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CFD Analysis of Pulsatile Flow and Non-Newtonian Behavior of Blood in Arteries

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Abstract: CFD analysis plays an important role in the area of analysis of blood flow as in-vivo measurements of blood flow is costly and easily not accessible. This paper presents simulation of blood flow in healthy and stenosed coronary artery 2-D models. The simulation was done considering non-Newtonian behavior of blood and pulsatile nature of blood flow which is close to physical scenario. Pressure distribution, velocity distribution and wall shear were examined to understand their effect on Atherosclerosis.

Keywords: Pulsatile flow, non-Newtonian fluid, coronary artery, stenosis.

1 Introduction

Blood is an invincible fluid which contains many enzymes and hormones, and nurtures life. It is also responsible for nutrient and oxygen transport. It is a non-Newtonian fluid and its constitutive equation plays an important role in hemodynamics and hemorheology [1]. The systemic flow of blood is characterized mainly by its pulsatile nature and various levels of branching of the vascular network [2]. The pulsatile nature of blood flow and geometrical features such as bifurcation junctions, curved sections and flow constrictions results into flow complexities such as flow separation, recirculation and stagnation at these critical sections. The presence of these abnormal flow conditions leads to the development of coronary heart disease (CHD) [2].

Atherosclerosis is the most common form of CHD. Atherosclerosis is caused due to deposition of plaque in one or more of the coronary arteries resulting in restriction of blood flow. This deposited plaque is termed as stenosis.

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Numerical blood flow simulation is gaining favor as a tool for predicting cardiovascular disease and aiding decision making process during the treatment of these diseases [3, 4].This is because in-vivo measurements of blood flow in an artery is costly, easily not accessible and are usually not accurate enough to predict pressure distribution and wall shear stress. CFD models of blood flow in artery may help researchers in testing how flow conditions leads to CHD, deciding the severity of disease and how to prevent the disease.

In the present study, numerical simulations of two-dimensional unsteady flow in a representative atherosclerosis (stenosed) model and one healthy model of the coronary artery is presented. The purpose of this study is to report the effect of stenosis and its severity on pressure and wall shear stress, and to establish comparison of these flow parameters with a healthy coronary artery.

In this work, the differences between steady and pulsatile flow conditions for the flow field within a diseased coronary artery with rigid walls were also examined. This is done with purpose to understand whether flow pulsatility is important for the development of CHD in the coronary arteries.

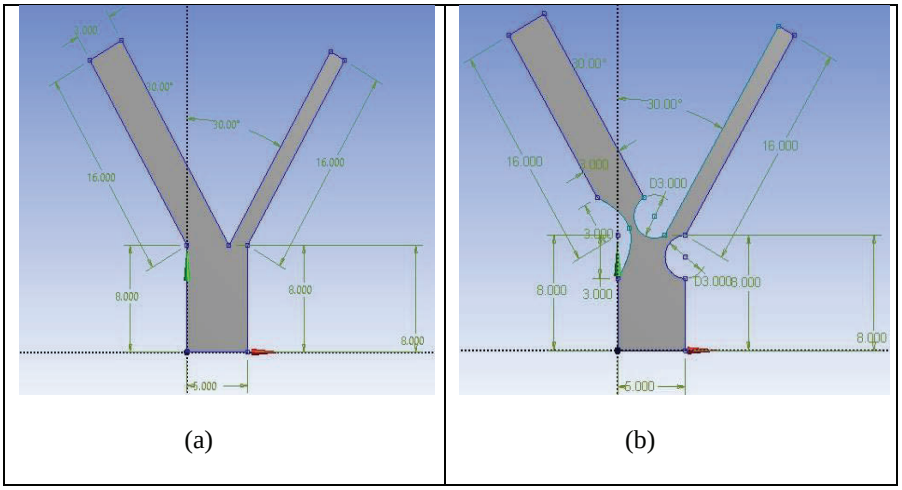
2 Materials and methods

A comparative two-dimensional numerical simulation of an idealized coronary artery was developed to predict flow behavior within healthy and stenosed coronary arteries. Dehlagi, Shadpoor and Najarian in their experiments on hemodynamic effects in elastic silicon rubber models had shown that geometry, artery wall structure and hemodynamic plays an important role on deposition of plaque i.e. creation of stenosis [5]. Pressure gradients, velocity distribution and shear stress on the wall are very important parameters in blood flow analysis. Clinically important plaque deposits are most common in areas of complex flow in the arteries. These complex flow regions often occur due to branching, bifurcations and curvature of the arteries [6]. The analysis of models with varying percentage of stenosis and at different number of locations was carried out. However, in this paper only two models representing a healthy artery, and artery with multiple stenosis (83 % stenosis at thicker section of bifurcation & 60 % stenosis at thinner section of bifurcation) are discussed in detail. Hereafter, stenosed model refers to above mentioned stenosis description. The models were built and meshed using ANSYS FLUENT 13.0 (ANSYS Inc., USA).

