

Attributes of oscillatory physiological blood flow through 3-D geometry of single stenosed artery

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Abstract. The present study investigates the significant changes of flow behavior for 65 and 85 percentages severities of stenosis by area. The blood is assumed to be incompressible, homogeneous and Newtonian, while artery is assumed to be a rigid wall. The transient analysis is performed using ANSYS-14.5. Oscillatory physiological and parabolic velocity profile has been imposed for inlet boundary condition. The investigation has a Reynolds number range of 96 to 800. Pressure based solver and finite volume method are used for calculations. The flow pattern, wall shear stress (WSS), pressure, velocity, streamline contours, cross sectional and Centre-line velocity distribution are observed at early-systole, peak-systole and diastole for better understanding of arterial disease. Wall Shear Stress distribution shows that as severity increases, shearing of flow also increases for all cases. Thus maximum stress is exerted in throat region at peak systole. Pressure distribution also demonstrates that at all cases 85% stenotic artery creates more force than 65% stenotic artery at their pre-stenotic region. Interestingly, a recirculation region is visible at the post stenotic region in 85% stenotic artery for all cases and recirculation region increases with increasing minimum velocity at the inlet flow. From streamline contours it can be interpreted that 85% stenosis can create vortex at any time phase. So 85% stenosis is very harmful for its high probability of creating vortex.

1. Introduction

Cardiovascular diseases are a class of diseases that involve with heart or blood vessels (arteries and vein). On the other hand, stenosis is a constriction in the blood vessels and a leading cause of stroke and even fatal death in most of the countries. This happens due to our relatively limited understanding about stenosis. Therefore, a study of stenosis can be helpful to understand the biomechanics of vascular diseases. In general the blood flow does not face any obstacles on the way and the flow is regular and smooth. But sometimes due to presence of cholesterol, calcium and other substance in blood, a fatty substance called plaque develops near the inner wall of the artery. As the time passes this plaque grows resulting in hardening the arterial wall and narrowing the blood vessels. This can cause severe diseases such as the development of atherosclerosis. As a result the arterial wall loses its elastic property which limits the area of blood flow. Therefore, the flow turns to be abnormal in the reduced cross sectional area of the artery according to Khader and Shenoy [1]. One of the interesting studies in this area suggested that pulsatile blood flow through artery vessel implicated in several types of hemodynamic forces that could impact in vessel wall structure. These forces are also the cause of development of vascular pathologies and an important factor in atherosclerosis. The regions of high shear stress, which implicates in a direct mechanical harm of the vessel wall, are regions where



atherosclerosis occurs. At present, there is no standard procedure to measure the physical severity of the stenosis. Doctors often judge the severity based on a patient's physical symptoms as well as growth rate, constriction size and pressure drop often at high risk to patient's lives as stated by Chua and Shread [3]. But the results of numerical simulation can be better than MR, CT and Ultrasound techniques in analyzing the severity of stenosis. Chua & Shread et al [3] proved that the flow through the constricted tube is characterized by high velocity jet generated in constricted region and flow separation downstream to the stenosis. Khader & Shenoy et al. [1] found the results from numerical simulation that demonstrate that velocity and stenotic jet length increases during increasing the severity of stenosis. Their results also demonstrated that the 3D stenotic CFD model is capable to predict the changes in flow behavior for increased severity of stenosis. Young et al. [4] also studied the wall shear stress and pressure gradient in the stenosis and evaluated the cause of plaque rupture. They found pulsatile blood flow through the stenosis with elastic wall to observe the lumen movement. According to their study the peak WSS occurred just before minimum lumen position. Pinto et al. [5] conducted numerical simulation assuming a physiological pulsatile flow through different models of stenosis. In case of subject-specific anatomically realistic stenosed carotid bifurcation subjected to pulsatile inlet condition, the simulation results demonstrated the rapid fluctuation of velocity and pressure in post-stenotic region by S. Lee et al [7]. Ahmed and Giddens [6] studied both steady and pulsatile flow through 25%, 50% and 75% constriction of a rigid tube where Reynolds number ranges from 500 to 2000. Some of the above mentioned previous experimental and computational studies analyzed the flow aspects in proximal and distal end of stenosis including a detailed observation. Apart from this, an attempt is made in this present study to demonstrate the significant changes of flow behavior for 65 and 85 percentages severities of stenosis. For this a CFD method is used for detailed investigation of flow parameters like velocity, Wall Shear Stress, pressure and streamline contours. The working domain and the boundary conditions of the geometry were defined in pre-processor software ANSYS Workbench. The finite volume analysis was performed using ANSYS Fluent-14.5.

