Numerical simulations of the pulsatile blood flow in narrowing small vessels using different rheological models

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Abstract. The study of the blood flow through the circulatory system vessels depends on the rheological properties of the blood, the pulsatile nature of the blood flow and the vessel diameter. Blood behaves as a non-Newtonian fluid in small vessels, in situations of complex geometry and in certain cardiovascular diseases such as hypertension, coronary artery diseases, etc. In this article, we present two-dimensional computational simulation of blood flow in an axisymmetric narrowed small vessels (stenosis artery) taking into consideration the non-Newtonian fluid properties. The study is based on the numerical solution of the Navier-Stokes equations for Pulsatile flow using three constitutive relations for modeling the rheological blood behavior, ie; the Cross law; the Generalized power law; Gpl, and the Herschel-Bulkley law. We examined the flow characteristics particularly the velocity, shear rate distribution, wall shear stress and time-dependent vortex structures with respect to critical flow conditions in small vessels. Results indicate that the instantaneous flow characteristics are significantly affected by the blood rheological models considered, especially, in zones of important shear rate.

1. Introduction

Blood behaves like a Newtonian fluid in large arterial vessel, where the typical shear rate exceeds 100s⁻¹ [1], [2], [3]. However, in the microcirculatory system, small arteries and capillaries, the strain rate is low and blood exhibits non-Newtonian effects, ie. the viscosity is dependent on blood shear rate. This behaviour is explained by the deformation and aggregation (roulleaux formation) of red blood cells (RBCs). The rheological properties of blood are also influenced by various cardiovascular diseases such as diabetes, myocardial infarction, hypertension, cholesterol, etc. The effects that are usually common to all these diseases are changes in the volume of red blood cells (RBCs) or even in RBC deformability, due to the blood abnormalities, and their tendency to align with the flow field at high shear rates. Moreover, the viscosity of the blood shows a shear-thinning property; the viscosity decreases with increasing shear rate. Several rheological models were investigated for blood to simulate this behavior [4],[5], [6], etc. However, it still not totally agreed upon the scientific community in which one best represents the blood viscosity behaviour.

A pulsatile flow consists of a mean flow to which an oscillating component is added. The cyclic nature of heart pump creates pulsatile conditions in all arteries. The heart ejects and fills with blood in alternating cycles called systole and diastole. The study of the pulsatile blood flow in narrowed arteries has attracted the interest of many researchers, treating blood as Newtonian and non-Newtonian fluid. Womersley [7] was one of the first to analyse the phase relationship between the pressure
gradient and the resulting velocity field for the pulsatile flow in mammalian blood vessels. Tu et al. [8] considered the blood obeying Herschel-Bulkley, Bingham and Power law fluid models to simulate the blood flow through arterial stenosis. They compared non-Newtonian results to those obtained with the Newtonian fluid for both steady and pulsatile flow in the case of moderate and severe stenosis degrees. Ishikawa et al. [9] investigated the effect of pulsation and the rheological properties of blood on flow patterns. They used the bi-viscosity model as a constitutive equation for blood. More recently, Razavian et al. [10] compared for various rheological models, the hemodynamic flow parameters in pulsatile nature of blood flow. They examined in particular the effect of the frequency of pulsation on the flow field and location of the vortex formation distal to the stenosis for different Womersley numbers. Details of a numerical comparison, based on the finite element method, between a Newtonian, Cross and generalised power-law models are to be found in the paper of Achab et al. [11]. in the case of the pulsatile blood flow through an arterial stenosis. Gambaruto et al. [12] presented a sensitivity study on the influence of both the blood viscosity model and the geometry variability in a carotid model.

In this paper, we study numerically the pulsatile blood flow in a narrowed small artery, assuming axial symmetry. We aimed to investigate the effects of rheological blood viscosity on the blood flow distribution and hemodynamic parameters. To account for the Non-Newtonian behavior of the blood, we consider the Cross, generalized power law (Gpl), regularized Herschel-Bulkley, and Newtonian models.

