FSI Simulation of Arterial Stenoses

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BACKGROUND

The partial occlusion of arteries due to mural thrombosis and embolization is one of the most frequently occurring abnormalities, like unstable angina, myocardial infarction, and sudden ischemic death [1] in man. Once an obstruction has developed, the flow of blood will be disturbed, so does a series of rheological and fluid dynamic parameters, which plays an increasing important role as the stenosis continues to develop. The study of physiologically pulsatile flow through thrombosis has a profound implication for diagnosis of vascular disease. Numerous studies performed in the past assumed geometry models generated from *in vivo* data, demonstrated on a simple tube stenotic model [2]. Axisymmetrical and nonsymmetrical models with different stenosis portion were analyzed by *in vitro* experiments [3]. In this project, numerical simulation will be carried to analyze different models.

PROBLEM FORMULATION

Although the actual geometry of a stenotic artery may be varied and complex, it can be simplified as axisymmetrical or nonsymmetrical constriction in a cylindrical tube [4]. The geometry of the blood vessel is shown in Figure 1.

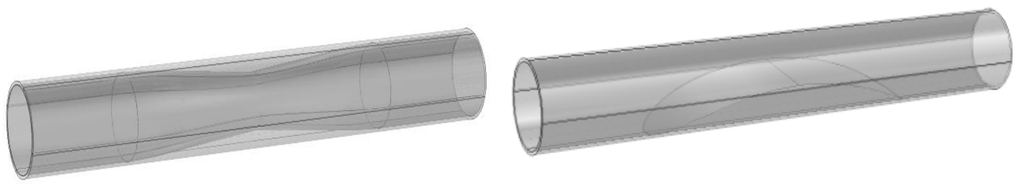


Figure 1. Axisymmetrical and nonsymmetrical geometries.

The geometry of the axisymmetrical stenosis was produced in [3],

$$R(z) = R_0(1 - \frac{\sigma}{2R_0}(1 + cos(\pi(\frac{z - Z_0}{Z_0}))))$$

where Z_0 is half length of the constriction, R_0 is radius of vessel, σ is maximal height of stenosis. $R(z)$ is radius in stenotic part. Nonsymmetrical thrombosis is generated by lateral intersection of two cylinders.

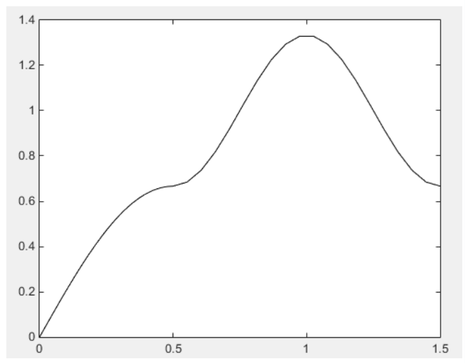


Figure 2. trigonometric function

In this project, the inlet and outlet boundary condition is set by pulsatile pressure for a period of 1.5 sec, established by a trigonometric function varies from maximal value and minimal value of pressure as shown in Figure 2. Mean pressures of inlet and outlet are 126.09 mmHg and 125.1 mmHg separately.

33%, 66% axisymmetrical, and 54%, 83% nonsymmetrical models with different stenosis length were analyzed.

Model Type	$R_0(mm)$	$\sigma(mm)$	$\frac{Z_0}{R_0}$	$\zeta(mm)$	Percent Stenosis (%)
Axisymmetrical	9.5	3.1	4		33
	9.5	6.2	4		66
	9.5	6.2	2		66
Nonsymmetrical	9.5	10.3	4	74.7	54
	9.5	15.7	4	53.3	83
	9.5	15.7	2	19.3	83

