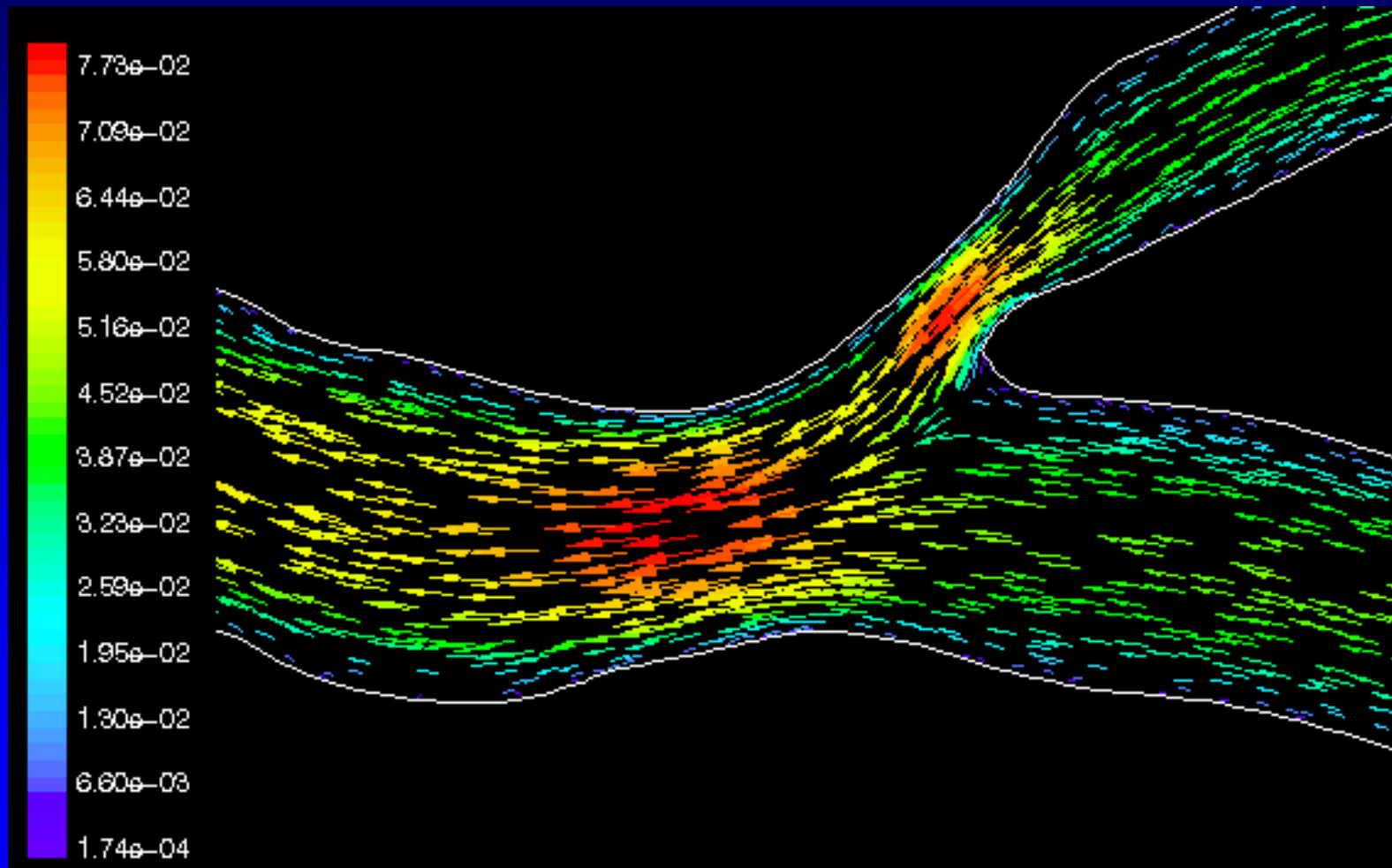


Velocity Vectors Colored By Velocity Magnitude (m/s) (Time=6.0550e-01)