2. Problem formulation

We consider the problem of blood flow in an artery and in the presence of stenosis. We use an axisymmetric, cylindrical coordinate system, \((r; z)\), to model the flow dynamics, in which \(r\) and \(z\) denote the radial and axial coordinates, respectively. The flow geometry and the coordinate system are illustrated in figure 1. The shape of the boundary stenosis segment versus \(z\) is given by the equation of Young [13]:

\[
r(z) = R - \frac{\delta}{2} \left[ 1 + \cos \left( \frac{2\pi z}{L_0} \right) \right] \quad \text{for} \quad L_1 \leq z \leq L_0 + L_1, \quad (1)
\]

where \(R\) denotes the radius of the non-stenosed part of the artery \((R = 0.5 \text{ mm})\), \(\delta\) denotes the maximum width of the restriction, \((\delta = 0.75R; 75\% \text{ stenosis degree})\). \(L_1\) and \(L_2\) the respective upstream and downstream lengths. \((L_1 = 20 D \text{ et } L_2 = 30 D)\), \(L_1, L_2\) are assumed to be large in comparison to its radius \(R\), so that the end effects can be neglected.

\[\text{Figure 1. Geometry of artery with stenosis}\]

2.1. Governing equations

The governing equations of the problem correspond to those of mass and momentum conservation, written in velocity-pressure form, for the two-dimensional unsteady conditions:

\[
\frac{\partial \rho}{\partial t} + \nabla \cdot (\rho \mathbf{V}) = 0
\]
\[ \frac{\partial (\rho V)}{\partial t} + \nabla (\rho V V) = \nabla \cdot \left( -p I + \tau \right) + F \]  

(3)

In the above equations, \( V = (u; w) \) denotes the velocity field in which \( u \) and \( w \) represent the radial and axial velocity components, respectively, \( p \) is the pressure field, \( \rho \) is the density of the blood, \( t \) denotes time, and \( F \) is the body force per unit volume, which is neglected. \( I \) is the unit tensor. \( \tau \) represents the strain tensor of the fluid: \( \tau = \dot{\gamma} \), with \( \dot{\gamma} \) is the rate-of-strain tensor given by \( \dot{\gamma} = \left( \nabla V + \nabla V^T \right) \). The arterial wall was assumed rigid and blood is modeled as incompressible fluids with density \( \rho = 1060 \text{ kg/m}^3 \).

In the present simulation the following boundary conditions:

- At the inlet boundary a laminar Poiseuille velocity with the time-varying function (presented in figure 2) is prescribed. This inlet velocity presents an acceleration phase (systole) and a deceleration phase (diastole).
- The vessel walls are assumed to have a no-slip boundary condition applied: \( V = 0 \)
- Along the line axis of symmetry, both the normal velocity and the first-order derivative of the axial velocity in the radial direction are assumed to be zero.
- For the outlet boundary, an outflow (no gradient) boundary condition is applied.

![Figure 2. Boundary conditions imposed at the inlet](image)

2.2. Blood viscosity model

Four different rheological models are chosen for the current study: Newtonian, Cross, generalized power law (Gpl) and Herschel-Bulkley, whose corresponding function and parameters are summarised below in Table 1, whereas a plot of the viscosity and strain rate relationships is presented in figure 3.

| Model               | Viscosity function                                      |
|---------------------|--------------------------------------------------------|
| Newtonian           | \( \mu = 0.0045 \text{ Pa.s} \)                        |
| Cross [14]          | \( \mu(\dot{\gamma}) = \mu_0 + \frac{\mu_0 - \mu_\infty}{1 + (\beta |\dot{\gamma}|)^s} \) |
|                     | \( \mu_0 = 114.5 \text{ mPa.s}, \mu_\infty = 4.29 \text{ mPa.s}, \beta = 5.29, s = 0.73 \) |
| Generalised power law [15] | \( \mu(\dot{\gamma}) = k (\dot{\gamma})^n |\dot{\gamma}|^{n-1} \) |
\[
\begin{align*}
  k(\dot{\gamma}) &= \mu_\infty + \Delta \mu \exp \left[-(1 + \frac{|\dot{\gamma}|}{a}) \exp(-\frac{b}{|\dot{\gamma}|})\right] \\
n(\dot{\gamma}) &= n_\infty - \Delta n \exp \left[-(1 + \frac{|\dot{\gamma}|}{c}) \exp(-\frac{d}{|\dot{\gamma}|})\right]
\end{align*}
\]

where \(|\dot{\gamma}|\) is the flow shear rate magnitude which is defined by \(|\dot{\gamma}| = \sqrt{\frac{1}{2} \{ \dot{\gamma} : \dot{\gamma} \}^2}\). The parameters of the different models are given by curve-fitting to experimental data [17] for hematocrit value of 48% using a non-linear least-squares method [18].

