

Influence of non-Newtonian Viscosity of blood on Wall Pressure in Right Coronary Arteries with Two Stenoses

Biyue Liu
Department of Mathematics
Monmouth University
West Long Branch, NJ07764
E-mail: bliu@monmouth.edu

Abstract: Three dimensional mathematical models are developed to simulate the blood flows in patient specific right coronary arteries with two stenoses. Simulations are carried out with various flow parameters under physiological conditions. Both Newtonian and non-Newtonian blood viscosity models are applied in the simulations to examine the influence of non-Newtonian viscosity of blood on the wall pressure. The numerical observations show that the non-Newtonian viscosity of blood does not have a significant effect on the time averaged wall pressure and pressure drop coefficient.

Keywords: right coronary artery, blood viscosity, Non-Newtonian, pressure drop coefficient.

1. Introduction

It is well known that blood is a complex suspension that demonstrates non-Newtonian rheological characteristics. Many numerical investigations have been conducted to examine the non-Newtonian effect on the blood flows in right coronary arteries and curved tubes [1-4]. Some studies found non-Newtonian rheology important [1, 3], while others suggested that under normal physiological conditions, the non-Newtonian effect may not be significant [2, 4]. However, most of the work in literature focused on examining how the non-Newtonian blood viscosity affects the flow velocity and the wall shear stress. Not much work has been done on investigating the influence of the non-Newtonian blood viscosity to the wall pressure and the pressure drop coefficient.

The objectives of the present work are to study the blood flows in patient specific right coronary arteries with two stenoses and to investigate the effect of the non-Newtonian blood viscosity on the local wall pressure (WP), the pressure drop

coefficient (CDP) and the magnitude of the local spatial gradient of wall pressure (WPG).

2. Flow Model and Numerical Method

A three dimensional model with a patient specific geometry is developed to simulate the blood flow. The blood is assumed to be laminar, incompressible and unsteady. The artery wall is treated to be inelastic and impermeable. The flow is governed by the three-dimensional Navier-Stokes equations. The geometry (see Figure 1) of the artery is reconstructed based on the lumen contour curves extracted from a dataset of IVUS slices [5]. The radius of the cross section at the inlet and outlet is 0.001628m and 0.001477m, respectively. The area stenosis severity for the proximal stenosis and the distal stenosis is 66% and 47%, respectively.

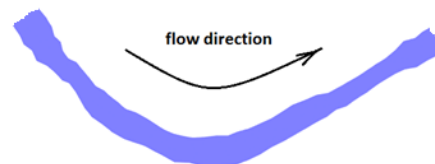


Figure 1 Geometry of the stenotic artery

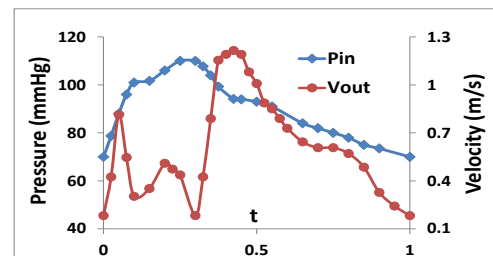


Figure 2 Pulse waveforms of velocity and pressure

The boundary conditions are imposed as following: A no-slip condition is applied to the velocities at the wall boundary. At the inlet boundary, a time dependent pressure with waveform (Figure 2 P_{in}) and no viscous stress

condition are imposed. At the outlet boundary, a fully developed velocity profile with pulse waveform (Figure 2 Vout) is imposed as the normal outflow velocity.

The blood is treated as a non-Newtonian fluid. Many non-Newtonian viscosity models have been used by researchers [1- 4, 6, 7]. The Carreau model and the Power-Law model are among the most reputable models to simulate the blood flow through arteries. Cho and Kensey showed that the Carreau model with the following viscosity-shear rate relation is well adopted with experimental data [3]:

$$\eta = \eta_{\infty} + (\eta_0 - \eta_{\infty}) [1 + (\lambda \dot{\gamma})^2]^{\frac{n-1}{2}}, \quad (1)$$

