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Using Computational Fluid Dynamics Model to Predict Changes in Velocity properties in Stented Carotid Artery

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Abstract:
Atherosclerosis is a disease that narrows, thickens, hardens and restructures a blood vessel due to substantial plaque deposit. In the Carotid Artery, the decision to treat using endarterectomy and stenting is determined by the velocity as measured by Doppler flow in the common Carotid Artery. The measured Doppler velocity as compared with the contra-lateral side has been correlated with the degree of stenosis. In some patients, the Doppler velocity after stenting is equivalent to or higher than before treatment. The hypothesis we are testing is that higher velocity measured after stenting may be due to the decreased compliance of the artery wall at the stent region. Geometric models of blood flow through an idealized carotid artery of different types and several degrees of constriction ranging from 0% to 100% were modeled.

The overall objective of this paper is to create a computational model with pulsatile flow and walls with physiological compliance. The pressure variations in the compliant stenotic artery are subjected to prescribed pulsatile waveform. The computational simulations were performed in which the physiological flow through compliant axisymmetric stenotic blood vessel (carotid artery) was solved using commercial software COMSOL. COMSOL multiphysics[1] is used to solve this challenging problem involving fluid-structure interaction. Here we have demonstrated comparisons of the centerline velocities V_0=0.40 m/s and V_{55}=0.53 m/s with stenosed rate 0% and 55% respectively with the fluid-structure interaction and their velocity profiles under the physiological flow conditions in the compliant stenotic artery. With these values, flow prefers to avoid the stenosed region which causes additional load on the section with the stenosis. Therefore the structure seemed to translate marginally towards the stenosed section at bifurcation junction. Huge compliance mismatches though, would not be desirable especially due to greater deformations experienced by the arterial walls which affected the centerline velocity.

1 Introduction

Atherosclerosis tends to develop in preferred sites, such as the bifurcations and flow divisions of the arteries. The development and progression of the atherosclerosis are related to the complex flow field occurring in the bifurcations of the arteries. It is widely known that there is the correlation between atherosclerosis lesion location and low or oscillating wall shear stress (Karino et al., 1988; Ku et al., 1985).

Many cardiovascular diseases are closely associated with the flow conditions in the blood vessel. One major type of arterial disease is atherosclerosis which is characterized by localized accumulation of cholesterol and lipid substances. It is commonly referred to as a “hardening” of the arteries. It is caused by the formation of multiple plaques within the arteries.

The symptom of atherosclerotic cardiovascular disease is heart attack or sudden cardiac death. The most artery flow disrupting events occur at locations with less than 50% of lumen narrowing (~20% stenosis is average) (Glagov et al., 1987)[2]. According to cardiac stress testing, traditionally the most commonly performed non-invasive testing method for blood flow limitations generally only detects lumen narrowing of ~75% or greater. From clinical trials, 20% is the average stenosis at plaques that subsequently rupture with resulting complete artery closure (Glagov et al., 1987). Most severe clinical events do not occur at plaques that produce high-grade stenosis. Only 14% of heart attacks occur from artery closure at plaques producing 75% or greater stenosis prior to the vessel closing. “FSI (Fluid Structure Interaction) occurs when a fluid interacts with the solid structure, exerting pressure on it which may cause deformation in the structure and thus alter the flow of fluid itself”. A fully coupled fluid-structure interaction means that the response of the solid is strongly affected by the response of the fluid, and vice versa. Thus, basic understanding of fluid-structure interaction is essential to understand these effects in cardiovascular system.

In the Carotid Artery, the decision to treat using endarterectomy and stenting is determined by the velocity as measured by Doppler flow in the common Carotid Artery. The measured Doppler velocity as compared with the contra-lateral side has been correlated with the degree of stenosis. In some patients, the Doppler velocity after stenting is equivalent to or higher than before treatment. The hypothesis we are testing is that higher velocity measured after stenting may be due to the decreased compliance of the artery wall at the stent region.

An FSI model becomes essential to achieve a basic understanding of effects of various mechanical forces involved in arterial disease development. The present study is concerned with physiological flow both in elastic and rigid wall vessels with 0 and 50 % constriction of cross-sectional area in stenosed arteries. The objective of this study is to understand how the flow features and wall shear stress field change with the development of stenosis by numerically analyzing the interactions between a blood flow and a stenosed wall. In the present study, we investigate deformation along the wall and velocity field, in the different type of constriction of the blood vessel.

2 Formulation of the Problem

Under certain conditions blood behaves as a Non-Newtonian substance. Therefore, when modeling the flow of blood it is inherently critical that the fluid properties are properly formulated. Studies show that the fluctuating blood viscosity is of negligible significance where the inertance of the blood is relatively large compared to the resistance [6]. Thus keeping constant viscosity through the model. Using the generalized Navier-Stokes equations which define a differential change in momentum within the blood is given by:

$$\rho\partial u/\partial t - \nabla[\eta(\nabla u+(\nabla u)^T)]+ \rho(u\nabla)u + \nabla p=F \quad (1)$$

Where ρ is the density of blood, η is the dynamic viscosity, and u is the velocity. This relation coupled with the field continuity equation $\nabla u=0$, completely defines the dynamics of blood flow through the artery. They are valid for the assumptions of the blood being Newtonian, incompressible and having a constant viscosity throughout. This relation is implemented within Comsol using the Incompressible Navier-Stokes (ns) mode. To solve the Navier-Stokes equations we introduce the boundary conditions being the known values of pressure at both the end of the blood domain.