The Papanastasiou [19, 20] regularization method is adopted to avoid computational problems of the Herschel-Bulkley model due to singularity close to zero shear rates. Thus, at all shear rates, the original form of the Herschel-Bulkley model is replaced by the following equation:

\[
\mu(\dot{\gamma}) = \left(\frac{\tau_0}{|\dot{\gamma}|} \left[1 - e^{-m|\dot{\gamma}|} \right] + k |\dot{\gamma}|^{m-1}\right),
\]

where \(m\) is the stress growth exponent, which must be large enough to approach the Herschel-Bulkley model. In this study \(m\) is taken equal to 10^5.

\[
\begin{align*}
  &\mu_\infty = 5.09 \text{ mPa.s}, \ n_\infty = 1.0 \text{ mPa} \Delta \mu = 24.4 \ \text{mPa.s}, \\
  &\Delta n = 0.48 \text{ mPa}, a = 48, b = 6, c = 30, d = 3.0
\end{align*}
\]

**Figure 3.** Shear stress plots versus the shear rate for different blood viscosity models; comparisons with experimental data at Htc=48%.

The apparent viscosity curves as functions of the shear rate are shown in figure 3 for all of four rheological models used. A comparison with the experimental blood viscosity data given by Brooks et
al. [17] for hematocrit ($Htc$) rate of 48% is also presented. For the Newtonian case, the viscosity is constant and takes a value of 0.0035 Pas. For the Cross, Gpl and Herschel-Bulkley models, the viscosity fits the experimental data as well as at all values of shear rate in the range of $2 \times 10^{-1} - 10^{3}$ s$^{-1}$. However, the significant differences between the models are located at the extreme values of the shear rates, mainly at low shear rates (below $2 \times 10^{-1}$ s$^{-1}$). Important viscosities values are observed for the Herschel-Bulkley model. At high shear rates (above $10^{3}$ s$^{-1}$), the Newtonian, cross and Gpl models seem to have similar viscosities curves and hence, tend to constant asymptotic viscosities with different values. The Herschel-Bulkley model diverges further from the other models and predicts lower viscosities values.

2.3. Numerical solution methodology

The governing differential equations have been solved numerically using the commercial CFD package ANSYS Fluent 13.0 based on a finite volume approach. The second order upwind scheme is used for discretization of the momentum equation. To insure the velocity-pressure coupling, the SIMPLEC (Semi-Implicit-Consistent for Pressure Linked Equation) algorithm, based on a predictor-corrector approach, has been used. The convergence criterion for numerical study residuals is set as $10^{-6}$ for mass continuity. The time step size is set at 0.001 s. To obtain the periodically convergent solutions five cardiac cycles were performed. The GAMBIT is used for the generation of the fluid domain mesh.

3. Results and discussion

Due to the pulsatility of blood flow, the hemodynamic features vary periodically with respect to time. Therefore, our results are observed at three specific instants of pulse cycle: $t = 0.1$ s (early systole), 0.3 s (peak systole), and 0.6 s (diastole).

We begin the discussion of our results by examining the shear rate distribution. For a non-Newtonian fluid the viscosity depend on the local shear rate values. Which in turn depend upon the fluid flow conditions (flow rate, geometry of the artery, stenosis severity, etc). We have presented in figure 4 the general aspects of the shear rate distribution in the flow domain at systolic time 0.1 s, for the case of Newtonian fluid and for stenosis severity of $S= 75\%$. The blue color corresponds to areas of low shear rates, while the red color refers to areas of high shear rates. Indeed, the qualitative analysis shows that low shear rate occurs close to centerline region, along the z axis, outside the stenotic zone with values in the range $8.8 \times 10^2 - 5.4 \times 10^2$ s$^{-1}$. Other regions of low shear rates occur in the recirculation area near the artery wall post-stenosis and around the reattachment point. However, high values of shear rate are localized near the wall along the artery, with values in the range of $10^3 - 1.2 \times 10^3$ s$^{-1}$. Furthermore, the shear rate is stronger around the stenosis zone and particularly at the post stenotic region near the throat, with peak shear rates of $9 \times 10^{-4}$ s$^{-1}$. This region of high shear rate often corresponds to the location of the lowest viscosity. Also, it is interesting to note that the magnitudes of shear rate for the value of Reynolds number considered (at $t=0.1$ s) are noticeable for the majority of the flow, and hence the viscosity of different rheological models will be affected only by this range of values.
Figure 4. Shear rate distribution for the Newtonian model at t = 0.1 s