Sep 26, 2000

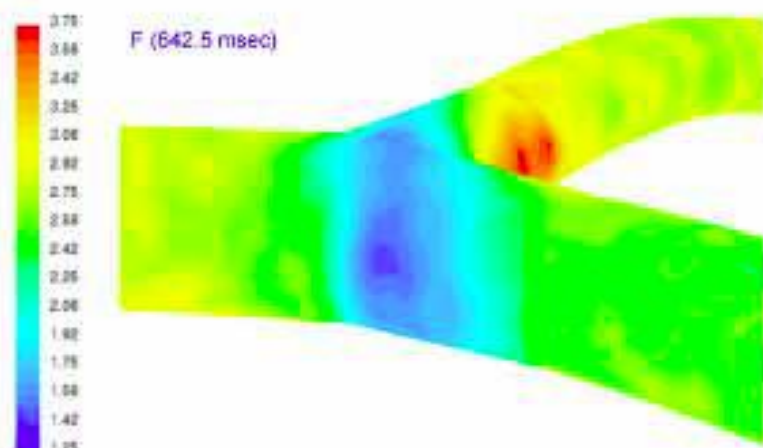
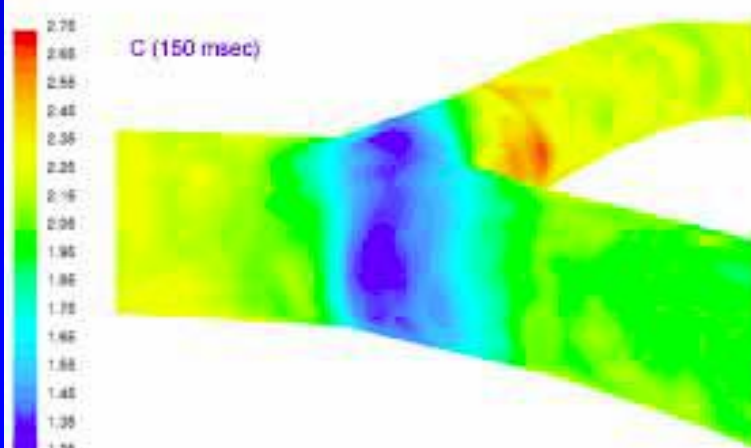
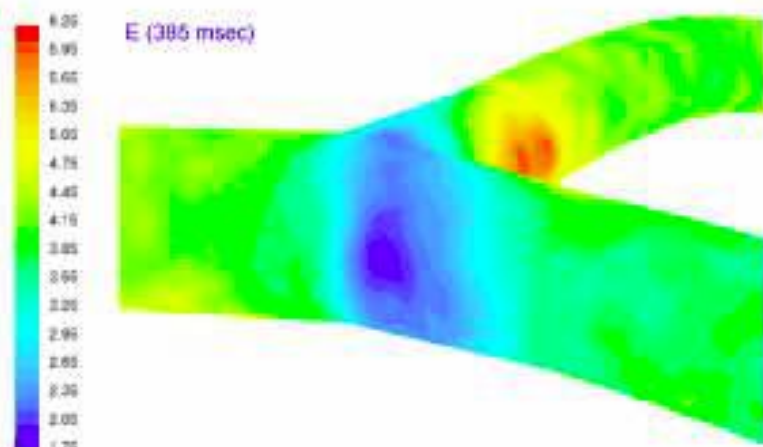
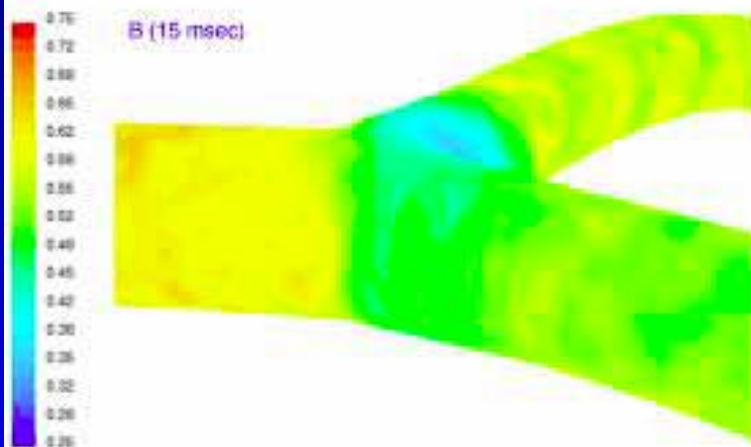
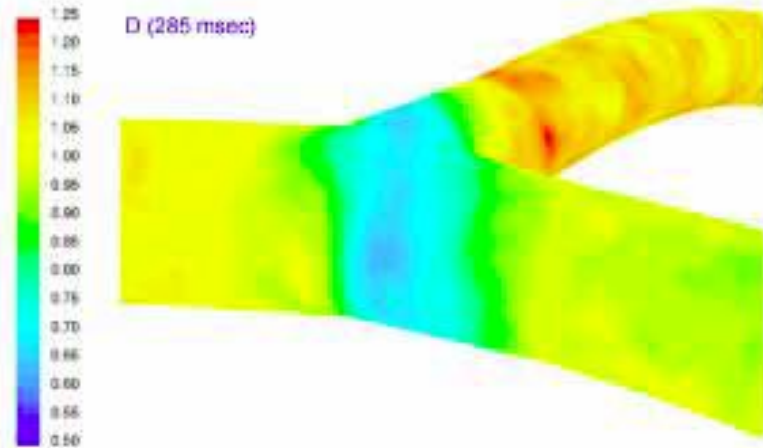
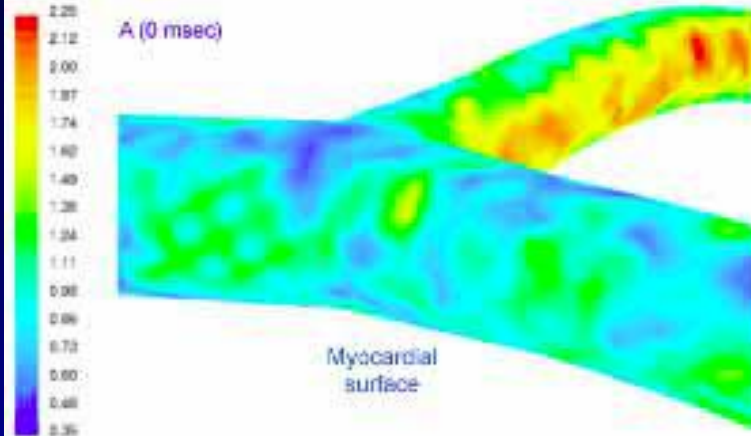
FLUENT 5.3 (2d, dp, segregated, lam, unsteady)

T= 905 msec



Velocity Vectors Colored By Velocity Magnitude (m/s) (Time=9.2659e-01)

Sep 26, 2000
FLUENT 5.3 (2d, dp, segregated, lam, unsteady)



CLINICAL IMPLICATION: Betablockers

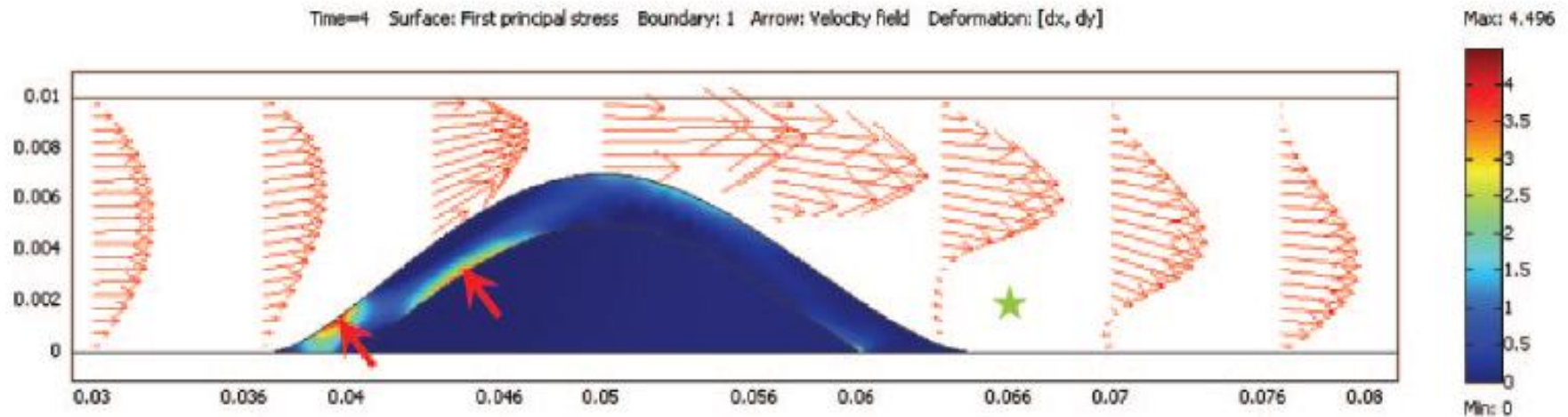
1. slow the heart rate,
2. prolong diastole,
3. constrict distal arteries by leaving the alpha receptors unopposed (so patient complain of cold hands and feet)
4. and so increase the wall shear stress

Observation 3

Acceleration and deceleration

A

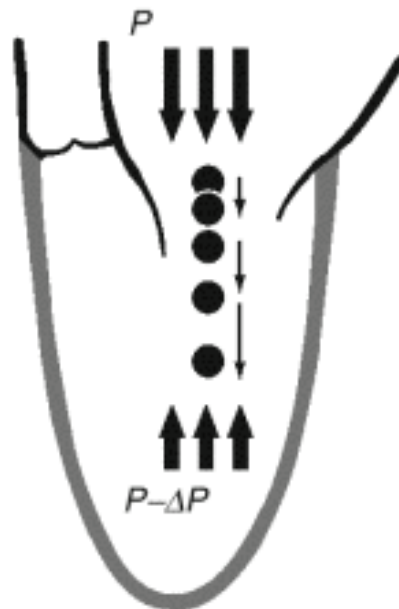
Time=4 Surface: First principal stress Boundary: 1 Arrow: Velocity field Deformation: [dx, dy]



