Fluid-Structure Interaction Studies of Coronary Artery Disease Biomechanics

M. Fandaros¹ , E. Steadman¹ , YY. Li² , JJ. Cao² , W. Yin¹

Department of Biomedical Engineering, Stony Brook University, Stony Brook, NY, USA
 Cardiac Imaging, DeMatteis Center for Cardiac Research and Education, St. Francis Hospital, Roslyn NY, USA

Coronary Artery Disease kills 1 in 7 in the United States

 Coronary artery disease is the most common form of heart disease[1] which can result in heart attack or other serious thrombotic events



- Cells in the blood and blood vessels are known to respond to alterations of the mechanical forces of their environment
 - Wall shear stress
 - Tensile strain
- Computational Models of the coronary arteries are commonly used to estimate these parameters to evaluate disease development

Background of Typical Models media Superior vena cava Left pulmonary artery plaque Left pulmonary veins Aorta Left coronary artery Right atrium Left circumflex artery Endothelial **Right coronary** artery lining Left marginal artery Posterior descending artery Left anterior descending (or interventricular) artery LAD diagonal branch Right marginal artery Left ventricle **Right ventricle** [3] 100mm 010

General FSI Set Up and Boundary Conditions



Modeling the Arterial Wall Material Properties

5-Parameter Mooney Rivlin model	C ₁₀ (kPa)	C ₀₁ (kPa)	C ₁₁ (kPa)	C ₂₀ (kPa)	C ₀₂ (kPa)	Tunica media Tunica extern
Media	9.26	3.50	1183.00	305.46	504.50	Endothelial cell
Fibrous Plaque	28.49	8.63	56.75	150.48	2721.00	Smooth muscle of Perivascular adig tissue cell

Mooney Rivlin constants for LAD media layer [4]

Endothelial cell Smooth muscle cell Perivascular adipose tissue cell Fibroblast cell Collagen fiber Nerve endingr Vascular Wall Cross section [5]

Lumen Tunica intima

Model 1: Mesoscopic Stenosis FSI Model

- Key Features and Improvements
 - Maximum resolution in the stenosis ROI:
 26 μm; avg resolution 56 μm.
 - Comparable to vascular endothelial cells (20-100µm).
 - 2 materials: Media material in the healthy segments, and fibrous plaque material in the stenosis region representing increased stiffness of stenotic vessels.
 - A physiologically accurate stenosis morphology
 - 71% occlusion severe stenosis, candidate for clinical intervention



from histological cross-sections. [6]

Model 2: Macroscopic Normal FSI Model

- Key Features and Improvements
 - Detailed geometry extracted from patient-specific CTA scan
 - Total length = 7.56cm with increased total volume compared to other coronary artery models
 - Average element size = 166µm



Model 1: Mesoscopic Stenosis Model Results





Model 1: Stenosis Model Results Cont.

time (s)	initial velocity (m/s)	Cardiac Squeezing Pressure (kPa)	peak vel (m/s)	avg vel (m/s)	peak WSS (Pa)	average WSS (Pa)	peak tensile strain	average tensile strain
0.025	0.044	0	1.1393	0.07941	82.786	0.8749	0.02841	0.00367
0.2	0.12	0	2.7834	0.23461	328.05	2.6736	0.07046	9.6499E-4
0.6	0.02	1.86	0.5756	0.03173	30.639	0.3422	0.08044	4.6427E-4



Model 2: Macroscopic Normal Model Results





Model 2: Normal Model Results Cont.

time (s)	initial velocity (m/s)	phase	Cardiac Squeezing Pressure (kPa)	peak vel (m/s)	avg vel (m/s)	peak WSS (Pa)	average WSS (Pa)	peak tensile strain	average tensile strain
0.025	0.11	Early diastole	0	0.30552	0.096227	11.047	0.91334	0.15848	0.0026173
0.2	0.3	Mid diastole	0	0.76613	0.26878	40.544	2.6236	0.13185	7.68E-04
0.6	0.038	Mid systole	1kPa	0.11924	0.032947	3.3544	0.30594	0.16379	0.0029305

Conclusions

- Successful use of 3D patient-specific geometries to perform FSI modeling in COMSOL on both macro and meso scale
- Using MR material constants from the media layer created reasonable deformations in the arterial wall during normal and stenosis conditions
 - With and without compression
- Higher than expected WSS results in stenosis model
- In normal model, fluid velocity, WSS, and tensile strain are within expected values



Future Directions

- Patient-specific 3D models can be created with patient-specific inlet and outlet pressures serving as BC's for rigorous validation
- Assessing vulnerability of plaques
- Particle tracing of platelets in the stenosis model
 - High spatial resolution intended to allow for modeling of cellular-level interactions which affect thrombosis.



Thank you!

For additional questions or comments, please contact us at:

- Marina Fandaros: <u>marina.Fandaros@stonybrook.edu</u>
- Elisabeth Steadman: <u>elisabeth.Steadman@stonybrook.edu</u>
- Dr. Wei Yin: <u>wei.yin@stonybrook.edu</u>



References

[1] Benjamin, Emelia J., et al. "Heart disease and stroke Statistics-2019 update a report from the American Heart Association." Circulation (2019).

[2] CDC Interactive Atlas of Heart Disease and Stroke, deaths 2019,

https://nccd.cdc.gov/DHDSPAtlas/?state=County

[3] "File:Coronary arteries.png", <u>https://commons.wikimedia.org/wiki/File:Coronary_arteries.png</u>
[4] Teng et. al., "The influence of constitutive law choice used to characterise atherosclerotic tissu material properties on computing stress values in human carotid plaques", JBioMech, 48, pp. 3912-3921, 2015

[5] Zhao, Yingzi, Paul M. Vanhoutte, and Susan WS Leung. "Vascular nitric oxide: Beyond eNOS. Journal of pharmacological sciences 129.2 (2015): 83-94.

[6] Brown, Bolson & Dodge. "Dynamic mechanisms in human coronary stenosis." *Circulation.* Vol 70 pp 917-922. 1984.