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Analysis of the effect of hematocrit on blood flow in a model of coronary artery stenosis

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***Abstract*—In this paper, the effect of hematocrit on increasing viscosity and consequently on related changes in blood flow patterns in coronary artery with stenosis is evaluated. A numerical procedure using the Galerkin finite element method is developed to simulate the pulsatile blood flow under physiological conditions. The non-Newtonian shear-thinning character of blood is modelled by inhibiting the generalized power-law, as a constitutive equation, in the governing equations of motions to be solved. Results from simulations indicate that hematocrit has considerable effects on instantaneous flow patterns and wall shear stress. Comparing flow patterns for different level of hematocrit shows marked differences between them especially at deceleration phases.**

***Keywords*—blood flow, hematocrit, finite element, coronary artery.**

I. Introduction

Blood is a complex suspension of formed elements suspended in an aqueous polymeric and ionic solution of low viscosity, the plasma, containing electrolytes, organic molecules and other proteins. It consists principally of red blood cells (erythrocytes) which constituted about 97 percent of the total particule volume, the white cells (leukocytes) and platelets occupy the remaining 3 percent volume. Because of their high concentration, the red blood cells exert a strong influence on the flow properties of blood. Hematocrit (He), defined as the proportion by volume of the blood that consists of red blood cells, is the most important determinant of whole blood viscosity. For normal human blood, the hematocrit is between 40-50 percent [1], and it should be emphasized that blood flow is non-Newtonian at all rates of shear for hematocrits above about 10% [2]. This behavior arises primarily from an increase in rouleaux density, length, and cell-cell interaction with increasing RBC concentration [3], particular, in some diseased conditions, and at low shear rates, as in the pulsatile flow case, in which blood is subjected to cyclic low velocities for a major part of the time period [4,5].

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Increasing blood viscosity by increasing hematocrit is one factor in hypertension and atherosclerosis. It is considered to be potentially pathological as shown by the study of Tohgi et al. [6] who report that the risk of cerebral infraction increases remarkably when hematocrit values exceed 45%. Salazar-Vazquez et al. [7], for their part, investigated the effect of hematocrit on blood pressure in diabetic patients, and stated that high hematocrit values can be associated with hypertension.

Furthermore, the shear-thinning character is the dominant non-Newtonian property of the blood [8], meaning its viscosity decreases as the shear rate increases, as a consequence of rouleaux dispersion at low shear rate. Extensive experimental and numerical approaches for flow through stenosis have been carried out, treating blood as a non-Newtonian fluid; Tu and al. [9] considered the blood obeying Hershell-Bulkley, Bingham and Power law fluid models to simulate the blood flow through arterial stenosis. The models predictions were compared to those obtained with the Newtonian fluid law for both steady and pulsatile flow. They acclaimed that the disturbances are stronger by their vorticity intensity and persist after the geometrical obstacle. Buchanan and al. [10] employed the Quemada and power-law models in pulsatile laminar flow through an axisymmetric stenosed tube; they reported that different flow patterns formed for the highest Womersley number under consideration, and found that the rheological models could affect wall shear stress quantities. More recently, M.R. Modarres Razavi and al. [11] compared for the same rheological models with Newtonian one, the hemodynamic wall parameters in pulsatile nature of blood flow. They examine in particular the effect of the frequency of pulsation on the flow field and location of the vortex formation distal to the stenosis for various Womersley numbers. These studies have all indicated the significant role of non-Newtonian behavior of blood in flow characteristic through stenotic artery. One of the successful models which has been able to capture the shear-thinning behavior of blood over a wide range of shear rates is the model proposed by Ballyk et al. [12] a generalization of the power law model. The purpose of this study is to investigate the effects of hematocrit on several hemodynamic factors, such as velocity, wall shear stress and pressure in a stenosed coronary artery. This analysis was conducted using the finite element method, the two dimensional Navier-Stocks equations coupled with the non-Newtonian constitutive model are solved by a finite element method, to investigate the influence of non-Newtonian behavior and hematocrit on the qualitative behavior of blood flow. The blood is assumed to be shear thinning and is modelled using a generalized power-law model with hematocrit values varying from 20% to 80%.

II. Numerical modelling

A. Mathematical model

In this work, blood is modeled as an incompressible non-Newtonian fluid. The governing equations, corresponding to the conservation of mass and linear momentum, can be written in velocity-pressure form as follows:

Continuity equation

$$\nabla.(\rho.v)=0 \tag{1}$$

Momentum equation

$$\rho\frac{\partial v_i}{\partial t}+\rho v_i\nabla v_j=\frac{\partial \tau_{ij}}{\partial x_j} \tag{2}$$

τ_{ij} the strain tensor:

$$\tau_{ij}=\mu\left(\frac{\partial v_j}{\partial x_i}+\frac{\partial v_i}{\partial x_j}\right)-p\delta_{ij} \tag{3}$$

Where ρ is the blood density; p and v denote the pressure and velocity of blood respectively, μ is the dynamical viscosity.

