NUMERICAL ANALYSIS OF HEMODYNAMIC FORCES THROUGH STENOTIC ARTERY

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Abstract: *A numerical analysis is carried out to demonstrate the variation of two hemodynamic forces: wall shear stress and pressure in the flow field of stenotic artery. The artery has 75% stenosis severity with axisymmetric cosine shaped stenosis. The wall of the vessel is considered to be rigid. A sinusoidal pulsatile flow is assumed as inlet boundary condition. During the simulation both standard k-ω model of low Reynolds correction and standard k-ε model are used. Some analysis with standard k-ω model with low Reynolds correction show better agreement with previous experimental results. The peak wall shear stress is found to be located proximal to the stenosis throat. The results from the standard k-ε turbulence model illustrates oscillating wall shear stress downstream the stenosis throat throughout the time period. The pressure gradient at the throat is highest at peak flow condition. These factors contribute to further growth and rupture of plaques.*

Keywords: *Atherosclerosis; wall shear stress; pressure gradient; compressive force.*

NOMENCLATURE

D	Diameter of the artery, m
R	Radius of the artery
WSS	Wall Shear Stress, Pa
Z	Normalized distance found by dividing the positional value by diameter
r	Radial distance, m
u	Velocity at a point, m/s
u_{avg}	Average Velocity
u_i	Mean velocity, m/s
I	Turbulent Intensity
ρ	Density of the fluid, kg/m^3
μ	Viscosity of the fluid, Pa-sec
ω	Angular frequency
τ_w	Axial Wall Shear Stress, Pa

INTRODUCTION

Atherosclerosis, the leading cause of death in the developed world, involves artery wall thickening due to accumulation of fatty material such as cholesterol and lipid substances. These fatty depositions are called plaques. Atherosclerosis can occur body-wide, in the arteries to brain, intestines, kidneys, legs etc. Formation of plaque is a continuous process and grows in a slow and controlled manner. The worst situation is the sudden rupture of the plaque. This rupture allows blood to clot inside the affected artery. In the heart it causes the heart attack and in the case of brain it leads to a stroke. Plaque stress is important factor when the mechanism of plaque rupture is considered. The arterial wall continuously interacts with hemodynamic forces, which include wall shear stress (WSS), hydrostatic pressure, pressure gradient, gravity and kinetic and potential energy provided by the cardiac pump. Plaque stress is the result of these external hemodynamic forces. Plaque rupture itself represents structural failure of a component of the diseased vessel, and it is therefore reasonable to propose that the biomechanical properties of atheromatous lesions may influence their vulnerability to rupture according to Young et al.[1] Properties of blood that affect its flow characteristics through the heart, arteries and veins includes- viscosity, inertial mass and volume of blood to be moved. Other factors that affect the motion of blood are - size of blood vessel, condition of blood vessel, smoothness of lumen, elasticity of muscular layer and destination of blood. Studies show that pulsatile blood flow through artery vessel implicates in several types of hemodynamic forces that can impact in vessel wall structure. These forces are also the cause of development of vascular pathologies and an important factor in atherosclerosis Li et al.[2]. Studies suggest that regions of high shear stress, which implicates in a direct mechanical harm of the vessel wall, are regions where atherosclerosis occurred. Conversely, Caro et al.[3] stated that atherosclerosis occurred in the site of low shear stress because of low mass diffusion of lipids. It is also associated with low and oscillatory wall shear stress in sites where blood flow is slow and reversal. On the other hand, vessel with steady blood flow and high shear stress are comparatively disease free.