Table 1. Axisymmetrical and nonsymmetrical geometries parameters

The model consists of three parts: blood, artery and muscle. Muscle presents a stiffness which resists artery deformation due to applied pressure. The blood is considered to be Newtonian since the in large arteries, velocity and shear rate will be high [5]. The density and dynamic viscosity of the blood are considered to be 1060 kg/m^3 and 0.005 Ns/m^2. Since the displacements and strains are so small that it is sufficient to consider the artery to be linearly elastic material. The material data is given for a Neo-Hooken hyperelastic material, but due to small strain limit, the material constants is the same for a linear elastic material [6]. The artery has density of 960 kg/m^3, with Poisson's ratio of 0.45, Neo-Hookean hyperelastic coefficient μ of $6.20 \cdot 10^6$ N/m^2, and elastic modulus is $1.0 \cdot 10^7$ N/m^2. The muscle has density of 1200 kg/m^3, with Poisson's ratio of 0.45, Neo-Hookean hyperelastic coefficient μ of $1.16 \cdot 10^6$ N/m^2, and elastic modulus is $7.20 \cdot 10^6$ N/m^2.

We want to study how fluid flow can deform structures and how a continuously deforming geometry will influence fluid flow.

Solution

Fluid-Structure Interaction (FSI) method is used to conduct blood flow analysis. The FSI solves fluid and structure domains independently. After the fluid domain is solved, the surface of structure domain is transferred by fluid domain surface, and structure domain is solved. Consequently, the fluid mesh has to be changed using Arbitrary Lagrangian Eulerian (ALE) [8], which provides a way to use both Lagrangian and Eulerian descriptions with reference to the third coordinate system. While for structure part, Lagrange description is used. The problem of seam at the interface between the structure and fluid caused by mesh moving is solved by using Laplacian mesh smoothing.