2.1 Model geometry and mesh

In most of the earlier research papers artery was modeled with the assumption that the geometry of a healthy coronary artery is almost tubular and symmetrical [7]. In

the present work also a 2-D model of coronary arteries were also modeled with this assumption. Since clinically relevant plaque deposits are most common in areas of complex flow which often occur due to branching or bifurcation, the artery was modeled with bifurcation so as to compare the flow parameters of healthy artery with diseased artery. The length and diameter of the part of artery which is not bifurcated are 8 mm and 5 mm respectively. The length of the part of artery after bifurcation is 16 mm for both thicker and thinner part of bifurcated artery. However, diameter of thicker part is 3mm and that of thinner part is 2 mm. The geometry of healthy coronary artery is shown in Fig. 1 (a). The geometry of the stenosed artery is similar to that of healthy artery. The only difference between the two model is reduction in area of flow near the point of bifurcation . The geometry of diseased artery is shown in Fig. 1 (b).



(a) (b)

Figure 1: (a): Geometry of healthy artery, (b): Geometry of stenosed artery.

The geometry of healthy artery was discretized into 554 elements, while that of diseased artery consisted of 499 elements. Grid independency test was performed to decide the density of mesh so that computational error and computational time could be minimized.

2.2 Fluid properties and boundary conditions

The literature on rheology of blood strongly indicates that the non-Newtonian behavior of blood flow cannot be neglected in a variety of geometries (e.g. [8,9]). Blood is a shear thinning fluid as it gets less viscous with the increase in shear rate.

The Carreau-Yasuda (C-Y) model is most commonly used to simulate blood as it has certain advantages over the Casson model and the Power Law model [3, 10]. As per C-Y model:

$$\mu_{eff}\left(\dot{\gamma}\right) - \mu_{\inf} = \left(\mu_0 - \mu_{\inf}\right)\left(1 + \left(\lambda\,\dot{\gamma}\right)^{a}\right)^{\frac{n-1}{a}} \tag{1}$$

where μ_0 and μ_{inf} are the dynamic viscosities at zero and infinite shear rate respectively and μ_{eff} is the effective viscosity. The shear rate is represented by $\dot{\gamma}$ and λ is a characteristic viscoelastic time of the fluid. The power law index parameters 'a' and 'n' can be determined from the experimental data [3]. In the simulations presented in this work, the reduced form of C - Y model i.e. Carreau model is used where value of 'a' is fixed to 2, since this model is available in solver Fluent. The values of parameters of Carreau model used in this simulation are: μ_0 = 0.056 Pa s, μ_{inf} = 0.0035 Pa s, λ = 3.313 s, n = 0.3568. Blood was modeled as a homogenous and incompressible fluid with a density value of 1060 Kg/m^3. Blood flow was assumed as laminar as in Valencia and Solis [6]. The incompressible Navier - Stokes equation and continuity equation were used as the governing equations.

Continuity equation:

$$\nabla.u = 0 \tag{2}$$

Navier-Stokes equation:

$$\rho\left[\frac{\partial u}{\partial t} + \left(u.\nabla\right)u\right] = -\nabla p + \mu_{eff}\,\nabla^2 u \tag{3}$$

The applied boundary conditions on the fluid domain are a time dependent inlet velocity and a constant pressure of 100 mm Hg at the outlet. The walls of the artery models are assumed to be rigid as in He et al [11].

Pulsatile flow was included in the simulation by imposing a time - varying velocity boundary condition at the inlet. Sinusoidal profile of inlet velocity during systole [2, 11, 12] was used as an approximation to a physiological pulse. The pulsatile flow profile is shown in Fig. 2. The pulsatile flow in a period is considered as combination of two phases i.e. systolic and diastolic. During systolic phase, the sine wave has a peak velocity of 0.5 m/s and minimum velocity of 0.1 m/s. A constant velocity i.e. a steady flow during diastole phase represents the cut-off in supply from the heart. Assuming a rapid heartbeat of 120 per minute, the duration of each period is 0.5 s. This pulsatile blood flow model was proposed by Sinnott, Clearly and Prakash [2]. An user defined function (udf) for the proposed inlet velocity model was written. For a comparison between steady flow and pulsatile flow, an inlet velocity of 0.36 m/s [13] was taken in the boundary condition of the steady flow case.