Some studies showed that WSS promoted oxidative stress inflammation, vascular smooth muscle cell migration, differentiation and proliferation. It also affected vessel re-modeling and development of vulnerable plaque. High WSS acting on the endothelium had a regressive effect on the balance that controls plaque stability at the stenosis region according to some researchers[1]. Young et al.[1] studied the shear stress and pressure gradient in the stenosis and evaluated the cause of plaque rupture. They used a physiologically realistic, pulsatile flow with a 70% carotid stenosis. Their study showed that the WSS reached maximum just distal to the stenosis followed by a negative local minimum. A pressure drop across the stenosis varied significantly during the systole and diastole. They stated that the magnitude of shear stress was very low compared to the pressure gradient and therefore pressure might be the main cause of plaque rupture. Li et al.[2] also studied the hemodynamic forces. They used pulsatile blood flow and an elastic wall to observe the lumen movement. Their stenosis model was of cosine shaped and 30%, 50% and 70% diameter reduction were considered. They also considered wall shear stress and the hoop stress. According to their study the peak WSS occurred just before minimum lumen position. Jayme et al.[4] obtained numerical simulation assuming a physiological pulsatile flow through different models of stenosis. Physiological waveform considered as inlet pulsatile velocity was of the femoral artery of a dog. They used Fourier series to adopt the inlet wave form. They also assumed in the modeling rigid tube, laminar flow and Newtonian fluid. They studied an idealized normal artery with no diameter reduction and three different axisymmetric stenosed arteries; 33%, 50% and 75% of diameter reduction. In the case of normal lumen some change occurs in the velocity profile, according with the instant of cardiac cycle. As for stenosed artery unsteadiness in the flow field was observed. They said changes of the velocity field were due to the development of reversal pressure gradient. The wall shear stress distribution was observed for the whole domain. These values of their simulation depended on the axial position, cardiac cycle phase, and stenosis geometry. The range of normal shear stress in their physiological normal pattern was between 1 to 7 Pa. the low shear stress observed here was of order of 0.1 Pa, which was associated with atherogenesis and highest value of WSS found was of the order of 240 Pa, which was in the range of direct endothelial injury occurrence according to their study.

Table 1. Flow parameters for present study

U_0 (m/s)	U_m (m/s)	ω (rad/s)	D (m)	ρ (kg/s)	μ (Pa. s)	C_p (J/kgK)
0.04254	0.02808	0.314	0.0508	1000	0.0036014	0.3594

Ahmed and Giddens[5] studied both steady and pulsatile flow through 25%, 50% and 75% constriction of a rigid tube and Reynolds number ranges from 500 to 2000 using Laser Doppler anemometry (LDA). Sinusoidal pulsatile turbulent flow through a rigid walled stenotic vessel has been numerically modeled using the Reynolds averaged Navier-Stokes equation approach by Varghese and Frankel[6]. Four different turbulence models were employed tin their study and the models influence on the results are presented. Their study showed that low Reynolds number k-ω turbulence model was in much better agreement with the experimental results of Ahmed and Giddens[5] than RNG (renormalization-group theory) k-ε model and the standard k-ε model, with regard to predicting the mean flow distal to the stenosis. These entire turbulence models were also used to predict the peak wall shear stress at the throat of the stenosis with minimum values observed distal to the stenosis where flow separation occurred. They used two stenosis models: smooth and sharp edged of rigid wall. Depending on the constriction severity of the atheroma, the flow behavior in the post stenotic region could be turbulent and/ or in transition to turbulent, although the presence of a laminar flow condition in the pre stenotic region.

In this study, a numerical simulation is carried out in an axisymmetric two dimensional stenotic artery as a simplified case to analyze wall shear stress and the pressure gradient. The study shows that the peak shear stresses at different time phase are located proximal to the throat. Pressure gradient is high at the peak flow condition as well as the WSS. The blood is considered to be incompressible, Newtonian fluid. The vessel wall is

considered rigid. The assumption of a rigid walled vessel is not far from reality, as the atherosclerosis promotes some reduction in the elastic properties of the vessel wall. For simulation standard k-ω model with low Re correction and standard k-ε model are used. Due to the better agreement with some experimental results, only Standard k-ω model with low Re correction is used for some present studies.

MATHEMATICAL MODELING:

The flow field is governed by incompressible flow the Reynolds-averaged Navier-Stokes equations in conservation form. It is expressed as:

$$\frac{\partial u_i}{\partial x_i} = 0 \quad (1)$$

$$\rho \frac{\partial u_i}{\partial t} + \rho \frac{\partial}{\partial x_j}(u_j u_i) = -\frac{\partial p}{\partial x_i} + \frac{\partial}{\partial x_j}(2\mu s_{ij}) \quad (2)$$

where, the strain-rate tensor s_{ij} is given by,

$$s_{ij} = \frac{1}{2}\left(\frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i}\right) \quad (3)$$

Pressure gradient is defined as a pressure drop per unit length.

$$\text{Pressure gradient} = {(P_1 - P_2)}/{L} \quad (4)$$

where, P_1 is entry pressure, P_2 is exit pressure and L is the length of stenosis.