where $\eta_0 = 0.056 \text{ Pa}\cdot\text{s}$ and $\eta_{\infty} = 0.00345 \text{ Pa}\cdot\text{s}$ are the zero and the infinite shear rate viscosity respectively, $\dot{\gamma}$ is the shear rate, $\lambda = 3.313 \text{ s}$ is the relaxation time constant, and $n = 0.3568$ is a dimensionless parameter. To investigate the effect of non-Newtonian viscosity of blood on the *CDP* and the *WPG*, we also use other two models: 1) treating the blood as a Newtonian fluid with a constant viscosity $\eta = 0.00345 \text{ Pa}\cdot\text{s}$; 2) treating the blood as a non-Newtonian fluid obeying the Power Law with the viscosity-shear rate relation [3, 7]:

$$\eta = \eta_0 (\dot{\gamma})^{n-1} \quad (2)$$

where η_0 is the consistency index and n is the Power Law index. Various values of η_0 and n are chosen in computation to find a better fitting Pow Law model for the right coronary artery. All of the three models have been commonly used to simulate blood flows in recent years [3, 4, 6].

3. Notations and Use of COMSOL

When carrying the computer simulation, the inlet and the outlet of the artery were extended in length by .4 cm and .75 cm, respectively, in the direction normal to the inlet/outlet cross-sections to reduce the influence of the boundary conditions in the region of interest. Numerical computations were performed using COMSOL5.2. Four consecutive cardiac cycles were simulated to ensure that the flow is truly periodic and the computations were repeated over tetrahedral meshes with different sizes to

confirm the independence of the numerical solutions on spatial mesh.

The pressure drop (*PD*) along the artery length is defined as $P - P_{in}$, where P_{in} is the blood pressure at the inlet of the artery segment (see Figure 2). The pressure drop coefficient (*CDP*) is defined as

$$CDP = \frac{\overline{(P - P_{in})}}{0.5 \rho \overline{U}}, \quad (3)$$

where ρ is the density of the blood; $\overline{(P - P_{in})}$ is temporal mean of pressure drop from the inlet along the artery axial length; \overline{U} is the spatial and temporal mean blood velocity at the inlet of the artery. The spatial gradient of the wall pressure (*WPG*) is defined as

$$WPG = \sqrt{\left(\frac{\partial p}{\partial x}\right)^2 + \left(\frac{\partial p}{\partial y}\right)^2 + \left(\frac{\partial p}{\partial z}\right)^2} \quad (4)$$

4. Results

Numerical results obtained from the Newtonian model and the non-Newtonian models obeying the Power Law and the Carreau model are compared and analyzed to examine the effect of non-Newtonian viscosity of blood on the computation of the pressure drop coefficient in blood simulations of stenotic right coronary arteies. Figure 3 includes the contour plots of the *WP* and the *WPG* on the lumen surface at the peak flow rate ($t = 0.425$). Figure 3 shows that both the wall pressure and the wall pressure gradient change markedly as the fluid moves through the artery.

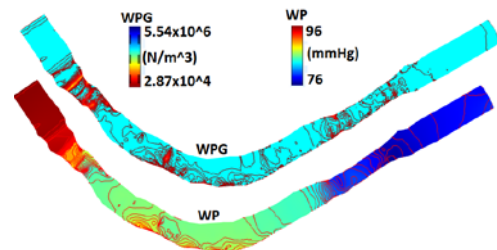


Figure 3 Contour plot of *WP* and *WPG*

Figure 4(a) presents the plots of slice-averaged *CDP* from the inlet to the outlet of the

artery. *C*-model and *N*-model represent the Carreau model and Newtonian model, respectively. *P1*-, *P2*- and *P3*-models are Power Law models with different choices for η_0 and n in equation (2) as following: For *P1*-model, $\eta_0 = 0.035\text{kg/m}\cdot\text{s}$, $n = 0.708$; For *P2*-model, $\eta_0 = 0.035\text{kg/m}\cdot\text{s}$, $n = 0.65$; For *P3*-model, $\eta_0 = 0.017\text{kg/m}\cdot\text{s}$, $n = 0.708$ [7]. Figure 4(b) plots the phasic waveforms of the *PD* at a point near the inner wall of the neck of the distal stenosis for different blood viscosity models. Figure 4(c) shows the slice averaged *WPG* along the artery.