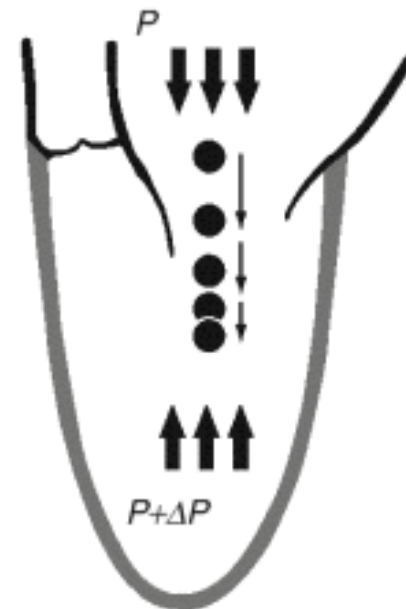
Connecting the Dots: During Diastole, With a Negative Pressure Gradient, the Coronary Flow Accelerates, while in Systole, the Flow Decelerates or Reverses Direction

Fig. 1.4 A fluid particle accelerates in presence of a negative pressure gradient (*left picture*) and decelerates in presence of an adverse pressure gradient

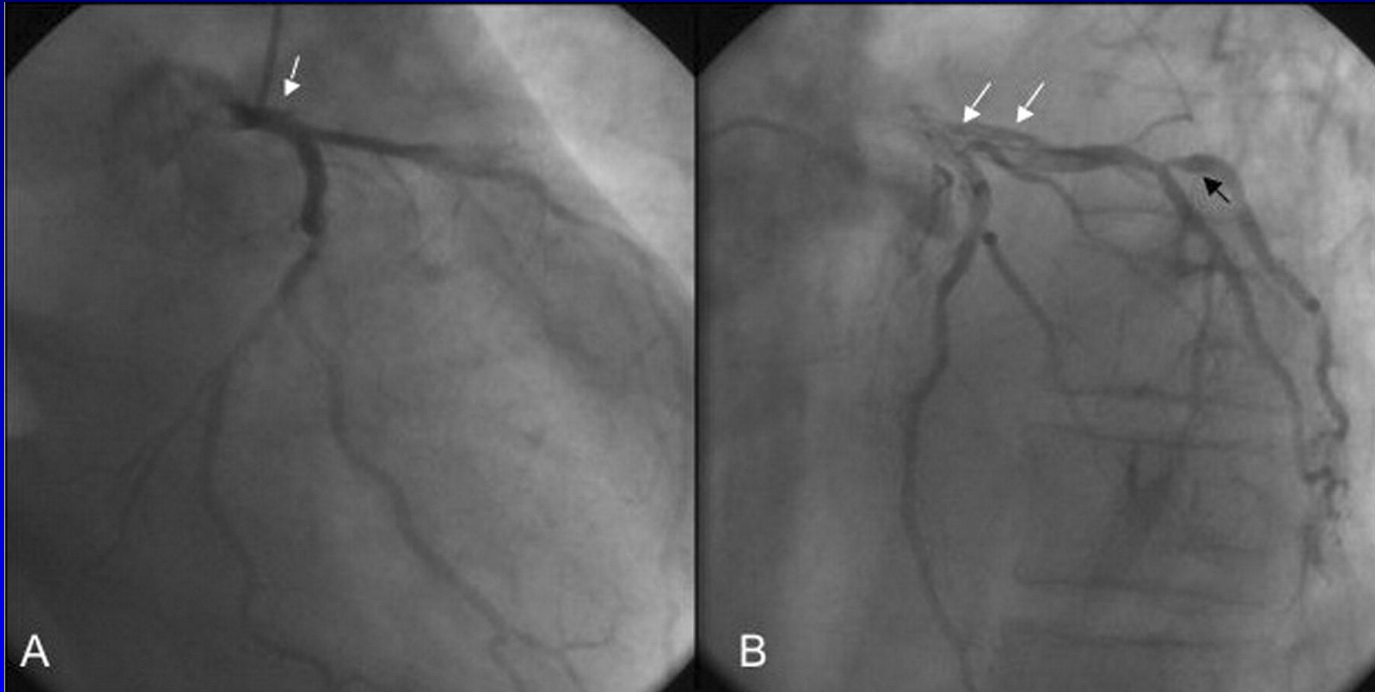
Negative pressure gradient
(*favourable, acceleration*)



Positive pressure gradient
(*adverse, deceleration*)



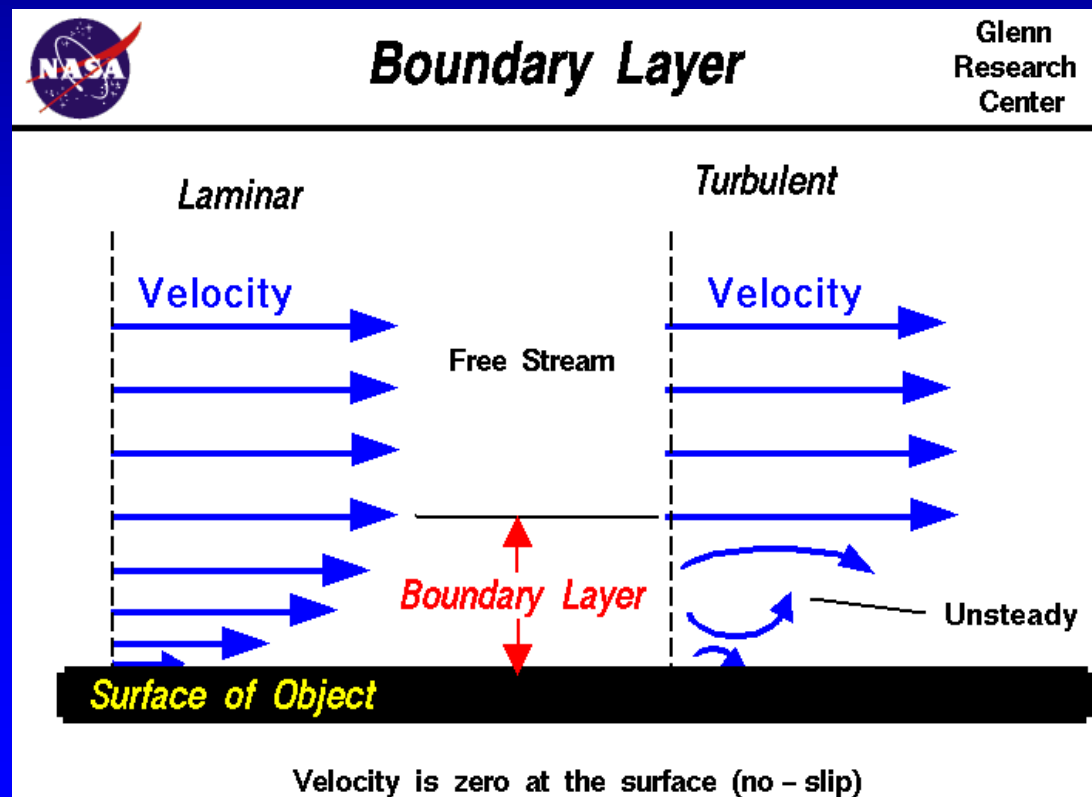
CONNECTING ALL THE DOTS 2: Coronary arteriograms show the dissection, which extended from the left main trunk to the proximal circumflex artery, the resultant 50% stenosis of the left main trunk (white arrows), and significant stenosis (90%) of the posterolateral branch (black arrow)