Poiseuille's Law:

$$Q = (P_1 - P_2)\frac{\pi r^4}{8\vartheta L} \quad (5)$$

Q is the volume flow rate; r is the radius, L is the length of the tube and ϑ is the viscosity of the fluid.

NUMERICAL MODELING

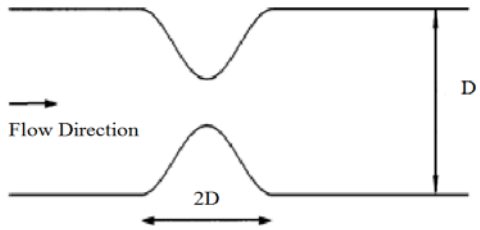
Stenosis model

The geometry of the stenosis is shown in Fig. 1 (a). As the plaque formation is continuous and localized, the assumption of cosine shaped stenosis resembles with the real cases. Stenosis level is considered 75% by area. The length of the vessel is considered 5D upstream and 30D downstream the stenosis throat.

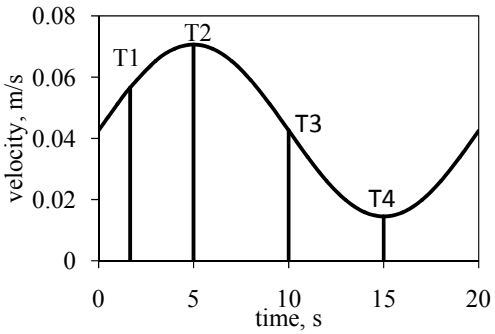
Flow modeling

Two turbulence models: standard k-ε and low Reynolds k-ω model are used. The PISO (pressure implicit splitting of operators) algorithm is used for pressure-velocity coupling. It is useful for stability in unsteady flow calculations[6]. For spatial discretization second order upwind scheme is used for pressure, moment, turbulent kinetic energy, turbulent dissipation rate and specific dissipation rate. For turbulence modeling the near wall treatment is an important consideration. Wall function approach is less reliable in the case of separated flow and enhanced wall treatment usually requires a very fine near-wall mesh capable of resolving the viscous sub layer. Here, the first grid point off the wall needs to be within the region where $y+ < 1$, $y+ \equiv (u/\mu)$ (where u, is the friction velocity). Latter is more suitable for low-Reynolds number flows[6]. Here for modeling, enhanced wall treatment approach is employed all through the study. The time period, T is calculated by the Eq. (6). The time step was taken as 0.3335 seconds and 20 iterations is done per time step. Here blood is considered incompressible and Newtonian fluid. The properties of blood and other key parameters are given in Table 1. Some parameters are taken from Ahmed and Giddens[5] for the sake of proper validation.

$$T = 2\pi/\omega \quad (6)$$



(a)



(b)

Figure 1. (a) Geometry of the stenosis. (b) Inlet velocity profile for present case; here, T1 = 1.665s, T2 = 5s, T3 = 10s, T4 = 15s and the time period is 20s.

Boundary conditions

A flat velocity profile varying sinusoidally with time is formed at 5D upstream the stenosis, using the Eq. (7). A user-defined function is written for this case.

$$U = U_0 + U_m \sin(\omega t) \quad (7)$$

Here, U_0 and U_m values are taken according to the flow field Reynolds number which varies from 200 to 1000. The inlet velocity profile is shown in Fig. 1(b) along with the different time phases which are used for post-processing calculations. The turbulence intensity and hydraulic diameter are also specified as inlet boundary conditions and are calculated by the Eq. (8) & (9). Hydraulic diameter is considered the diameter of the non-stenotic part of the vessel.

$$I \equiv \frac{u'}{u_{avg}} \cong 0.16 \left(Re_{D_H}\right)^{-1/8} \quad (8)$$

$$Re_{D_H} = \frac{\rho U_0 D}{\mu} \quad (9)$$

RESULTS AND DISCUSSION

Before starting of present investigation the numerical simulation is needed to be validated. Once the simulation results are satisfactory, comparing with the experimental results, the computer code is used for present computation.