The correct specification of the viscosity model is crucial to capture the correct rheological behavior of blood. In this study, the generalised power law is adopted. Our choice of this constitutive equation was dictated by the physical considerations of capturing the viscosity variation in lower shear rates. It is more accurate than the Bingham and Casson models in regions of separated flow. This model is a developed form of the known Power law and encapsulates the behaviors of many of the other blood models. Specifically, it behaves Newtonian at high strain rates and has Casson and Carreau models as special cases. For the Generalised Power law the shear stress is given by the expression:

$$\begin{cases}\tau=\lambda(\dot\gamma)\dot\gamma^{n(\dot\gamma)}\\ \lambda(\dot\gamma)=\mu_\infty+\Delta\mu\exp\left[-(1+\frac{\dot\gamma}{a})\exp(-\frac{b}{\dot\gamma})\right]\\ n(\dot\gamma)=n_\infty-\Delta n\exp\left[-(1+\frac{\dot\gamma}{c})\exp(-\frac{d}{\dot\gamma})\right]\end{cases} \tag{4}$$

Here, λ and n are the consistency coefficient and power-law index for the shear-thinning fluid and are assumed to be functions of the local strain rate $\dot\gamma$, μ_∞ is the limiting Newtonian viscosity, a, b, c, d, $\Delta\mu$, Δn, and n_∞ are the generalized power-law model parameters. We obtain these unknown parameters by curve-fitting to experimental data [13, 14] using a non-linear least-squares method.

Figure 1 illustrates the comparison between theoretical (solid lies), and experimental data (symbols) for range of hematocrit from 20% to 80% and revealed the good ability of this model to fit a shear rate dependence of the blood viscosity [15].

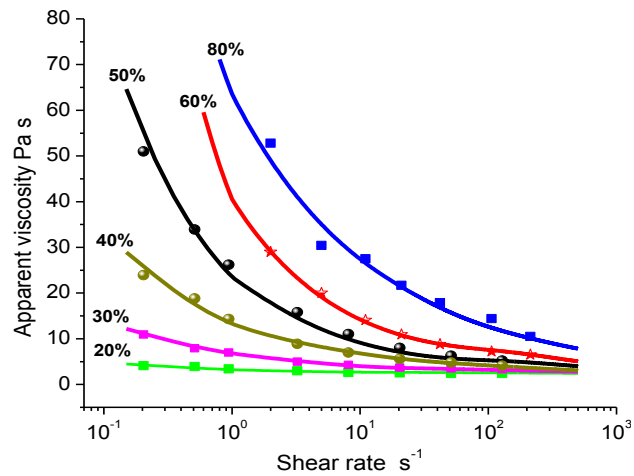

Figure 1. Fitting of the blood viscosity by the generalised power law model for various hematocrit values

B. Physical model

For the sake of simplicity, it is assumed that the geometry of the stenosis artery is axisymmetric and the vascular wall is considered as a rigid tube. Geometry of the artery in the presence of stenosis is constructed mathematically of which the shape is described by the cosine model suggested by Young [16]:

$$\frac{r(z)}{R_0}=1-\frac{\delta}{2R_0}\left(1+\cos\frac{\pi(z-z_0)}{L}\right) \tag{5}$$

Here L is the width of stenosis, δ the maximum width and R_0 being the unconstructed radius of the stenosed vessel, z_0 is the center position of stenosis region. This work is concerned with the right stenosed coronary artery. The 75% stenosed artery is modeled as a straight tube, with dimensions of, $\delta=0.75$, L=2, z0=16, (Fig. 2).

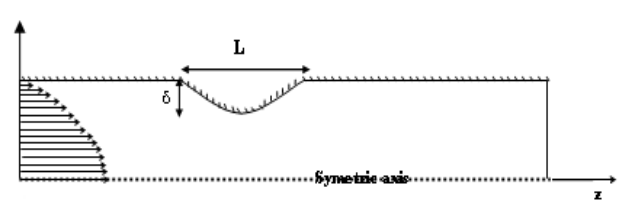

Figure 2. Flow geometry of blood vessels

C. *Boundary conditions*

The blood flow in the coronary arteries is very pulsatile with zero or even reversing (negative) flow in systole. The boundary conditions required to solve the governing equations are as follows.

- The pulsatile flow was generated by means of axial velocity inlet profile imposed as function of the time (figure 3):
- The radial velocity is set to zero at the inlet.
- At the outlet of the artery, the fully developed flow condition is applied; we arbitrarily prescribed a zero pressure.
- On all rigid walls, all velocity components were set to zero according to the no-slip condition.
- In the plane of symmetry, both the normal velocity and the first-order derivative of the axial velocity in the radial direction are set to be zero.