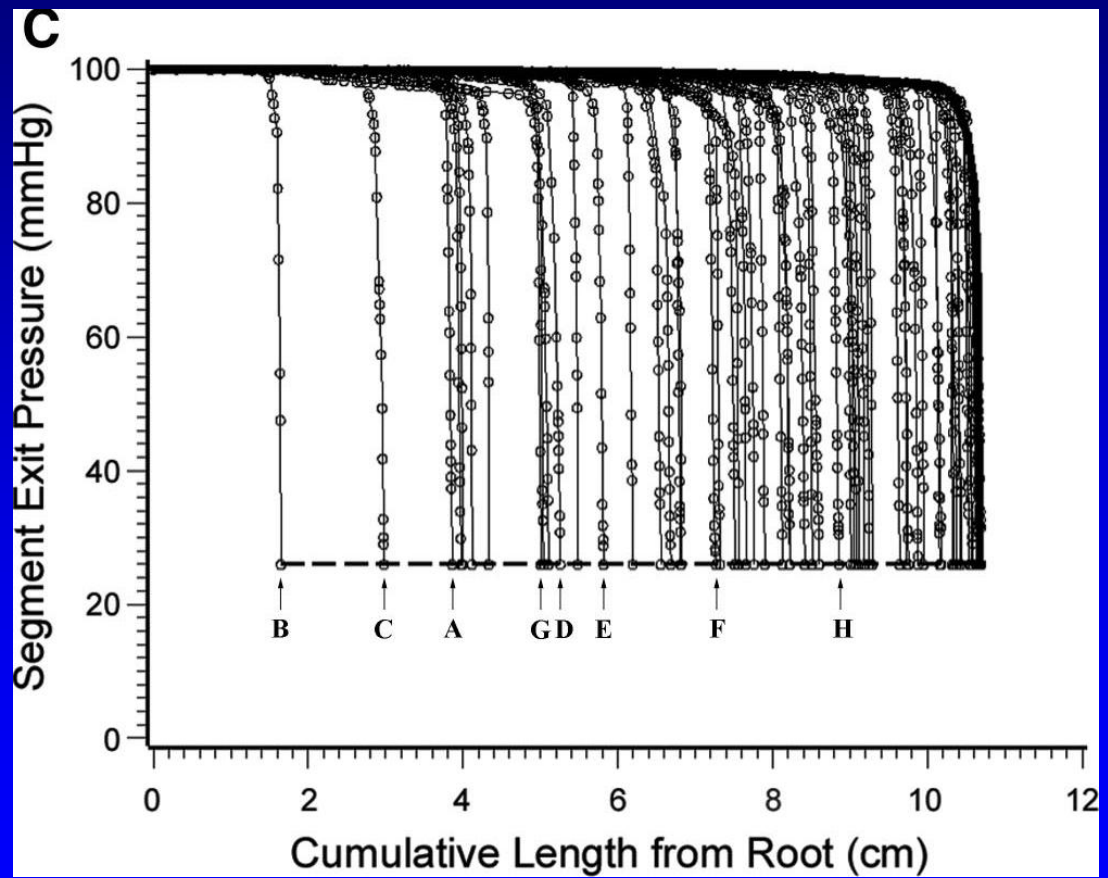
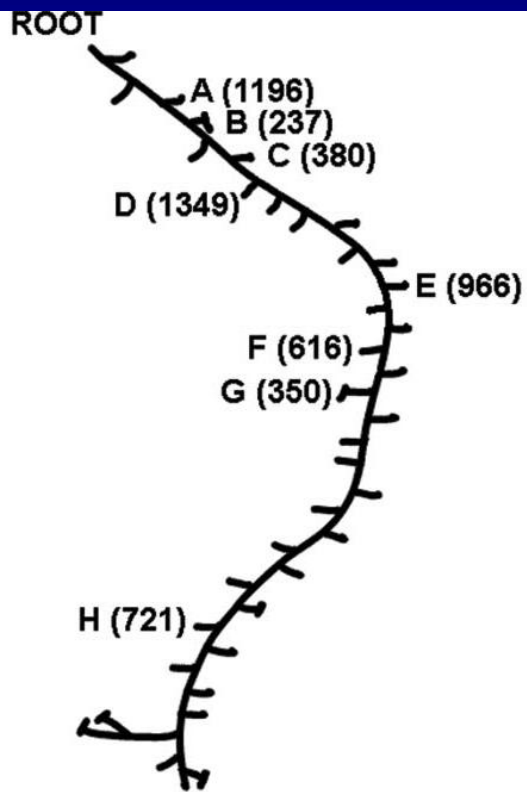


Aoyagi S. et al.; Ann Thorac Surg 2007;83:289-291

Observation 4

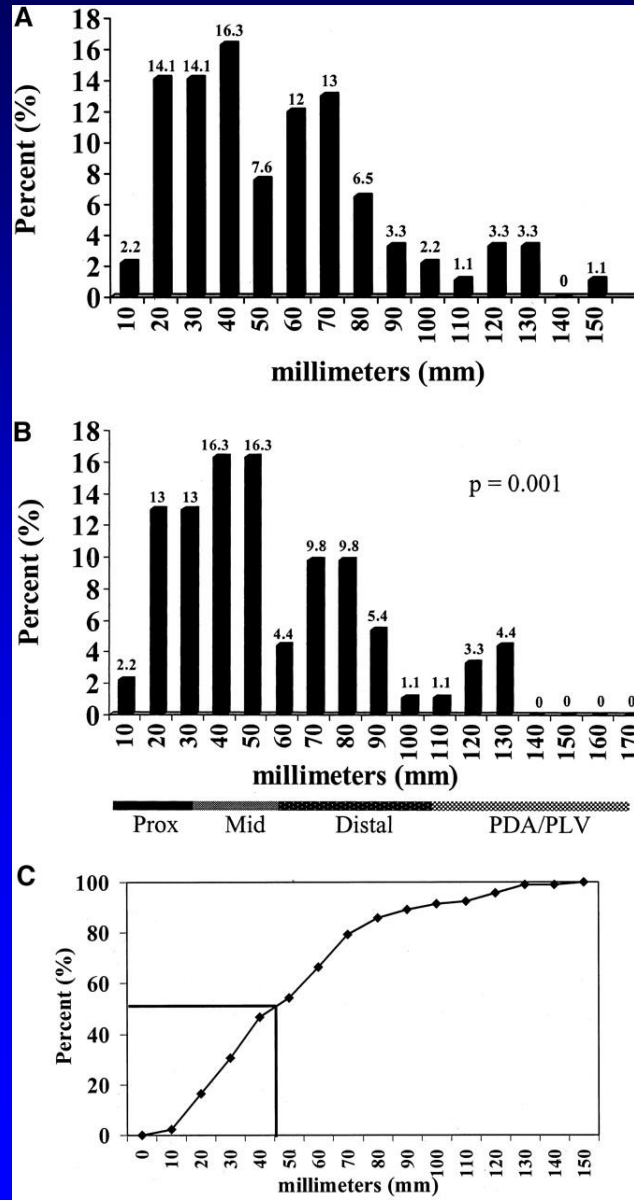
Boundary layer is the thin region between the wall and the external flow. When the thickness of the boundary layer is equal to the radius of the vessel, there is no more room to grow. Because the coronary flow is pulsatile, the boundary layer develops during on heart beat and restarts from zero in the next ones.