Validation

Validation of the present numerical computation is done by plotting the steady velocity profile at 2.5D downstream from the stenosis throat and comparing it with the velocity profile of Varghese and Frankel[6]. For this case, a parabolic velocity profile is assumed as inlet boundary condition and developed at 5D upstream of the stenosis. The mean inlet velocity corresponds to Reynolds number 500 and the flow is assumed to be steady. The results are shown in the Fig. 2, where a good agreement can be found with Varghese and Frankel[6]. Small discrepancy near to wall and at center is found. The reasons of this discrepancy might be due to the consideration of two-dimensional geometry and the position of inlet velocity profile. Varghese and Frankel[6] located their inlet velocity profile at 2.5D upstream to the stenosis. However, the present result agrees qualitatively with Varghese and Frankel[6].

Second validation is done with Ahmed and Giddens[5] as shown in Fig. 3. For this case, inlet velocity as shown in Fig. 1(b) with flat profile is created 5D upstream to the stenosis throat. Radial variations of velocity at different axial location downstream to the stenosis are observed at peak flow condition, T2. Each profile is offset by 10 units in horizontal direction as shown in Fig. 3. Two turbulence models: standard k-ε model and standard k-ω model with low Reynolds correction are used. Standard k-ε model shows poor agreement with the experimental results but standard k-ω model with low Re correction agrees qualitatively with the experimental results of Ahmed and Giddens[5]. Standard k-ω model predicts the jet like behavior of the experimental velocity profile better than standard k-ε model. The difference in profiles may be due to 2 dimensional geometry consideration and difference in inlet waveform. A length of 96D prior to stenosis was considered in the experiment. They used this long length so the flow will be fully developed long before the stenosis region. The present simulation has not considered any gravitational effect in the flow field; this may also leads to variation in profiles.

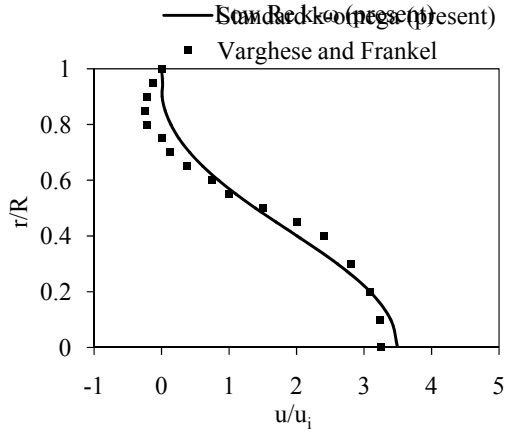


Figure 2. Comparison of computed steady velocity profile.

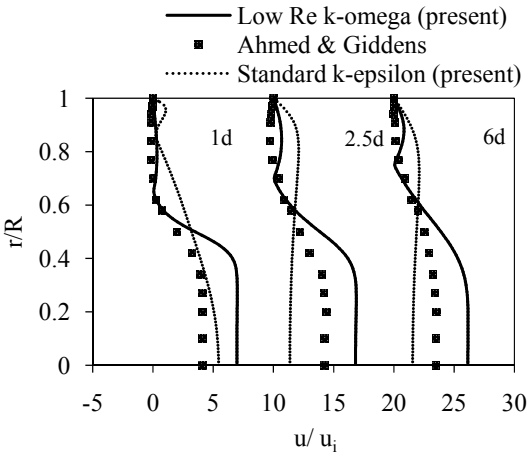


Figure 3. Comparison of computed velocity profile at different axial locations distal to the stenosis at peak inlet flow condition (T2).