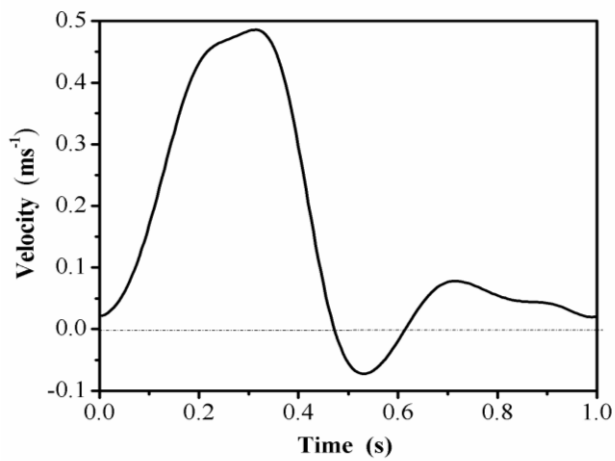

Figure 3. The inlet velocity waveform of blood vessel.

D. *Numerical method*

The momentum and continuity equations are solved numerically by a finite element method [17, 18]. For the continuity equation, the penalty function approach is introduced to eliminate the pressure from the constraint condition, to reduce the degrees of freedom for each node and to save computational coasts. The discretization of these equations follows the standard Galerkin's finite element formalism. This discretization leads to the following non-linear partial differential equations, written for each element in a matrix form as:

$$w^2[M]\{\dot{v}\} = Re[N]\{v\} + \lambda[P]\{v\} + [S]\{v\} = \{f\} \qquad (6)$$

Where: $[M]$ is the mass matrix $[N]$ is the convective matrix, $[P]$ is the penalty matrix, $[S]$ is the diffusion matrix, $\{v\}$ the time derivative of velocity, $\{v\}$ velocity vectors, $\{f\}$ is the boundary forces, λ is the penalty parameter.

The non-linear terms in equation (6), resulting from the advection in the inertial term and the non-Newtonian viscosity behaviour in the constitutive relationship, need to be solved at each time step over a cardiac pulsation. Consequently, a direct iterative technique, in which an initial value is assumed for velocity vector $\{v\}$ is used. The time integration is performed by an implicit first-order time step scheme. The time step is chosen small enough that the stability conditions on the convective and diffusive terms are preserved.

The computational domain extends from z=0 to z=46, in order to have a sufficient development length in the axial direction. Motion equations are integrated in time for several periods until a periodic solution is reached, in the periodic steady state of period T, the solutions at instants t and t + T had to be equal. Typically, transient calculations over five cycles were sufficient to satisfy this condition (error within 10^{-5}). Moreover, the importance of non-Newtonian properties of blood on the blood flow in artery is studied by changing the level of hematocrit from 20% to 80 %.

III. Results and discussion

Numerical results concerning the flow characteristics are presented for axial velocity, wall shear stress and pressure. In figure 4, the time variation of axial velocity on a given point on the tube centerline is displayed for different level of the hematocrit for non-Newtonian models. The difference in the velocity profile is pronounced at the systolic phase for time between 0.2s and 0.4s with the high velocities associated with flow acceleration, and the difference diminishes with the low velocities at diastolic phase. From this figure we observe that an increase in hematocrit decreases the axial velocity.

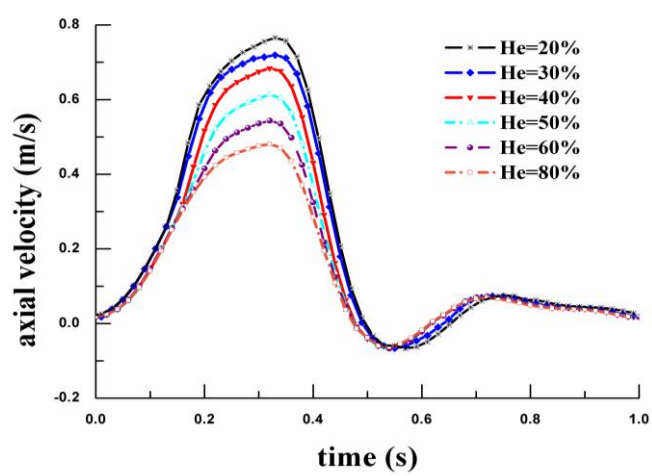

Figure 4. Variation of axial velocity in a time cycle for different rate of hematocrit