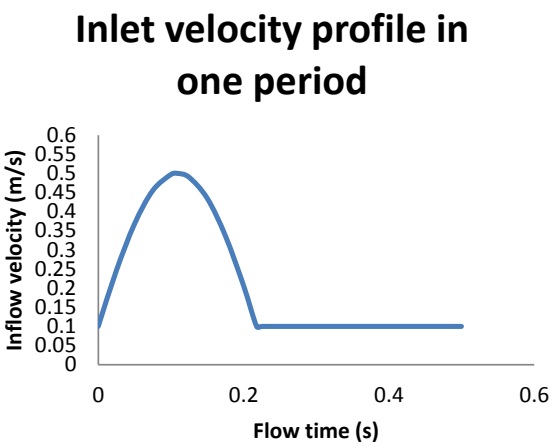


Figure 2: Inlet velocity profile.

3 Results and discussion

To observe the variation in pressure distribution, velocity distribution and wall shear stresses of the artery due to pulsatile nature of flow, simulation was done for 10 time steps of 0.05 s. These time steps attributes to one cycle of pulsatile flow, where up to 0.218 s is systolic phase and from 0.218 s to 0.5 s is diastolic phase as shown in Fig. 2.

3.1 Healthy artery vs stenosed artery

The pressure and wall shear stress distributions at systole (t = 0.05 s) for the healthy bifurcated coronary artery are shown in Fig. 3(a, b). As shown in Fig. 3(a) pressure is maximum at the point of bifurcation and downstream the bifurcation it develops higher wall shear stress [Fig. 3(b)].

Velocity vectors for healthy and stenosed arteries model for t = 0.05 s are shown in Fig. 4(a, b). Evidently, velocity gradients are higher downstream the bifurcation point for the healthy artery as shown in Fig. 4 (a). Velocity gradient for stenosed artery is maximum at section of 83% stenosis due to flow constriction as shown in Fig. 4 (b). It can also be observed from Fig. 4 that velocity distribution fairly remains same within the core of coronary artery for both cases. Reduction in velocities near the wall (almost zero at the wall of artery confirming no-slip condition) and downstream of stenosis region can also be observed from Fig. 4.The increase in maximum velocity due to 83 % stenosis is almost 3 times of maximum velocity of healthy artery for the same time interval. This sudden increase in velocity

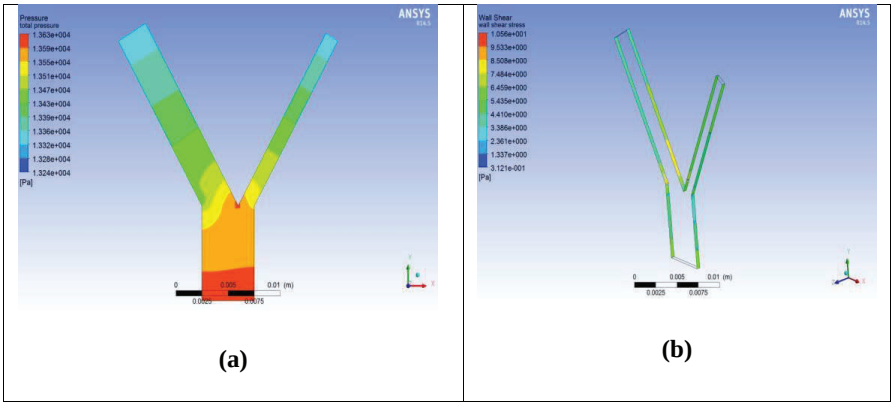


Figure 3: (a): Pressure distribution of blood flow, (b): wall shear stress distribution for healthy artery at t = 0.05 s.