Figures 5(a) - 5(f) reveal the cross-sectional velocity profiles at two different axial positions: z/R=40 (stenosis throat), and z/R=44 (post stenotic region) for Non-Newtonian (Cross Gpl and Herschel-Bulkley) and Newtonian models at early systole; t=0.1s, peak systole; t=0.3s and diastole; t=0.6s respectively. At early systole, the axial velocities present, for all models, a relatively flat profile at the throat of the stenosis, due to the greater shear force acting on the fluid in this region. The Cross and Gpl models are similar and have a slightly higher axial velocity than the Newtonian model and noticeable difference with the Herschel-Bulkley model. The normalized maximal axial velocity values are 10.25, 10.2, 9.6 and 8.4 for Gpl, Cross, Newtonian and Herschel-Bulkley respectively. At the post stenotic region, a parabolic profile is observed for the Newtonian case and slightly flattened profiles for Gpl Cross and Herschel-Bulkley models. Furthermore, the Herschel-Bulkley model shows a more accelerated flow with normalized velocity of 0.8 compared to Newtonian with 0.73, Cross with 0.68 and Gpl with 0.69. This result indicates that the Newtonian viscosity is the lowest than the viscosity of all non-Newtonians model, and so, the corresponding shear rate is below 10x10³ s⁻¹, according to figure 3. At the systolic acceleration the axial velocities show almost flattened profiles at the stenosis throat for all models. The non-Newtonian effect is similar as at t=0.1 s with considerable velocities values because of high inlet velocity. The normalized maximal velocities are, 28, 28.3 for the Gpl and Cross models respectively, 26.2 for the Newtonian model. Whereas the Herschel-Bulkley model underestimates significantly the velocity which has a value of 21.2. In the post stenotic region (figure 5 (d)) reversal flow is present, with negative values in the axial velocity, occurring near the wall for all models. The axial velocity is even more sensitive to the rheological models used. Large differences in maximal velocity values are found between Herschel-Bulkley (19.2), Newtonian (15.7) and Cross-Gpl (12). This is a consequence of the viscosity property of blood at high shear rate. At diastolic phase (t = 0.6s) the flow change direction, and negative axial velocities are observed. As can be seen from figures 5(e), 5(f) insignificant differences in axial velocities corresponding to Cross, Gpl and Newtonian because of reduced inlet velocity.

The complex non-Newtonian viscosity impacts the flow characteristics, especially in the vicinity of stenosis region. To investigate the effects of different rheological models applied to blood on flow patterns, we examine the instantaneous streamlines, in particular, the recirculation regions extent, during cardiac cycle. Appearance of these regions in the flow field has some pathological
Figure 5. Instantaneous radial velocity distribution at the stenosis throat and post stenotic region for: (a), (b) $t=0.1$ s; (c), (d) $t=0.3$ s; (e), (f) $t=0.6$ s
consequences, as it increases the residence time of blood constituents (RBCs), which in turn will increase the formation of blood clot by RBCs aggregation. Figures 6 (a) - (c) illustrate the recirculation regions extent plots for different blood viscosity models at t=0.1 s, t=0.3s and t=0.6 s respectively. To give more detail, only the part of the artery near the stenosis is shown. It is noted that streamlines are formed for smaller inlet velocity because of the high stenosis degree. Noticeable variations in the recirculation extent are observed throughout the cycle. At t=0.1 s, the Cross and Gpl models provide the shortest recirculation extent in the post stenotic zone. The flow pattern looks similar for both Newtonian and Herschel-Bulkley models, but with larger recirculation. As the flow accelerates during systolic phase, the extent of recirculation zone increases for all blood viscosities models and reaches its maximum at t=0.3 s. At this time, the flow field is largely influenced by the viscosity. The Cross and Gpl models are indistinguishable for almost all times with recirculation lengths located at (z/R-z_s/R) = 6.6 from stenosis throat. The recirculation extent of Newtonian model is less than that of Herschel-Bulkley model; it reaches the value of 9.8 from stenosis throat. Whereas the Herschel-Bulkley is found, as expected, to show a more disturbed pattern flow downstream the stenosis with a recirculation length located at (z/R-z_s/R)=14 from stenosis throat. During the decelerating phase, at t= 0.6s, the recirculation zone is observed in the distal of the stenosis for the Newtonian blood model, as its viscosity is the lowest for moderate shear rates; caused by reduced inlet velocity at this instant. The flow behaviors for non-Newtonian models are similar with non-recirculation.