CONNECTING ALL THE DOTS

The majority of atherosclerosis happens in the proximal segment of any arteries (coronary arteries, aorta, etc)



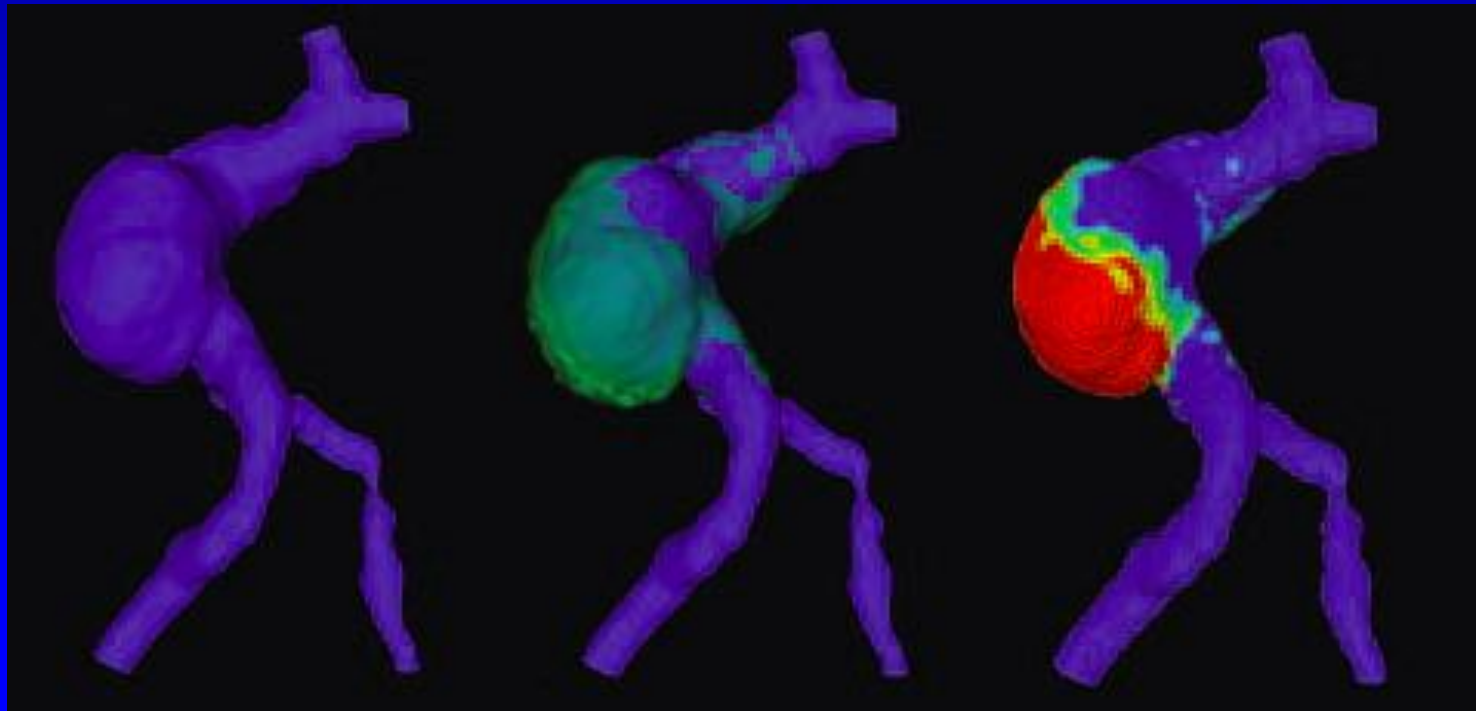
Wang, J. C. et al. Circulation
2004;110:278-284

Observation 5

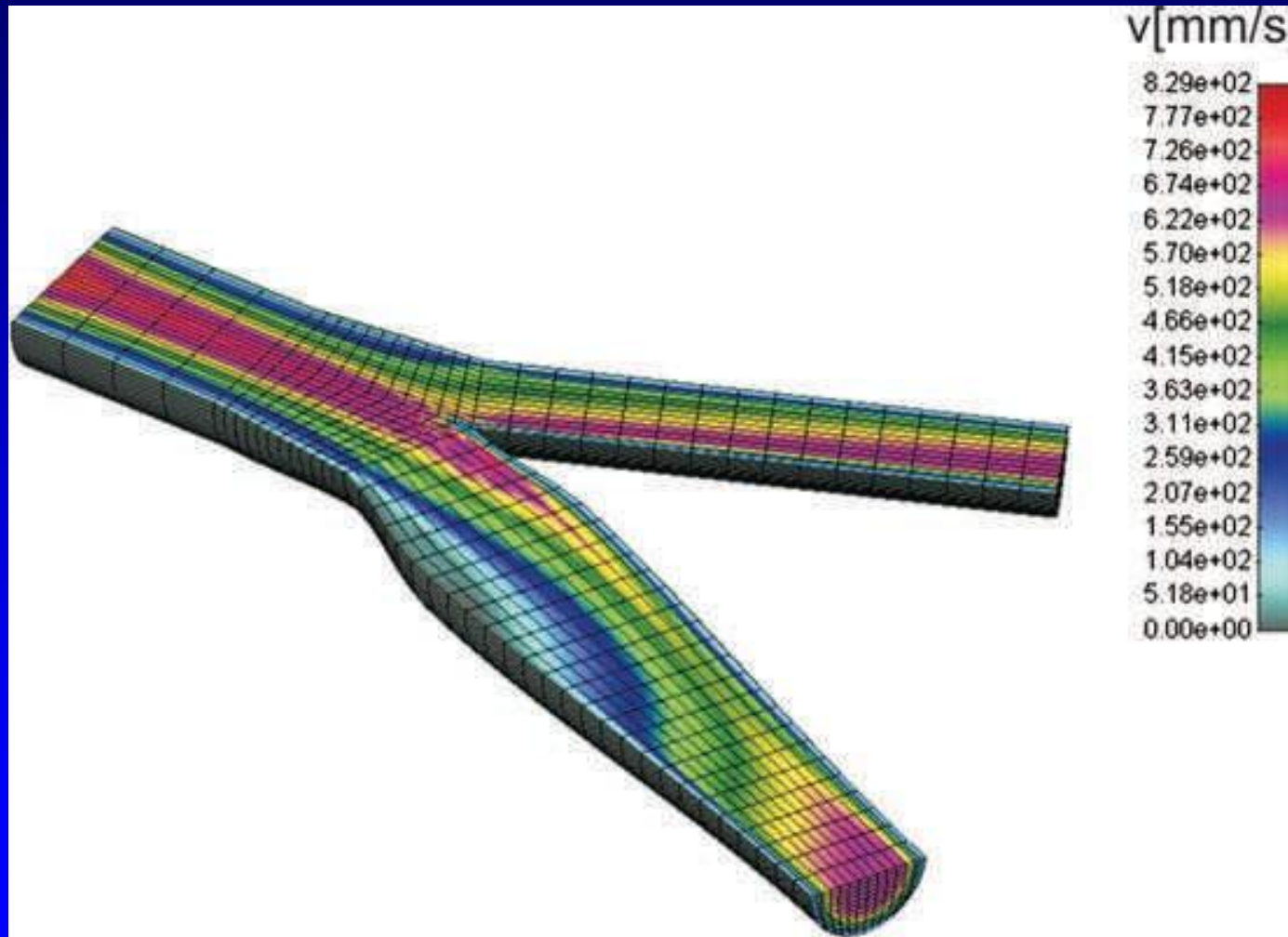
- **QUESTION:** What happens to the low shear stress areas in the cerebral vascular system (with antegrade flow during systole and diastole?)