Wall Shear Stress Variation

Variations of axial wall shear stress along the length of the 2D vessel in the vicinity and distal to the stenosis are shown in Fig. 4, at four different time phases (T1, T2, T3 and T4) of the flow cycle. The peak value of WSS is higher in the accelerating phase (T1 to T2) and lower at decelerating phase (T3 to T4). It is highest at the peak flow condition (T2) and lowest at minimum flow condition (T4). The negative values of WSS at further downstream are consistent with the flow reversal found at downstream close to the stenosis throat. During the decelerating phase, at downstream to the stenosis throat, vortices develop.

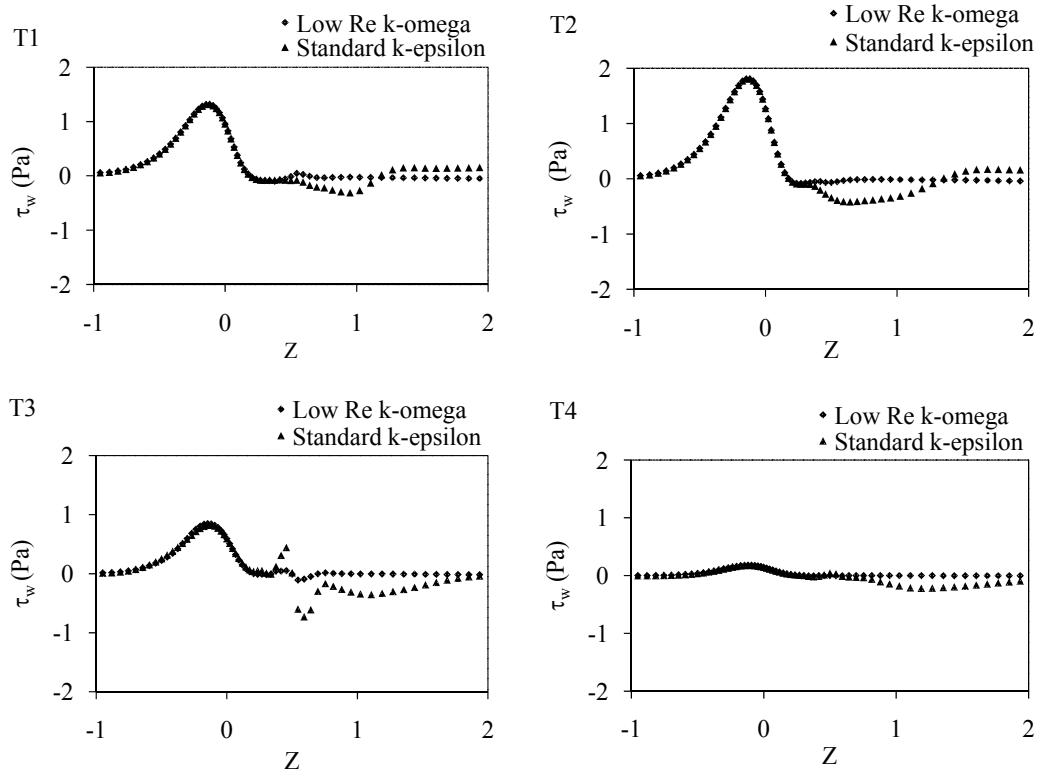


Figure 4. Axial wall shear stress variation at different time phases of the flow cycle. The axial distance is indicated in normalized units, Z.

At T3 an oscillatory form of WSS is found after the first peak is due to the generation of flow disturbance at downstream to the stenosis and this phenomenon can be observed well by the standard k-ε model. The location of zero shear stress indicates the re-attachment and flow reversal. In each time phase the maximum WSS is formed just prior to the stenosis throat due to the presence of high velocity in that region. It is seen that rupture normally occurs before the minimum lumen position[7]. The WSS fluctuates throughout the flow cycle and results in arterial wall thickening. High WSS elongates the endothelial cells and force to align in the direction of the flow. Low WSS had negligible effect on the cell but increases intercellular permeability and consequently increases the plaque formation in the region[9].