![Figure 6. Instantaneous stream lines plots for different blood viscosity models at three different times: (a) t=0.1 s, (b) t=0.3 s, and (c) t=0.6 s](image)

Temporal maximal axial velocities during the whole cardiac cycle were shown in figure 7 for different rheological models. The maximal axial velocities are located at z_s/R=40 corresponding to a
maximal constricted zone. All the curves appear to follow the inlet pulsatile velocity form. As can be seen from this figure, the differences between maximal axial velocities are relatively small for different models in the systolic phase. Also, no significant changes are found when the inlet velocity is reduced during the rest of cardiac cycle.

The variation of the normalized wallshear stress (with respect to the value of the wall shear stress at inlet under fully-developed flow) with time at z/R = 40 has been plotted in figure 8. Unlike the characteristics of the maximal axial velocity profile, blood viscosity model has a profound impact in the results of the wall shear stress in during flow acceleration (systolic phase). As can be seen, wall shear stress is greatest for both Cross and Gpl models and lowest for the Herschel-Bulkley model.

![Figure 7. Variation of the maximal axial velocity with time at z/R=40, for different blood viscosity models](image)

![Figure 8. Variation of the wall shear stress with time at z/R=40, for different blood viscosity models](image)

The wall stresses versus the axial variable for both Newtonian and non-Newtonian fluid flow cases are shown in figures 9 (a), 9 (b) at systolic acceleration phase (t=0.3 s) and diastolic deceleration time (t=0.6 s) from the cycle. The wall shear stress is normalized to its value at the artery inlet for developed flow. These figures show that, in general, there is a stronger increase in shear stresses in the region of maximum stenosis constriction for all fluid viscosity models, followed by an abrupt decrease in the post stenotic region with negative values at the site of recirculation zone. The wall shear stresses show higher absolute values (in modulus) for the Cross, Gpl and Newtonian models when compared with Herschel-Bulkley model. Here too, the results obtained by all blood viscosity models show only minor differences outside the stenosis zone.
Figure 9. Distribution of shear stress along the wall for different blood viscosity models; (a) t=0.3 s, (b) t=0.6s

4. Conclusion

In this paper, we have examined numerically the impact of blood viscosity on pulsatile blood flow through a small narrowing artery. We have focused on comparing results obtained by various blood rheology models on hemodynamic flow patterns. In particular, velocity distributions, recirculation lengths and the wall shear stress distributions. Four rheological models were used to represent the blood viscosity variation with shear rate; Cross, Gpl, Herschel-Bulkley, and Newtonian. Our numerical results indicate that the high values of shear rate are located around the stenotic and post-stenotic regions. Accordingly, the instantaneous hemodynamic flow field is highly dependent on the local viscosity, given by various rheological models, in these locations. The Cross and Gpl models produce similar results with respect to velocities, wall shear stress and recirculation lengths. The Newtonian model presents larger recirculation zones when compared to Cross and Gpl results. However, the Herschel-Bulkley shows considerable deviation in results compared to corresponding Cross, Gpl and Newtonian. Therefore, the use of this model causes flow patterns more disturbed downstream of the stenosis because of its lowest viscosity for high shear rates. We conclude that the choice of the blood viscosity model is important for predicting the correct flow patterns for the stenosed small artery.

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