2. Model description:

2.1. Geometry :

This study is considered pulsatile flows of Newtonian fluid in a three dimensional axisymmetric stenotic arteries. For this study, 65% and 85% stenotic artery (by area) are taken. The flow geometry comprises a tube of diameter (D) of 6 mm and a length (L) of 96 mm and divided into three regions, pre stenotic, throat and post stenotic where the length of pre stenotic, throat and post stenotic region are $4D$, $2D$ and $10D$ respectively. The wall is considered to be rigid. Fig.1(a) shows the schematic diagram of arterial stenosis. The flow field mesh consists of 43657 nodes and 91561 elements for 65% stenotic artery and of 45496 nodes and 95592 elements for 85% stenotic artery. Figure 1(b) shows the mesh in cross sectional inflow plane of a stenotic artery

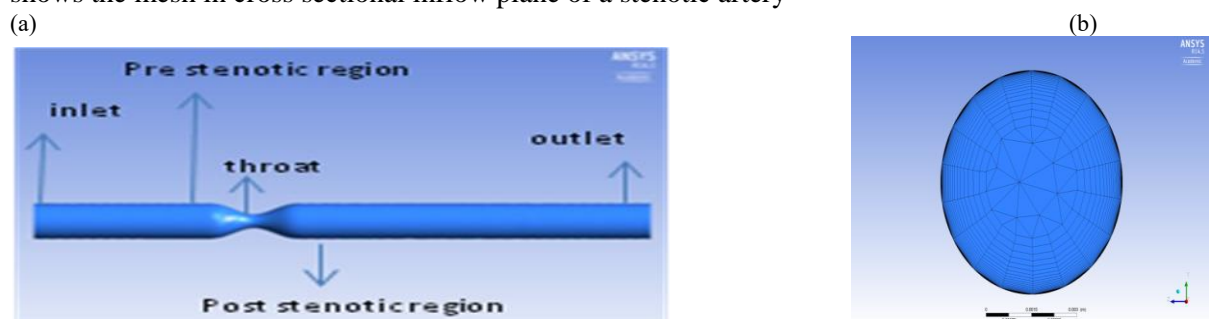


Figure 1. (a) Model of a stenotic artery; (b) mesh in cross sectional inflow plane of a stenotic artery.

2.2 Governing equation and Numerical Method

Blood is taken as fluid where the blood is considered incompressible. The density of the blood is 1050 kg/m^3 . In a Newtonian model for the blood viscosity, the value of μ is treated as a constant

