***Paper ID: FM-02***

# Effects of Fibrous Cap Thickness and Stenosis Severity on the Fluid and Structural Behavior in a Model Stenosed Artery

M. Rejaul Haque, M. Emran Hossain Bhuiyan and Dr. A.B.M. Toufique Hasan
Department of Mechanical Engineering
Bangladesh University of Engineering and Technology, Dhaka-1000
*E-mail: md.rejaulh@yahoo.com*

## Abstract

*The study of blood flow through a stenosed artery is very important because of the fact that the cause and development of many cardiovascular diseases are related to the nature of blood movement and the mechanical behavior of the blood vessel walls. The interaction between the blood and the plaque may causes the rupture of the plaque. In the present research, study of the blood –plaque interaction using a fluid -structure interaction (FSI) model has been done. The degree of stenosis has been varied from 10% to 70% (by area) and fibrous cap thickness varies from 0.1mm to 2.0mm. Vortex rings are developed at the pre and post stenotic region. Deformation of stenosis, Wall shear stresses (WSS), Peak principle stress and Vonmises stress increases with increasing the degree of stenosis. The peak principle stress found to be maximum (≈2000 pa)at the instants of peak velocities of the phasic flow for the case of 70% of stenosis with 0.1mm fibrous cap thickness.*

***Keywords:*** *stenosis, fibrous cap thickness, rupture, principle stress, fluid-structure interaction*

## 1. Introduction

Blood flow through the artery has the similar behavior with the fluid through a channel. This type of phenomenon can be described by the fluid mechanics engineering. This is known as Hemodynamics. Hemodynamics plays a great role to explain the fluid (Blood) flow behavior through the artery which is inherently unsteady. Structure of the blood vessel includes three layers**:** Tunica intima (collagen and smooth muscle), Tunica media(elastic fibers, collagen and smooth muscle), and Tunica adventitia (connective tissues consisting of elastic and collagen fibers). There lies a small lumen at the center of artery through which blood flows at a very high pressure. However, due to bad food habit, lacking of physical exercise, oversleeping may cause serious plaque formation in the blood vessels.

The stenosis is defined as a partial constriction of the blood vessel due to the accumulation of cholesterols and fats and the abnormal growth of tissue. Plaque which actually constricts the blood vessel or cause of stenosis consists of Lipid core and fibrous cap. Fibrous cap which covers the lipid core is a thin domain and consists essentially of collagen and smooth muscle cells. The lipid core is formed by fatty composites. The disease caused by this is known as Atherogenesis . Atherosclerotic plaques may rupture without warning under physiological conditions and cause fatal sub sequential diseases such as heart attack and stroke. The exact mechanism causing plaque rupture is not well understood. Stenosis severity has been widely used as a measure of seriousness of stenosis and basis for surgery decision. The stenosis causes the most frequent anomaly in blood circulation .Once the constrictions is formed ,the blood flow is significantly altered and fluid dynamical factors play important roles as the stenosis continues to enlarge leading to the development of cardiovascular diseases such as heart attack and stroke.

The degree of stenosis is quantified by the diametric occlusion in the artery as a percentage of the unobstructed diameter. Pressure losses in stenosed arteries become significant for severe stenosis leads to stroke or infraction, considered as one of the major causes of death and disability in humans. Plaque interacts with the blood flow creating mechanical stresses which can lead to its rupture and which can produce recirculation downstream the plaque. Plaque rupture and recirculation can yield the formation of a thrombus and eventually lead to a heart attack. In this study, development of a FSI model in order to study the interactions between the blood flow and the plaque has been conducted.

In this research, a 2-D simplified model is considered to investigate the several fluid flow and structural behavior such as streamlines, vortex formation, and deformation of plaque, wall shear stress, peak principle stress and vonmises stress for different cap thickness and different degree of stenosis at different time. Both the plaque material is modeled as hyper elastic material guided by Mooney-Rivlin model [1].

In this present study, a plaque with a thin fibrous cap with a moderate Arterial stenosis could also be vulnerable. Therefore, Fibrous cap thickness could be considered as a useful indicator for plaque vulnerability in addition to the traditional measure of stenosis.

## 2. Modeling Details

The evaluation of the shape of stenosis in real case is not an easy task, as different complex shapes are found which cannot be modeled easily. In order to avoid difficulty 2-D analysis has been considered in the present research. Blood is a non-Newtonian fluid. We have considered it as a Newtonian, homogeneous and incompressible fluid. The apparent viscosity is nearly a constant in large Arteries with diameter ($\approx$ 5mm) and therefore the non-Newtonian effects can be avoided. Here modeled Artery's diameter is 10mm as shown in Fig.1.The Governing equations for fluid domain are 2D Navier strokes equations and for solid domain structural properties are included under the FSI study.