-

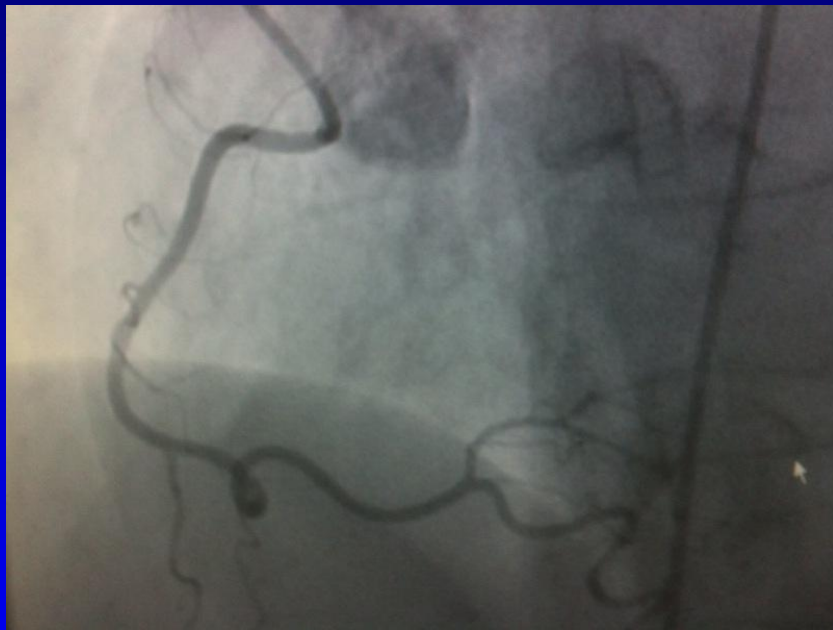
- Displacement map: The baseline shape (blue, left) is coregistered with the shape at the follow-up time point (green, middle). red indicates larger displacement; blue corresponds to an unchanged surface from one time point to the next.



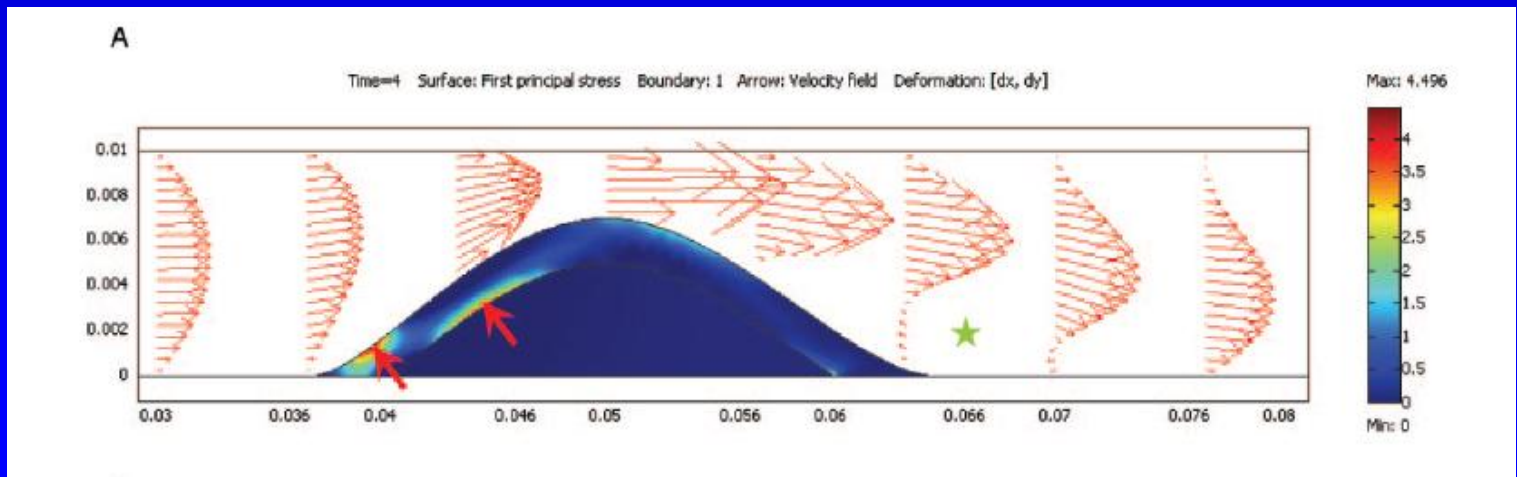
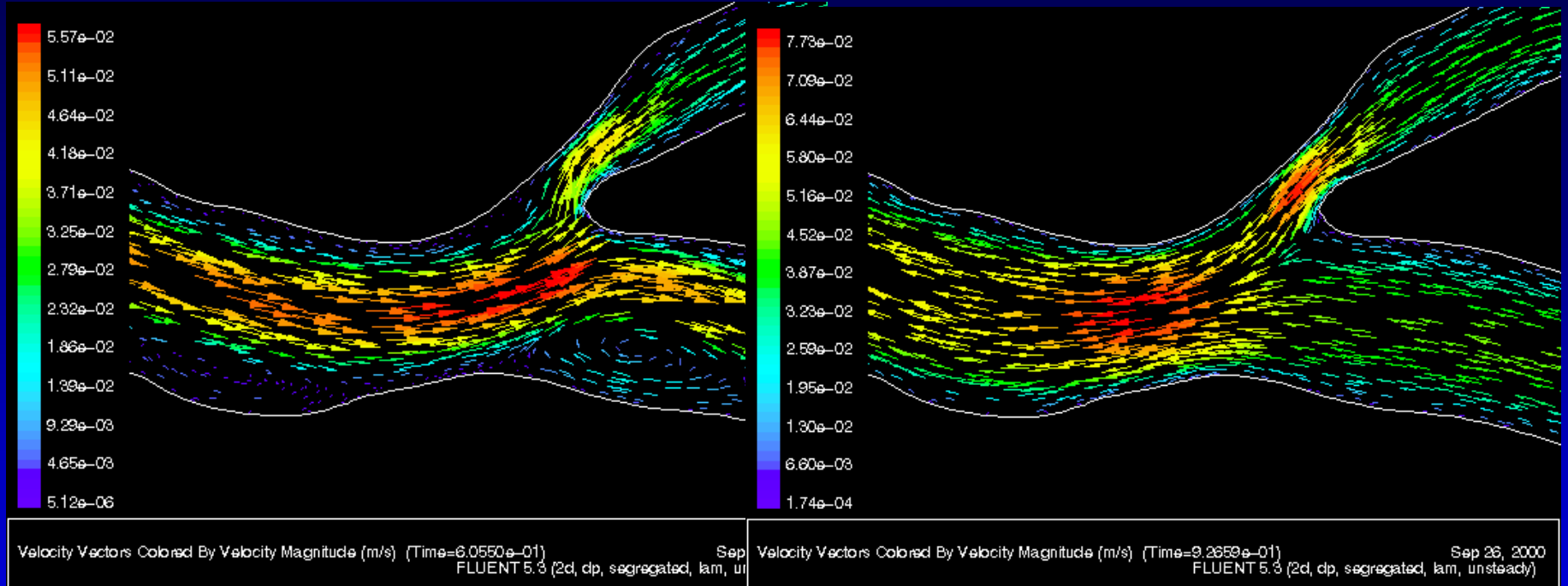
CONCLUSIONS 1