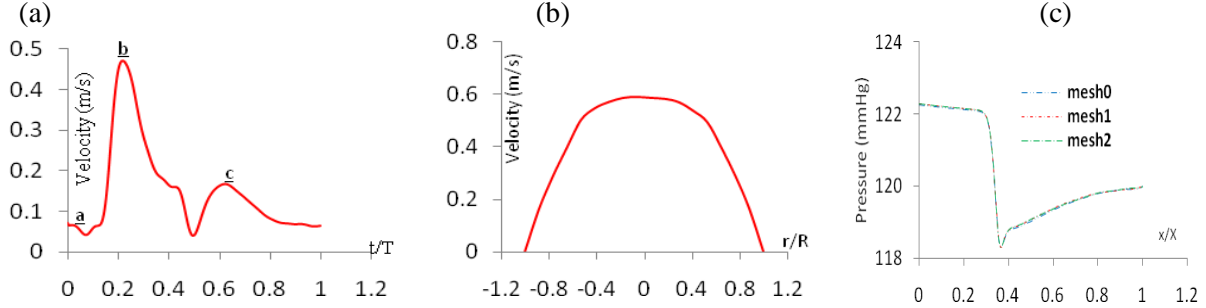


Figure 2. (a) Oscillatory physiological waveform, (b) parabolic inlet velocity profile, and (c) pressure distribution in 65% stenotic artery from different mesh sizes.

usually set to $\mu = 3.45 \times 10^{-3} \text{ Pa.s}$. Due to constriction blood passes through the throat and post stenotic region with high velocity. Flow velocity at post stenotic region increases but pressure of that decreases. For this region, turbulent flow is expected at post stenotic region. Again, low Re $k-\omega$ model is more acceptable for flow analysis by Shahed et al [8]. Therefore, low Re $k-\omega$ turbulent mode is taken for calculation.

Now, the Navier-Stokes equation can be given by-

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

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

$$\text{where, the strain-rate tensor } s_{ij} \text{ is given by, } s_{ij} = \frac{1}{2} \left(\frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i} \right) \quad (3)$$

$$\text{The } k \text{ equation: } \frac{\partial}{\partial t} (\rho k) + \frac{\partial}{\partial x_i} (\rho k u_i) = \frac{\partial}{\partial x_j} \left(\Gamma_k \frac{\partial k}{\partial x_j} \right) + G_k - Y_k + S_k \quad (4)$$

$$\text{The } \omega \text{ equation: } \frac{\partial}{\partial t} (\rho \omega) + \frac{\partial}{\partial x_i} (\rho \omega u_i) = \frac{\partial}{\partial x_j} \left(\Gamma_\omega \frac{\partial \omega}{\partial x_j} \right) + G_\omega - Y_\omega + S_\omega \quad (5)$$

The numerical simulations are performed by well known software ANSYS Fluent 14.5. A pressure based algorithm is chosen as the solver type. This solver is generally selected for an incompressible fluid. An extensive test is carried out with different sizes of mesh such as mesh0 (75511 element), mesh1 (82580 element) and mesh2 (90227 element) respectively. Figure 2(c) shows the pressure distributions for 65% stenosis artery with mentioned mesh sizes. In all cases, the pressure distributions are same. It implies that the solution is grid independent. Before starting of present investigation the numerical simulation is needed to be validated. 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 [2].

2.3. Boundary condition:

It assumed that the arterial walls are rigid and no-slip condition is imposed at the walls. Since the blood flow through arterial stenosis is an unsteady phenomenon and the blood flow to be fully developed at inlet region, Oscillatory physiological parabolic velocity profile is imposed for inlet

boundary condition. For this purpose an user defined function is written in C compiler for the following equation

$$u_x = u \left(1 - \frac{y^2 + z^2}{radius^2} \right), \text{ where } u = \sum_{n=0}^{16} (A_n \cos(\omega t) + B_n \sin(\omega t)) \quad (6)$$

Where A_n and B_n are the coefficients which demonstrate the unsteady parabolic nature of velocity at inlet. Sixteen harmonic coefficient are used, which results approximately same physiologically realistic flow wave form shown in figure2(a) and parabolic inlet velocity profile are shown in figure 2(b). In figure 2(a), a, b, and c represent the positions of early systole (0.041sec), peak systole (0.205 sec), and diastole (0.615 sec) respectively. In this study Reynolds number varies from 96 to 800. Since cardiac pulse cycle is 0.82sec, is found from the calculation $\omega = \frac{2\pi}{0.82} = 7.66 \text{ rad/sec}$.