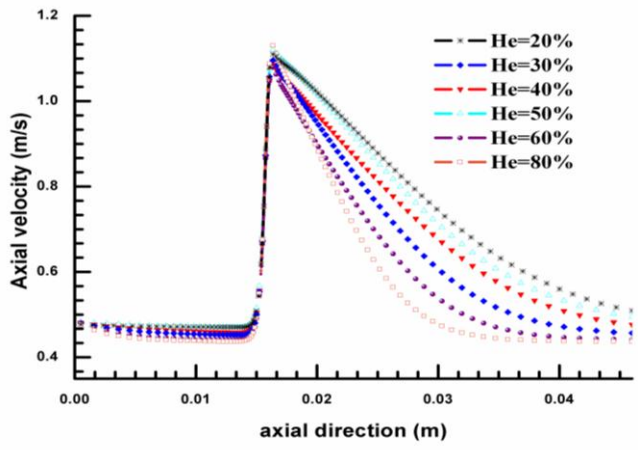

Figure 5. Axial velocity profiles on symmetric axis for different rate of hematocrit

We represent in figure 5 a comparison of the axial velocity profiles along the symmetric axis, for the six levels of hematocrit in the same flow condition as previous. The maximum velocity in a cycle occurs at the throat of stenosis. As the fluid moves further downstream, the velocity profile becomes stabilized, i.e. fully developed flow. This figure clearly shows that the difference in axial velocity profiles is pronounced at and downstream the stenosis. Peak velocities are increased with increasing hematocrit at the constriction, while velocities are reduced with the hematocrit far from the constriction.

Figure 6 illustrates the pressure distributions at the centerline of the artery. Note that zero pressure at the outlet of the artery is specified because the pressure is determined only up to a constant in the model. In all cases a, pressure drop caused by the stenosisis observed. The pressure reaches a slight local minimum at the throat and then recovers downstream from there. It is also observed that hematocrit level influences the pressure. The increase of hematocrit increases the pressure drop along the artery.

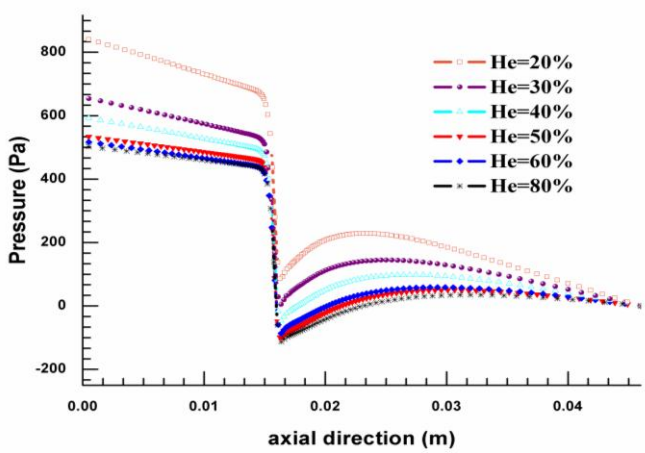

Figure 6. Pressure variation along the symmetric axis for different rate of hematocrit

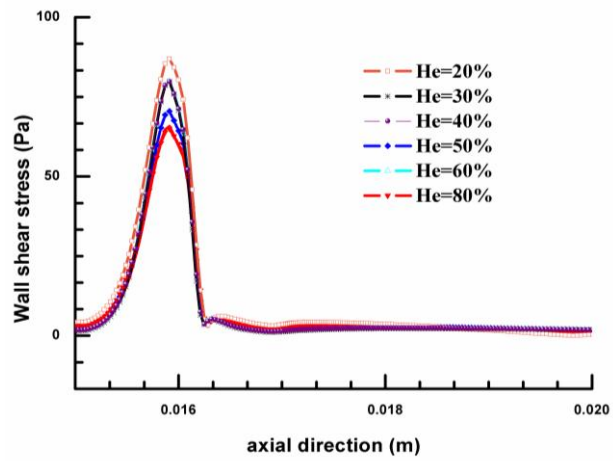

Figure 7. Variation of shear stress along the wall for different rate of hematocrit

Wall shear stresses as a function of axial distance are presented in Figure 7 for 20–80% hemarocrit rates, only at the constriction area. It is well known that at this location the WSS values are the highest because of the jet effect due to the constriction. Downstream from the point of the maximum the wall shear stress decreases rapidly. As indicated the peak wall shear stress decreased with the hematocrit which explains the increase in the degree of blood deceleration with hematocrit in the wall vicinity.

IV. Conclusion

The pulsatile flow of blood in stenosis artery is simulated by finite element method. It was concluded that using a non-Newtonian model for blood viscosity was important over the whole cardiac cycle. And this modifies the flow structure, even beyond the contracted region. The analysis of obtained calculation results suggest that the hematocrit may be an important factor to predict the main characteristics of the physiological flows and may have some interest in biomedical applications.

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