### 2.1 Fluid Domain

Governing equations are Navier-Stroke equation(1):

$$\frac{\rho \partial V}{\partial t} + \rho(V.\nabla)V = -\nabla P + \mu \nabla^2 V \tag{1}$$

At solid-fluid interface boundary, the condition is governed by equation (2) and equation (3):

$$U_{fluid} = U_w \tag{2}$$

$$U_w = \frac{\partial U_{solid}}{\partial t} \tag{3}$$

A simple model of the artery with one-sided cosine shaped stenosis is considered in the present study. The experimental investigation of Ahmed and Giddens [2] considering pulsatile flow through an axisymmetric smooth stenosis was studied here .The geometry used was similar to theirs with the stenosis shape given by a cosine functioned equation (4) .If $H$ is the height of the nonstenotic part of the tube , *S(x),*gives the shape of the stenosis.

Where $S_0$ is the % stenosis severity with $x_1$ and $x_2$ specifying the position and length of the stenosis.

$$S(x) = \frac{S_0 H}{2}[1 - cos\{2\pi(x - x_1)/(x_2 - x_1)\}]/2 \tag{4}$$

The Height of the artery $H$ is 10 mm. The viscosity of the blood is taken as $\mu = 3.4\times10^{-3}$Pa.s with a mass density of $\rho$ =1035 Kgm$^{-3}$. At the inlet boundary the input velocity expression is given by equation (5):

$$v = v_0 \sin\left(\frac{2\pi t}{T}\right) \tag{5}$$

Where,$T$=pulse period,$v_0$=0.12 m/s

(Corresponding to Reynolds number $Re$= 366)

The Womerseley parameter ( $\alpha = H/2 \times \sqrt{2\pi\rho/T\mu}$ ), for our investigation is found 8.26. Observation has been made at the point of maximum acceleration, maximum velocity and maximum deceleration. All the Arterial boundaries are given no-slip boundary conditions.In the present study, the simulations are carried out using a available CFD software which FSI is handled by means of the ALE formulation. The entire domain is subdivided into 7000~12424 triangular elements.

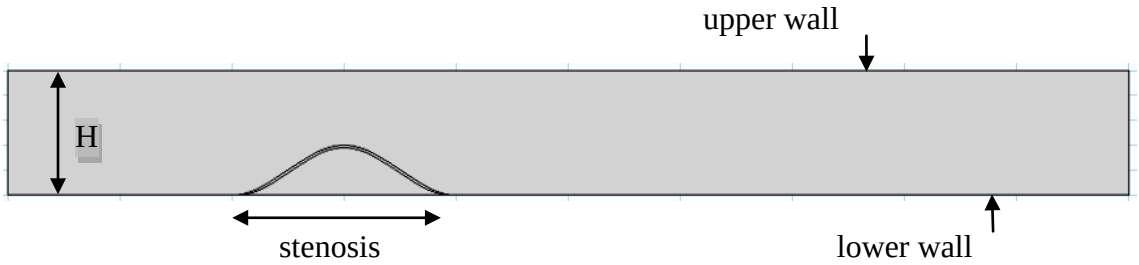


**Fig. 1.** Schematic of an arterial stenosis

### 2.2 Solid Domain

The lipid pool and the fibrous cap are modeled as isotropic nearly incompressible hyper elastic materials. For hyper elastic materials, the stress-strain relationship is nonlinear and derives from a strain energy function w. Due to the isotropy of the materials, this function is expressed in terms of invariants of deformation tensors.
For near incompressible material, the energy function w is splited into distortional and volumetric partsas shown by equation(6):

$$W = w(c) + U(j) \tag{6}$$

In the present study w is defined according to the Mooney-Rivlin model. Hence the energy function is stated by equation (7):

$$W_s = C_{10}\,(\bar{I}_1 - 3) + C_{01}\,(\bar{I}_2 - 3) + \frac{1}{2}\kappa(J_{el} - 1)^2 \tag{7}$$

**Fibrous cap**
$C_{10}$=9200 $Nm^{-2}$
$C_{01}$=0 $Nm^{-2}$
κ=3000 MPa
**Lipid deposit**
$C_{10}$=500 $Nm^{-2}$
$C_{01}$=0 $Nm^{-2}$
κ=200 MPa
The fibrous cap and lipid deposit densities are taken as 1000 $Kgm^{-3}$
These data are taken from papers [1].