CONCLUSIONS 2



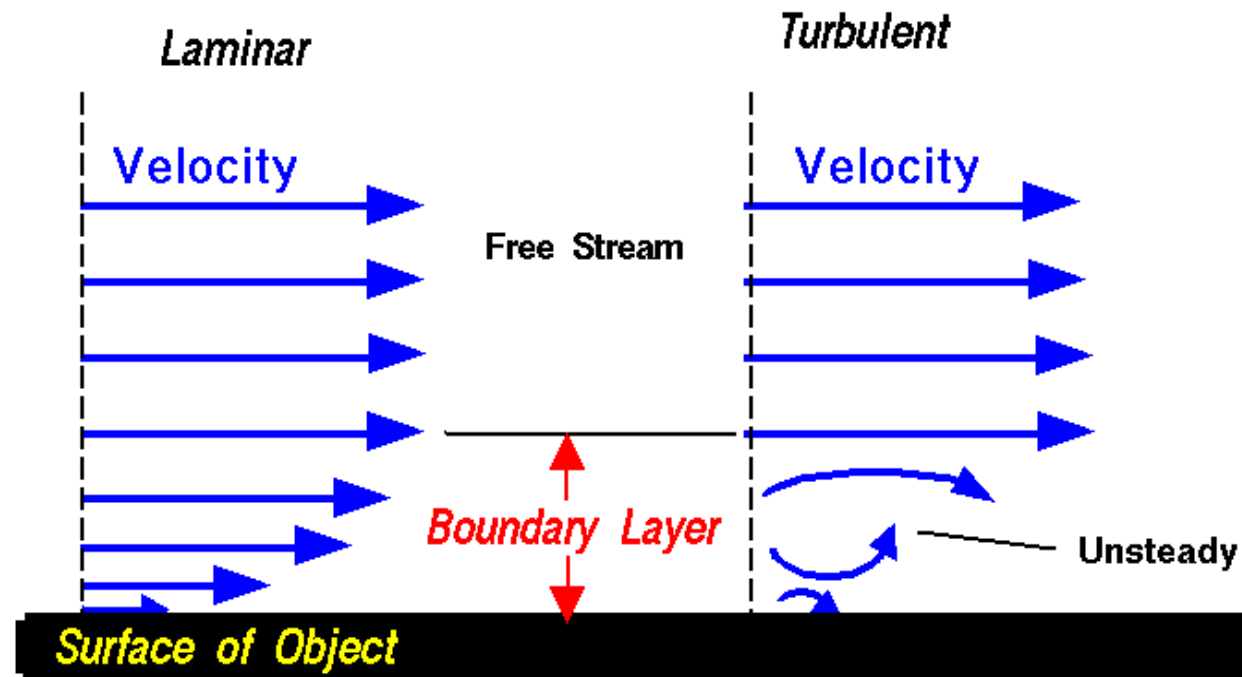
CONCLUSIONS 3



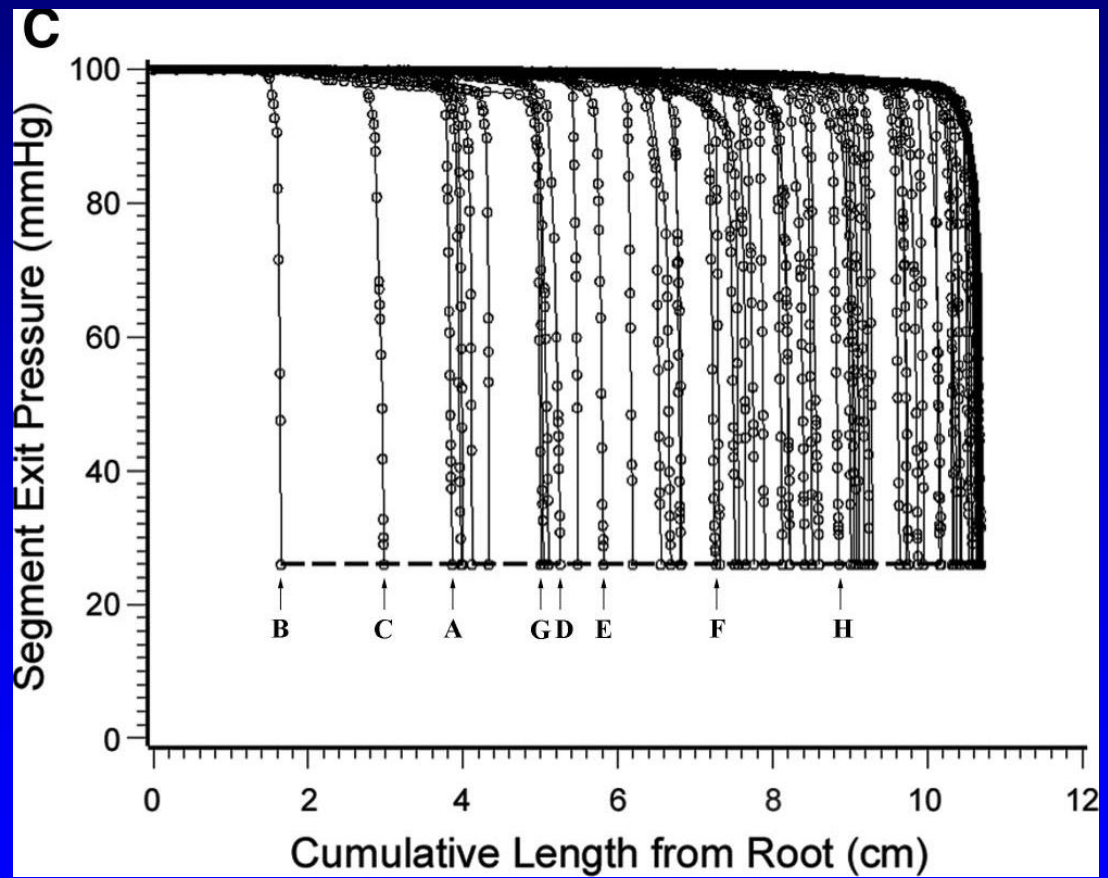
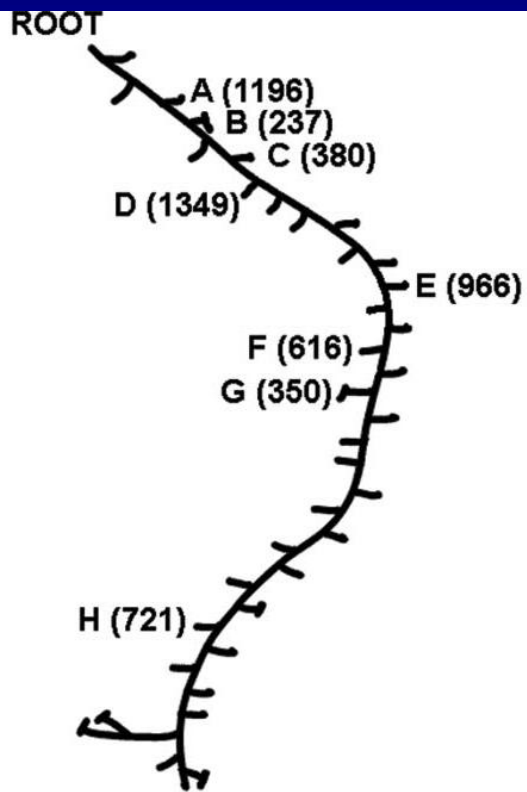