The structural mechanics of the artery is defined by the global equilibrium equations based on stress components. In a general form, this is given by:

$$-\nabla\sigma = 0 \quad (2)$$

where σ is the stress tensor. The artery itself was taken to consist of a Neo-Hookean hyperelastic material. This extension of Hooke's law is thought to better approximate the stress-strain relationship that exists within the artery. In the Comsol model this is realized by the Axial-Symmetric stress-strain mode. This is an approximation to the full three dimensional formulation and assumes loads only in the radial and axial directions. The boundary conditions introduced within the structural mechanics module take into account the coupling of the two physics. This is done by taking the computed pressure obtained from the Navier-Stokes equations and utilizing this as a load onto the artery walls

3 Comsol Model

Figure 1 show the axial 3D symmetric model used in this study. The stented carotid artery consists of three major segments: stent, proximal and distal. The general dimensions are chosen to represent our model. Ls = 0.025 m, hs=0.0025m, h=0.0005m and Do = 0.0055, which results in hs/Do= 0.5. The entire model was similarly defined as extended Y model. Modulus of elasticity for artery wall is Ea= 10^7 Pa [7]. Poisson's ratio for the stented portion was taken as 0.33 as of stainless steel which is considered to be homogenous and isotropic.

The model is built using an axis symmetric stress-strain analysis type along with the time dependent incompressible Navier-Stokes module

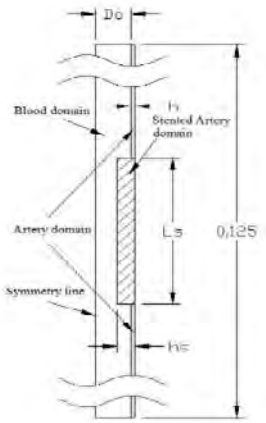

Figure 1: General Dimensions of the Model

Flow is assumed to be Newtonian, incompressible, viscous and laminar. The dynamic viscosity of blood is taken as 0.005 Ns/m^2, and the density of the blood is taken as 1060 Kg/m^3. Flow through the vessel is driven by a pressure difference between the inlet and the outlet of the system in a pulsatile fashion. The boundary

conditions are assumed to be fluctuating pressure distribution as defined in figure 2.

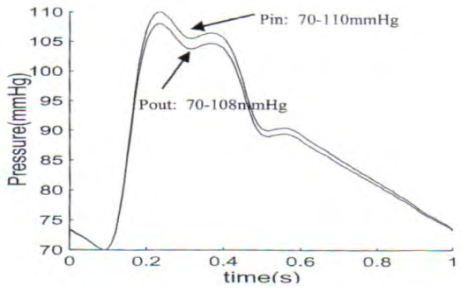

Figure 2: A typical pressure profile for human carotid artery.

The fluid-structure interaction is taken into account with COMSOL by coupling the structural mechanics of the wall and fluid dynamics of the blood then simultaneously solving the system. The model with two different stenosed rate were solved in order to study the effects of the compliance - mismatch

4 Results

The boundary deformations at all times for both the geometry configurations show that the maximum deformation does not exceed 10 μ m, which corresponds to a strain of less than 1%. In such a case, the one-way fluid-structural coupling assumption is a reasonable approximation. The maximum deformation was observed at t=0.52s, just after the time when the pressure load must have been the maximum, which is at the peak of the pressure profile.

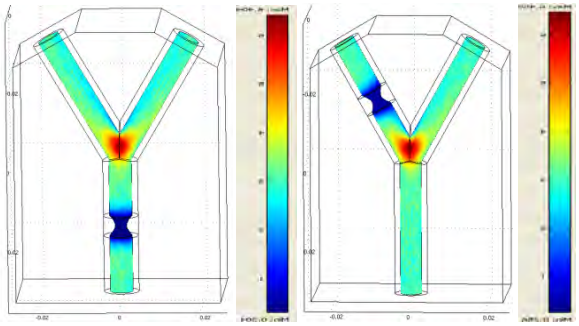

Figure 3 **Figure 4**

Figure 3 & 4 Typical deformed arterial boundaries plot at t = 0.52s. The deformation is in microns for inlet and oulet arterial section respectively.

On a broad level, it is observed that if the stenosed region is in the outlet section, it undergoes more deformation. The deformation is also non-uniformly distributed when the stent is in one of the outlet sections. This is a natural consequence of the asymmetric geometry which causes an asymmetric flow field. The flow field is the same for all the cases with different stent properties for a given geometry configuration. When the stent is in the inlet section the flow is uniformly distributed among the two outlet sections. This is not the case when there is a stent placed in one of the outlet sections. In the latter situation, the stenosed region offers an obstruction to the flow and as is visible in the plot below, the flow prefers to go through the non-stenosed region rather than the stenosed one.