## 3. Results and Discussion

The streamline flow pattern is disturbed due to the presence of stenosis. As the flow is sinusoidal time dependent, significant flow variations on the pre and post stenotic region are observed.The features of interests are the recirculations and the stress over the plaque. From Fig. 2. and Fig. 3. it is clearly observed that ,with the increase of percentage of stenosis the vortex rings are seen not only at lower boundary but also affects the upper boundary at the same instant of time due to large area constriction provided by the accumulation of the the fatty decomposites.

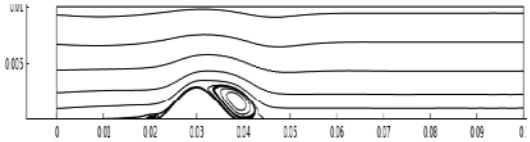

**Fig. 2.** vortex rings for $S_0$ =30% at $t/T$ =0.37

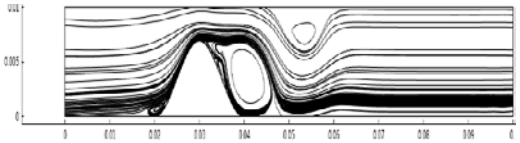

**Fig. 3.** vortex rings for $S_0$ =70% at $t/T$ =0.37

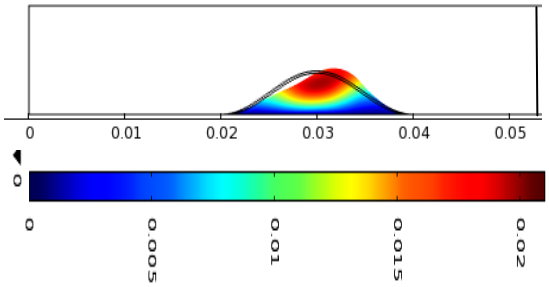


**Fig. 4.** Displacement field for 30% stenosis and 0.1 mm fibrous cap thickness

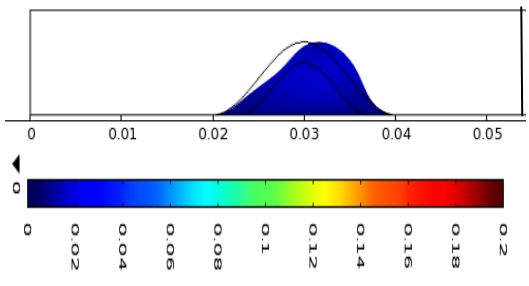


**Fig. 5.** Displacement field for 70% stenosis and 2 mm fibrous cap thickness

The displacement field have been shown with color range in Fig.4. and Fig.5.In the first figure the red portion shows a displacement value ranging from 0.015-0.02mm while in case of second fig the deep blue portion shows a range of values near to the first one. This clearly implies that, 30% stenosis with 0.1 mm fibrous cap thickness can be considered as the same threat to that of offered by the 70% stenosis with 2mm fibrous cap thickness.

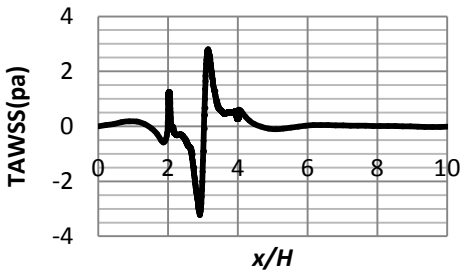


**Fig. 6.**Time Averaged Wall Shear Stress for 70 % stenosis with 0.1mm fibrous cap thickness at lower boundary

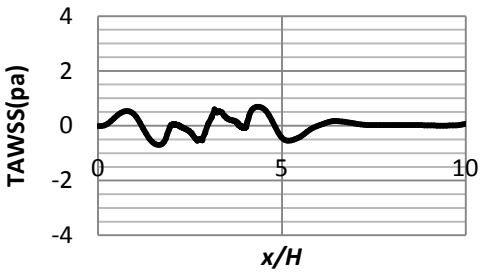


**Fig. 7**.Time Averaged Wall Shear Stress for 70 % stenosis with 2mm fibrous cap thickness at lower boundary

Fig. 6. and Fig. 7. represent the Time averaged wall shear stress as the wall shear stress varies sinusoidal with the inlet inflow. However, TAWSS are plotted along the length of the stenosis in a dimensionless form and for same degrees of stenosis( 70% here) ,it shows a lower peak with the increase of fibrous cap thickness.