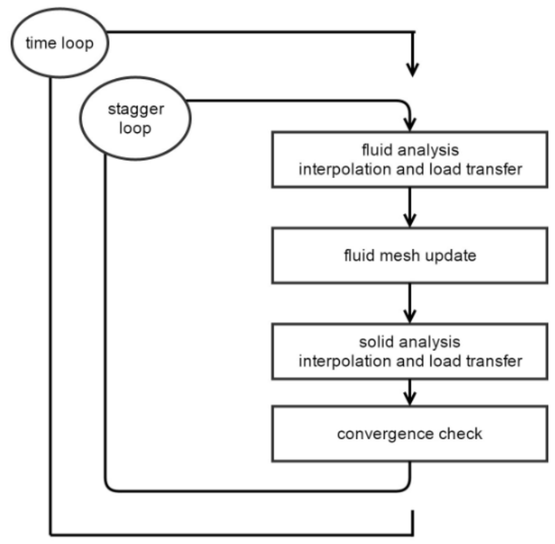


Figure 3. FSI algorithm

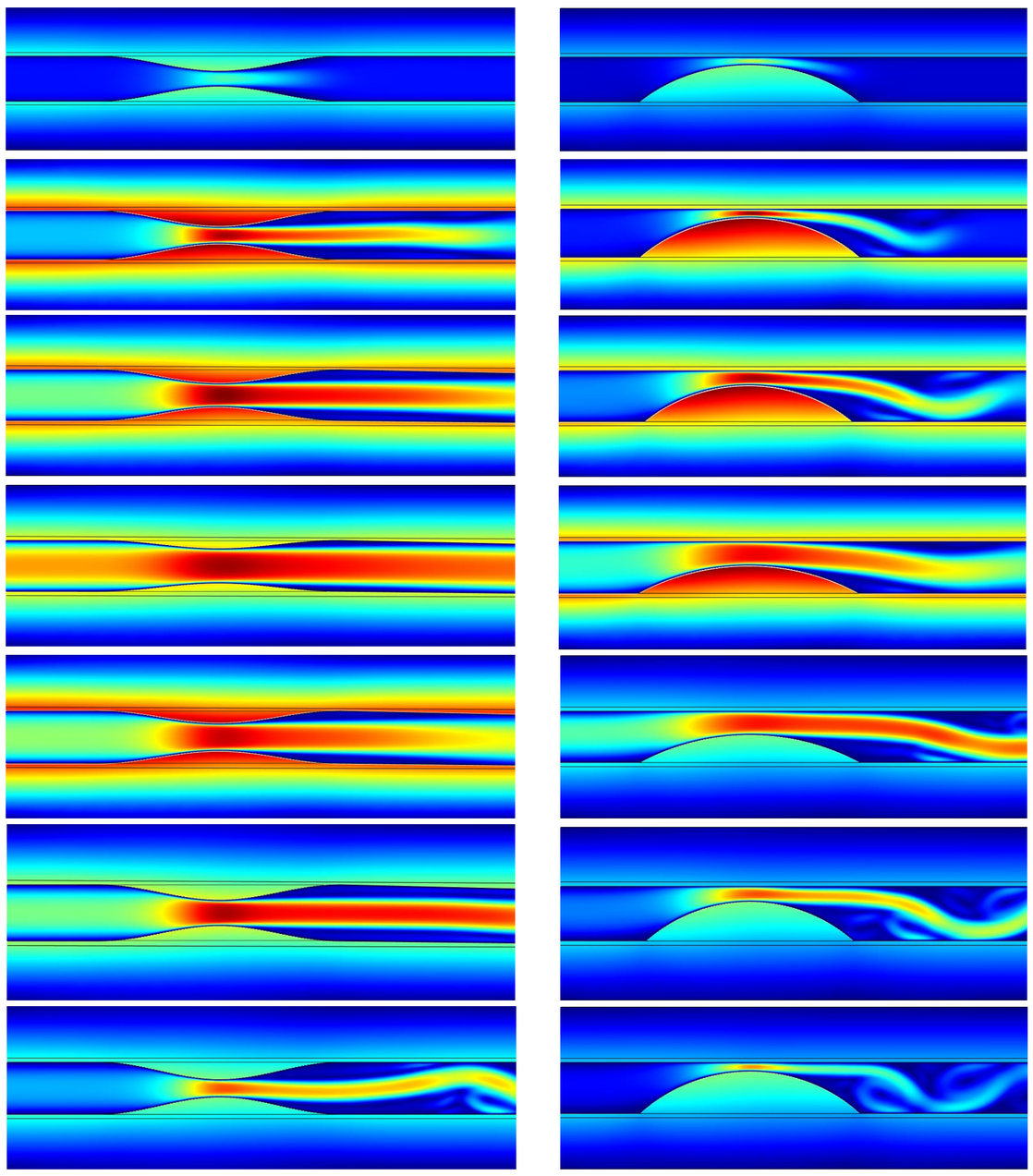
The blood flow in artery is governed by the Navier-Stokes equations of incompressible flow. [7]

$$\rho\frac{\partial \mathbf{u}}{\partial t} - \nabla \cdot [-p\mathbf{I} + \eta(\nabla \mathbf{u} + (\nabla \mathbf{u})^{\mathbf{T}})] + \rho((\mathbf{u} - \mathbf{u}_m) \cdot \nabla)\mathbf{u} = \mathbf{F}$$

$$-\nabla \cdot \mathbf{u} = 0$$

where **u** is the velocity field and $\mathbf{u_m}$ is the coordinate system velocity with the pressure p, the volume force affecting the fluid **F**.

RESULTS



Summary and Source

As velocity and displacement contour shown in results, which explains the difference between axisymmetrical and nonsymmetrical thrombosis. Only models with largest portion stenosis are shown due to the poster size. The velocity field varies in each time step. Area with stenosis, where flow gain momentum to keep a high speed, is characterized with high velocity. The deformation of thrombosis and artery is found to be maximum during the peak systole at the throat area. And there will be turbulent during diastole part, which will lead sedimentation of platelet and particles, increasing thrombosis formation. This will be left for future study.

Reference:

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