3. Results and discussion:

3.1. Wall shear stress distribution:

WSS is an important factor in determining the severity of arterial stenosis. WSS depends on the viscosity of the fluid and velocity gradient and, is defined as $\tau = \mu \frac{du}{dr}$, where μ is the viscosity and $\frac{du}{dr}$ is the velocity gradient.

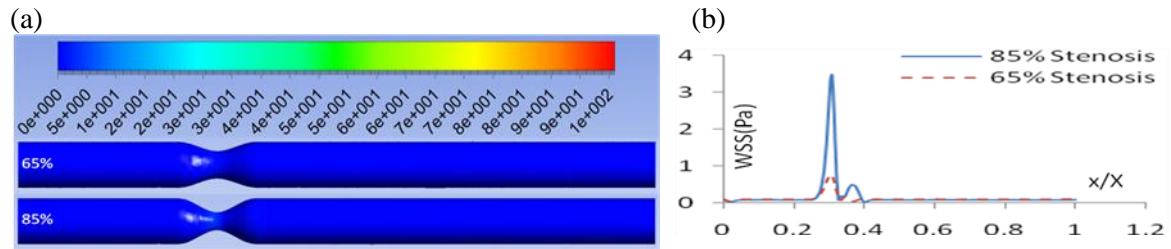


Figure 3. (a) Distribution of WSS in 65% and 85% stenotic arteries at early systole and, (b) comparison of 65% and 85% stenotic arteries for WSS distribution at early-systole.

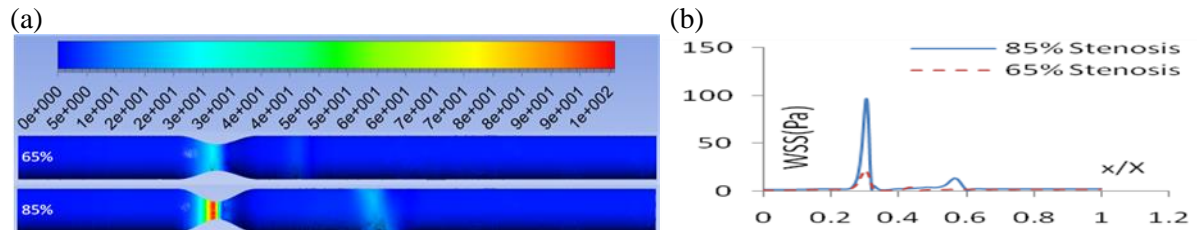


Figure 4. (a) Distribution of WSS in 65% and 85% stenotic arteries at peak systole and, (b) comparison of 65% and 85% stenotic arteries for WSS distribution at peak-systole.

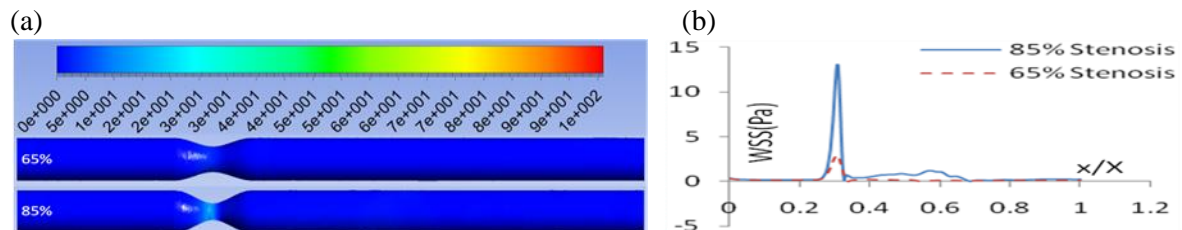


Figure 5. (a) Distribution of WSS in 65% and 85% stenotic arteries at diastole and, (b) comparison of 65% and 85% stenotic arteries for WSS distribution at diastole.