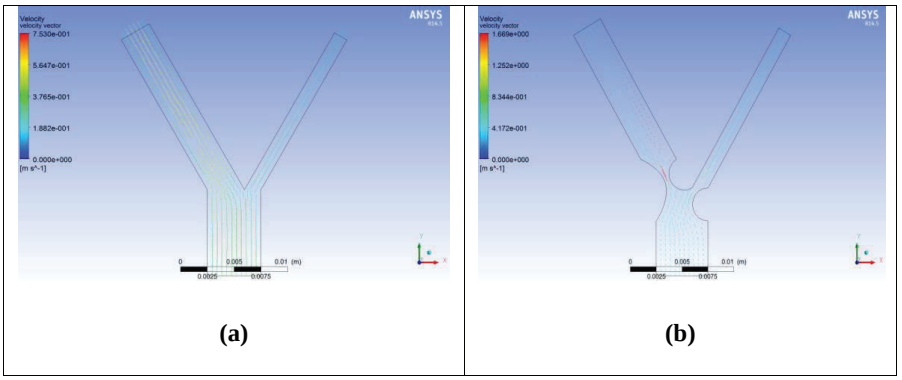


Figure 4: (a): velocity vectors of healthy artery, (b): velocity vectors of stenosed artery for t = 0.05 s.

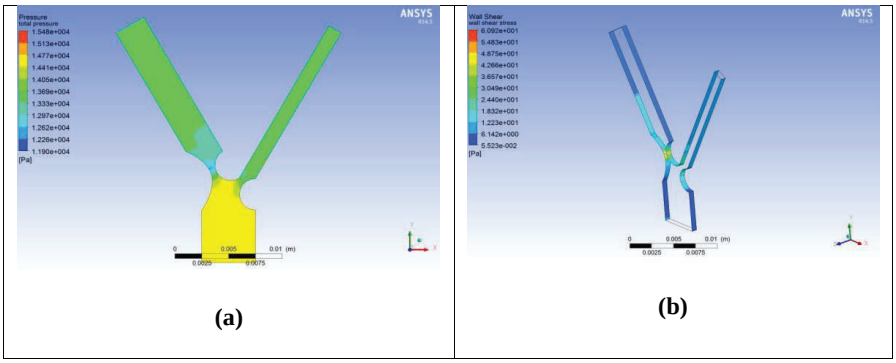


Figure 5: (a): Pressure distribution, (b): WSS distribution of stenosed artery at t = 0.05 s.

imposes more pressure at the inlet which means heart has to pump out more blood rapidly in same period resulting in high blood pressure.

The pressure distribution and wall shear stress distribution for the stenosed artery at systole (t = 0.05 s) are shown in Fig. 5 (a & b). As shown in Fig. 5 (a) the maximum pressure is located at the point of bifurcation and in Fig. 5 (b) maximum wall shear stress (WSS) is located at the point of maximum stenosis. Also, the flow in stenosed artery does not have stagnation point, so it exhibits more extended region with highest pressure [6] compared with the pressure distribution in the healthy bifurcate artery, see Fig. 3 (a). The contour of pressure distribution closely resembles the waveform of inlet velocity. The high velocities at the stenosis generates there a low pressure region as can be seen in Fig. 4(b) and 5(a) The maximum values of WSS in the stenosed artery model are almost 6 times higher as compared to the healthy model. High WSS is considered as a major factor in the development and growth of stenosis.

The results of each time steps for both healthy and stenosed coronary arteries are presented in Table 1. A trend can be observed from Table 1 that maximum values of flow parameters are initially increasing with time till t = 0.1 s, then it starts decreasing till time t $\approx$ 0.25 s and remains constant thereafter. This is because the pulsatile nature of blood flow is sinus (systolic phase) till t = 0.218 s and reaches its peak value of inlet velocity 0.5 m/s at t $\approx$ 0.1 s. In diastolic phase (t = 0.218 - 0.5 s) representing the cut-off in supply from heart, the inlet velocity becomes constant (0.1 m/s) which results steady flow of blood, and so values of flow parameters becomes constant in diastolic phase.

An increase in wall shear stress was also observed with increase in percentage of

Table 1: Flow parameters at different time interval.

Time (s)	Maximum Velocity (m/s)		Maximum Pressure (mm Hg)		Maximum Wall Shear Stress (Pa)	
	Healthy	Stenosed	Healthy	Stenosed	Healthy	Stenosed
0.05	0.56	1.67	102.23	110.78	8.51	48.75
0.1	0.75	2.2	102.23	116.10	10.56	60.92
0.15	0.65	1.9	102.23	113.48	8.51	48.75
0.2	0.38	1.1	100.43	102.68	4.41	24.40
0.25	0.19	0.55	100.43	102.68	3.39	18.32
0.3	0.19	0.55	100.43	102.68	3.39	18.32
0.35	0.19	0.55	100.43	102.68	3.39	18.32
0.4	0.19	0.55	100.43	102.68	3.39	18.32
0.45	0.19	0.55	100.43	102.68	3.39	18.32
0.5	0.19	0.55	100.43	102.68	3.39	18.32