Boundary Layer

Glenn
Research
Center

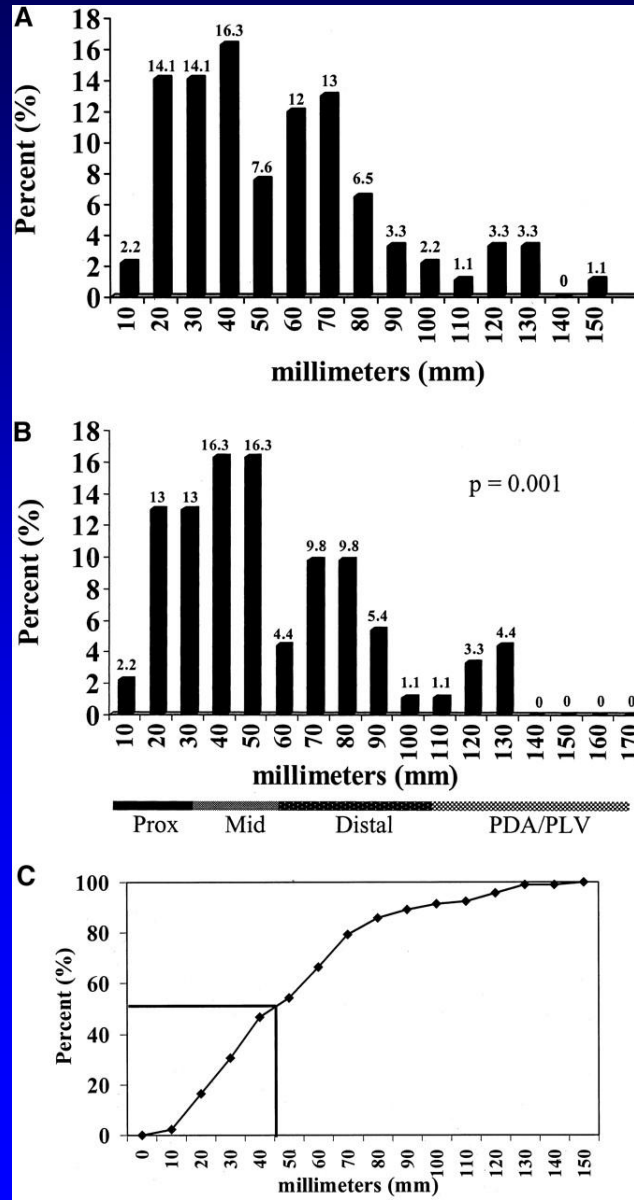


Velocity is zero at the surface (no - slip)

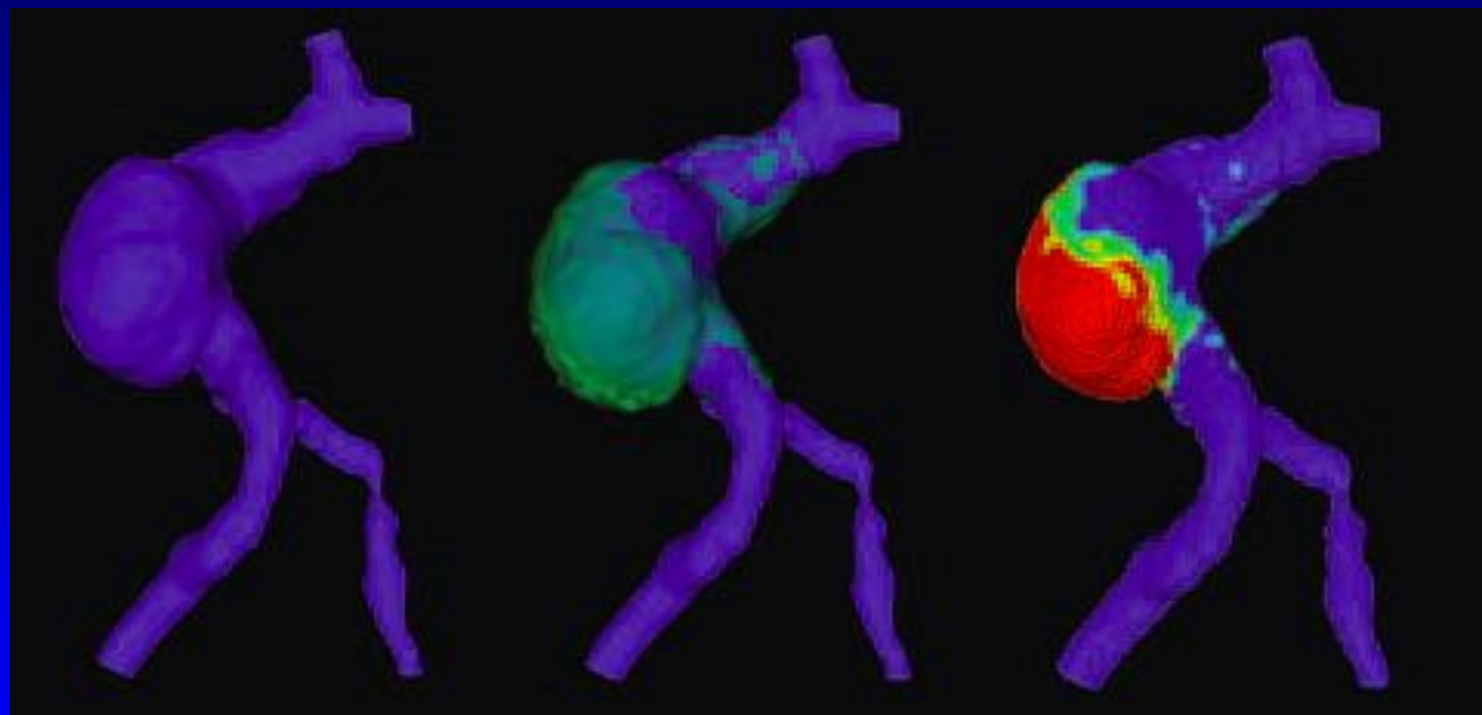


CONNECTING ALL THE DOTS 4

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CONCLUSIONS 4

- 1. Low cholesterol treatment
- 2. Exercise
- 3. Betablockers (prolong diastole, and distal vasoconstriction) Digoxin prolongs diastole however does not cause vasoconstriction. Alpha receptor blockers are worse