Figure 3(a) shows the distribution of WSS in 65% and 85% stenotic arteries at early-systole. WSS is uniformly distributed throughout the geometries. But in 3(b), a sudden climb to the maximum value of WSS and then sudden decline of the WSS can be noticed in the throat. Fig 3(b) depicts the maximum WSS of 65% and 85% stenotic arteries are 0.76 Pa and 3.48 Pa respectively. Figure 4 shows the distribution of WSS at peak systole. Figure 4(a) shows that, in both geometries, WSS is uniformly distributed at the pre stenotic region but significant change in WSS is occurred in throat and little variation in WSS is seen at post stenotic region. At peak systole both of the geometries increase their maximum WSS in the stenosis throat. From 4(b), we can notice the maximum WSS of 65% and 85% stenotic arteries are 21.98 Pa and, 96.78 Pa respectively. Figure 5(a) depicts that, 85% stenotic artery creates little change in WSS at the throat. From 6(b), we can see the maximum WSS of 65% and 85% stenotic arteries are 2.97 Pa and, 13.03 Pa respectively.

3.2. Pressure distribution:

Pressure distribution in both stenotic arteries at early-systole, peak-systole, and diastole is shown in figure 6.

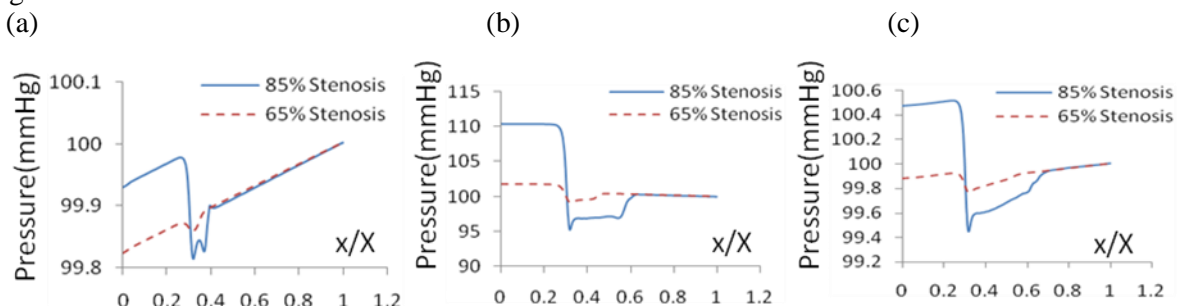


Figure 6. comparison of pressure for 65% and 85% stenotic arteries at (a)early systole (b) pick systole and (c) Diastole

Figure 6(a) shows the distribution of pressure in 65% and 85% stenotic arteries at early-systole. In both geometries the pressure is uniform and varying with a small variation throughout the geometry. No significant change of pressure for 65% stenotic artery but sudden decline of pressure for 85% stenotic artery at the throat region. Figure 6(b) shows the distribution of pressure in 65% and 85% stenotic arteries at peak-systole. High pressure is observed at pre stenotic region but suddenly a large decline of pressure is occurred in the throat region in 85% stenotic artery, and little variation of pressure is noticed in 65% stenotic artery. It describes that the pressure at inlet and throat for 65% stenotic artery are 101.77 mmHg and 99.11 mmHg and for 85% stenotic artery are 110.42 mmHg and 95.27 mmHg respectively. Figure 6 (c) shows that the comparison of pressure distribution of 65% and 85% stenotic arteries at diastole. We see that, pressure is uniform and varying with a small variation at the throat. We know that pressure difference may provide some kind of extra force that may drive blood through the stenosis with high velocity. Since The pressure difference in 85% stenotic artery is higher than that of 65% stenotic artery. So 85% stenotic artery will create more force than 65% stenotic artery at their pre-stenotic region.