The asymmetry also appeared to influence the maxima of the flow field in response to the pressure drop. It was observed that in the case when the stenosed region was in the inlet section, the maximum velocity magnitude was around the instant of the maximum pressure drop across the flow boundaries. On the other hand, when the stenosis was in the outlet section of the artery, it was observed that the maximum velocity magnitude was 0.04 s after the instant when the pressure drop was at its maximum (t = 0.5 s). This maximum value even sustained for around 0.08 s after which the velocity field started reducing in magnitude in accordance with the downward slope of the sinusoidal pressure function at the flow boundaries. This lag can be attributed to the asymmetry in the flow geometry causing flow disturbances due to which the non-uniform velocity field is out of phase with the pressure-drop across the flow boundaries. The lag is sustained for some time due to the viscous damping effects. There can also be a partial contribution of the asymmetric deformation. Owing to our one-way fluid-structure coupling simplification, this effect has not been taken into account.

Analysis: Centerline Velocity Calculation

Flow field comparison for the two geometric configuration around the instant of maximum pressure drop.

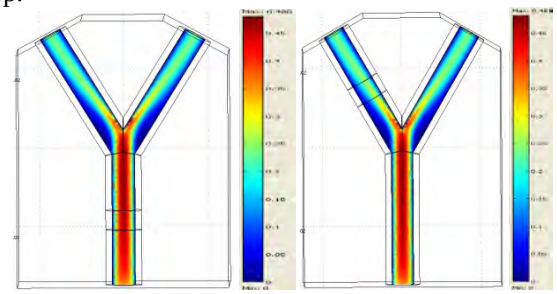

Figure 5: Flow field at 0%stenosed rate

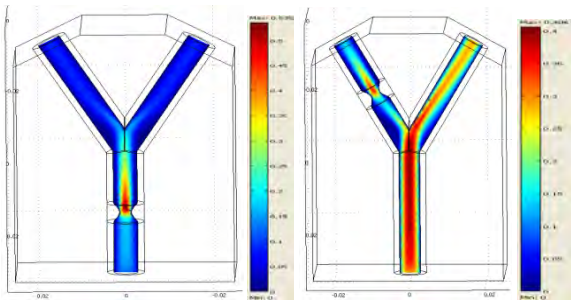

Figure 6: Flow field at 55%stenosed rate

Figure 7 shows the comparison of the centerline velocities simulated for both 0% and 55% stenosed rate, constriction in the inlet arterial cross-sectional area by referring Fig 5 and 6. The centerline axial velocity profile has the same pattern of the inlet velocity waveform. During accerleration and early flow deceleration, there is small difference of the velocity profiles between the two model.

Similarly, Figure 8 Shows the comparison of the centerline velocities for both 0% and 55%, but in oulet arterial section.

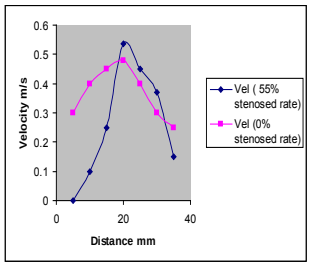

Figure 7

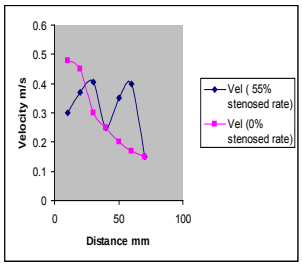

Figure 8

Figure 7 and 8 Matched Centerline velocity for Inlet and oulet arterial Section arterial section.

According to the clinical point of view, shear stress is more essential. When blood force distribution acts on the boundary surfaces of stenosed area, higher shear stress occurs at the throat section of the stenosed area and rapidly drops reversing its direction. If the stenosed rate is increased, higher stress occurs at the vicinity of the stenosed area, which means the blood flow could be closure leading to higher pressure drop and increasing the centreline velocity at higher stenosed rate. That results in decreasing compliance effect.

5 Conclusion and Future Recommendation:

The present work illustrated the implementation of a one-way coupled fluid-structure interaction (FSI) based computational model to study the blood flow through an arterial section with a stenosed region having a stent placed in it. The stenosed region with the stent was considered as a new subdomain with the application of the lumped parameter model. The stent region's mechanical properties were varied during the computational study.

This parametric study yielded that a compliance mismatch can prevent strokes that typically occur in the stenosed region due to the reduction of static pressure. Huge compliance mismatches though, would not be desirable especially due to greater deformations experienced by the arterial walls.

The stenosed region was placed at different positions to see the effect of asymmetry on the arterial wall deformation. The asymmetry caused non-uniformity in the flow field which influenced the response of the arterial walls and the stented region. It was observed that flow prefers to avoid the stenosed region which causes additional load on the section with the stenosis. The structure therefore, seemed to translate marginally towards the stenosed section at the bifurcation junction.

These findings need to be confirmed by either a two way coupled FSI study or a fully coupled multiphysics application supported by COMSOL Multiphysics. The computational effort for the same would be huge and could not be implemented within the framework of the current project.

The promising trend in increase in the computational resources available on a desktop holds potential for such a fully coupled study to be performed in the near future.

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