Wall Pressure

Figure 5 shows the wall static pressure distribution in the neighborhood of the stenosis during four time phases of the flow cycle. For each time phase there is a pressure drop at the stenosis throat. This phenomenon is consistent with Bernoulli's principle. At the peak flow inlet condition the pressure drop is maximum at the throat and minimum at the minimum inlet flow condition. At time T1 the vortices starts to diminish and at T3 the vortices and reversed flow starts to form at downstream and near to the stenosis. Due to this disturbance in the flow field at T1 and T3 pressure distribution after the stenosis contain a peak and a low pressure respectively according to Fig. 5. Within a flow cycle the pressure gradient varies from high to low at the throat as shown numerically in Table 2. This periodic change in pressure

gradient is vulnerable to plaque rupture. The pressure drop introduces a compressive stress in that region and as the pressure gradient fluctuates with time, a cyclic compressive stress is generated around the throat. This leads to rupture of plaques. According to Poiseuille-Hagen formula (Eq. 5), the flow volume varies directly and the pressure gradient inversely with the fourth power of the radius. Blood flow and pressure gradient in vivo are markedly affected by small changes in the radius of the artery. It can be stated that if a small amount of plaque forms and reduces the diameter in a small percentage, the flow rate will increase tremendously which will lead to higher pressure gradient across the throat. This higher pressure gradient will enforce more plaque rupture in that region.

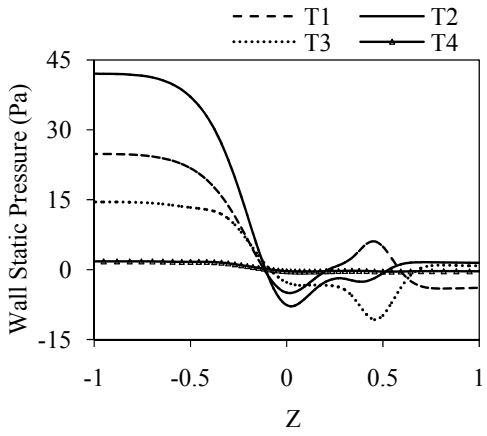


Figure 5. Wall static pressure variation with the different time phases of the inlet flow cycle.

Table 2. Pressure Gradient at the stenosed region

Time	Pressure Gradient, Pa/cm
T1	3.99
T2	2.83
T3	1.35
T4	0.21

The arterial wall is in constant interaction with hemodynamic forces, which includes the WSS and the blood pressure. The plaque rupture is caused due to the force applied on the atheroma. The forces in the form of stress tend to deform the plaques which are rigid in nature. The axial wall shear stress and the compressive stress due to pressure drop at the throat are both cyclic in nature. These cyclic stresses lead to fatigue failure of the plaque material. In the first stage of fatigue failure a crack initiates, this reduces the smoothness of the lumen surface and generates local vortices. Again the cracks may store blood or blood particles in them increasing the deposition of oxidized cholesterol in that area. This phenomenon will lead to rapid crack propagation and then to sudden rupture of plaque. The present study shows that the value of peak shear stress is around 2Pa at the peak flow condition as shown in Fig. 4(T2) and pressure gradient is nearly 4Pa/cm at peak inlet flow condition as shown in Table 2. As both the pressure gradient and WSS is peak at the region of throat, both of the forces may be the cause of ultimate failure of the plaque. Comparing the value of peak stress with the values of pressure gradient, the role of pressure gradient in the phenomenon of plaque rupture is more dominating than the wall shear stress.

CONCLUSIONS

The variation of hemodynamic forces: wall shear stress and pressure gradient are numerically analyzed in this study. Low Reynolds k-ω and standard k-ε turbulence models are used for this analysis. Comparison of the numerical results shows qualitative agreement with the experimental results. The static pressure drop at the minimum lumen position is shown and a huge pressure drop is found which provides a compressive force at the throat. The oscillatory behavior of the pressure just downstream to the throat at accelerating and decelerating phases of the inlet velocity is due to flow reversal. The peak wall shear stress in found prior to the stenosis throat. The negative values of wall shear stress represent the reversed flow at downstream to the stenosis. The standard k-ε model gives oscillating wall shear stress at downstream to the stenosis. Both wall shear stress and pressure gradient are cyclic in nature and contributes to plaque formation and rupture.

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