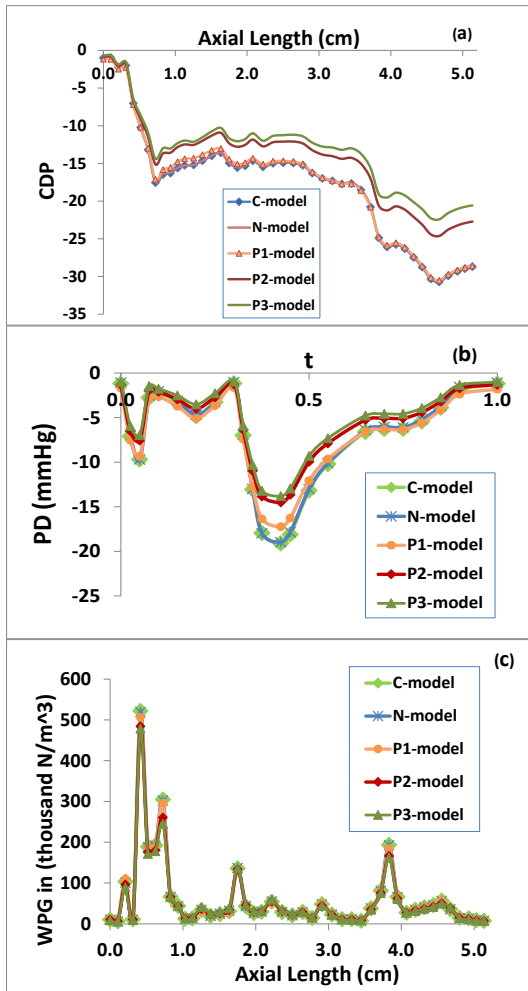


Figure 4. (a) Slice-averaged *CDP* along the artery, (b) phasic *PD*, (c) Slice-averaged *WPG*

From Figure 4(a) and 4(c) we can see that the plots of the slice-averaged *CDP* and *WPG*

resulted from the Carreau model, Newtonian model and *P1*-model are overlapped and there is no significant difference between the three curves. This indicates that under the setting of the parameters as in equation (1) for the Carreau model and the setting of the parameters of *P1*-model in equation (2) there is no significant effect of the non-Newtonian viscosity of blood on the slice-averaged *CDP* and *WPG* when the blood viscosity constant of the Newtonian model is chosen as the same value as the infinite shear rate viscosity η_∞ in the Carreau model.

Compared to the slice-averaged *CDP* resulted from the Carreau model, the maximum relative errors of the slice-averaged *CDP* resulted from Newtonian and *P1*-Power Law models are less than 5%. Figure 4(b) shows that the *P1*-model underestimates the *PD* at the diastolic peak flow ($t = 0.425$) while it reaches a good agreement with *C*-model and *N*-model during the remaining parts of a cardiac cycle. The plots for *P2*- and *P3*-models in both Figure 4(a) and Figure 4(b) are significantly different from those for *C*-, *N*- and *P1*-models. This indicates the *CDP* of the blood simulation in a stenotic right coronary artery resulted from the Power Law is sensitive to the choice of the η_0 and n .

5. Discussions

Cho et al. investigated the effect of shear-rate-dependent non-Newtonian viscosity of blood on flow in large arterial vessels with various degrees of atherosclerosis [3]. They compared the numerical solution calculated using the modified Powell-Eyring non-Newtonian model with the data of Newtonian fluid. Their results show that the effect of non-Newtonian viscosity on the overall pressure drop is very small for flows of high Reynolds number, while the non-Newtonian viscosity effect of blood on the pressure drop could not be neglected at the flow condition with Reynolds number ≤ 100 . In the present work we compare the simulation results using Newtonian model, the Carreau and Power-Law non-Newtonian models and find that there is no significant effect of non-Newtonian viscosity of blood on the slice-averaged *CDP* and *WPG*. The Power Law underestimates the pressure drop at the peak flow ($t = 0.425$), while it results in a

slice-averaged *CDP* with insignificant difference compared to that from the Carreau model.

6. Conclusions

The blood flow in human right coronary arteries with serial stenoses is complex. The wall pressure, the pressure drop coefficient and the wall pressure gradient change markedly on the lumen surface. A comparison indicates that non-Newtonian viscosity of blood does not have a significant effect on the computation of the slice-averaged *CDP* and the mean *WPG* in the patient specific stenotic right coronary artery model used in this study.

7. References

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