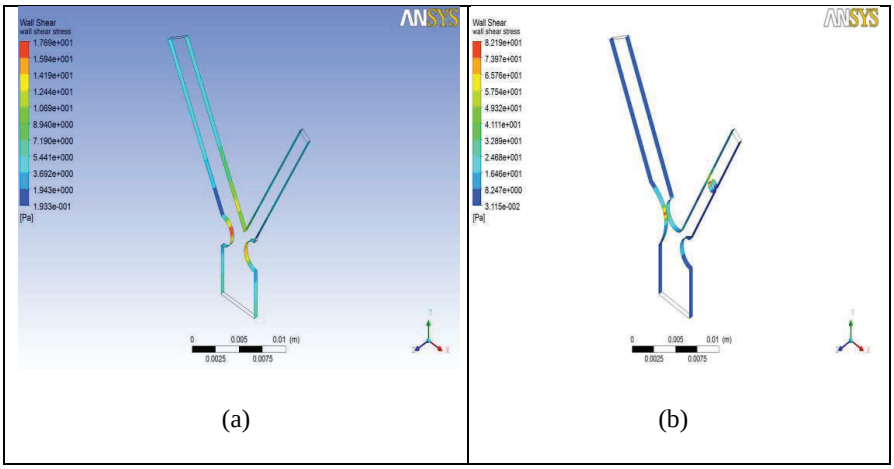


Figure 6: (a): WSS for single stenosis (60%), (b): WSS for multiple stenosis (83%, 60% & 75%).

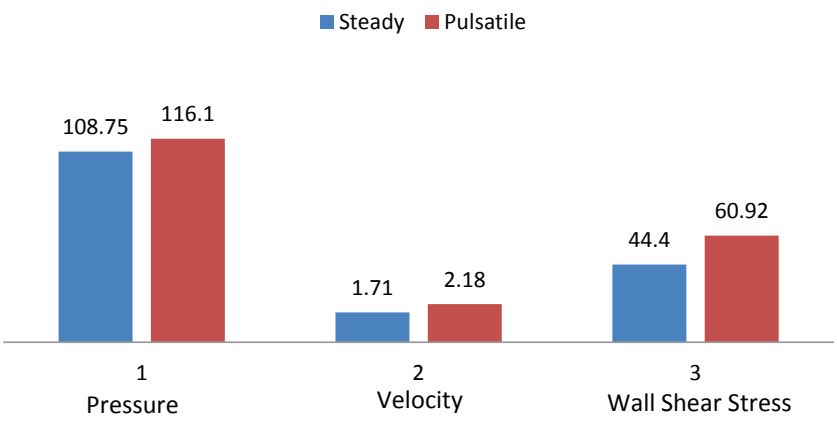


Figure 7: Comparison between steady and pulsatile flow.

stenosis and more number of locations having plaque deposition while analyzing different diseased arteries. This analysis is not explained in detail in this paper, however end results are shown in Fig. 6 (a, b). This trend clearly indicates that severity of stenosis is directly related to wall shear stress.

3.2 Steady flow vs pulsatile flow

A separate analysis of stenosed artery for the boundary condition of steady flow was carried out. Velocity at the inlet was kept constant at 0.36 m/s and pressure at the outlet same as of pulsatile flow boundary condition. A comparison between these two boundary conditions can be made for the same inlet velocity. In pulsatile flow boundary condition, at t = 0.05 s inlet velocity is approximately 0.36 m/s. Fig. 7 shows the values of flow parameters (such as pressure, velocity and wall shear stress) for the two boundary conditions. It clearly depicts that the values of flow parameters particularly wall shear stress are low in the steady flow as compared to pulsatile flow. This means an underestimation of flow parameters results, when pulsatile nature of blood flow is neglected.

4 Conclusion

This paper present the numerical investigation of blood flow in the coronary artery. The flow parameters of healthy bifurcated and stenosed bifurcated coronary artery for pulsatile nature of blood flow and non-Newtonian behaviour of blood were compared for one cycle. Increase in the magnitude of pressure, velocity and wall shear stress was observed due to stenosis in the artery. These factors attributes to in-

crease in blood pressure, decrease in downstream flow and further increase in the percentage level of stenosis.

The transient flow field from the pulsatile case differ markedly from the steady flow field. The steady flow field underestimates the values of flow parameters (pressure, velocity and WSS). Further studies more close to physical scenario are necessary to investigate the effects of flow parameters on the development of stenosis.

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