3.3 velocity distribution:

In Figure 7, inlet velocity in both geometries begins with same magnitude for all cases. Velocities at the throat and post stenotic region for all cases of 85% stenotic artery are relatively higher than that of 65% stenotic artery. Recirculation regions are observed at post stenotic region in 85% stenotic artery for all cases and recirculation region increases with increasing minimum velocity at the inlet flow which have been shown in Figure 8. On the other hand, velocity is laminar in 65% stenotic artery at post stenotic region for all cases. The highest velocity (about 1.97 m/s) is observed in 85% stenotic artery at peak systole and the lowest velocity is observed in 65% stenotic artery at early systole.

4. Conclusion

A numerical study on pulsatile blood flow through 3-D stenotic artery is performed. Pressure and velocity flow field and wall shear stress occurred in the flow field are analysed. Wall shear stress distribution shows that as severity increases, shearing of flow also increases for all cases and maximum stress is exerted in throat region at peak systole. Pressure distribution demonstrate that at every case 85% stenotic artery creates more force than 65% stenotic artery at their pre-stenotic region and 85% arterial stenosis can cause fatal death by blasting the pre stenotic arterial region of the patient of high blood pressure. Numerical simulated results of this study demonstrate that velocity increases in

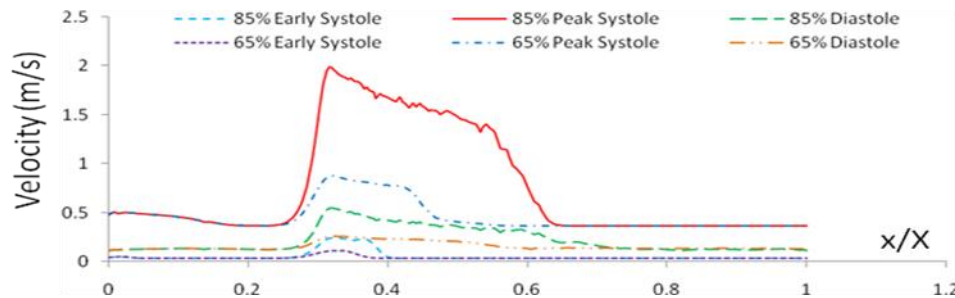


Figure 7. Comparison of velocity of 65% and 85% stenotic arteries

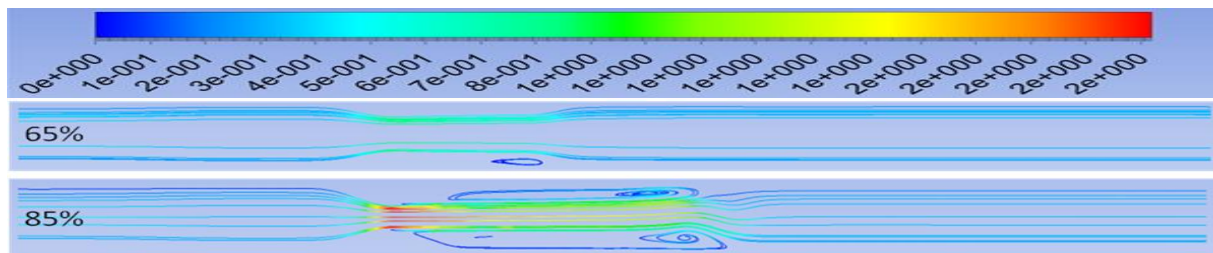


Figure 8. Streamline of velocity at pick systole for 85% stenotic arteries

throat region with the increase in severity for all cases. Due to stenotic constriction, the flow behavior changes abruptly in the downstream of the stenosis. During peak systole the flow increases at the throat region forming a jet and later disrupts suddenly forming eddies due to pressure drop in downstream side. During diastole, due to flow deceleration, the traces of the jet formed prevail for short period and there are no significant changes of flow behavior for both geometries. A recirculation region is visible at the post stenotic region at 85% stenotic artery for all cases and recirculation region increases at minimum velocity of the inlet flow. From this figure it can be interpreted that 85% stenosis can create vortex at any time phase. So 85% stenosis is very harmful for its high probability of creating vortex.

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