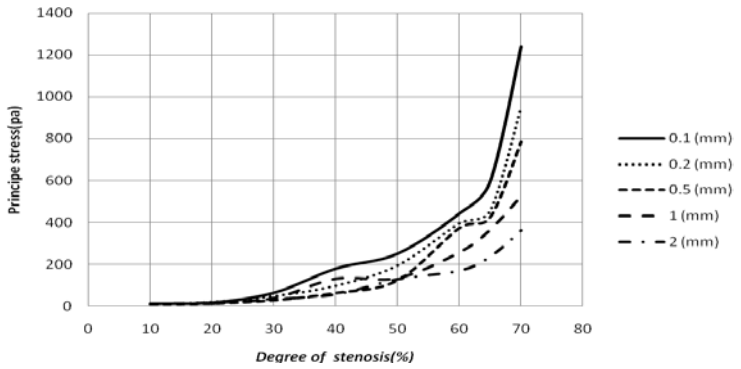


**Fig. 8.** variations of Principle stress with degree of stenosis for different fibrous cap thickness

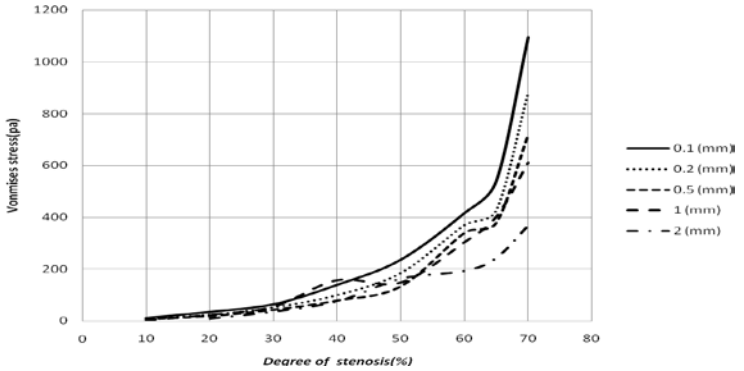


**Fig. 9.** variations of Vonmises stress with degree of stenosis for different fibrous cap thickness

Variations of principle stress and vonmises stress for different degrees of stenosis and fibrous cap thickness are shown in Fig. 8. and Fig. 9. Fig. 8 implies that 200 Pa principle stress can be occurred within the plaque shoulders for different combinations such as : 1)40% stenosis with 0.1mm fibrous cap thickness 2)50% stenosis with 0.2mm fibrous cap thickness 3) 65 % stenosis with 2 mm fibrous cap thickness. As a result, for determining the rupture risk, all poses the equal amount of threat to the patient's evaluation. So, the main theme of the present research is to examine the patient not only by the presence of degree of stenosis but also by the thickness of fibrous cap thickness which is often be neglected within the moderate range of stenosis (30%-70%). Stenosis higher than 70% actually impose a higher threat to plaque vulnerability whatever the fibrous cap thickness is.

## 4. Conclusion

This paper is devoted to the study of the fluid-structure interactions between the blood flow and the plaque in a 2D geometry of a modeled Arterial stenosis. The computations show that Plaque stress/strain analysis is important for a better understanding of plaque rupture process. Critical stress/strain conditions are very sensitive to changes in pressure conditions, material properties, plaque structure, lipid pool size, shape and position and various model assumptions. However,to determine the plaque vulnerability Patients in the future may be examined not only by the degree of stenosis but also by the appearance and thickness of fibrous cap thickness.

## 5. References

[1] N.EI Khatib,S.Genieys,A.M.Jine and V.Volpert, Non-Newtonian effects in a fluid-structure interaction model for atherosclerosis.*Journal of technical physics.*50,55-64.2009
[2] Ahmed S.A. and Giddens , D.P., Pulsatile post stenotic flow studies with laser Doppler anemometry, *journal of Biomchanics.*17,695-705.1984.
[3] Dalin Tang, Chun Yang, Shunichi Kobayashi, David N. Ku., Effect of a lipid pool on Stress/Strain distribution in Stenotic Arteries: 3-D Fluid- Structure Interactions ( FSI) Models,126,363-370,2004.
[4] Zhi-Yong Li, Simon P. S. Howarth, Tjun Tang and Jonathan H. Gillard. How critical is fibrous cap thickness to carotid plaque stability?: A flow-plaque interaction model, 37,1195-1199.2006.
[5] Bathe. M. and Kamm. R. D. A fluid-structure interaction finite element analysis of pulsatile blood flow through a compliant stenotic artery, 121,361-369.1999.
[6] S. Chakravarty, Effect of stenosis on the flow behavior of blood in an artery, *International journal of engineering science*, 25, 1003-1018.1987.